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Obstructive Sleep Apnoea in Aotearoa/New Zealand

An objective and questionnaire-based approach to population prevalence estimation and clinical screening

A thesis presented in partial fulfilment of the requirements for the degree of

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in
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Ngāti Kahungunu ki Wairoa, Ngāti Rakaipaaka, Rangitāne ki Tamaki-nui-ā-Rua

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E tipu e rea mo ngā rā o tou ao
Ko te ringa ki ngā rākau a te Pākehā, hei oranga mo tou tinana
Ko to ngākau ki ngā taonga a o tipuna, hei tikitiki mo to māhunga
Ko to wairua ki te Atua, nāna nei ngā mea katoa

Grow up and thrive for the days destined to you.
Your hands to the tools of the Pākehā to provide physical sustenance,
Your heart to the treasures of your Māori ancestors as a diadem for your brow,
Your soul to God, to whom all things belong
Sir Apirana Ngata

ABSTRACT

The goals of this thesis were to objectively assess the prevalence of obstructive sleep apnoea syndrome (OSAS) among Māori and non-Māori adults in a community-based sample, and to develop a questionnaire-based multivariate predictive tool for OSAS, to help improve referral of patients to specialist sleep services, and prioritise waiting lists. This research was situated within the wider scope of ethnic inequalities in health between Māori and non-Māori, and was conducted within a Kaupapa Māori Research (KMR) framework.

Between August 1999 and June 2001 letters and information were progressively sent out to 1200 (600 Māori, 600 non-Māori) Wellington residents aged 30-60 years selected randomly from the electoral rolls. Participants were asked to wear a small sleep monitoring device (MESAM4) for one night in their own homes and to fill out a sleep questionnaire. Contemporaneously, sleep and questionnaire data were collected from 510 consecutive patients aged 30-60 years, who were referred to the regional sleep clinic for suspected OSAS.

In the community sample, OSA was found to be more prevalent among Māori. Among men, 21.98% of Māori had OSA (RDI≥ 5) compared with 11.37% of non-Māori. Among women, 6.28% of Māori and 3.02% of non-Māori respectively had OSA (RDI≥ 5). The higher risk among Māori appeared to be due to well-recognised risk factors such as higher body mass index (BMI) and larger neck circumference, rather than ethnicity per se.

Using the combined data from the community and clinical samples, two clinical prediction models were developed using logistic regression modelling. One model (Model 1a) included age, sex, observed apnoeas, self-reported habitual snoring, subjective excessive daytime sleepiness, and BMI. The second model (Model 2a) included neck circumference instead of BMI. Model 1a correctly classified 82.50% of participants (sensitivity 72%, specificity 87%). Model 2a correctly classified 81.10% of participants (sensitivity 80%, specificity 82%).

This research indicates that OSA is a common problem among New Zealand adults and that ethnic disparities exist. The results provide important guidance for planning to

meet population needs, by identifying differential needs of specific groups. The prediction models provided reliable estimates of *a priori* probability of OSA, and therefore may be useful tools for screening patients for OSAS.

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As I look back at my life, I am both humbled and relieved to have reached this amazing milestone. There are a number of people who have assisted me through this journey, and I would like to take this opportunity to express my appreciation.

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GLOSSARY OF TECHNICAL TERMS AND ABBREVIATIONS

%	Percentage
AIC	Akaike Information Criterion
ALAC	Alcohol Advisory Council of New Zealand
Apnoea	Cessation of airflow
Apnoea Hypopnoea Index (AHI)	the number of apnoeic events plus hypopnoeas per hours of sleep as determined by polysomnography.
AUC (Area under the curve)	A measure of accuracy of the ROC curve
BMI (Body Mass Index)	Weight in kilograms divided by height in metres squared (kg/m^2).
BP	Blood Pressure
CI	Confidence Interval
CSA	Central Sleep Apnoea
CSC	Community services card
CVD	Cardiovascular disease
DF	Degrees of freedom
DHB (District Health Board)	Organisations established to protect, promote and improve the health and independence of a geographically defined population. Each District Health Board will fund, provide or ensure the provision or services for its population.
ECG	Electrocardiogram
EDS	Excessive daytime sleepiness
EEG	Electroencephalogram
EMG	Electromyogram
EOG	Electrocculogram
Epoch	a measure of duration of a sleep recording
ESS	Epworth Sleepiness Scale

False Negative	The ratio of the number of events incorrectly classified as non-events over the sum of all observations classified as non-events
False Positive	The ratio of the number of non-events incorrectly classified as events over the sum of all observations classified as events
First night effect	the effect of the environment and sleep recording equipment on the quality of the subject's sleep during the first night of recording
GHQ	General health questionnaire
HR	Heart Rate
HRI	Heart Rate Variation Index
IRI	International Research Institute for Māori and Indigenous Education
KMR	Kaupapa Māori Research
MESAM4	Madaus Electronic Sleep Apnoea Monitor 4
MSLT	Multiple sleep latency test
MOH	Ministry of Health
MVA	Motor vehicle accident
nCPAP	Nasal continuous positive airway pressure
NPV (Negative Predictive Value)	The probability of not having the disease when the test result is negative
NZDEP	New Zealand Deprivation Index
ODI	Oxygen Desaturation Index
OR	Odds Ratio
OSA	Obstructive Sleep Apnoea
OSAS	Obstructive Sleep Apnoea Syndrome
OSAHS	Obstructive Sleep Apnoea-Hypopnoea Syndrome
Polysomnography (PSG)	Gold standard for measuring sleep
PPV (Positive Predictive Value)	The probability of disease in a person with an abnormal/positive test result
Prevalence	The number of instances of a given disease or occurrence in a given population at a specific point in time
P-value	A statement of the probability that the difference observed could have occurred by chance, reflecting the statistical significance of the result
RDI	Respiratory Disturbance Index
REM	Rapid eye movement sleep
RERA	Respiratory Effort Related Arousal
ROC (Receiver Operator Characteristic) Curve	Non-parametric plot of the true positive (sensitivity) and false positive rates (1-specificity)
RR	Relative Risk
RTS	Return to sender

Sa02	Level of oxygen saturation in blood
SAS	Sleep Apnoea Syndrome
SD	Standard Deviation
SDB	Sleep disordered breathing
SE	Standard Error
Sensitivity	The ratio of correctly classified events over the total number of events
SNZ	Statistics New Zealand
Specificity	The ratio of correctly classified non-events over the total number of non-events
TRRHAEP	Te Rōpū Rangahau a Eru Pōmare
UPPP	Uvulopalatophyrangoplasty
US	United States

GLOSSARY OF MĀORI TERMS

Aotearoa	Māori name for New Zealand often translated as "land of the long white cloud
Whānau	Family, extended family
Iwi	Tribe
Hapu	sub-tribe
Tino rangatiratanga	Māori self-determination; sovereignty
Pākehā	Person of predominately European descent; not Māori
Māori	The indigenous people of New Zealand
Mauri	Māori vitality; life force

CHAPTER 1
INTRODUCTION

Breathing and sleeping are two very important vital processes. It is therefore surprising that only in the past 25 years has disordered, even disrupted, breathing during sleep been recognised as a substantial medical and public health problem (Drazen 2002).

Obstructive sleep apnoea syndrome (OSAS) is the most commonly diagnosed sleep breathing disorder, both in New Zealand and overseas. OSAS is generally caused by the upper airway collapsing as people relax into sleep. People with severe OSAS are often very sleepy, because the quality of their sleep is disturbed by the frequent brief and often forgotten arousals to initiate breathing (Bassiri and Guilleminaut 2000). Of all sleep disorders, OSAS has been found to be the most serious with respect to morbidity and mortality. Given its associated morbidity, and the effectiveness of treatment, identification of patients with OSAS is an important public health issue. (Baumel et al. 1997, Young et al. 2002a).

In New Zealand, sleep disorders medicine is in its infancy with the number of overnight beds for the diagnosis and treatment of sleep disorders totalling around 20 (Gander 2003). At present there is no systematic national approach to the recognition, diagnosis and treatment of sleep disorders (Neill et al. 2000). It is therefore likely that the majority of New Zealanders with sleep disorders are undiagnosed, misdiagnosed or inappropriately treated. Anecdotal evidence indicates that a disproportionately small number of Māori and Pacific peoples attend sleep clinics (Frith and Cant 1985, Baldwin et al. 1998), with an indication that Māori and Pacific patients suffer more severe OSAS (Baldwin et al. 1998). This has raised concerns about accessibility of services and possible differences in prevalence between ethnic groups.

This thesis presents a study that was carried out in the Wellington region to objectively assess the prevalence of OSAS in Māori and non-Māori adults aged 30-60 years in a community population sample, and to develop a clinical prediction tool for screening OSAS, based on data collected in a community sample and a sleep clinic sample. It was a joint project between the Sleep/Wake Research Centre, Te Rōpū Rangahau a Eru Pōmare, and Wellsleep sleep clinic, and it forms part of a group of studies, which aim to

assess the impact of sleep problems in New Zealand. It was primarily designed as a companion study to the national sleep survey of 10 000 adults (5500 Māori, 4500 non-Māori), which examined the prevalence of OSAS symptoms and risk factors among Māori and non-Māori adults aged 30-60 years (Harris 2003). The combination of these two studies will allow more accurate estimates of prevalence information in New Zealand, which will allow an assessment of the public health impact of OSAS and enable planning for population health care needs.

My role on this study was as the primary researcher. I led all aspects of the study, from recruitment of participants and data collection through to the analysis and interpretation of all data. It is beyond the scope of this thesis to report on all the findings from the study, and whilst the prevalence of OSAS is examined in this thesis, the main analyses are based on the development of a clinical screening tool to assist in the identification of patients with OSAS. A tool of this nature may be useful in a primary care setting, to assist in the referral of patients to specialised sleep services. This would be expected to reduce economic costs, by reducing the number of inappropriate referrals, and thus reduce pressure on limited available resources. Furthermore, more reliable and systematic identification of OSAS may assist in the management of adverse outcomes that affect Māori disproportionately, including hypertension, cardiovascular disease, stroke, and motor vehicle accidents (Pōmare et al. 1995, Ministry of Health 2000, Sargent et al. 2004).

This thesis extends current sleep disorders research in New Zealand by providing the first objective prevalence estimates of OSAS in a population-based sample. It utilises Kaupapa Māori Research (KMR) methodology to examine health issues where needs and risks for Māori can be assessed to the same level of analysis as non-Māori. The development of the clinical screening tool is unique in the combination of variables examined, and in the inclusion of a community-based comparison group as well as a large clinical population. This thesis also offers new information on the role of ethnic and socio-economic factors in OSAS, about which little is known.

The findings of this study will contribute to strategic planning to improve the management of sleep disorders in New Zealand, by recognising the needs of specific groups in the community. This thesis will also provide critical guidance for the

provision of adequate and appropriate diagnosis and treatment facilities for Māori and non-Māori.

1.1.1 Summary of chapters

Chapter Two (Background) – This chapter is divided into two main sections. As this study was designed within the scope of KMR and has a particular focus on Māori health outcomes, the first section of this chapter provides an overview of Māori health. It examines Māori health status, Māori health research, and explains the specific function of KMR in the present study. The second section of this chapter provides a comprehensive overview of OSAS, from pathophysiology through to diagnosis, along with a review of literature pertaining to population prevalence studies and clinical prediction tools. This chapter concludes with a brief background of the study along with its aims and hypotheses.

Chapter Three (Methods) – This chapter describes the methods utilised in this study, with an extensive description of the collection of data in the community and clinical samples, along with a description of the study protocol, data management and statistical analyses.

Chapter Four (The Community Sample) – This chapter provides a description of the data collected in the community sample. The response rates are evaluated and potential biases inherent in the sample are examined. Questionnaire data are analysed along with the objective data. Population adjusted prevalences are presented, along with significant predictors of OSA.

Chapter Five (The Clinical Sample) – This chapter provides a description of data collected in the clinical sample. Demographic, questionnaire and objective data are analysed along with significant predictors of OSA.

Chapter Six (Development of a Screening Tool) – The primary focus of this chapter is on the development of a mathematic model to predict OSA using data combined from the clinical and community samples. This chapter is divided into three main sections. The first section examines the demographic profiles and objective sleep data of the combined sample. The second section assesses the best fitting and most parsimonious models to describe the relationship between OSA and a set of predictor variables. The

final section evaluates the performance of each model, with a close examination of the nature of misclassified results (false negatives and false positives).

Chapter Seven (Discussion) - This chapter is divided into three main sections. The first section provides a summary and explanation of results. Univariate results are examined simultaneously for both community and clinical samples in order to provide an overview of the variables tested in each predictive model. Population prevalence estimates are then provided for varying severities of OSA and OSAS. The results of the development and evaluation of the prediction models are then discussed. The second section considers the strengths and weaknesses of this study, which provides the necessary context for considering the findings of this study. The final section discusses the implications and recommendations of this study along with the further research needs that it highlights.

CHAPTER 2
BACKGROUND

MĀORI HEALTH

2.1 Introduction

This thesis was undertaken within the wider scope of Kaupapa Māori Research (KMR), it therefore assumes Māori norms, and prioritises Māori needs. This section on Māori health aims to provide the necessary context to the overall study. Māori health status is discussed along with Māori health rights in regards to the Treaty of Waitangi. Issues relevant to Māori health research are then discussed, including the classification of ethnicity and the research of ethnic disparities. The final section provides a brief synopsis of KMR methodology and its relevance to the present study.

2.2 Māori Health Status

Māori are the indigenous people of New Zealand, and comprise approximately 15% of the population. Although improvements have been seen in Māori health status over the decades, compared to other New Zealanders, major disparities still remain and are widening in almost all health indicators (Pōmare et al. 1995, Ministry of Health 2002a, 2002b).

Data from 1980–1999, show that Māori had the highest rates of mortality. Whilst the rates were stable and modestly decreasing, the gap between Māori and non-Māori non-Pacific peoples widened significantly. Similarly, Māori life expectancy increased from 64.6 to 65.8 years for males (1.2 years) and from 69.4 to 71.0 years for females (1.6 years). But once again the gap between Māori and non-Māori non-Pacific people increased. The gap between Māori and non-Māori males increased 57%. The corresponding gap for females increased 26% (Ajwani et al. 2003).

The data from this period also demonstrate that while Māori have experienced decreasing rates of mortality from three major causes of death (cardiovascular disease, unintentional injury, respiratory diseases). Unfortunately however these gains have largely been offset by increasing cancer mortality (all major types). Furthermore, the relative inequalities between ethnic groups have tended to increase over time. These

findings are consistent with three special reports compiled by Professor Eru Pōmare, which provide a comprehensive overview of Māori health trends for the period spanning 1955 to 1991. All three have served to highlight the fact that while Māori life expectancy has improved, large disparities between Māori and non-Māori exist for most disease categories (Pōmare 1981, Pōmare and de Boer 1988, Pōmare et al. 1995).

While conventional measures such as life expectancy, death rates, fertility rates, and hospitalisation give some indication of Māori health, it is argued that they are imperfect measures in that they focus on sickness and illness and fail to capture Māori vitality or mauri (Durie 1998). However, such measures have strengths in terms of availability and ability to provide commentary on trends over time. They are therefore useful tools to monitor the impact of government policy on Māori health and the performance of services for Māori (Pōmare et al. 1995).

Explanations for ethnic inequalities between Māori and non-Māori have frequently focused on individual risk factors such as lifestyle, behavioural factors and genetic predisposition. However, it is now recognised that individual factors are often influenced by wider determinants of health such as social, political, economic, cultural and historical factors, all of which are usually outside the control of the individuals and groups affected (Ministry of Health 2002b). The role of these additional factors in the existence and maintenance of disparities between Māori and non-Māori is now receiving more attention.

Socioeconomic factors are considered major determinants of health, and given that the Māori population is disproportionately distributed among the most deprived sectors of New Zealand society, socioeconomic determinants of health have been largely implicated in the health inequalities seen between Māori and non-Māori (Crampton et al. 2000b). However, there is evidence indicating that Māori outcomes in health are consistently worse than those of Pākehā at all levels of deprivation (Reid et al. 2000). Furthermore, data for 1980-1999 demonstrate that despite higher levels of deprivation, Pacific peoples have lower mortality rates than Māori, both overall and for many specific diseases (although trends are similar). It has been suggested that part of the reason for these findings may be that the indicators used, do not fully capture the extent of the real differences in socioeconomic position. Other reasons include the cumulative effects of disadvantage over the life course, barriers to health care, and racism (Howden-Chapman and Tobias 2000, Blakely and Pearce 2002).

The imbalance of health care utilisation and health care need can contribute significantly to the poorer level of health experienced by Māori (Ministry of Health 2002b). There is increasing evidence indicating differential access to both primary and secondary care services for Māori (Te Puni Kōkiri 2000, Baxter 2002). A number of factors have been implicated as potential barriers to health care access for Māori. These range from financial barriers through to a lack of cultural comfort with mainstream providers (Crengle 2000). Discrimination within healthcare services against Māori is also seen to play an important role in health inequalities. For example, while Māori are 1.6 times more likely than non-Māori to die from ischaemic heart disease, non-Māori are 1.5 times more likely to receive a coronary bypass and 3.5 times more likely to receive an angioplasty, which are the surgical interventions most likely to prevent premature death (Pōmare et al. 1995). There is also evidence of unfavourable attitudes amongst general practitioners. In particular, Māori are often blamed for their poor health and some general practitioners do not support changes to policies and practices that might bring about population-level health gains for Māori (McCreanor et al. 2002). It has been suggested that the quality of care provided may also be systematically inferior for Māori patients (Baxter 2002).

Racism is also an important factor in health inequalities, while also having a direct impact of health. In New Zealand it is acknowledged as a contributing factor in the existence of health disparities between Māori and non-Māori (Ministry of Health 2001). Jones (2001) provides a three-levelled model of racism as a framework to explain potential effects of racism on health, which involves institutional, personally-mediated, and internalised racism. Institutionalised racism refers to differential access to the goods, services and opportunities of society by race. In addition to material conditions, institutionalised racism is seen to manifest itself in terms of access to power and information, access to resources, and access to voice. In New Zealand, it is suggested that this type of racism provides a vehicle by which the distribution gap of deprivation between Māori and non-Māori is maintained. Like institutional racism, personally-mediated racism is reflected as prejudice and discrimination by race, which can be both intentional and unintentional. Finally, internalised racism is characterised by the acceptance of negative messages about one's own abilities and intrinsic worth, which is often manifested as resignation, helplessness and hopelessness. Of these three levels of racism, institutional racism is seen as the most fundamental, and it is hypothesised that

if institutional racism is addressed, the other levels of racism will diminish over time (Jones 2000).

2.3 The Treaty of Waitangi

The Treaty of Waitangi is the founding document of New Zealand. It was signed in 1840 between Māori and the British Crown, and in some part was a response to the poor health status report of Māori (Reid 1998). Prior to 1800, while the population of Māori were relatively low, the population was at least increasing (Durie 1998). Colonisation caused a significant decrement in Māori health, not only by the introduction of epidemics and muskets, but also by the undermining of tradition Māori social structures, beliefs and values. Consequently, during the nineteenth century, the Māori population was nearly decimated.

The Treaty is a living document and it continues to define the relationship between the Crown and Māori (Durie 2000, Ministry of Health 2000). Although widely debated, the basic tenets of the Treaty revolve around the governance agreement for Pākehā settlement, and a guarantee of protection of Māori interests against negative impacts from settlement, both immediate and ongoing (Blakely et al. 2002). Thus the current health disparities between Māori and other New Zealanders are evidence that Māori interests are not being protected and therefore can be seen as a breach of Māori rights under the Treaty (Reid 1998).

The current government is focused on reducing health inequalities between ethnic groups, and has directed the health sector to acknowledge the principles of the Treaty and its relationship to the social, physical and economic well being of Māori. Underpinning the governments health strategies are the following Treaty principles (Ministry of Health 2002a):

> **Partnership**: working together with iwi, hāpu, whānau and Māori communities to develop strategies for Māori health gain and appropriate health and disability services.
>
> **Participation**: involving Māori at all levels of the health and disability sector in the planning, development and delivery of health and disability services.

> **Protection**: working to ensure Māori have at least the same level of health as non- Māori and safeguarding Māori cultural concepts, values and practices.

Given the impact of colonisation on Māori health and the breaches of the Treaty, it is important that contemporary Māori health is examined within a framework that takes into account the impact of our colonial history and the Treaty of Waitangi. A Treaty framework addresses structural barriers to equity in health and engages with Māori rights.

2.4 Māori Health Research

Monitoring health status plays an important role in the effort to reduce health disparities between ethnic groups. However, accurate recording of ethnicity is needed to provide useful information about the utilisation of health services, to plan and evaluate public health services, allocate health resources, and to monitor trends in the health status of peoples from different ethnic groups (Reid and Robson 1998).

2.4.1 Classification and measurement of ethnicity

The primary analytical comparisons in the present study are between Māori and non-Māori, as defined by ethnicity, using the 1996 census question. Ethnicity is a fundamental component in measuring disparities between Māori and non-Māori, and for developing appropriate services and policies, ensuring the needs of specific groups in the population are met. Therefore it is important to discuss the issues surrounding the classification and measurement of ethnicity. A number of health researchers in New Zealand commonly use ethnicity data in an uncritical manner, giving little attention to the problems of measurement that exist (Thomas 2001).

The collection of ethnicity data has been a requirement for hospital services and some primary and community services for some years, however, ongoing problems have been noted with the quality and comprehensiveness of ethnicity data. This issue has been exacerbated by changes in the ethnicity question in official statistics. The Crown has a Treaty responsibility (of good governance) to maintain Māori health data to at least the same quality as that of non-Māori (Ajwani et al. 2003). Currently there is poor consistency across data sets, in terms of missing data (ethnicity is not asked of all persons) and because an alternative ethnicity question (other than the census ethnicity

question) is used. While this information does not affect the quality of data for the total population, it marginalises Māori information, making planning and evaluation of policy interventions difficult (Robson and Reid 2001).

In the census, the definition of Māori has changed considerably over time. Early definitions of Māori were based on race and classified according to quantum of blood. It was not until the 1991 census that the biological or racial concept of ethnic origin was explicitly replaced by the socio-cultural concept of ethnic groups, measured by self-identification. Māori ancestry was also separately measured. The definition of ethnicity used by Statistics New Zealand (SNZ, cited in Allan 2001) is as follows:

> *Ethnicity is the ethnic group that people identify with or feel they belong to. Ethnicity is seen as self-perceived and people can belong to more than one ethnic group. An ethnic group is defined as a social group whose members have the following characteristics:*
>
> - *Share a sense of common origins*
> - *Claim a common and distinctive history and destiny*
> - *Possess one or more dimensions of collective cultural individuality*
> - *Feel a sense of unique collective solidarity*

Despite the definition above, ethnicity may also be influenced by a number of other factors including ancestry, culture, race, country of birth (nationality), or religion. It is however difficult to gauge how people form their responses to the ethnicity question. For example, some people may regard the terms 'race' and 'ethnicity' as synonymous or may even regard 'ethnicity' as a euphemism for 'race' (Allan 2001).

The wording of the question also plays an important role in the understanding of the concept of ethnicity. This is highlighted in the 1996 census where the wording used in the previous census was altered to make it clearer to respondents that they could tick more than one ethnic box. This led to a much higher proportion of multiple ethnic responses, with many respondents interpreting the instruction to 'tick as many boxes as you need' in terms of ancestry rather than current ethnic (cultural) affiliation. Consequently, a significant change in the size and demographic composition of the Māori ethnic population was seen for this particular census. This issue was rectified in subsequent censuses (Alan 2001).

From 1991 onwards, the census describes three Māori populations: the Māori descent or ancestry group, the *Māori ethnic group* comprising those who indicated Māori as at least one of their ethnic affiliations; and the *sole Māori* group comprising of those who indicated Māori as their only ethnic affiliation.

The production of these different groupings has led to some debate as to which Māori population should be used as the reference population for comparison and commentary (Robson and Reid 2001). Significant socioeconomic and cultural as well as health differences are known to exist between sole Māori and Māori ethnic groups (although the pattern if not the magnitude of disadvantage is the same for both) (Reid et al. 2000). The use of the Māori ethnic group may underestimate the inequalities between Māori and the New Zealand European ethnic group. On the other hand, use of the sole Māori group concept may overestimate the differences as well as greatly reduce the size of the Māori population. The ancestry/descent population is linked to a number of constitutional rights, for example the ability to enrol in a Māori electorate. However, ancestry has very strong biological overtones. The global pattern of health disparities challenges the assumption of the concept of race as a genetic biological measure and suggests that ethnic health disparities and genetics have little to do with each other (Sankar et al. 2004), therefore the biological concept of 'race' has been argued to have little scientific value when applied to the human population and is described as a social construct (Goodman 2000). Ethnicity on the other hand, is thought to better reflect lived social reality and cultural affiliation, which have been found be more closely aligned to a variety of outcomes, including health (Senior and Bhopal 1994).

A disparities analytical framework

The present study adopts a disparities analytical framework, comparing Māori and non-Māori. The primary purpose of monitoring disparities is to inform appropriate interventions and to eliminate inequality, which is in line with the guarantees of the Treaty of Waitangi. It is also consistent with the strategy of the current government to address ethnic health inequalities (Ministry of Health 2000).

However, research of this type is often seen as controversial. Reid and colleagues (2000) provide critical commentary on some prevailing myths surrounding ethnic disparities research. One common belief is that interventions based on ethnicity are racist. It is argued however, that the presence of ethnic disparities per se is what is racist, and non-intervention perpetuates racist outcomes, especially given the fact that

ethnic disparities in health outcomes exist along the entire socioeconomic gradient. Another common belief is that this type of research promotes Pākehā levels of health as the Māori goal, but it is argued that Māori only seek to eliminate ethnic disparities, not to become Pākehā. It is further argued that the adoption of a universal approach to service provision legitimises the non-recognition of ethnic disparities and privileges non-Māori, which in turn promotes institutional racism (Robson and Reid 2001).

Reid et al. (2000) also warn against the reporting of disparities without seeking explanation. This approach assumes that the basis for the differences is already completely understood and thus bolsters ideologies of biological flaws, which is a form of racism, whereby poor health is attributed to internal deficiencies and wider determinants such as structural factors are held blameless (Ajwani et al. 2003).

2.5 Kaupapa Māori Research

Research in New Zealand has developed unambiguously within the scope of Western culture and the history of the dominant culture, which has disadvantaged Māori. The failure of research to inform the elimination of disparities between Māori and non-Māori and contribute positively to Māori health has seen the emergence of Kaupapa Māori Research (KMR), which is the underlying methodology or process of enquiry in the present study (IRI and TRRHAEP 2000). Kaupapa Māori literally means a Māori way or agenda (Henry and Pene 2001).

KMR is located within the wider scope of Māori struggles towards decolonisation and tino rangatiratanga (self determination). Therefore it involves challenging Pākehā hegemony and reclaiming a Māori reality (Smith 1999). There is no one definition of KMR, but it has been described as being related to being Māori, and is associated with Māori principles and philosophies (Moewaka-Barnes 2000). It is therefore based on the assumption that Māori culture, language and beliefs are valid and legitimate (Smith 1999 cited in IRI and TRRHAEP 2000).

There is ongoing debate about who can undertake this type of research. While KMR is often described as research "by Māori, for Māori and with Māori", non-Māori researchers may be involved in supporting roles (Smith 1999).

There is no specific set of methods, but rather KMR is an approach to research that brings Māori to the centre, and prioritises Māori needs. Therefore KMR embraces both

quantitative and qualitative approaches, but interrogation of these methods is required in relation to cultural sensitivity, cross-cultural reliability and useful outcomes for Māori (IRI and TRRHEP 2000).

Te Roopū Rangahau Hauora a Eru Pōmare (TRRHAEP) is the Māori health research centre who formed part of the collaborative team in the present study. TRRHAEP have a Māori health focus and commitment to research that contributes positively to Māori health development. For TRRHAEP, KMR means (cited in Harris 2003):

> *Prioritising Māori in the questions asked and the processes chosen. Not a set of methods, but rather an approach to the way Māori research is framed. Research controlled by Māori. This applies to research that may also involve the general population but is driven by Māori priorities for the purpose of improving Māori outcomes.*
>
> - *Using culturally safe processes.*
> - *Generating solutions and aspirations from within Māori realities.*
> - *That there is a notion of action and commitment to change.*
> - *A growing and evolving methodology.*

The KMR principles outlined above form the foundations of the present study. In addition, a number of concepts outlined by TRRHAEP for the use of quantitative methods in KMR are utilised in the present study. Firstly, accurate classification and description of ethnicity data are considered as fundamental in undertaking a disparities analysis between Māori and non-Māori.

Secondly, the principle of *equal explanatory power*, which refers to *equal study power* and *equal power of explanation* or *equal analytical power*, is seen as vital in the quality, outcomes and effectiveness of this kind of research for Māori. This principle recognises the statistical needs of Māori as having equal status with those of the total New Zealand population (Robson 2002).

To achieve equal study power for Māori and non-Māori, the simplest method is to seek equal numbers of Māori and non-Māori responders, which requires stratifying a sample by ethnicity. This method provides a means of producing information for Māori health development to at least the same depth and breadth as obtained for non-Māori health development. If the present study did not stratify for ethnicity in the sampling, the sample would have included approximately 85% non-Māori and only 15% Māori. The

overall findings of the study therefore would have typically favoured the numerically dominant, and would not adequately reflect Māori realities (Robson 2002).

The other essential component of equal explanatory power is equal power of explanation (*equal analytical power*), which refers to the power of definition, explanation and meaning (Robson 2002). Māori researchers are therefore required to have a key role in all determining aspects of the study, which was the case in the present study. It is suggested that research of this nature may have greater legitimacy in Māori communities (Smith 1999).

In summary, KMR in the present study positions Māori as central in the research design, analysis and utilisation of findings and sets Māori health in a broader historical, social, economical and political context. Adopting this methodology contributes positively to Māori health development by providing information to inform health policy, health service configuration, and practices that benefit Māori.

OBSTRUCTIVE SLEEP APNOEA SYNDROME

2.6 Introduction

Obstructive sleep apnoea syndrome (OSAS) is characterised by various signs and symptoms, but specifically by the occurrence of repetitive episodes of airflow reduction (hypopnoea) or cessation (apnoea) due to upper airway obstruction during sleep. Loud snoring, excessive daytime sleepiness and a reduction of blood oxygen levels usually accompany these episodes of upper airway obstruction (American Sleep Disorders Association 1997). OSAS is not a condition that develops spontaneously, but rather it is a progressive disease (Pendlebury et al. 1997, Lindberg et al. 1999), and it forms part of a spectrum of sleep-related breathing disorders, which includes the upper airway syndrome (UARS), central sleep apnoea (CSA) and simple snoring. It is estimated to affect at least 2-4% of middle-age men and women (Young et al. 1993), and it is associated with substantial comorbidity, including obesity, hypertension, diabetes, and cardiovascular disease, highlighting its broad public health importance (Nieto et al. 2000, Peppard et al. 2000b, Newman et al. 2001).

Although OSAS is being increasingly recognised as an important problem in children, and there are similarities in some aspects of pathophysiology and consequences, the aetiology and associated morbidity are considerably different (Young et al. 2002a). Therefore the primary focus of this thesis is OSAS and its consequences among adults.

2.7 Classification and Diagnosis

The International Classification of Sleep Disorders (ICSD) (American Sleep Disorders Association 1997) is the classification system most widely used to diagnose OSAS (Buysse et al. 2003) and is endorsed by the American Academy of Sleep Medicine and other professional sleep societies. More recently the American Academy of Sleep Medicine (1999) produced some recommended standard definitions, criteria and severity ratings for OSAS to facilitate comparability between research and clinical practice.

The diagnostic criteria for OSAS according to the American Academy of Sleep Medicine (1999) are as follows:

> An individual must fulfil criterion A or B, plus criterion C.
>
> A. Excessive daytime sleepiness that is not better explained by other factors;
>
> B. Two or more of the following that are not better explained by other factors:
>
> - Choking or gasping during sleep
> - Recurrent awakenings from sleep
> - Unrefreshing sleep
> - Daytime fatigue
> - Impaired concentration;
>
> C. Overnight monitoring demonstrates five or more obstructed events per hour during sleep. These events may include any combination of obstructive apnoeas/hypopnoeas or respiratory effort related arousals (RERAs[1]).

The severity of OSAS has two components: the severity of daytime sleepiness and the frequency of respiratory events during sleep. For sleepiness, the following severity criteria are recommended:

[1] A RERA is defined as a sequence of breaths characterized by increasing respiratory effort leading to an arousal from sleep, but which does not meet the criteria for an apnoea or hypopnoea. RERAs are not included in the ICSD. Note: Prevalence and disease outcome data are scarce for RERAs as it is only a recently described event.

> **Mild**: Unwanted sleepiness or involuntary sleep episodes occur during activities that require little attention. Examples include sleepiness that is likely to occur while watching television, reading, or travelling as a passenger. Symptoms produce only minor impairment of social or occupational function.
>
> **Moderate:** Unwanted sleepiness or involuntary sleep episodes occur during activities that require some attention. Examples include uncontrollable sleepiness that is likely to occur while attending activities such as concerts, meetings, or presentations. Symptoms produce moderate impairment of social or occupational functions.
>
> **Severe:** Unwanted sleepiness or involuntary sleep episodes occur during activities that require more active attention. Examples include uncontrollable sleepiness while eating, during conversation, walking, or driving. Symptoms produce marked impairment in social or occupational function.

The following severity categories for sleep-related obstructive events are recommended:

Mild = 5-15 events/hour of sleep
Moderate = 15-30 events/hour of sleep
Severe = > 30 events/hour of sleep

In contrast to the identification of OSAS, which requires daytime sleepiness, OSA is identified on the basis of sleep-related obstructive events alone. The appropriate number of sleep-related obstructive breathing events for diagnosing a clinically significant entity has been the subject of much controversy and is still being debated in the literature. The use of five events per hour as a minimum threshold value is informed by epidemiological data (Young et al. 1993), which suggests minimal health effects, such as hypertension, sleepiness, or motor vehicle accidents. Currently there are no data to indicate an appropriate distinction between mild and moderate degrees of obstructed breathing events during sleep, therefore the recommended level of 15 events per hour is based purely on consensus opinion (American Academy of Sleep Medicine 1999)

In most sleep laboratories and research studies, sleep-related obstructive breathing events are defined by the number of obstructive apnoea and hypopnoea episodes per hour of sleep, as measured by the apnoea-hypopnoea index (AHI) or the respiratory disturbance index (RDI). The AHI is the average number of apnoeas and hypopnoeas

per hour of sleep and is most often used when referring to data from gold-standard polysomnographic monitoring. Similarly, the RDI is the average number of apnoea and hypopnoeas per hour of sleep and sometimes also includes RERAs. While the definition of *apnoea* is generally agreed upon, the definition of *hypopnoea* is not. A duration criteria of 10 seconds is generally agreed upon in adults, however, more variable definition features include the degree of airflow or respiratory effort reduction, inclusion and degree of oxygen desaturation, and inclusion of arousal from sleep. To compound the problem further, each of these is dependent on the method of detection. The implications of differing hypopnoea definitions and methods of detection have been extensively explored (Meioli et al. 2001). For example, a recent multicentered, community-based longitudinal study reported up to a 10-fold difference in the prevalence of OSA according to varying definitions of hypopnoea (Redline et al. 2000).

Across the literature, various terminologies are often used synonymously, including OSA, OSAS, sleep apnoea syndrome (SAS), and obstructive sleep apnoea hypopnoea syndrome (OSAHS). These conditions all fall within the broader category of sleep-disordered breathing (SDB). In the present study, the term OSA is used when referring to the number of sleep-related obstructive events and when combined with daytime sleepiness, the term OSAS is used to indicate a clinically relevant entity.

2.7.1 Measurement of OSA

Standard polysomnography (PSG) is the accepted gold standard for the diagnosis of sleep-disordered breathing. PSG consists of monitoring brain electrophysiological activity, eye movements, muscle tone (usually from the chin), heart rate and rhythms, respiration, blood oxygen levels, and leg movements. It has however been criticised as a method of evaluation due to its cost and inaccessibility (Pack and Gurubhagavatula 2003).

Increased awareness among the general public and health care professionals of the clinical and physiologic importance of SDB has led to an increased demand on limited clinical resources. Therefore a number of alternative strategies have been developed to decrease the number of PSG studies conducted including split-night PSG, which utilises the first half of the night to evaluate the presence of OSA and the second half to implement treatment (Rodway and Sanders 2003). Other strategies include the use of portable monitoring devices, ranging from devices that can record as many signals as

does attended PSG, to only one signal such as oximetry (Flemons et al. 2003). The different types of studies used in the evaluation of OSA are classified according to their recording ability, with Level 1 (standard PSG) considered the reference standard to which the other devices are compared (Figure 2.1).

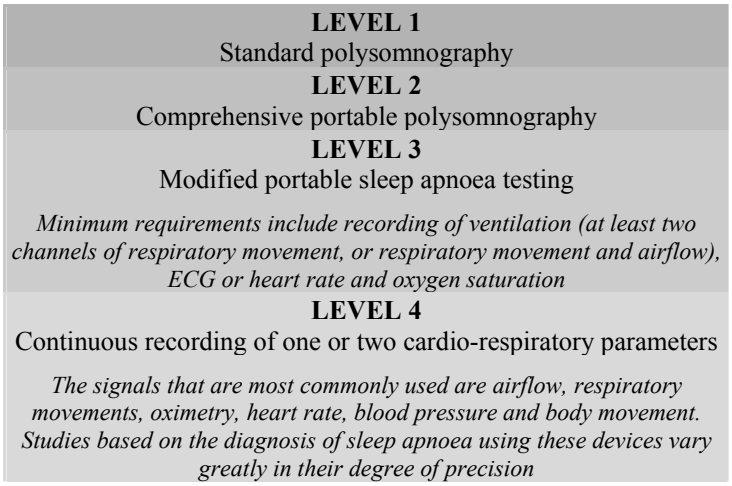


Figure 2.1 Types of studies for OSA evaluation
(Source: American Sleep Disorders Association 1994).

One advantage of portable devices, aside from cost effectiveness, is having the patient/participant in a familiar environment, which may provide a better appraisal of night time pathology then can be obtained in the unfamiliar laboratory setting. However, one downside of portable monitoring is that it is more susceptible to artefact (Whittle et al. 1997). In general, the risk of misdiagnosis is less likely with standard PSG than with other methods. A recent review of home diagnosis of sleep apnoea highlighted the need for more rigorous research to properly evaluate these alternative diagnostic strategies. It is suggested that this research should also consider more diverse populations of patients, including primary care populations, subjects with co-morbid conditions, different ethnic populations other than White, and women (Flemons et al. 2003).

In the community sample of the present study, the MESAM4 portable device (MAP, Martinsried, Germany) was used to measure OSA. It is classified as a Level 3 device. However, because it does not measure respiratory effort, it is not a classic Level 3 device, but it is considered under this category because it does involve monitoring

several channels of breathing variables. With this level of device, and those below it, a greater degree of subjectivity is needed in the classification of respiratory events. Level 3 monitors have been shown to reduce and increase the probability that a patient may have sleep apnoea in an attended setting. However, their reliability in an unattended setting is not well established (Flemons et al. 2003).

According to the American Sleep Disorders Association (1994) practice standards, portable devices in the assessment of OSA are an acceptable alternative only in the following situations: 1) when initiation of treatment is urgent and standard PSG is not available; 2) for patients not able to be studied in the laboratory; or 3) for follow-up when a diagnosis has been established. They also recommend that only Level 2 and 3 studies are acceptable for the diagnosis and assessment of therapy of OSA, as long as they include a body position sensor, as body position often affects the severity of obstruction, thus failure to identify this positional component may lead to inappropriate treatment (Ferber et al. 1994).

2.8 Pathophysiology

The pathophysiology of OSA is highly complex and still incompletely understood. The site of the upper airway obstruction lies in the pharynx. The specific site varies among OSAS patients, however the three primary sites commonly implicated are the oropharynx (tongue), hypopharynx, and the nasopharynx (soft palate) (Malhotra and White 2002).

During sleep, there is a considerable loss of tonic activity in the muscles of the upper airway, which predisposes it to collapse. Anatomical abnormalities of the pharynx and its associated structures are also common in patients with OSA. These abnormalities tend to decrease the cross-sectional area of the upper airway and/or increase the pressure surrounding the airway, both of which predispose the airway to collapse (Kuna and Remmer 2000, Sanders 2003).

The relative contribution of each of these pathophysiological mechanisms underlying OSA is also influenced by predisposing risk factors, including male sex, age, and obesity, of which obesity appears to be the most important (Neill and McEvoy 1997). Other factors such as sleep deprivation, sedatives, and sleep position have also been shown to aggravate OSA (Cartwright 1984). The supine position in sleep has been

found to cause more severe and frequent obstructions in the upper airway, by way of gravitational forces (Itasaka et al. 2000, Oksenberg et al. 2000, Nakano et al. 2003).

The pathophysiological changes associated with disrupted breathing lead to oxygen desaturation and an increased effort to breathe, and these are believed to lead to subsequent arousals, which cause sleep to be fragmented. One night of sleep fragmentation in normal subjects has been shown to significantly impair subjective assessment of mood and decrease mental flexibility and sustained attention (Martin et al. 1996). Furthermore, the cumulative effect of sleep restriction has clear measurable effects on neurobehavioral markers of alertness, sleepiness, mood disturbances, stress, and performance (Dinges et al. 1997). Therefore the impairment of sleep due to OSA accounts for the associated symptoms and complications (Neill and McEvoy 1997, Basirri and Guilleminaunt 2000, Verse and Pirsig 2003).

2.9 Clinical Symptoms and Presentation

Symptoms of OSAS that appear during sleep include snoring, witnessed apnoeas, disturbed nocturnal sleep, choking, drooling, nocturia, dyspnoea, and reflux. Daytime symptoms include profound sleepiness, waking unrefreshed, fatigue, depression and morning headaches. Compared to daytime symptoms, the nocturnal symptoms are more specific to OSAS (Bassiri and Guilleminault 2000).

Loud snoring in all positions and excessive daytime sleepiness (EDS) are the most common symptoms of OSAS in adults (Neill and McEvoy 1997). Snoring is a hallmark symptom of OSAS because it reflects the basic pathophysiology underlying the disorder. However not all OSAS sufferers snore, and not all snorers have OSAS. Furthermore, the relationship between EDS and nocturnal symptoms is not clear (Young et al. 1993, Bennett et al. 1998, Stradling et al. 1999). Some patients with severe OSA may present with minimal sleepiness. While at the extreme end of daytime sleepiness, the inability to control sleepiness may cause the patient to fall asleep while in conversation, eating, walking, or driving. A number of explanations have been postulated for such a poor association, including individual variability in the affect of arousals (Stradling et al. 1999) and individual resistance to sleep fragmentation (Bennett et al. 1998). Daytime sleepiness has also been shown to be a morbid characteristic of severely obese patients, most likely due to a metabolic and/or circadian abnormality of the disorder (Vgontzas et al. 1998).

OSAS symptoms also overlap with a number of other disorders including depression (Szuba 2001), hypothyroidism (Rajagopal et al. 1984, Kapur et al. 1998, Skjodt et al. 1999), and periodic limb movements (Stoohs et al. 2001). This highlights the need for careful inquiry about other symptoms in order to differentiate OSAS from these disorders.

2.10 Epidemiology

2.10.1 Prevalence studies

Understanding disease prevalence is important in assessing the health status and needs of the population. This section provides a review of OSAS studies in adults, along with a review of studies that have examined the prevalence of OSAS in different ethnic groups.

A number of studies have examined the prevalence of OSAS. However due to variation in methodology, direct comparisons are limited. Previous reviews have taken into account methodological issues by comparing studies with similar study design, or roughly adjusting for differences in definition. Davies and Stradling (1996) examined 12 studies of OSAS prevalence in Western populations, and using conservative approaches to account for methodological differences in study designs, they estimated that 1 to 5% of adult men have OSAS. Similarly, in a review by Lindberg and Gislason (2000) of 10 studies that used two-stage sampling methods, OSAS prevalence was estimated at 0.3%-5% for adults.

While these reviews provide prevalence estimates of OSAS, they do not account for the large number of adults who have OSA but do not report sleepiness. OSA regardless of the presence of sleepiness has been shown to have adverse health outcomes. It is therefore argued that the prevalence of OSA (as determined solely by abnormal breathing during sleep) is extremely important in understanding the potential burden of SDB in the population (Young et al. 2002a).

Estimates of at least mild OSA (AHI≥ 5) range from 3 to 28%, and for at least moderate OSA (AHI≥ 15), estimates range from 1 to 14%. Young et al. (2002a) conducted a refined review, including only studies that used standard polysomnography, had relatively large samples, and utilised two-stage stratified probability sampling methods with appropriate weighting techniques. These studies were the Wisconsin Sleep Cohort,

the Vitoria-Gasteiz Spanish Cohort, and the Southern Pennsylvania Cohort. The prevalence estimates for both OSA and OSAS were much in closer agreement than previous estimates.

The Wisconsin Sleep Cohort study (Young et al. 1993) is an on-going longitudinal study of the natural history of cardiopulmonary disorders of sleep. A questionnaire was distributed among a large group of male and female state employees aged 30 to 60 years. From questionnaire responders, a random sample of 602 participants was studied by overnight polysomnography. The estimated prevalence of SDB (AHI≥ 5) was 9% for women and 24% for men. With the addition of daytime hypersomnolence[2], 2% of women and 4% of men were found to have met the minimal diagnostic criteria for the sleep apnoea syndrome.

The Southern Pennsylvania Cohort study (Bixler et al. 1998, 2001) was conducted in a random sample from the general population. In phase 1, 12219 women and 4364 men ranging in age from 20 to 100 years were interviewed by telephone. In phase 2, 1000 women and 741 men from the Phase 1 participants were selected for one night of sleep laboratory PSG evaluation. The results of this study indicated that, for OSA (AHI≥ 5), men had a prevalence of 17% and women 7%. For OSAS (AHI≥ 10 and daytime symptoms), the prevalence estimates for men and women were 3.9% and 1.2% respectively.

The Vitoria-Gasteiz Spanish cohort (Duran et al. 2001) also consisted of participants from the general population aged 30-70 years. The first phase of the study was completed by 2148 participants (76.9%), and included a home survey, blood pressure measurement, and overnight monitoring with the MESAM4 monitor. In phase two of the study, participants with suspected OSA (n=442) and a subgroup of those with normal results (n=305) were invited to undergo polysomnography, of which 555 agreed. OSA (AHI≥ 5) was found in 26% of men and 28% of women. The authors suggest that the higher prevalence among women in this study compared with the Wisconsin study

[2] All three of the following symptoms were criteria for hypersomnolence: How often participants felt excessively sleepy during the daytime; woke up unrefreshed, regardless of how long they had slept; and had uncontrollable daytime sleepiness that interfered with daily living – responses of 'frequent' and 'habitual' (≥ 2 days per week) were considered to indicate hypersomnolence.

may be explained by differences of the health status of the population (healthy worker effect in Wisconsin study), and the difference in the age span used.

Of particular importance to the present study is the population study of SDB in Australian men (Bearpark et al. 1995), which utilised identical monitoring equipment (MESAM4) and similar scoring criteria. Two hundred and ninety four (60% response rate) men aged between 40-65 years were recruited from the Busselton Health survey. Twenty six percent of men were found to have SDB (RDI≥ 5). With the addition of daytime sleepiness criteria[3], the prevalence of OSAS was estimated to be 3.1%.

From these four studies it is estimated that up to 4% of men and 3% of women may have OSAS. In terms of abnormal breathing events alone, it is estimated that roughly 20% of adults have at least mild OSA (AHI≥ 5) and 6% have at least moderate OSA (AHI≥ 15). However, all these studies, are restricted to predominately White populations, and therefore may not be applicable to other ethnic groups.

Prevalence estimates for different ethnic groups

Differences in prevalence of OSAS by ethnicity are important for furthering the understanding of this syndrome and in the delivery of appropriate diagnostic and treatment services.

Ancoli-Israel et al. (1995) conducted a study in a group of randomly selected adults from a community dwelling population aged 65 years and older. The sample included 346 Caucasians and 54 African-Americans. African-Americans reported less satisfaction with sleep ($p = 0.017$), and also more difficulty falling asleep ($p < 0.001$). African-Americans were twice as likely to have RDI≥ 30 than Caucasians, independent of age, sex and BMI. Furthermore, the mean RDI for African-Americans with severe SDB (RDI≥ 30) was significantly greater than that for Caucasians (72.1 vs. 43.3, $p = 0.014$). The authors postulate that the elevated risk of severe SDB may be related to higher prevalence of hypertension or other conditions. However, they acknowledge that larger sample sizes are needed to accurately determine the reason for ethnic differences.

[3] Falling asleep during the day when you are not busy, not including planned naps –at least 'often'

In the Cleveland Family study (Redline et al. 1997), an ongoing community-based genetic epidemiological study, 225 African-Americans and 622 Caucasians aged 2 to 86 years were recruited as members of families with an individual with known OSAS (85 index families) or as members of neighbourhood control families (63 families). Participants were studied with a portable study (Level 3 study). African-Americans with SDB were found to be younger than Caucasians with SDB (37.2 yrs vs. 45.6 yrs, $p < 0.01$), independent of BMI, alcohol exposure or smoking status. The authors therefore concluded that the reason for racial differences may be due to differences in upper airway anatomy and possible physiological differences. Craniofacial risk factors in a subset of the sample (95 Caucasians, 41 African-Americans) were therefore tested. In Caucasians, both bony and soft tissue risk factors were identified. In contrast, in African-Americans, only increased upper airway soft tissue dimensions (tongue size and soft palate width) rather than bony features were identified. The small sample size may have contributed to the inability to detect bony risk factors among African-Americans. Furthermore, as the population used in this study was derived from neighbourhood controls and index, they may not be representative of the general population.

Other studies have focused on craniofacial form as explaining ethnic differences, which include an extension of the Cleveland Family study (Crakirer et al. 2001), and a study undertaken in New Zealand (Coltman et al. 2000) in a small group of clinical patients, which is discussed in Section 2.15.1. While these studies have found differences in correlations of craniofacial features to OSA, the significance of the results is difficult to interpret given that both studies were not necessarily representative of people with OSA in the general population.

In the Sleep Heart Health study (Young et al. 2002b), a longitudinal study of the cardiovascular consequences of SDB, comprising community dwelling adults from eight established cohort studies, data were collected by questionnaire, clinical examination and in-home polysomnography (Level 2 study) in 5615 men and women aged between 40-98 years. The sample consisted of 4330 White Americans, 418 African-Americans, 586 Native Americans and 281 'others'. The proportion of White Americans with SDB ($AHI \geq 15$) was 17%, compared with 20% of African-Americans, and 23% of Native Americans. After controlling for age and sex, SDB was not higher in African-Americans compared with White Americans, while the prevalence in Native Americans compared with White Americans was significantly higher (OR 1.70, 95% CI

1.37-2.11), however when BMI was controlled for, the difference between the two groups disappeared (OR 1.09, 95% CI 0.87-1.37).

Among clinic populations, ethnic differences in severity of OSAS have also been identified. In the US, a study of sleep clinic patients in a university based sleep disorders clinic found that African-Americans were younger, heavier and had more severe disease. The authors concluded that these results may indicate racial differences in facial structure, upper air-way muscle tone, or respiratory control in African-Americans (Scharf et al. 2003). In New Zealand, Māori and Pacific peoples referred to sleep clinic have been shown to be more likely to have OSAS and to be more severely affected than Pākehā (Baldwin et al. 1998).

Ip and colleagues (2001, 2004) provided the first estimates of OSAS prevalence in an Asian population of men and women aged 30 to 60 years, using two–stage sampling methodology and in lab polysomnography (Level 1 study). In the first phase of the study, sleep questionnaires were distributed to 1542 men and 1532 women, of which 784 men and 854 women responded. All questionnaire responders were then invited to undergo full polysomnography and 153 men and 106 women agreed. For men, the prevalences of OSA (AHI≥ 5) and OSAS (AHI≥ 5 and daytime sleepiness criteria[4]) was 8.8% and 4.1% respectively. For women, the prevalence estimates of OSA and OSAS were estimated to be 3.7% and 2.1% respectively. The prevalence estimates for OSA are considerably lower than those reported in predominately White populations (Young et al. 1993). On the other hand, the prevalence of OSAS was much closer. Increasing BMI and age were associated with SDB, however, the correlations were weaker than those reported in the Wisconsin study. The authors postulated that craniofacial risk factors may therefore explain the prevalence of OSAS in this population (Ip et al. 2002).

More recently, Udwadia et al. (2004) carried out the first epidemiological study estimating the prevalence of OSAS in India. A two-phase cross-sectional prevalence study was conducted in healthy urban Indian males aged 35-65 years, who attended

[4] (1) felt excessively sleepy during the daytime; (2) felt unrefreshed or tired during the day, regardless of how long they had slept; (3) fell asleep or dozed off momentarily while watching TV, reading, or at meetings/church; and (4) felt sleepy while driving. The answer was considered positive if the score was ≥2. Participants were identified as having excessive daytime sleepiness (EDS) if they gave a positive response to three of the four questions.

hospital for a routine health check. In the first phase of the study, 658 subjects (94%) returned completed questionnaires regarding their sleep habits and associated medical conditions. In the second phase, 250 of these underwent an overnight home sleep study (Level 3 study). The prevalence of OSA (AHI≥ 5) was estimated to be 19.5% (95% CI 16.50-22.50), and 7.5% for OSAS (AHI≥ 5 and daytime hypersomnolence[5]). While OSA prevalence estimates were similar to those from the benchmark study of Young et al. (1993), the prevalence of OSAS was significantly higher and is one of the highest rates reported in any epidemiological study. While the authors acknowledge that the cause of the high prevalence is unclear, they suggest that Indian facial and anthropometric characteristics might be responsible. However, the differences in criteria for sleepiness may also account for some of the difference in prevalence estimates.

This review raises a number of issues regarding ethnic differences in the prevalence of OSA and OSAS. Firstly, it highlights a lack of comprehensive data to assess the prevalence of OSAS among non-White populations. Although the studies of Ip et al. (2001, 2004) and Udwadia et al. (2004) are comprehensive studies, they are limited in their ability to inform ethnic disparities, as they were conducted among Chinese and Indians participants. In those studies that have indicated SDB to be more prevalent and severe among minority ethnic groups, the disproportionate numbers of non-White participants included in the samples do not allow enough statistical power to adequately inform ethnic inequalities. The explanation for ethnic differences in health are many and multilayered (Blakely and Dew 2004). Some of the hypothesised reasons for ethnic differences in OSA include a higher prevalence of co-morbid conditions which may influence prevalence and severity of SDB (Ancoli-Israel et al. 1995), differences in BMI (Young et al. 2002b), and genetic factors influencing craniofacial morphology (Redline et al. 1997, Cakirer et al. 2001). However, apart from BMI, evidence to support these hypotheses is lacking.

Although genetic research has mostly discredited the belief in a biological basis of racial groups, genetic variation continues to be used to explain racial or ethnic differences in biological risk for particular diseases in both epidemiology and medicine

[5] At least 3 or more days/weeks during the past 3 months in one or more of the following: after awakening, during free time, at work or driving, or during daytime in general.

(Pfeffer 1998). It is not surprising then, that reports of racial or ethnic differences in OSAS are most often genetic causes. Overemphasis on genetics as a major explanatory factor in ethnic health disparities may lead to a neglect of other important factors that contribute to disparities more substantially, and may also reinforce racial stereotyping, which may contribute to disparities in the first place. It also runs the risk of particular ethnic groups being seen as inherently biologically inferior to groups who enjoy better health (Sankar et al. 2004).

In light of these issues, more research is needed to examine ethnic prevalence patterns and the possible reasons for differences between groups. Meanwhile, regardless of why ethnic inequalities exist, the provision and response of services to meet differing needs amongst the community are extremely important public health issues (Young et al. 2002a).

2.10.2 Risk factors

The identification of risk factors is important in the recognition of groups most vulnerable to OSAS. It forms an important part of clinical case finding and provides important aetiological clues (Young et al. 2004). The most commonly reported risk factors for OSAS include sex, age, obesity, smoking, and alcohol intake. These risk factors often co-exist and therefore may collectively affect the severity of OSAS. Other suggested risk factors include craniofacial features, familial predisposition, nasal congestion, and race or ethnicity.

Sex

OSAS was previously thought to be a disease predominately of men, with reports of ratios as high as 10-90:1 for men compared to women presenting at sleep clinics (Bassiri and Guilleminault 2000). However, as shown in the above review, population-based studies indicate that the male:female ratio is approximately 2-3:1 (Young et al. 1993, Young et al. 2002b). The discrepancies between clinic and population estimates indicate that a bias favouring men for diagnostic evaluation has been operating. It is postulated that the sex disparity in OSAS diagnosis may be due to health care providers disregarding typical symptoms in women and/or perhaps underreporting of snoring among women (Young et al. 1996, Young and Finn 1998, Jordan and McEvoy 2002).

Considering the male predominance of OSAS, relatively few studies have investigated the specific role of sex. Past research has primarily focused on the role of sex hormones

as risk factors. It is well established that men have a greater tendency than women for android fat distribution that results in a greater degree of central and upper body fat distribution, which accounts for some of the increased prevalence of OSA in men (Millman et al. 1995). Furthermore in men, testosterone levels have also been shown to be associated with upper airway collapsibility in OSA patients. Others have suggested that the remaining sex differences may be accounted for by different upper airway muscle function during sleep (White et al. 1985, O'Connor et al. 2000). Although many factors have been identified as playing a role in sex differences, more research is needed to provide a better understanding of sex differences in aetiology, presentation, clinical management, and outcomes (Young et al. 2004).

Menopause

For women, the risk of OSAS has been shown to increase postmenopausally independent of BMI and other confounding variables (Bixler et al. 2001, Young et al. 2003). Recently, two large epidemiological studies have indicated that menopause is a significant risk factor for OSA in women and hormone replacement therapy (HRT) appears to be associated with reduced risk. It is postulated that progesterone levels may play a role in protecting women from OSA before menopause (Popovic and White 1998, Bixler et al. 2001). In the Southern Pennsylvania cohort study, the prevalence of OSA was low in pre-menopausal women (0.6%) as well as postmenopausal women with HRT (0.5%). In these women, obesity was the primary risk factor. Postmenopausal women without HRT had a significantly higher prevalence of OSA than pre-menopausal women (2.7 vs. 0.6%, $p = 0.02$) (Bixler et al. 2001). In the Wisconsin Sleep cohort study, women of post-menopausal status were four times more likely to have OSA (AHI>15) than pre-menopausal women (Young et al. 2003).

Age

It is suggested that there is a clear lessening in quantity and quality of sleep with age that appears to be more rapid in males (Walseben et al. 2004). The prevalence of OSA has been shown to increase with age, with a 2 to 3 fold higher prevalence in older people (≥ 65 years) compared with those in middle age (30-64 years) (Young et al. 2004). Using home monitoring (Level 3 study), Ancoli-Israel et al. (1991) found that 62% of 427 randomly selected elderly people aged 65 year and over had SDB (RDI≥ 10). While among middle-aged adults the prevalence of OSAS appears to steadily increase with age, the increase in prevalence has been shown to plateau in older people

(Young et al. 2004). The basis for strong relationships between aging and increased apneic activity is not well understood, it may be related to changes in sleep quality, cerebral function, muscle tone, obesity, cardiac function and lung function associated with aging. Bixler and colleagues (1998) found in a large group of randomly selected men aged 20-100 years, that the prevalence of OSA (AHI≥ 10) increased monotonically with age from 3.2% (95% CI 1.6-6.4%) to 23.9% (95% CI 15.7-34.9%) for the middle and older age groups (OR=9.4 95% CI 3.9-23.1). In terms of OSAS (AHI≥ 10 and daytime sleepiness, hypertension or other cardiovascular complication), a steady rise was seen in each 10-year age increment from 20-59 years (0.4%, 1.5%, 2.8% and 5.4% respectively). However, the age-specific prevalence declined in the 60-69 years and ≥ 70 years groups (4.2% and 2.5% respectively). These results suggest that the clinical significance of OSA may decrease among the elderly. The authors suggest that the diagnosis of OSAS should therefore be adjusted for age.

On the basis of these findings, it is suggested that OSA in older adults may be a condition distinct from that of middle age (Young 1996). There are a number of plausible explanations for the age trends reported, including cohort effects and measurement errors. A better understanding of sleep-related breathing disorders in older adults is still needed (Young et al. 2004).

Excess body weight

Obesity is highly prevalent among patients with OSAS. A number of studies have shown that increasing values of most measures of body habitus including body mass index, waist, hip and neck circumference, circumference ratios, and skin-fold thickness, are strongly related to SDB (Young and Finn 1998). Excess body weight has been hypothesised to alter breathing during sleep via multiple mechanisms including alterations in upper airway structure or function, and distribution of the relationship between respiratory drive and load compensation (Young et al. 2002a).

It is however unclear which measure of body habitus most closely relates to SDB. Several studies using multiple regression analyses have found neck circumference to be a better predictor of the AHI than general measures of obesity such as BMI (Stradling and Crosby 1991, Davies et al. 1992, Hoffstein and Mateika 1992, Flemons et al. 1994, Baldwin et al. 1998). However, others have found waist circumference to be a better predictor than either BMI or neck circumference in men (Grunstein et al. 1993, Deegan and McNicholas 1996).

Hoffstein and Mateika (1992) measured the neck and abdominal circumferences in a large group of patients suspected of having OSAS (n=670). When matched for BMI and age (n=156), abdominal circumferences were similar, but the neck circumference was significantly higher in OSA patients (41.2cm vs. 39.1cm, $p < 0.0001$). In contrast, in a study by Grunstein et al. (1993), of 1464 consecutive men who underwent sleep studies, waist circumference ($r^2 = 0.156$, $p < 0.001$) was found to be a better predictor for OSA than either neck circumference or BMI, which suggests that the link between obesity and OSA cannot be explained solely by neck fat deposition. Similarly, Deegan and McNicholas (1996) found that, after controlling for BMI and age, waist circumference correlated more closely with the AHI than neck circumference among men, while the opposite was true among women.

The variation of findings across these studies may relate to the fact that measures of body habitus are correlated, some highly so. Differences in findings may therefore reflect varying degrees of measurement accuracy or perhaps statistical problems with the variables being strongly interrelated, especially in smaller samples.

Alcohol consumption

Alcohol consumption has been demonstrated to reduce the activity of the muscles that maintain the patency of the upper airway, which consequently predisposes the upper airway to collapse (Bassiri and Guilleminaunt 2002). Experimental studies have shown that alcohol aggravates OSA, especially when consumed around bedtime (Scrima et al. 1982, Tsutsumi et al. 2000). Population-based studies have not consistently demonstrated significant associations between self-reported alcohol consumption and OSA. While some have shown associations (Jennum et al. 1992), others have not (Bearpark et al. 1995, Olson 1995a). In addition, the effect of long-term alcohol use patterns on the development or progression of OSA is not yet known (Young et al. 2004).

Cigarette smoking

Smoking is often mentioned as a possible risk factor for OSA, and while there are several plausible mechanisms for a role of smoking in OSA, there are only a few studies that have shown an association (Wetter et al. 1994). The upper airway inflammation and associated airway disease caused by smoking is hypothesised to increase vulnerability to OSA. In addition, declining blood nicotine levels have been shown to affect sleep stability (Young et al. 2004). In a Tucson Epidemiological study (n=2187),

current cigarette smoking was found to be an independent risk factor for snoring, even after control for male sex, age and obesity. Snoring prevalence was also found to remain elevated in subjects who had recently quit smoking, but declined in ex-smokers to the level of non-smokers within four years of smoking cessation. In the Wisconsin Sleep Cohort, current smokers were three times (95% CI 1.4-6.4) more likely to have OSA than those who had never smoked. Former smokers on the other hand, were not more likely to have OSA (AHI≥ 5) than those who had never smoked. Interestingly however, findings from the Sleep Heart Health Study showed an inverse association between current smoking and OSA after controlling for several factors including age and BMI. Smokers had significantly fewer respiratory disturbance events. The authors speculate that the participants with severe OSA may have been more likely to quit smoking (Newman et al. 2001).

2.11 Consequences of OSAS

There are a number of adverse consequences associated with OSAS. The examination of the outcomes of OSA is important in assessing the cost of the untreated disease to society, as well as to the sufferer (Young and Finn 1998).

2.11.1 Excessive daytime sleepiness, cognitive function and quality of life

Daytime sleepiness is one of the most commonly recognised co-morbidities of OSA, and it appears to result from the recurrent arousals from sleep, triggered by repetitive episodes of partial or complete obstruction to the upper airway (Bennett et al. 1988). It is now recognised that sleepiness is also related to milder forms of OSA. As shown in the Sleep Heart Health Study (SHHS) cohort, excessive daytime sleepiness (defined as an Epworth sleepiness scale[6] ≥ 11), was 33% more likely in those with mild OSAS (AHI 15-29) and 67% in those with moderate to severe OSAS (AHI >30) (Gottlieb et al.1999).

The recognition of reduced quality of life (QOL) as an important outcome stems from the recognition that excessive daytime sleepiness, abnormalities in mood, and physical limitations are common in OSAS and therefore may contribute significantly to impaired

[6] This scale requires the participant to rate their likelihood of dozing in eight common, soporific situations. Each question is scored on a Likert scale from 0 (would never doze) to 3 (high chance of dozing). The scores are then added together and a sleepiness score is derived ranging from 0 to 24.

QOL. QOL refers to an individual's perception of their overall well-being based on functional ability, health and satisfaction with important dimensions of their lives (Reimer and Flemons 2003). Findings from the Wisconsin Sleep Cohort suggest poor QOL may be observed across the range of OSAS severities (Finn et al. 1998). However, participants in the SHHS with mild to moderate SDB were found to have reduced quality of life, but only in one specific area, while those with severe SDB had significantly poorer scores on all QOL scales (Baldwin et al. 2001).

A wide range of cognitive functional deficiencies have been demonstrated in clinic-based studies in patients with severe OSAS, which include general intellectual ability, learning and memory, sustained and focused attention, information processing efficiency, and visual and psychomotor performance. However, it is now recognised that earlier studies may have overestimated the effects of OSAS on neurocognitive behaviour, due to limitations in study design (Young et al. 2002a, Redline 2002). Population-based studies suggest that milder to moderate levels of SDB may have little impact on cognitive performance (Boland et al. 2002). Thus the public health impact of impaired cognitive function among OSAS suffers is not yet clear (Young et al. 2002).

Treatment with continuous positive airway pressure (CPAP) appears to improve QOL, mood disturbances, neurocognition and also appears to reduce sleepiness (Flemons and Tsai 1997, Bennet et al. 1999, Yu et al. 1999, Moyer et al. 2001).

2.11.2 Motor vehicle accidents (MVAs)

A number of population and clinical-based studies have found that patients with OSAS have an increased risk of automobile accidents. The increased risk has been attributed to a number of different factors including daytime somnolence, decreased vigilance, and impaired psychomotor reaction time (Young et al. 1997a, Barbe et al. 1998, Teran-Santos et al. 1999, Risser et al. 2000, Masa et al. 2000, Yee et al. 2002, Sassani et al. 2004). Patients with moderate to severe OSAS have been shown to have up to a fifteen-fold increase in risk of motor vehicle accidents compared to controls (Horstmann et al. 2000). Overall, the evidence for increased risk of MVA in patients with OSAS is robust and there is reasonable evidence to suggest that accident risk can be reduced by effective treatment with CPAP (Marshall et al. 2003).

2.11.3 Co-morbid conditions

Obstructive sleep apnoea has been shown to be associated with a number of medical diseases including diabetes, hypertension, coronary artery disease, myocardial infarction, congestive heart failure, and stroke. These associations however, may be due in part to risk factors common to all these conditions, or they may reflect a role of OSA in the aetiology of these conditions (Young et al. 2002a). Prior to diagnosis and treatment, patients with severe OSAS have been shown to be heavy consumers of health care resources compared with matched controls. Cardiovascular disease (CVD) and especially hypertension accounted for most of the increased utilisation (Ronald et al. 1998, Bahammam et al. 1999, Smith et al. 2002).

OSA has been associated with a number of cardiovascular changes including morning headaches, systemic and pulmonary hypertension, cardiac arrhythmias, and non-organic impotence (He et al. 1988). However, despite the large number of cross-sectional or case-controlled studies describing the associations, the issue of whether OSA independently increases the risk of CVD has been a contentious one (Leung and Bradley 2001). The difficultly in unravelling the relationship relates to the numerous potential confounding variables (such as obesity), the difficulty in comparing studies, and until recently there has not been a suitable animal model to study the long-term cardiovascular effects of OSA.

Hypertension is one of the most extensively studied outcomes of OSAS, however, until recently the relationship between the two has been ambiguous (Redline 2002). The mechanisms through which OSA promotes hypertension are not fully understood, but evidence suggests that the intermittent hypoxia and sympathetic nervous system activation play central roles (Leung and Bradley 2001). The prevalence of OSA is considerably high among patients with hypertension. More than 40% of patients with OSA are reported to have daytime hypertension, while about 30% of middle aged men with primary hypertension are thought to have occult OSA (Bassiri and Guilleminaut 2000). Recent findings from the Wisconsin Sleep Cohort study offers the most convincing evidence to date that OSA may have an independent effect on daytime hypertension, but the results suggest that the relationship is only modest. Furthermore, the potential for reducing blood pressure by treating OSA is still unclear (Young et al. 2002a).

OSAS has also been shown to be independently associated with increased risk for insulin resistance and glucose intolerance, which may explain part of the increased cardiovascular morbidity and mortality associated with OSAS (Coughlin et al. 2004). In a study of 270 consecutive patients (197 men, 73 women) referred for suspected OSAS without known diabetes, SDB parameters (AHI and minimum oxygen saturation) were significantly associated with an increased risk of insulin resistance, which was independent of obesity (Ip et al. 2002). In another study, which included 150 healthy men from the community without diabetes or cardiopulmonary disease, OSA (AHI≥ 5) was independently associated with an increased risk of having impaired or diabetic glucose tolerance (OR 2.15, 95% CI 1.05-4.38, $p<0.0001$) after adjusting for BMI and body fat percentage (Punjabi et al. 2002).

2.11.4 Mortality

Retrospective studies of clinical cohorts have suggested that people with untreated OSA are at greater risk for early mortality that is secondary to CVD or MVA. These studies however suffer from a number of methodological weaknesses, including small sample sizes, lack of controls, and problems with study design (Redline 2002, Young et al. 2002a). In a recent cohort study of 444 OSAS patients followed for at least 4 years post-diagnosis, mortality in treated patients was significantly lower than in those who did not follow treatment. In addition, the untreated patients showed excessive mortality compared with the general population, after adjusting for age and sex. Stratification by age showed a greater mortality rate ratio in patients less than 50 years of age, independent of mortality from cardiovascular causes (Marti et al. 2002).

In general, the available data suggests that increased mortality is likely in people with severe OSAS, particularly in those in whom OSAS first appears in middle life or earlier. However, precise estimates of the magnitude of the association are still unknown (Redline 2002).

2.12 Treatment and Management

There are a variety of different treatment options available in the management of OSAS. These can be divided into general and specific treatments. General treatments tend to be more conservative and include weight loss, change in sleep position, smoking cessation and avoidance of alcohol and sedatives. Specific treatments on the other hand, aim at directly treating the cause of the obstructive events.

As obesity is one of the major risk factors for OSA, weight loss is considered an important component of treatment (Smith et al. 1985, Peppard et al. 2000a). However, focusing purely on weight loss has been shown to be ineffective, especially in those who have severe OSAS. Aside from weight loss, increased exercise may also decrease the severity of OSAS. A study by Peppard and Young (2004) found that a lack of exercise was associated with increased severity of SDB independent of body habitus. Sleep position modification (e.g., sewing a golf ball into the back of a patient's sleepwear to encourage a lateral sleep position) can be useful in patients where OSA is present predominately in the supine position (Neill and McEvoy 1997).

The most common and effective method of treatment for OSAS is nasal Continuous Positive Airway Pressure (CPAP). This treatment maintains a patent airway during sleep by splinting the airway with positive pressure through a nasal mask, which has been shown to eliminate apnoeas, sleep fragmentation, and consequent hemodynamic changes. The optimal pressure is normally determined during the course of a diagnostic study, where a technician is available to adjust the CPAP until the pressure is found that best relieves the patients disordered breathing events. Although CPAP is an effective treatment, compliance can be a problem. Studies have shown compliance ($\geq$ 4.5 hours per night) rates from 40-80% (Grunstein and Sullivan 2000, Verse et al. 2003). Adverse effects of CPAP are generally related to the pressure or airflow or the mask-nose interface.

Other methods of treatment include dental devices, which are generally used in patients with mild OSA and those with moderate to severe OSA who are intolerant of, or refuse, CPAP. These devices fall into two main categories: those which hold the tongue forward and those which reposition the mandible forward during sleep (Lowe 2000). Surgical procedures are generally used in patients who are unable or unwilling to comply with medical management. Surgical procedures include nasal reconstruction, uvulopalatopharyngeoplasty (UPPP), maxillomandibular advancement, and tracheotomy (Neill and McEvoy 1997, Riley et al. 2002).

2.13 Public Health and Sleep Service Issues

A number of broader issues also exist in the management of OSAS, which relate to public health approaches to OSAS and service provision. OSAS is common in the general population and is seen to be steadily increasing. A study in the US estimated that 93% of women and 82% of men who met the recommended criteria for moderate to severe OSAS (AHI≥ 15 and often or almost always extremely sleep in daytime) remain undetected (Young et al. 1997b).

Given the known morbidity and mortality associated with OSAS, untreated OSAS is estimated to impose significant economic and social costs to society. Costs are associated with diagnosing and treating the condition, costs of treating medical conditions that may be exacerbated by OSAS, diminished work productivity due to direct effects of OSAS or due to complications of associated co-morbidities, and the cost of accidents (Fischer and Raschke 1997, Ronald et al. 1998, Bahammam et al. 1999, Kapur et al. 1999, Redline 2002). As treatment of OSAS provides many benefits to patients and society, it is imperative that strategies are developed to address the recognition and management of OSAS.

Current sleep services for adults are under resourced and are inadequate to meet clinical demands (Flemons et al. 2004). Concerns have also been raised about possible selection bias that favours men (Young and Finn 1998), and limited access to sleep medicine services for minority groups and the poor (National Commission on Sleep Disorders 1993).

The primary care setting plays an important role in the identification of OSAS. However, research indicates that OSAS is grossly under recognised in this setting. Primary care physicians are relatively under informed about the clinical features and medical and social ramifications associated with OSAS (Kramer et al. 1999). Preliminary results from a study in New Zealand indicate similar findings (Dr Jai Sood 2004, pers. comm.). It is therefore likely that the majority of New Zealanders with sleep disorders are undiagnosed, misdiagnosed or inappropriately treated or referred. The failure to recognise sleep disorders in primary care constitutes a major personal and public health crisis that must be addressed (Kramer et al. 1999, Dement and Netzer 2000). Tools have been developed which may help primary care physicians in

recognising sleep disorders, but few have been validated in this setting (these are discussed in the following section).

Of particular importance, from a public health perspective, is identifying cost-effective prevention and intervention strategies (Young et al. 2002a). Current population-based strategies for weight reduction may decrease OSA severity and progression, and may prevent its occurrence. Nasal congestion, hormonal change with menopause, and smoking are also promising modifiable risk factors, however more investigation is required. In addition, widespread education of health professionals and the general public will go far in solving the problems of OSAS (Dement and Netzer 2000).

2.14 Clinical Prediction Tools for OSAS Screening

Screening in medicine and epidemiology has taken on somewhat different applications. Population screening refers to the organised application of a diagnostic test in largely asymptomatic or unrecognised symptomatic individuals. Clinical screening on the other hand refers to a series of intermediate tests performed on a symptomatic patient for whom a diagnosis has not yet been established (Baumel et al. 1997). The validity of a screening test is measured by its utility to do what it is intended to do. Generally, it is highly desirable to have a screening test that is both highly sensitive and highly specific, but this is not always possible and generally there is a trade-off between sensitivity and specificity (Henneken and Buring 1987). This trade-off has to do with the fact that, for many clinical tests, there are some people who are clearly normal, some who are clearly abnormal, and others who fall somewhere between the two, which is often termed the grey-zone (Coste and Pouchot 2003).

In regards to identifying OSAS, the decisions faced by clinicians are different depending on the setting. In a primary care setting, the required decision is usually whether or not to refer the patient to a sleep specialist. On the other hand, in a sleep clinic setting, the decision is whether the patient requires immediate treatment, PSG, portable home monitoring, or whether no further testing is required. The threshold that has to be crossed to do something lies on the spectrum of the probability that a patient has OSAS, and will be influenced by an estimation of the impact that OSAS is having on the patient's quality of life, as well as by the presence or absence of coexisting illness (Flemons and Whitelaw 2002).

One of the goals of this thesis was to develop a clinical prediction model to screen for OSAS in a primary care setting. This section therefore provides a review of other studies that have formulated clinical prediction models. This review is restricted to models that utilise primarily self or partner reported variables, and prediction models requiring extensive physical examination are excluded (e.g., Kushida et al. 1997, Tsai et al. 2003). A number of methodological issues impact on the comparability of these studies, including different analytical methods, differences in variables tested, different sample populations, and different goals.

As many of the variables tested in these studies are interrelated, it follows that many different potential statistical models are possible. Essentially, the prediction models can be considered as clinical screening tests that provide an estimate of the likelihood that a patient has or does not have OSA, as defined by some arbitrary AHI or RDI cut-off (Flemons and McNicholas 1997).

Crocker et al. (1990) developed a prediction model using data from 100 consecutive patients who had been referred for suspected OSAS. The prevalence of OSA (AHI> 15) in this group of patients was 27%. Logistic regression modelling identified observed apnoeas, hypertension (past or present), increasing BMI, and increasing age as significant independent predictors of OSA. A probability cut-off point of ≥ 0.15 was selected to minimise the number of subjects with false negative predictions. When tested in an independent group of patients (n=105), the model correctly classified 33 of 36 patients with OSA (sensitivity = 92%) and 35 of 69 patients without OSA (specificity = 51%).

Viner and colleagues (1991) examined whether clinical history and pharyngeal examination could serve as a sensitive screening test for OSA in a sleep clinic setting. Data were collected from 410 patients (388 men, 72 women) referred for suspected OSAS. The prevalence of OSA (AHI> 10) in these patients was 46%. Using logistic regression modelling, increasing age, BMI, male sex, and snoring were identified as significant independent predictors of OSA (AHI$\geq$ 10). For patients with a predicted probability of less than 0.20, the model yielded a sensitivity and specificity of 94% and 28% respectively. The authors concluded that clinical features did not reliably predict OSA in patients suspected of having the disorder. However, among patients assigned a low probability of having OSA, the model was sufficiently sensitive to allow about a 30% reduction in the number of unnecessary sleep studies.

Flemons and colleagues (1994) randomly selected a series of 180 patients (134 men, 46 women) referred to a tertiary sleep clinic for suspected OSAS. Using linear regression, a sleep apnoea clinical score (SACS) was derived using neck circumference, hypertension, habitual snoring, and reports of nocturnal choking or gasping. Patients with all four predictive variables had a likelihood ratio and post-test probability of OSA (AHI≥ 10) of 5.17 (95% CI 2.54-10.51) and 81% respectively. In contrast, patients with the lowest SACS (<5) had a likelihood ratio of 0.25 (95% CI 0.15-0.42) and a post-test probability of OSA of 17%.

The advantage of likelihood ratios used by Flemons et al. (1994) is that they can be used to convert pre-test probability to post-test probability using a simple nomogram[7]. Likelihood ratios can therefore be more stable when applied to different populations, as the pre-test probability of disease is taken into account. While this approach has merit, it requires the clinician to have a broad enough experience to enable them to derive an accurate value for the likelihood that the patients do have the disease (pre-test probability) (Flemons and Whitelaw 2002). It has been suggested that in a primary care population, population prevalence estimates (i.e., 2% women and 4% men) may be used as pre-test probability estimates. However, based on US and European estimates, the prevalence of OSA in primary care is approximately 35% (Netzer et al. 2003).

More recently, Flemons (2002) provided a simplified prediction rule based on neck circumference, to estimate a patient's probability of having a positive diagnosis of OSA. Neck circumference is adjusted if the patient has hypertension (4 cm is added), is a habitual snorer (3 cm is added) or is reported to choke or gasp most nights (3 cm is added). A low clinical probability corresponds to an adjusted neck circumference of less than 43 cm, an intermediate probability (4-8 times as probable as a low probability) to a neck circumference of 43 to 48 cm and a high probability (20 times as probable) to a neck circumference of more than 48 cm. The advantage of this rule is that the likelihood of disease can be easily calculated without requiring auxiliary devices.

Maislin et al. (1995) developed and assessed a prediction tool in a multi-centred sleep study with a group of self-referred patients. Using confirmatory factor analysis, a self-

[7] A nomogram is based on Baysiean Theory and is used to convert pre-test probability to post-test probability, using likelihood ratios

report symptom frequency index for apnoea was derived (Index 1). Multiple logistic regression analysis was then used to develop a multivariable apnoea risk (MAP) index, which included Index1, age, sex, and BMI. Using a cut-off point of 0.50 to discriminate between those with high and low risk of OSA (RDI≥ 10), the sensitivity and specificity of the model were 88% (95% CI 84-92) and 55% (95% CI 48-62) respectively. The authors concluded that operating characteristics of the models were in a range consistent with potential clinical utility. The MAP index has been utilised in other studies to stratify participants according to their risk for OSA. George et al. (2003) in a study to assess the prevalence of SDB in football players, used the MAP to stratify football players into high (MAP >0.50) and low (MAP <0.5) risk for SDB prior to additional tests. The MAP has similarly been used in a study of OSA prevalence in commercial truck drivers (Gurubhagavatula et al. 2004).

The receiver operating characteristic (ROC) curves[8] of Cocker et al. (1990), Viner et al. (1991) and Flemons et al. (1994) have previously been compared using the same patient data set (Flemons et al. 1994). Similar diagnostic characteristics and predictive power were found across models. More recently, Rowely and colleagues (2000) prospectively evaluated the clinical utility of the prediction rules of Crocker et al. (1990), Viner et al. (1991), Flemons et al. (1994), and Maislin et al. (1995) in 370 patients (191 men, 179 women) referred to a sleep clinic for suspected OSAS. The prevalence of AHI≥ 10 and ≥ 20 was 67% and 49% respectively in this sample. The probability cut-off points from the original studies were used to differentiate between patients with or without OSA. All of the models demonstrated reasonable sensitivity (76%-96%), but they were not very specific (13%-54%). The positive predictive values ranged from 69%-77%. The clinic prediction rule of Flemons et al. (1994) yielded the highest specificity. Interestingly, all four models performed better for men than for women. The authors postulated that because the overall test population were extremely obese, the probability of OSA in the non-OSA group was most likely overestimated by these models, which led to a higher number of false positives. Furthermore, the lack of discriminatory ability shown amongst women was hypothesised to be due to the fact that the women were more obese than the men, but had a lower prevalence of OSA.

[8] A graphical presentation of the relationship between true positives and false positives at different thresholds.

In an earlier study by Kapuniai et al. (1988), the usefulness of questions from the Hawaii Sleep Questionnaire was assessed. Using stepwise multivariate discriminant analysis, witnessed apnoeas and loud snoring were identified as potential predictors of OSA (AHI > 10). While the authors acknowledged that BMI would also be a useful variable, it was not included because they were concerned that the additional computational steps might deter use in a clinical setting. A score of 1 was assigned for the presence of a predictor and a score of 2 indicated probable OSA. When tested in an independent group of patients (n=53) referred for suspected OSA, a score of 2 correctly identified 100% of the cases with severe sleep apnoea (AHI > 40) and 70-76% of those with AHI> 5. However, this was offset by a substantial number of false positives. This study is limited in that data from only 22 volunteers and 23 sleep clinic patients were used to construct the model, and because the predictive variables were assumed to have equal weightings.

All the models discussed thus far have been developed in populations with symptoms suggestive of OSAS. While several studies in the general population, and in referred patients, have explored the relationship between OSA and clinical features, these studies have not specified the results of the statistical analyses in sufficient detail to allow a clinical prediction model to be created (Stradling and Crosby 1991, Grunstein et al. 1993, Hoffstein and Mateika 1992). One such study was by Kump and colleagues (1994), who collected data from 465 participants who were part of an ongoing genetic-population study (The Cleveland Family Study, discussed previously). Logistic regression analysis demonstrated that increased apnoea activity was best predicted by three questions about intensity of snoring, roommate-observed choking, and having fallen asleep while driving. Use of symptoms with data on sex and body mass index (BMI) improved predictive ability by 10%. The authors concluded that their questionnaire could be used as a screening tool for assessing symptoms of OSA in population samples.

Similarly, Hoffstein and Szalai (1993) examined the predictive value of history and physical examination in the diagnosis of OSAS in a group of 594 (471 men 123 women) patients referred to the sleep clinic for suspected OSAS. Stepwise multiple linear regression analysis found that age, sex, BMI, bed partner observation of apnoea and pharyngeal examination were significant predictors of AHI, explaining 36% of the variability.

A study by Netzer and colleagues (1999) tested the utility of the Berlin Questionnaire as a means of identifying patients in primary care with a high or low risk of OSA. The Berlin Questionnaire was an outcome from a conference on sleep in primary care, which involved 120 US and German pulmonary and primary care physicians. Questions were selected based on consistent risk factors of OSA shown in the literature. Risk grouping was based on responses in three symptom categories: 1) the presence and frequency of snoring behaviour; 2) daytime sleepiness or fatigue; and 3) a history of obesity or hypertension. Seven hundred and forty four patients were recruited from five primary care sites in Cleveland, Ohio to complete the questionnaire. Of these, 279 (37.5%) were categorised as having a high-risk of OSA. To evaluate the risk-groupings, 69 patients classified as high-risk and 31 patients classified as low risk underwent portable sleep monitoring (Level 3 study). For patients assessed as high risk, RDI > 5 was predicted with a sensitivity of 86%, a specificity of 77%, a positive predictive value of 89%, and a likelihood ratio of 3.79. With increased severity (RDI> 15), the sensitivity was 54%, specificity was 97%, and the likelihood ratio was 16.62. The authors concluded that the Berlin Questionnaire is a useful method to detect important symptom distributions and permit risk groups to be identified in the absence of a physician patient encounter.

The usefulness of the Berlin questionnaire in a primary care setting has, however, been questioned. Based on an assumption that the pre-test probability of OSA in a primary care setting is 4% (the prevalence of OSAS in men), a positive response of the Berlin Questionnaire (indicating high risk) yields a post-test probability of only 11%, which is probably not high enough to warrant referral to specialist services (Nardone 2000). However, the prevalence in primary care has been shown to be much higher, about 35%, which means that the Berlin questionnaire would produce a post-test probability of 60%, which is within a moderately useful range for clinical decisions (Netzer and Strohl 2000, Netzer et al. 2003). The authors also commented that the questionnaire was not designed to replace clinical reasoning (Netzer and Strohl 2000).

This review highlights the variables that do and do not predict OSA. Most studies have found a relationship between the AHI and anthropometric variables such as BMI, neck circumference and some type of abnormal respiration during sleep (snoring, apnoeas, choking or gasping) witnessed by a bed partner. However, reports on the usefulness of features such as age, sex, hypertension, alcohol consumption and daytime sleepiness are

diverse (Flemons and McNicholas 1997). The inability of some pertinent risk factors to discriminate between patients with and without breathing disturbances may be attributable to selection bias in the clinical samples used.

In general, the majority of models have tended to produce an excess of false positive identifications, which is not surprising given that they were developed in symptomatic patients. This suggests that they would be overly cautious when used as screening tools, which is obviously safer than having an excess of false negative identifications. However, several of these models have been found to be superior to the judgement of expert clinicians, and are useful in deriving pre-test probability estimates (Crocker et al. 1990).

Although potentially useful measurement instruments, particularly in the context of directed clinical assessment, most of the models are inadequate as stand-alone diagnostic instruments. Furthermore, the majority of models have been developed in predominately White populations consisting mainly of men, so their accuracy requires validation in other populations, especially in different ethnic groups, women and those with lower expected prevalence rates of OSAS.

2.15 Sleep Research and Services in New Zealand

2.15.1 New Zealand studies

There are a limited number of studies in New Zealand specifically examining ethnic differences in OSAS. Firth and Cant (1985) provided the first commentary on the disproportionate number of Māori and Samoan patients presenting at one of the first sleep clinics in New Zealand.

Baldwin and colleagues (1998) further examined ethnic differences in 233 consecutive patients referred to the Sleep Disordered Breathing Unit at Green Lane Hospital. Clinical and physiological characteristics of Māori (n=48), Pacific (n=33) and European (n=152) patients were compared. Of these patients, 85% of Māori and 94% of Pacific Island people were diagnosed with OSAS (AHI≥ 10 and daytime sleepiness), compared with only 49% of Europeans. In addition, most severity markers for OSAS (AHI, apnoea time and oxygen saturation) were worse for Māori and Pacific patients than Europeans, despite Māori and Pacific Island people having the favourable factor of being younger. Using linear regression modelling, BMI, neck size and age, but not ethnicity, were independent predictors of severity markers of OSAS. Using logistic regression modelling, neck circumference was the only significant independent risk factor for OSAS (AHI≥ 15 and ESS≥ 12). The authors concluded that any racial predisposition for Māori was operating mainly through well-recognised risk factors, particularly obesity.

The findings presented in these two articles suggest that the prevalence of OSAS may be higher in Māori and Pacific peoples than in Pākehā, however to date no population-based prevalence studies have been carried out to confirm this. The skewing of Māori and Pacific Island patients towards the more severe end of the spectrum is of some concern, and may indicate barriers to access for Māori and Pacific peoples with mild to moderate disease.

As previously highlighted by Harris (2003), these studies are limited in their discussion and interpretation of ethnic differences in OSAS. Frith and Cant (1985) suggested that the disproportionate number of Māori and Pacific people with OSAS was most likely due to the high incidence of obesity in these groups, however, they imply that something inherent in Māori and Pacific cultures may play an important role in weight

problems ("Weight reduction seems particularly difficult in Polynesian patients, perhaps because of their dietary habits and cultural values" p.747). Similarly, Baldwin and colleagues (1998) attribute the increased ill-health of Māori and Pacific people to "cultural factors resulting in a high BMI, high alcohol consumption, and both poor self management skills and compliance with therapy" (p.254). While the authors do raise the issue of possible problems with access to health care services for Māori and Pacific people, the blame is reduced to cultural factors - "Māori and Pacific Islanders may be less likely to present themselves and therefore reduce their chances of referral" (p.258). Overall, the commentary provided by these two studies is deficient, as the primary focus is on alleged internal deficiencies, while structural factors are held blameless in explaining ethnic disparities (Valencia 1997).

Research of this nature requires a shift in focus, from identifying individual-level risk factors to identifying societal-level risk factors. As discussed previously, there is strong evidence of differential treatment and access to health care services for Māori compared to non-Māori (Pomare et al. 1995, Baxter 2002). These articles also fail to provide information about how the different ethnic groups were defined. Furthermore, the frequent use of the term 'race', implies a genetic component.

A study by Coltman et al. (2000) aimed to determine the relative contribution of craniofacial form, and anthropometric factors, in a group Māori and European male patients (26 Māori, 27 Europeans) with OSA (RDI≥ 15). Measurements of facial and cranial width, length and height, airway size, stature, weight, BMI, neck circumference, RDI and age were obtained. For Māori, small reductions in mandibular prognathism and a wider bony nasal aperture were major factors associated with OSA (adjusted r^2=0.359). In contrast, for Europeans, OSA was found to be associated with a larger neck circumference and a reduced retropalatal airway size (adjusted r^2=0.602). The authors concluded that the results indicated that OSA in these two racially distinct groups was due to different aetiological factors. However, they acknowledge that only part of the variation in RDI was explained by their results, and that further investigations are necessary to more clearly define differences.

As with the previous articles, this particular study also suffers from many deficiencies (Harris 2003). Firstly, race was assigned by a panel of three judges based on each subject's facial appearance on a standardised full-face photograph, although these did concur with patient's self-reported racial groups, race based on appearance supports

scientific racism, which assumes that people can be allocated to racial groups on the basis of a shared biology. Given this method of racial identification, it is not surprising that differences were found between Māori and European patients. Furthermore, given the small sample size, the results of this study are not necessarily representative of Māori and non-Māori in the general population.

In general, all these studies fall short of good research according to Kaupapa Māori Research methodology, suffering from a number of deficits such as small sample size, lack of equal explanatory power, and failure to adequately classify ethnicity, which in turn limits their ability to adequately inform and understand ethnic differences.

2.15.2 Specialist Sleep Services in New Zealand

At last count there were seven specialist sleep services located in the main centres in New Zealand (Auckland, Hamilton, Wellington, Christchurch and Dunedin), which provide approximately 17 beds in total for the diagnosis and treatments of sleep disorders, which includes both public and private beds. For a population of approximately 3.5 million, this equates to approximately 0.5 beds per 100000. Two of these services are solely privately funded, two are fully publicly funded, and the other three receive a mixture of private and public funding (Harris 2003). The publicly funded sleep clinics provide specialist services to several hospitals and are considered tertiary services.

Based on figures from 2001-2003, approximately 5 million dollars of public funding is allocated to sleep clinics per year by respective District Health Boards (DHBs) for the diagnosis and treatment of sleep disorders (Dr Sandy Dawson 2001, pers. comm.). These figures equate to approximately 1900 people per year attending publicly funded sleep clinics. The average cost of assessment and investigation involving gold standard PSG is approximately $1100 per patient, with the addition of $900 for treatment with CPAP.

However, the current level of funding is inadequate, which is evident in the exponential growth in waiting list times. The limited funding also means that clinical beds are unable to be utilised every night (Neill et al. 2000). Furthermore, the dissemination of the results of the present study and the national sleep survey (Harris 2003) are likely to lead to an increased level of awareness among the general public and health care

professionals, which may further increase the discrepancies between current resources and demand. In addition, the need to fund replacement CPAP machines (minimum life expectancy 6 years) has also not been accounted for and will become an escalating problem in future years (Neill et al. 2000).

Sleep services in New Zealand are also hindered by the lack of a systematic nationwide approach to the management of sleep disorders. There are marked variations in the funding of CPAP machines and also differences in terms of the type of clinical problems that services are being asked to investigate. The type and quality of sleep investigation services offered also varies. The Thoracic Society of Australia and New Zealand (TSANZ) and the Australasian Sleep Association (ASA) (2003) have established an accreditation[9] process to foster quality in the approach to the management of sleep disorders, which may address these issues. However, at present, accreditation of sleep disorders services in New Zealand is voluntary.

While a number of problems are evident with current services, an opportunity exists to develop these services according to population needs. The possible higher prevalence of sleep disorders among Māori is an important factor to be considered in the allocation of public funding to sleep services.

[9] A process whereby the professional standards and competence of a sleep disorders service is formally recognised by the TSANZ and the ASA)

BACKGROUND TO THE STUDY

In New Zealand, there are a number of factors hindering sleep services, including a lack of sufficient funding, a lack of homogeneity of approaches towards the management of sleep disorders, a lack of specific sleep medicine training among health care professionals, and finally, a lack of prevalence information for the New Zealand population.

A research programme was developed in partnership between the Sleep/Wake Research Centre, Te Rōpū Rangahau a Eru Pōmare, and Wellsleep sleep clinic, to address these issues. This thesis describes the second research study in this programme, a population based study using objective measures of sleep to examine OSAS prevalence, screening and co-morbidity among Māori and non-Māori. This study was designed as a companion study to the national survey of sleep problems and OSAS symptoms and risk factors among Māori and non-Māori adults (Harris 2003), which was the first research project from the collaborative programme. The combination of these two studies will allow more accurate estimates of prevalence information in New Zealand, which will allow an assessment of the public health impact of OSAS and enable planning for population health care needs. This area of sleep medicine provides a unique opportunity for the needs of Māori to be recognised and incorporated early into the planning of services.

In addition to prevalence estimates, this study was designed to develop a multivariate prediction tool for OSAS, which may be used to assist in the referral of patients from primary care to specialist sleep services. The following goals and hypotheses are specific to this thesis.

2.16 Goals and Objectives

The primary goals of this thesis are to objectively assess the prevalence of OSA and OSAS among Māori and non-Māori in the Wellington region, and to develop a mathematical prediction model for OSA that may be used as a clinical screening tool to assist in the referral of primary care patients to specialist services.

Specific objectives in the community sample:

- Estimate the prevalence of OSA and OSAS for Māori and non-Māori men and women aged 30-60 years, with overnight monitoring using the MESAM4 sleep monitoring system.
- Examine the differences between Māori and non-Māori for variables considered in the development of the predictive tool.
- Examine the prevalence of OSA in Māori and non-Māori for OSA, after controlling for other risk factors.

The specific objectives in the clinical sample:

- Estimate the prevalence of OSA and OSAS for Māori and non-Māori men and women aged 30-60 years in a consecutive sample of patients at the sleep clinic.
- Examine the differences between Māori and non-Māori for each variable used in the development of the predictive tool.
- Examine the prevalence of OSA in Māori and non-Māori after controlling for other risk factors.

The specific objectives in the combined sample:

- To find the best fitting and most parsimonious models to describe the relationship between OSA and a set of clinical features.
- Evaluate the performance of each prediction model.

2.17 Research Hypotheses

- OSA and OSAS are more common in Māori than non-Māori in men and women.
- OSA and OSAS are more common among males than female within Māori and non-Māori ethnic groups.
- Differences in the prevalence of OSA between Māori and non-Māori, will be explained by other factors such as socioeconomic deprivation, age, sex, smoking, alcohol, BMI, and neck circumference.
- A combination of clinical features of OSAS can serve as a reliable screening tool to identify individuals at high or low risk of OSA.

CHAPTER 3
METHODS

3.1 Introduction

This chapter outlines the methods used to conduct this study. It describes the design and practical aspects of the study in addition to the collection, management and analysis of the data. Because data from two different study populations (community and clinical samples) were collected with differing methods, separate descriptions are provided for each.

3.2 Sampling Strategies

3.2.1 The community sample

The target population in the community sample was Māori and non-Māori, men and women in the Wellington region aged between 30-60 years.

Sampling frame

The electoral rolls (General and Māori) in the Wellington region were used as the sampling frame. The electoral rolls provided good coverage of the target population, with an estimated 94% of New Zealanders aged between 30-60 years enrolled at the time of sampling (Harris 2003). It is therefore assumed that the study outcomes of interest would not be different for those not enrolled.

The sample was stratified for descent/ancestry (600 Māori, 600 non-Māori) rather than ethnicity, as the electoral roll does not provide information on ethnicity. Although it is well established that not every person of Māori descent self-identifies with the Māori ethnic group (Statistics New Zealand 1997b), a close relationship is known to exist between the two (Durie 1998). In the present study, all responders of Māori descent identified as being part of the Māori ethnic group. Stratification by descent enabled population averages to be calculated for each group, and in line with Kaupapa Māori research (KMR), allowed equal explanatory power for both Māori and non-Māori, enabling separate analyses of Māori and non-Māori data with the same level of power, while also allowing comparative analyses between the two.

The specific age range of 30-60 years used in this study was based on overseas research, which indicates a high prevalence of OSA in this age range. It was also chosen for comparability with the benchmark prevalence study of Young and colleagues (1993). As increasing age has been shown to be a risk factor for OSA, the sample was stratified into 10-year age groups (30-39, 40-49, 50-59) using 'year of birth' provided from the electoral rolls. However, given the two-year duration of the data collection period, a slight shift in the age range was inevitable, which meant that there were a number of participants who were slightly older than 60 years. Given the small numbers and minimal shift, this issue is likely to have minimal effect on the results of this study.

Given the differences in OSAS by sex, separate prevalence analyses were planned for men and women. However, the sample could not be stratified by sex, as this information is not available from the electoral rolls. It was expected, however, that random sampling would produce approximately equal numbers of men and women.

Sample size

It was agreed that there was no specific method for determining an appropriate sample size for a study of this nature, particularly because the prevalences of some of the dependent measures were not yet known. Therefore no specific power calculations were carried out in the determination of an appropriate sample size.

Based on the common rule of thumb that there should be at least 10 cases per variable to be entered into a multivariate equation, it was decided that a total number of 400 participants (200 Māori, 200 non-Māori) would need to be recruited. In order to achieve this response, a sample of 1200 (600 Māori, 600 non-Māori) was drawn.

Response bias

The occurrence of non-response created the potential for under or overestimation of the prevalence of obstructive sleep apnoea syndrome (OSAS), in that data collected from responders might differ significantly from non-responders. There were a number of potential sources of bias in this study including the inability to contact people by telephone, and also from individuals potentially being more likely to participate if they thought they might suffer from sleep related breathing problems.

To identify potential biases and assess the generalisability of results, sleep questionnaire data was also sought from people who did not wish to have a sleep study. These data were subsequently compared to questionnaire responses of sleep study participants, and

to the national survey of OSAS risk factors and symptoms (Harris 2003), which achieved a 72% response rate. In addition, age, descent and socioeconomic deprivation profiles were compared between *responders* and *non-responders* using information from the electoral roll. These comparative analyses are presented in Chapter 4.

3.2.2 The clinical sample

Consecutive patients aged between 30-60 years who had been referred to the Wellsleep sleep clinic for suspected OSAS were approached to take part in this study. The Wellsleep clinic is a two bed sleep unit located at Bowen Hospital, Wellington and is part of the Wellington School of Medicine and Health Sciences, Otago University. The clinic provides diagnostic and treatment services for several hospitals in the greater Wellington region and also sees privately referred patients. Although OSAS is the primary sleep disorder diagnosed and treated at the clinic, less common sleep disorders are also investigated.

3.3 Measurement of OSA

In the present study, the definition of OSA was based on three respiratory disturbance thresholds (RDI $\geq$ 5, $\geq$ 10, $\geq$ 15). These thresholds are commonly used in other population studies and studies where clinical prediction models have been developed (Young et al. 1993, Flemons et al. 1994, Bearpark et al. 1995, Maislin et al. 1995, Duran et al. 2001, Ip et al. 2001, Ip et al. 2004). However it is important to note that, to date, the clinical importance of any particular cut-off point has not been adequately determined. This section describes the objective measures used to assess OSA in each of the samples and the reasons for their use. Initial data processing is outlined, but detailed information on variables and analyses are presented in the relevant subsequent chapters.

3.3.1 MESAM4 portable monitoring device

In the community study, objective measures of sleep-disordered breathing (SDB) were obtained using the MESAM4[10] ambulatory monitoring system (MAP; Martinsried, Germany). This device consists of a small recording box powered by six AA batteries,

[10] The word MESAM4 is an acronym derived from the name "Madaus Elektronik Sleep Apnoea Monitor" with the '4' indicating that it is the four-channel version of the device.

which was set-up via computer to start recording at a specified time and continued recording until it was downloaded to a computer via a serial cable. The recording device was worn in a pouch with a shoulder strap while the participant was mobile, and placed under their pillow whilst in bed. This equipment specifically measured respiratory sounds, heart rate, arterial oxygen saturation and body position (Figure 3.1).

Figure 3.1 Volunteer wearing the MESAM4 equipment (while mobile and while in bed)

Nightly events, such as waking for longer than 10 minutes or going to the toilet, were referenced by the participant with an event marker button. Heart rate was measured with three chest electrodes applied with disposable 3M Red Dot™ gel adhesives. The red electrode was placed on the upper right side of the sternum, the black electrode on the lower sternum, and the yellow electrode in a modified V2 position. This electrode placement assumed normal orientation of the heart.

A small cyclical microphone was taped and secured with a band to the participants' larynx, directly above the jugular and below the Adam's apple, to record snoring sounds. The recorder determines the occurrence (yes/no) and quality (loud/quiet) of snoring in the frequency range from 100 to 800 Hz. It also detects loud sounds that are determined above a defined noise level, in the frequency range of 100 Hz to 15 kHz (Figure 3.2).

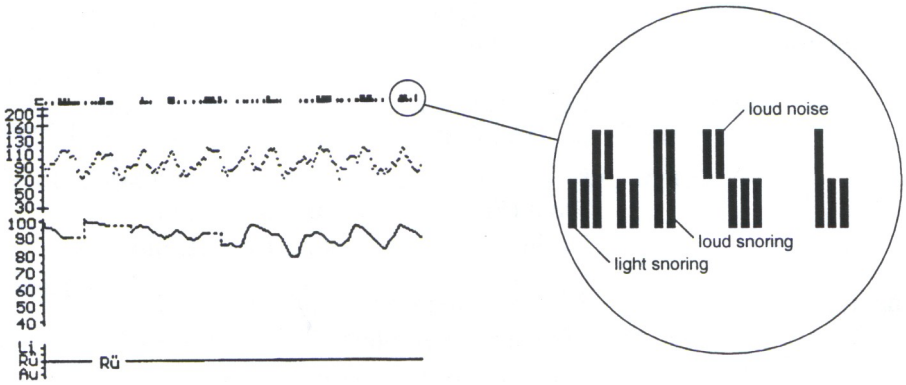


Figure 3.2 Recorded sound and snoring

(Reproduced from MESAM®IV Instruction manual p.86 with permission from MAP Medizin-Technologie GmbH Fraunhofer St. 16. 82152 Martinsried).

Oxygen saturation ($Sa0_2$) was monitored using a flex or clip oximetry sensor (Figure 3.3). The flex sensor was preferentially used, as it was reported by participants to be more comfortable over the duration of the night than the clip sensor, which is more suited for short-term usage. Both finger probes measure $Sa0_2$ within the range of 40-100% and have accuracy levels of ± 2%, which implies that at least 68% of measured values fall within the defined range. Oxygen saturation is measured every 2 seconds (MAP Instruction Manual, Edition 04/95). The sensor was fitted to the participant's index fingertip on the non-dominant hand. To avoid displacement, it was secured with 3M Micropore™ medical tape, assuring blood flow was not restricted and that no strain was placed on the cable.

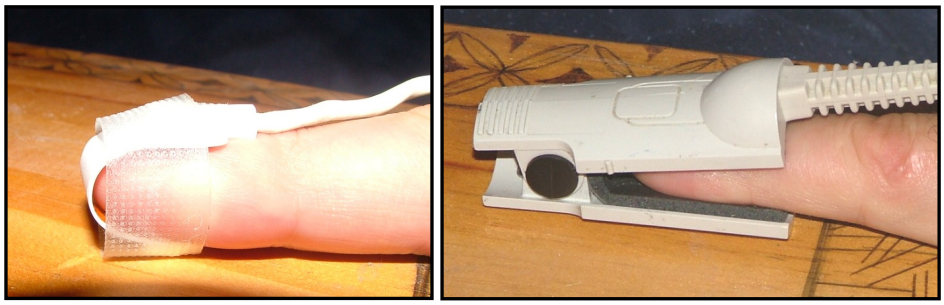


Figure 3.3 MESAM4 oximetry sensors (Right: flex sensor, Left: clip sensor)

The body position sensor was taped to a Velcro secured band strapped around the participant's body, secured with 3M Micropore™ medical tape, just below the sternum and parallel to the front of the body for accurate measurement. This parameter was, however, excluded from analysis due to inconsistencies observed when data were

transferred and viewed on different computers. Figure 3.4 displays the placement the MESAM4 equipment.

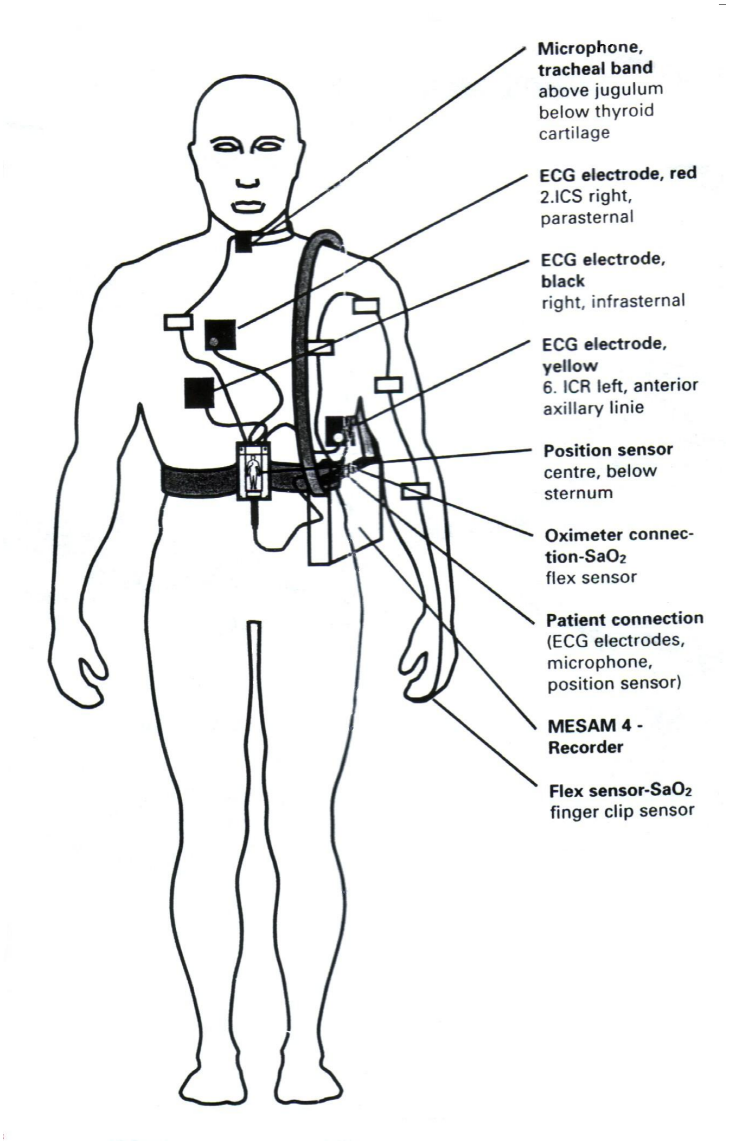


Figure 3.4 MESAM4 equipment placement

(Source: Reproduced from MESAM®IV Instruction Manual p.31 with permission from MAP Medizin-Technologie GmbH Fraunhofer St. 16. 82152 Martinsried).

One pertinent disadvantage of the MESAM4 and other Level 3 and 4 studies is that they do not allow direct determination of wakefulness and the stages of sleep. Without the presence of sleep data, an individual's respiratory disturbance index (RDI) is likely to be underestimated, as sleep is likely to be overestimated, especially if the participant is unknowingly awake for a significant part of the recording period. To address this

limitation, participants were given verbal and written instructions to press the event marker button when they began trying to sleep, if they woke during the night, and at their final wakening time (Appendix 1). In addition, participants were also asked to note their sleep/wake times throughout the night. In some cases, participants forgot to press the marker, or the marker did not coincide with their subjective reports. In this case, heart rate criteria for an awakening were used to assist the scorer. Another limitation of the MESAM4 is the inability to differentiate between the types of apnoeic events (central, obstructive, mixed).

In general, due to the limited physiological data available, the classification of respiratory events requires a greater degree of subjectivity using the MESAM4 device compared to PSG. Despite these shortcomings, and with practical constraints of time and money, the MESAM4 was seen as a suitable device to measure OSA in the community study. A number of other studies have used this equipment to measure sleep disordered breathing (SDB) (Richman et al. 1994, Bearpark et al. 1995, Philip et al. 1997, Sonka and Nevsimalova 1997, Hochban et al. 1999, Hui et al. 1999, Hui et al 2002, Zucconi et al. 1999).

Scoring of MESAM4 studies

The MESAM4 software (version 3.23) allowed data to be viewed on computer screen, and printed out as a paper record. Although automated analysis of the MESAM4 data was available, the software did not allow for removal of artefacts and was therefore judged not to be sufficiently accurate. All MESAM4 recordings were therefore printed via computer to a laser printer (for enhanced channel definition) and manually scored.

An experienced scorer, blinded to the identity of participants, performed detailed analyses for each five-minute epoch (5 minutes = 76 mm). Each epoch was evaluated for state (sleep/wake), respiratory disturbances (apnoea/hypopnoea), snoring, and excluded from further analyses if more than half the epoch contained artefact from any of the recorded channels. A magnifying glass and ruler were used to assist in judging signal deviations from baseline.

Sleep related obstructive respiratory events

Two methods of scoring respiratory events were used in the present study. The first method was derived from an Australian population study (Bearpark et al. 1995), which has been shown to correlate strongly with polysomnography (r_s =0.94, p<0.0001). The second method was derived from a study by Penzel and colleagues (1990), and sought to capture hypopnoeic events.

An apnoea was scored if there was an episode of oxygen desaturation of ≥ 4% from the preceding baseline in conjunction with (1) an increase in heart rate (HR) of at least 10 beats per minute; or (2) a burst of snoring associated with commencement and termination of a desaturation episode; or (3) both 1 and 2 (Bearpark et al. 1995). A hypopnoea was scored if there was a peak increase in HR by at least 10 beats per minute above the preceding baseline in addition to snoring (Penzel et al. 1990). In order to differentiate the two types of events, they were scored using different symbols (↓=apnoea □=hypopnoea). An example of this scoring method is provided in Figure 3.5. In this particular example, 12 apnoeic events and 2 hypopnoeic events were scored within the two 5-minute epochs.

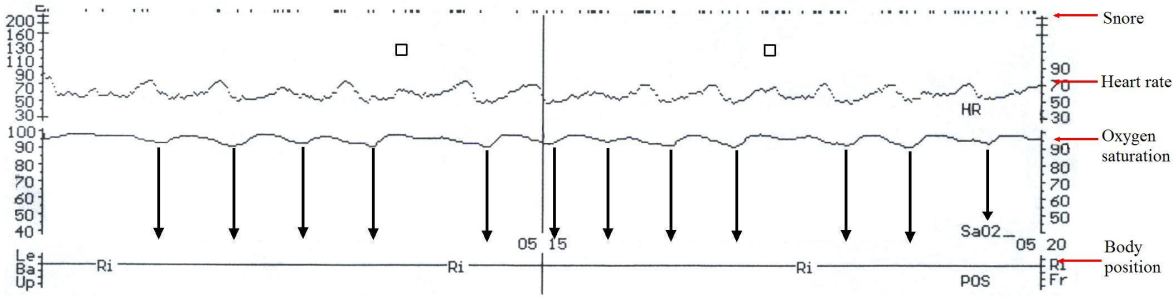


Figure 3.5 Example of MESAM4 scoring on printout (2x 5 min epochs)

From this scoring method, two respiratory disturbance indices were calculated. RDIa, was defined as the total number of respiratory events involving oxygen desaturation of ≥ 4% from the preceding baseline, divided by the total estimated sleep time in hours. RDIc was defined as the total number of all respiratory events (apnoeas and hypopnoeas) divided by the total estimated sleep time in hours.

Snoring

Snoring was scored according to a validated method (Bearpark et al. 1995). This method has not validated against PSG, but against audio recordings, with which it was found to be strongly correlated (r_s=0.92, p<0.0001).

An overall snore percentage was calculated by the assignment of a snoring grade from 0-9 to each 5-minute epoch according to the number of snores counted (Table 3.1).

Table 3.1 Snore grade criteria

No. of snores	Snoring grade (%)
<2	0
2 – 6	1 (10%)
7 – 13	2 (20%)
14 - 20	3 (30%)
21 - 27	4 (40%)
28 - 34	5 (50%)
35 - 41	6 (60%)
42 - 47	7 (70%)
48 - 54	8 (80%)
>55	9 (90-100%)

A score of 0 indicated no snores in a given epoch whereas a score of 9 indicated continuous repetitive snoring (Figure 3.6). A grade of 8 indicated either repetitive snoring or snoring associated with respiratory events for 80 - 90% of the epoch, and so forth.

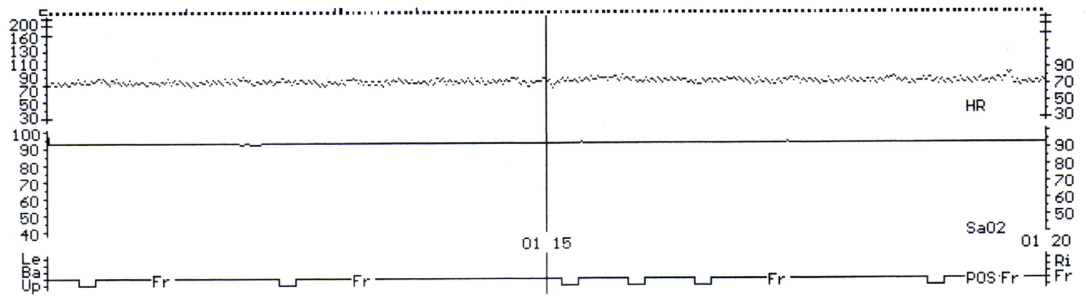


Figure 3.6 Continuous snoring

The overall snoring percentage for each participant was calculated with the following equation: s*um of snore grades for all sleep epochs X 100 /number of sleep epochs X 9.* Therefore a participant with a grade of 9 for each epoch would receive an overall snoring percentage of 100.

Exclusion from scoring

Epochs were excluded from further analyses if artefact was found in any of the four signals for more than half of an epoch. Artefact occurred for a variety of reasons, including participant movement, electrode displacement and technical failure (faulty sensors).

Given that snoring produced an intermittent signal, a continuous unbroken signal was presumed to be due to other noise rather than snoring (Figure 3.7). Similarly, sound signals that had an unusual morphology (e.g., were particularly long) or were associated with movement, were also considered as artefact.

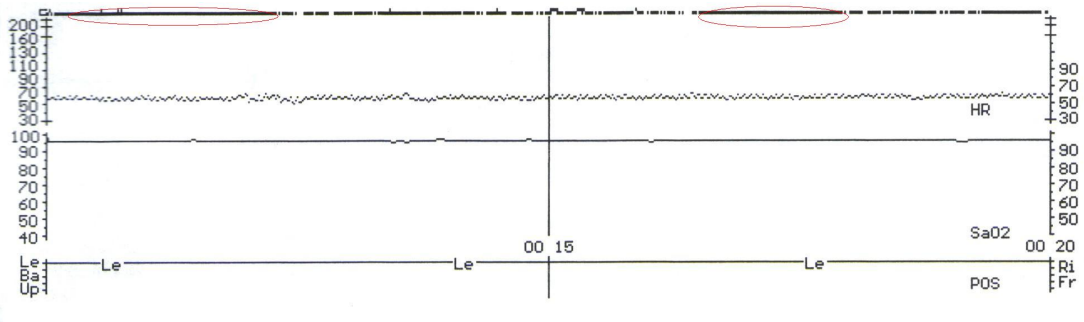


Figure 3.7 Snoring artefact

$Sa0_2$ artefact was identified by a broken signal line, which usually indicated poor probe contact. Additionally a vertical fall in the $Sa0_2$ signal, which indicated compression or movement, was classified as artefact (Figure 3.8).

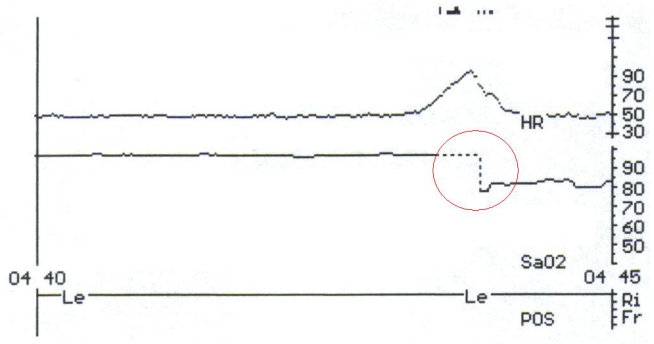


Figure 3.8 $Sa0_2$ artefact

Estimated total sleep time

Because actual sleep cannot be identified from the variables recorded using the MESAM4, the person scoring each study had to differentiate sleep and wake periods with the assistance of the event marker, subjective reports of sleep/wake times, and with changes in baseline heart rate within each epoch. If baseline HR changed abruptly (≥ 3bpm) and maintained the new rate, this was considered indicative of an awakening, and was scored as 'awake' if it persisted for more than half of an epoch.

MESAM4 versus 'gold standard' polysomnography

Previous studies have evaluated the diagnostic validity of the MESAM4 by performing simultaneous recordings with polysomnography (PSG) (Stoohs and Guilleminault, 1992, Roos et al. 1993 Bearkpark et al. 1995, Esnaola et al. 1996, Cirignotta et al. 2000). However these studies only report the correlation between the two, which can be misleading, since it is only a measure of the strength of a relationship. Two measurements may correlate perfectly but have different scales of measures, in which case they may not agree (Flemon and Littner 2003). Furthermore, all these studies were conducted in a clinical setting, and are not necessarily generalisable to a home setting (Flemons et al. 2003).

As part of the present study, a small validation study was conducted. Twelve volunteers selected from the community and clinical samples wore the MESAM4 equipment and PSG equipment simultaneously for a night either at the clinic or at home. Findings from the validation study showed that total sleep times were consistently overestimated with the MESAM4 manual scoring method, which is consistent with other findings (Bearpark et al. 1995). However, participants in the validation study were not required to press the MESAM4 marker button or to note their sleep and wake times, which was a requirement in the present study. It is assumed that this additional information would have improved the scorer's ability to estimate total sleep time in the validation study.

In line with other studies (Stoohs and Guilleminault 1992, Bearpark et al. 1995), the RDI scores derived from the manual scoring of the MESAM4 data correlated strongly with PSG values (r_s =0.99, 95% CI 0.96-1.00, $p<0.0001$), however agreement between the total number of scored respiratory events was highly variable. The MESAM4 frequently overestimated the number of respiratory events, particularly hypopnoeas. This suggests that in the community sample, RDIa is a more reliable measure of OSA

than RDIc. Despite these discrepancies, both respiratory indices displayed reasonable discriminatory ability, correctly classifying all participants into the different thresholds of respiratory disturbance (≥ 5, ≥ 10, ≥ 15) as identified by PSG. However given the small sample size (n=12) these results are limited. A detailed report of this validation study is included in Appendix 2.

Inter and Intra-scorer reliability

To assess the agreement in the interpretation of the MESAM4 signals, ten studies of varying severity were selected and re-scored by two trained scorers (KM and WW1). Re-scoring took place at least a year after the initial scoring. Reliability was calculated based on the number of apnoeas and hypopnoeas scored overall from the ten studies. Overall agreement controlling for chance was measured using the weighted kappa (*k*) statistic[11]. As the scoring was ordinal in nature, this method allowed disagreement between scorers to be weighted differentially depending on the distance between the two scores.

Overall, the agreement according to the weighted kappa statistic ranged between 0.63-0.87, indicating 'good' to 'very good' agreement for both inter and intra-scorer reliability (Table 3.2).

Table 3.2 Inter and intra-scorer reliability for scoring of MESAM4 studies

Scorer comparison	Variable	% Agreement	Kappa (*k*)	Weighted Kappa	95% CI
Interscorer					
WW1 vs. KM	Snore %	72%	0.62	0.87	0.85-0.89
	Apnoeas	85%	0.53	0.70	0.65-0.75
	Hypopnoeas	73%	0.45	0.63	0.59-0.67
WW2 vs. KM	Snore %	81%	0.74	0.90	0.89-0.92
	Apnoeas	92%	0.72	0.80	0.75-0.85
	Hypopnoeas	86%	0.69	0.81	0.78-0.84
Intrascorer					
WW1 vs. WW2	Snore %	58%	0.62	0.85	0.83-0.87
	Apnoeas	77%	0.51	0.68	0.63-0.73
	Hypopnoeas	63%	0.44	0.63	0.59-0.67

[11] The Kappa statistic produces values between 0 and 1. A value between 0.4 and 0.75 is regarded as 'fair to good agreement,' while >0.75 is regarded as 'excellent agreement' (Fleiss 1981).

Interestingly, intra-scorer reliability (WW1 vs. WW2) was lower than the inter-scorer reliability (WW1 vs. KM, WW2 vs. KM), and inter-scorer reliability was better when compared with scoring towards the end of data collection (WW2 vs. KM). These results suggest that there was some amount of learning by the primary scorer (WW) through the duration of the study, especially with respect to scoring snoring and hypopnoeas. Although the primary scorer was experienced in PSG sleep scoring, she was not specifically experienced in scoring MESAM4 studies. These results also highlight a higher degree of disagreement between scorers when scoring hypopnoeas. Despite these discrepancies, it was decided re-scoring of the studies was not warranted. However these issues should be an important consideration in future studies utilising the same scoring criteria for MESAM4 data.

3.3.2 Polysomnography

Overnight polysomnographic data were collected for clinic patients using the Compumedics™ computerized system. Studies included both attended clinical and unattended home PSG. The following measurements were recorded for clinic and portable home studies: electroencephalography (C3/A2 and C4/A1 placement); electrooculography (EOG); and chin electromyography (EMG) to identify sleep stages, electrocardiogram (ECG), thermistry and nasal prongs to measure nasal and oral airflow, oximetry to measure oxyhemoglobin saturation. Thoracic and abdominal bands were used to measure respiratory effort. A microphone was attached over the trachea (lower neck) to record snoring. A position sensor was attached to the thoracic band to measure body position. Leg paddles were attached to each of the outer calve muscles to measure anterior tibialis electromyograms, to enable screening for periodic limb movement disorder (PLMD), which has some daytime symptoms in common with OSAS (Guilleminault and Anagnos 2000, Stoohs et al. 2001).

Each 30-second epoch was scored for sleep stage according to standard criteria (Rechtschaffen and Kales 1968). An apnoeic event was defined as the cessation of nasal and oral airflow for at least 10 seconds and a hypopnoea was defined as at least 50% reduction of at least 2 out of 3 signals (airflow, thoracic, abdominal movements) for 10 seconds or more accompanied (American Sleep Disorders Association 1994).

For comparability with MESAM4 results, a comparable RDI measures were calculated for each clinic patient from the PSG data. Snoring percentage was assessed by

polysomnography at different decibel levels, which were summed together to get the total percentage of the night spent snoring, however the snoring channel was not available for home PSG studies.

3.4 Other Objective Measures

3.4.1 Body Mass Index

Body Mass Index (BMI) (weight (kg)/height (m)²) is one of the most commonly used measures of general obesity (Garrow and Webster 1985). There is good evidence showing that one principal driver of OSAS is the current obesity epidemic (Young et al. 2004). Specific anatomical and physiological properties of the airway are suggested to interact with obesity to predispose the development of airway collapse during sleep (Fogel et al. 2003). Minimal weight loss in moderately overweight OSAS patients has also been shown to significantly improve OSAS symptoms (Smith et al. 1985).

In the community study, height was measured to the nearest 0.5cm without footwear. Weight was measured in light clothing using SECA™ digital floor scales. These scales were periodically calibrated using a 5kg weight. In the clinical sample, height and weight were measured or taken from the patient's clinical notes, if available. However with home PSG studies, where height and weight were not available, patients were asked to estimate their height and weight. This method was adopted as only one sleep technician conducted the home PSG studies and it was not possible to carry the equipment necessary for weighing and measuring the height of these patients.

For non-Māori, standard BMI categories were used to define overweight and obesity. However different thresholds were used for Māori. The standard BMI benchmarks have previously been found to be inappropriate, as Māori tend to have a higher muscle to fat ratio than other New Zealanders (Swinburn et al. 1999). Therefore for non-Māori, a BMI of 25-30 kg/m^2 was used to classify overweight and a BMI of >30 kg/m^2 was used to classify obesity. For Māori, overweight was defined as a BMI of 26-32 kg/m^2 and obese was defined as a BMI > 32 kg/m^2.

3.4.2 Blood pressure

Due to equipment constraints, blood pressure was only measured in the community study. A Heine™ Gamma 4.5-sphygmomanometer was used to measure blood pressure. Unfortunately, time constraints only permitted one measurement per

participant. If however, the participant did have a particularly high or low reading, blood pressure was measured again at the end of the night to ensure readings were not caused by measurement error.

In line with recommended standards, most other studies take a series of blood pressure measurements over a period of time, from which an averaged measurement is derived (Hla et al. 1994, Bovet et al. 2002). The single measurement taken in the present study is somewhat crude and should be interpreted with caution. Where possible, the measurement was taken at a standard point during the protocol, after the participant had been seated completing the questionnaire and consent form. However, this did not always go to plan, as participants often got up during this *resting* phase to tend to household activities (e.g., answer the phone, tend to their children). The blood pressure data are not examined in this thesis.

3.5 Questionnaire Data

3.5.1 The sleep questionnaire

A one-page double-sided sleep questionnaire (Appendix 3) was administered to all community and clinic participants. The questionnaire focuses on general sleep, and symptoms, risk factors and outcomes associated with OSAS. It was developed and piloted prior to this study and was subsequently used in the national sleep survey (Harris 2003).

Demographics

Sex (Question 1)

OSAS and the features associated with it are known to vary between men and women (Ambrogetti et al. 1991, O'Connor et al. 2000, Bixler et al. 2001, Dancey et al. 2003, Larsson et al. 2003).

The first question in the questionnaire asked participants to identify their sex. As mentioned previously, this information was not provided in the electoral roll information. The term *sex* rather than *gender* is used throughout this thesis, which is informed by the field of sociology, where sex is seen as a biological term referring to a person's biological given state, and gender is a social term referring to a person's social roles.

Date of birth (Question 2)

Age has been identified as a risk factor in sleep disturbances in general (Krieger et al. 1997) and also in OSAS (Bixler et al. 1998). Date of birth (dd/mm/yyyy) was asked in the questionnaire to enable a more accurate calculation of the participant's age, rather than using *year of birth* available from the electoral roll. Age was calculated at the date the participant answered the questionnaire.

Ethnicity (Question 3)

The consideration of ethnicity is helpful in the planning of health services and has the potential to offer new insights into the causes of disease. It also allows for different realities to be captured that would not otherwise be captured using the biologically based concept of race (Senior and Bhopal 1994).

Participants were asked to self identify their ethnicity, using the ethnicity question from the 1996 census (Statisitcs New Zealand 1997a). This allowed collected data to be weighted back to the appropriate proportions for the general population. It also allowed the Māori population to be analysed as either sole Māori, which includes those who give Māori as their only ethnic affiliation, or Māori ethnic group (MEG), which includes those who indicated Māori as at least one of their ethnic affiliations. In line with the national sleep survey (Harris 2003), the MEG was analysed in this thesis.

Socioeconomic factors

Socioeconomic risk factors are recognised as important determinants of health at both a population and individual level (Salmond and Crampton 2001). Socioeconomic factors constitute a pathway linking ethnicity to health (Ministry of Health 2002b). While there is clear evidence that Māori are over-represented among the most deprived sectors of New Zealand society (Crampton et al. 2000b), evidence to date indicates that socioeconomic factors account for only part of the disparities that exist between Māori and non-Māori (Ministry of Health 2002b, Reid et al. 2000).

Little is known about the distribution of OSAS by SES, even though many of the co-morbidities associated with OSAS have been shown to have socioeconomic gradients, including obesity, smoking and co-morbid conditions such as hypertension and other cardiovascular diseases (Howden-Chapman and Tobias 2000). The present study utilised two measures of socioeconomic position.

1. Community services card (Question 17)

As a proxy measure of individual socioeconomic position, participants were asked whether they were eligible for a community services card (CSC). Individuals can access this card if they are over 18 years of age and on low to middle incomes, adjusted for family size. The primary benefit of the card is subsidised visits to the doctors and prescriptions (e.g., a visit to the doctor will cost $20 rather than $40) (WINZ 2003). This question was initially included in the questionnaire, as the more sensitive NZDep96 tool discussed below had not yet been developed. However this measure was useful in the clinical sample, as patients' addresses were not available to the researcher, so NZDep96 scores could not be attributed. This variable is also better suited to be tested in the screening tool than NZDep96, as it can be directly asked from patients whereas attaining NZDep96 rankings is more complex.

2. NZDep96

The New Zealand Index of Deprivation (NZDep96) was used as a measure of socioeconomic deprivation (Salmond et al. 1998). Socioeconomic deprivation refers to a state of social and economic disadvantage relative to the society to which an individual or group belongs (Howden-Chapman and Tobias 2000). NZDep96 is a theoretically robust index of deprivation for small areas based on the 1996 New Zealand census data (Statistics New Zealand 1997a). Each census meshblock contains a median of 90 people and is categorised between 1 (least deprived) and 10 (most deprived). Deprivation in each area is defined from a relative standing including material and social factors, and is based on eight elements that are clearly linked to poor health. These elements reflect lack of living space, income, employment, communication, support, qualifications, transport, and an owned home (Salmond et al. 1998). The specific variables are listed in Table 3.3.

Table 3.3 NZDep96 variables
(Source: Salmond et al. 1998)

Dimension of deprivation	Variable description (in order of decreasing weight)
Communication	People with no access to a telephone
Income	People aged 18-59 receiving means tested benefits
Employment	People aged 18-59 unemployed
Income	People living in equivalised* households within income below an income threshold
Transport	People with no access to a car
Support	People aged <60 living in a single parent family
Qualifications	People aged 18-59 without any qualifications
Owned home	People not living in own home
Living space	People living in equivalised* households below a bedroom occupancy threshold

*Equivalisation: methods used to control for family composition

As shown in Figure 3.9, the spread of deprivation is wider in some deciles than in others. The difference between deciles 2 and 5 is not huge, unlike the difference between deciles 7 and 10.

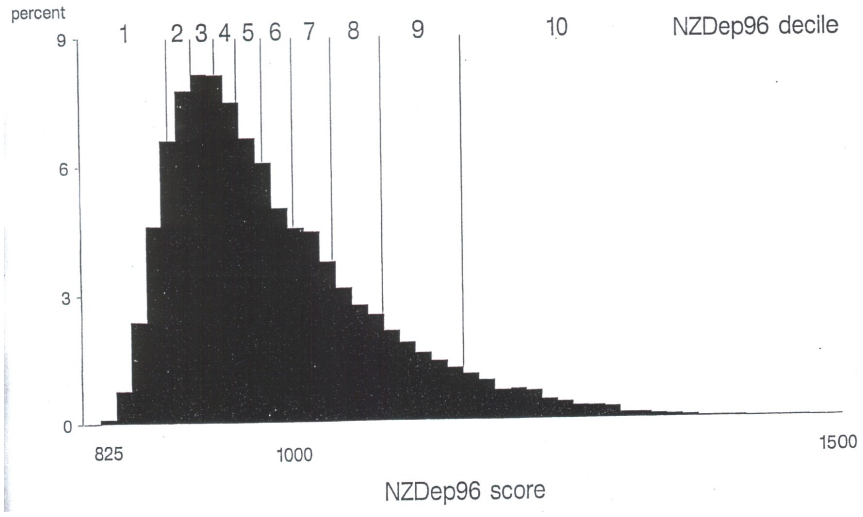


Figure 3.9 Distribution of NZDep96 scores, with the NZDep96 scale superimposed

(Source: Salmond et al. 1998)

The NZDep96 index is closely correlated with individual measures of deprivation, but is not necessarily reliable for any given individual. Not all deprived people live in deprived areas, and while area-level socioeconomic effects on health are important, it is suggested that they are probably not important as personal socioeconomic effects (Blakely and Pearce 2002). Therefore care needs to be taken when making inferences based on this aggregated data.

Using a SAS™ (Version 8.02) programme developed by the assisting statistician, individuals in the community study were assigned a deprivation index based on their home address provided in the electoral roll.

General sleep variables

A few general sleep questions were also asked as part of this questionnaire.

Self-reported quantity of sleep (Question 4)

A number of epidemiological studies have suggested an association between self-reported sleep duration and long-term health. Individuals who report both an increased (>8 hour per day) or reduced (<7 hours per day) sleep duration have been shown to have a modest increased risk of all-cause mortality, cardiovascular disease, and developing symptomatic diabetes (Alvarez and Ayes 2004). In the present study, participants were asked how many hours sleep they normally get in 24 hours.

Self-perceived quality of sleep (Questions 5 and 6)

Participants were also asked how often they thought they got enough sleep, and also how often they woke feeling refreshed. The question pertaining to unrefreshing sleep was derived from the 1991 National Sleep Foundation and the Gallup Organization survey on insomnia (Harris 2003). While unrefreshing sleep is a recognised symptom of OSAS (American Sleep Disorders Association 1997), given that there are a number of possible causes for unrefreshing sleep, it is often used as a general measure of sleep problems.

OSAS symptoms

Excessive daytime sleepiness (Question 9)

Sleep fragmentation in the form of numerous, brief arousals from sleep has been demonstrated to be a significant contributor to excessive daytime sleepiness (EDS) in people with OSAS. In the case of severe OSAS, people may experience hundreds of these arousals a night (Martin et al. 1996, Bennett et al. 1998).

In this study, daytime sleepiness (hypersomnolence) was measured using the Epworth Sleepiness Scale (ESS), which asks the likelihood of dozing in eight common situations. Each question is scored on a Likert scale from 0 (would never doze) to 3 (high chance of dozing). The scores are then added together and a sleepiness score is derived ranging from 0 to 24. This scale is widely accepted as a validated and reliable self-report measure of sleepiness among adults (Johns 1991, 1992, 1993, 1994), with scores above

10 generally being considered indicative of excessive sleepiness (Johns and Hocking 1997).

The ESS it one of the most commonly used tool for measuring self-reported sleepiness and is accepted as reliable, internally consistent and externally validated by comparison with the clinical 'gold standard' sleepiness measure, the Multiple Sleep Latency Test (Johns 1992, Johns 2000, Mitler et al. 2000). This measure is however, susceptible to participant deception, bias, and misinterpretation and therefore should generally not be viewed in isolation. Furthermore, findings from the national sleep survey indicate that age, ethnicity and socioeconomic status also have an independent effect on ESS (Gander et al. 2002).

In the present study participants who did not complete all eight items used to calculate this score were classified as missing for this particular variable.

Observed apnoeas (Question 8)

Generally the individual OSAS sufferer is unaware that they stop breathing during sleep. It is the bed partner who is alerted to the problem. Participants were therefore asked whether they had ever been told that they sometimes stop breathing during sleep. This question was taken from the Australian population study (Bearpark et al. 1995). A number of other studies have found observed/witnessed apnoeas to be a useful predictor of OSA (Kapuniai et al. 1988, Hoffstein and Szalai 1993, Flemons et al. 1994, Douglass et al. 1994, Kump et al. 1994, Maislin et al. 1995, Hui et al. 2002, Young et al. 2002b), but not all (Viner et al. 1994, Ip et al. 2001).

Habitual snoring (Question 7)

Snoring is a consequence of changes in the configuration of the upper airway occurring during sleep. It is one of the most commonly presented clinical symptoms of OSAS (Neill and McEvoy 1997). A number of studies have found self-reported snoring (either frequency or intensity) to be an independent predictor of OSA (Kapuniai et al. 1988, Viner et al. 1991, Flemons et al. 1994, Kump et al. 1994, Douglass et al. 1994, Ip et al. 2001).

Snoring frequency was ascertained by asking participants how often they snored with the options of never, rarely, often or always. This question was derived from a British

survey of male car drivers (Maycock 1996), where drivers were asked whether they snored at night on a 4-point scale (not at all, rarely, occasionally or every night).

OSAS risk factors

Neck circumference (Question 8)

Neck circumference was measured as a surrogate measure of upper body obesity. A number of previous studies have identified neck size as a stronger predictor of OSAS than body mass index or other measures of obesity (Baldwin et al. 1998, Millman et al. 1995, Ben-Noun et al. 2001). It has been postulated that the weight of fatty tissue in the neck represents an additional loading contributing to airway collapse (Stradling and Crosby 1991). Neck circumference has been shown to be good predictor of OSAS in overweight and obese patients (Ben-Noun et al. 2001). It is also plausible that OSA may increase the risk of obesity (Redline et al. 2003, Coughlin et al. 2004).

Neck size was first measured by the participants as required in the sleep questionnaire, using the paper tape measures that were used in the national sleep study. A subsequent measurement was taken by the researcher at the level of the cricothyroid membrane (Adam's apple) to assess how accurate people were at measuring their necks. This information was collected in order to validate the use of the paper tape measures in the national mail out survey.

Smoking status (Question 14)

The hypothesised mechanisms for the role of smoking in OSA include airway inflammation and smoking-related disease, in addition to effects of declining blood nicotine levels on sleep stability. Whilst epidemiological studies (Bloom et al. 1988, Wetter et al. 1994, Bearpark et al. 1995, Marin et al. 1997) have found positive associations with smoking and increased risk of SDB, it is still not firmly established as a risk factor (Young et al. 2004). In New Zealand, smoking rates are higher in Māori than non-Māori even after controlling for deprivation (Crampton et al. 2000), especially amongst women.

In the present study, participants were asked to describe themselves as either a regular smoker (*I smoke one or more cigarettes per day*), occasional smoker (*I do not smoke every day*), ex-smoker (*I use to smoke but not any more*), or a non-smoker (*I have never smoked regularly*). This question was modified from the 1996 census question regarding smoking (Statistics New Zealand 1997a).

Alcohol consumption (Questions 15 and 16)

A number of studies have shown that OSAS is exacerbated by alcohol consumption, which induces increased relaxation of the airway during sleep (Bassiri and Guilleminault 2000, Tsutsumi et al. 2000). Although the short-term effects have been well substantiated, the long-term effects of habitual alcohol consumption are less clear.

To obtain a picture of the different drinking patterns reported among Māori and non-Māori, two alcohol related questions were asked in this study. One related to the frequency of drinking (*Q15. How often do you drink alcohol?*), and the other to the amount typically consumed (*Q16. On a typical drinking occasion, how many drinks do you have?* (*One drink equals a glass of beer or a glass of wine or a nip of spirits*)). The options provided for each question allowed categorisation according to the Alcohol Advisory Council of New Zealand guidelines (1997) on the upper limits for responsible drinking.

Possible consequences

Co morbid disease (Question 13)

Potential consequences of untreated OSAS include increased risk of hypertension, coronary artery disease, myocardial infarction, stroke, psychiatric problems, impotence, cognitive dysfunction, and memory loss. It has been suggested that a significant proportion of all deaths and illness attributed to cardiovascular disease may actually be a result of OSAS. Sustained elevations of hypertension amongst OSA patients is so common that OSA syndrome itself may be a possible risk factor for hypertension (Krieger and Redeker 2002).

Participants were asked if they were currently receiving treatment for the following medical conditions: asthma, hypertension, heart trouble, diabetes, stroke, thyroid problems, psychological problems and sleep problems – with the options of yes, no or don't know. In this thesis, these co-morbid diseases are analysed as potential predictors of OSAS.

Driving and motor vehicle accidents (Questions 11 and 12)

The higher risk of motor vehicle accidents (MVAs) among untreated OSA sufferers is postulated to be primarily a result of the excessive daytime sleepiness resulting from disrupted sleep at night (Young et al. 1993, Marshall et al. 2003). Furthermore, MVAs are a major cause of mortality and morbidity for Māori (Sargent et al. 1994).

Participants were first asked to select an option that best described the number of hours per week, including the weekends that spent driving a motor vehicle on average. The categories were taken from The Auckland Car Crash Injury Study (Connor et al 2001). Participants were also asked how many times in the last three years they had been involved in a motor vehicle accident where they were the driver. This question was taken from a British mail survey of male car drivers (Maycock 1996). These variables were not analysed as part of this thesis.

Pre-sleep study questions

In the community sample, in addition to the sleep questionnaire, a number of other questions were asked. These were taken from Wellsleep clinic's standard set of patient questions, and addressed medical history, alcohol and caffeine consumption on the night of the study, and usual consumption (Appendix 4). This information provided important contextual information for each study, especially where significant respiratory disturbance was found.

Post-sleep study questions

The morning after their MESAM4 study, each participant was asked to rate their sleep on the night of the study compared to a "normal night's sleep" on a five-point scale (1=much worse, 3=typical, 5=much better). To evaluate the practical use of the MESAM4, participants were asked to comment about any difficulties they may have experienced while wearing the equipment, which may have impacted on the quality of their sleep (Appendix 4). The most common difficulty reported was with the finger clip oxygen sensor (shown in Figure 3.3). The constant continuous pressure on the finger from the clip caused a degree of discomfort for some participants over the course of the night. This is not surprising, given that that particular sensor is specifically designed to be worn for shorter durations (e.g., doctor's office). The flex sensor was therefore preferred, but due to equipment constraints, it could not be used for all participants.

3.6 Data Collection

Ethical approval for the study was received from the Wellington Ethics Committee. Between August 1999 and May 2001, letters and information sheets outlining the study were progressively mailed out to the randomly selected sample of 1200. During the same period, consecutive patients aged between 30 and 60 years of age who had been referred to the sleep clinic at Bowen hospital, Wellington, for suspected OSA were asked to take part in the study.

3.6.1 Community sample

Trial studies

Prior to the data collection phase, a number of trial nights were completed with a small number of family, friends and staff of the Sleep/Wake Research Centre, firstly to identify any problems with the proposed study protocol, and secondly to estimate the time required to set up the equipment for each study. These study nights were successful, so no changes were made to the community study protocol.

Recruitment

Telephone numbers were searched for each individual in the sample using the Internet white pages (www.whitepages.co.nz). Where telephone numbers were not available, a programme designed in SAS™ was used to match the addresses of people in the sample with people with the same address on the electoral roll. Phone numbers were then searched for under the names of people at the same address. Telephone directories from previous years were also searched, as sometimes people had only recently chosen to have their telephone numbers unlisted.

To allow time for contacting people in the sample and collecting data from participants, information packs were progressively sent out between August 1999 and May 2001. Information packs contained a covering letter (Appendix 5), information detailing the study (Appendix 6), a consent form (Appendix 7), and a paper tape measure. Letters for Māori and non-Māori samples differed slightly, with letters to Māori containing Māori salutations (Tēnā koe vs. Dear Sir/Madam and Yours sincerely vs. Nāku noa).

In the covering letter, individuals were informed that a researcher would contact them in the next few days to ask whether they would like to take part in the study. The telephone number found for each person was printed in their respective letters, and it

was stated that researchers would be contacting them on this number. If the number was incorrect, people were requested to contact the research team if they were interested in taking part in the study. Similarly, where no valid telephone number was found, people were given the option to contact the research team via telephone, email or mail if they were interested in taking part in the study. Unfortunately, only a few people whose telephone numbers were incorrect actually contacted us. This highlights the importance of being able to make verbal contact with potential participants.

Researchers contacted people in the sample with valid phone numbers, to elicit participation. If a person had not yet received the information pack another was sent to them or the study was explained over the telephone. If the person agreed to an overnight sleep study, a suitable night was arranged. In order to set the time for the MESAM4 equipment to start recording, participants was asked their approximate usual bedtime. Recorders were then set up via the laptop computer an hour before their stated bedtime. Address details were also checked with the participant and a letter of confirmation (Appendix 8) was sent to each participant stating the time and date of the study and outlining the necessary preparation required (despite the reminder letter, there were still a small number of people who forgot that they had agreed to participate on that particular night). On the night of the study, participants were requested to adhere as closely as possible to their normal daily routine and to be in some form of sleepwear when the researchers arrived. In order to make the study as convenient as possible to the participants and to avoid potential attrition, participants were encouraged to reschedule their study if the scheduled time became inconvenient.

All people who did not wish to take part in the overnight sleep study were asked to answer the sleep questionnaire over the phone. Only a small portion of these people declined (n=81, 19.20%).

The sleep study protocol

Participants were first required to read and sign the consent form if they had not already done so (as these were sent with the information packs), and to complete the sleep questionnaire. Following this, a single blood pressure measurement was taken, along with neck circumference, height, and weight. Additional questions were also asked regarding the participant's usual sleep and wake times, and alcohol and caffeine consumption on the day of the study, and their usual consumption. Current medication usage was also noted. If the participant was not already changed into sleepwear, they

were asked to do so. The MESAM4 equipment was then attached. At the end of the night, instructions regarding the equipment and protocol were provided (Appendix 1), and participants were given a contact telephone number in the case of an emergency.

In the morning, equipment was disconnected and subjective reports of sleep and wake times were noted. Participants were also asked to rate their sleep compared to a normal night's sleep and whether they had experienced any difficulties with the equipment. MESAM4 recordings were downloaded via a serial port connected to a computer, saved, and subsequently printed out using a laser printer for scoring. If however, the participant had to get up exceptionally early, they were instructed on how to safely remove the equipment, and alternative arrangements were made to collect the equipment and questionnaire information.

Ethical and cultural considerations

Cultural Safety

Collecting data from Māori participants raised a number of issues regarding cultural safety. Cultural safety is based within a framework of biculturalism, and is congruent with the tenets of the Treaty of Waitangi (Richardson 2004).

As a Māori researcher, I was involved in all aspects of the research process and data collection with Māori participants. It was noted that a few Māori participants commented that they would not have taken part in the study if I was not Māori. This may reflect attitudes towards the colonising aspects of research in New Zealand that have resulted in distrust and aversion to research among some Māori individuals and communities (Smith 1999).

The following points were raised as important when dealing with Māori participants in a study of this nature. These points were seen as guidelines rather than absolutes, bearing in mind that Māori are a heterogeneous group and therefore have diverse realities (Durie 1998).

1. The head is the most sacred part of the body for many Māori; therefore it should be treated with care and respect.

2. When entering a setting specifically set aside for Māori processes and meeting one should always remove shoes. This includes going to the homes of Māori

participants, who may be too polite to ask you to do this, but who will feel a lot happier about your attitude of respect if you do follow this basic courtesy.

3. When setting up the sleep equipment one should not set-up in areas that are typically used for food (i.e., kitchen, dining table) unless told otherwise. It is considered unhygienic and tapu (sacred) to put items that come into contact with the body in these areas.

In addition to cultural safety surrounding Māori participants, issues of cultural safety were considered for all participants, in terms of gender, sexuality, social class, occupation group, generation, ethnicity or a combination of variables. This expanded definition of cultural safety is informed by the work of the late Irihapeti Ramsden (2003).

Researcher safety

The personal safety of researchers was also a consideration of this study as data was collected in participants' homes. To minimise potential risks, researchers travelled in pairs and carried a mobile phone at all times.

Obligation to participants

If significant sleep disordered breathing (SDB) was indicated from a study, the participant was informed by phone and a letter was sent to their General Practitioner (Appendix 9), if requested. To categorise the severity of sleep disordered breathing for this purpose, the respiratory sleep physician used the following criteria:

1.	No significant sleep disordered breathing =	RDIc < 10
2.	Mild obstructive sleep apnoea syndrome=	RDIc 10-20
3.	Moderate obstructive sleep apnoea syndrome =	RDIc 20-40
4.	Severe sleep apnoea syndrome =	RDIc greater than 40

3.6.2 The clinical sample

As mentioned previously, data collection at the sleep clinic coincided with collection in the community. The sleep technician on duty briefed patients about the study, and written consent was obtained (Appendix 10). Patients also completed the sleep questionnaire. PSG studies were either conducted at the clinic or at the patients' home as unattended portable PSG studies. Essentially the data gathered from these two types of studies were the same, except that the portable studies did not provide snoring data.

The usual procedures of the sleep clinic were carried out with the clinical participants. Prior to their sleep studies, patients were instructed via letter that on the night of their study they should adhere to their normal daily routine, including alcohol, caffeine and medication usage, however napping is discouraged. Patients whose study was held at the clinic were encouraged to bring their *favourite* pillow, or they were provided with pillows of comparable height.

3.7 Data Management

All questionnaire data for community and clinic participants were double entered and checked independently in separate Epi-Info (Version 6.04a, World Health Organisation) databases. Data files were then converted to SPSS™ (Version 11) and SAS™ (Version 8.02) files for statistical analysis. MESAM4 raw scores were entered into a Microsoft Excel™ spreadsheet and systematically checked for data entry errors. The required sleep study variables were then calculated from the raw scores and subsequently merged with the respective questionnaire data using Microsoft Access™. Similarly, polysomnography variables were double entered and checked in Epi-Info and merged with respective questionnaire data.

3.7.1 Statistical analyses

While specific analyses are discussed in respective chapters, this section discusses the main statistical analyses used. The primary breakdown of data is by ethnicity and sex, and statistical significance was accepted at the level of $p<0.05$.

Univariate analyses

All data were graphically screened for normality according to the specific groupings required for each analysis (Māori men, non-Māori men, Māori women, non-Māori men). Where proportions are calculated, exact 95% confidence intervals are presented and comparisons between groups were conducted using chi-squared (χ^2) tests, with Yates continuity adjusted p-values. Where tests are not presented and 95% CI did not overlap, p was assumed to be less than 0.05. Where expected numbers in cells fell below 5, chi-square tests were not calculated as the tests were no longer valid. All continuous data in this study were non-normal, so Wilcoxon's rank sum tests were used to test for differences in medians. Alongside median values, interquartile ranges (IQR) are presented. To examine differences in continuous distributions between all four analysis groups, the Kruskal-Wallis non-parametric test was used

Population prevalence estimates

In Chapter 4 (The community sample), general population prevalence estimates are calculated for OSA, as defined by RDIa ≥ 5, ≥ 10, ≥ 15 for Māori and non-Māori, men and women. Adjusted prevalence estimates of general sleep variables and OSAS symptoms and risk factors, such as poor quality sleep, snoring always, observed

apnoeas, neck circumference, and excessive daytime sleepiness are not reported, as these have been presented in the national sleep survey (Harris 2003).

As the sample was stratified, data were weighted by the population proportions in 10-year age groups for 30-60 years olds in each sex and ethnicity group. Weightings were derived from the 1996 census using Māori ethnic group (MEG) numbers in the Wellington region (Statistics New Zealand 1997a). Population numbers are detailed in Appendix 11. Confidence intervals and p-values were calculated using variances derived from stratified sampling (Snedecor and Cochran 1967).

Logistic regression analysis

The primary multivariate statistical method utilised in this study was logistic regression modelling. Logistic regression modelling allows prediction of a binary or dichotomous outcome from a group of variables that can be continuous, discrete, dichotomous, or a combination. The specific details of the structure of each model are discussed in the respective chapters. Results of the logistic regression models are primarily presented as odds ratios and 95% confidence intervals. The variables considered in the logistic regression models are detailed in Table 3.4.

Table 3.4 Coding and variable names for possible predictive values

Possible predictive variable (Covariates)	Variable names and descriptions
Ethnicity	Dichotomous variable: Māori=1 non-Māori=0
Sex	Dichotomous variable: Men=1 Women=0
Age	***Age 1***: Continuous variable: 1 year increase ***Age 2***: Continuous variable: 10 year increments
CSC eligibility	Dichotomous variable: Yes=1 no/don't know=0
BMI	***BMI 1***: Continuous variable ***BMI 2***: Categorical variable (Reference group=normal/underweight) 1. Overweight 2. Obese
Neck circumference	***Neck 1***: Continuous variable: cm increase ***Neck 2***: Dichotomous variable: > national NZ average=1 <national NZ average=0 *Averages were calculated from the national sleep survey (Harris 2003) for each group (Māori men, non-Māori men, Māori women, non-Māori women)
Epworth Sleepiness Score	***ESS 1***: Dichotomous variable: ESS>10=1 ESS≤10=0 ***ESS 2***: Categorical variable (Reference group= 'ESS≤10'): 1. 11-15 2. 16+
Snore	***Snore 1***: Dichotomous variable: Often=1 Always/Rarely/Never=0 ***Snore 2***: Dichotomous variable: Often/Always=1 /Rarely/Never=0 ***Snore 3***: Categorical variable (Reference group= 'never'): 1. Rarely 2. Often 3. Always 4. Don't know
Observed apnoeas	***Apnoeas***: Dichotomous variable: Yes=1 No=0
Wake feeling refreshed	***Refreshed 1***: Dichotomous variable: Never/Rarely=1 Often/Always=0 ***Refreshed 2***: Categorical variable (Reference group= 'never'): 1. Rarely 2. Often 3. Always
Getting enough sleep	***Enough 1***: Dichotomous variable: Never/Rarely=1 Often/Always=0 ***Enough 2*****:** Categorical variable (Reference group= 'never'): 1. Rarely 2. Often 3. Always
Asthma	***Asthma***: Dichotomous variable: Yes=1 No/Don't know=0
Hypertension	***Hypertension***: Dichotomous variable: Yes=1 No/Don't know=0
Heart Trouble	***Heart***: Dichotomous variable: Yes=1 No/Don't know=0
Diabetes	***Diabetes***: Dichotomous variable: Yes=1 No/Don't know=0
Stroke	***Stroke***: Dichotomous variable: Yes=1 No/Don't know=0
Thyroid problem	***Thyroid***: Dichotomous variable: Yes=1 No/Don't know=0
Psychological problem	***Psych***: Dichotomous variable: Yes=1 No/Don't know=0
Sleep problem	***Sleep***: Dichotomous variable: Yes=1 No/Don't know=0

Table 3.4 Coding and variables names of possible predictive variables for OSA (cont ….)

Possible predictive variables (Covariates)	Description
Smoking	***Smoking 1***: Dichotomous variable: Regular/Occasional=1 Ex-smoker/Non smoker=0 ***Smoking 2***: Dichotomous variable: Regular=1 Occasional/Ex-smoker/Non-smoker=0 ***Smoking 3***: Categorical variable (Reference group=Non-smoker): 1. Regular smoker 2. Occasional smoker 3. Ex-smoker
Alcohol	Four independent categories were developed from Questions 15 and 16 with regards to frequency and amount of alcohol consumed: 1. Non-drinkers=participants who answered 'never' to question 15 (How often do you drink alcohol?) 2. Exceeding recommended limits=participants whose reported alcohol consumption was more than the recommended upper limit on an occasional or per week according to ALAC guidelines (1997). 3. Daily alcohol=participants who reported consuming alcohol daily, but not more than the recommended upper limit on an occasion or per week 4. Moderate alcohol= participants who reported consuming alcohol, but not daily and not more than the recommended upper limit. From these four variables three independent dichotomous alcohol variables were able to be tested: ***Alcohol 1***: Dichotomous variable: moderate drinker =1 non-drinkers=0 ***Alcohol 2***: Dichotomous variable: daily drinker =1 non-drinkers=0 ***Alcohol 3***: Dichotomous variable: exceed recommended limits =1 non-drinkers=0

CHAPTER 4
THE COMMUNITY SAMPLE

4.1 Introduction

This chapter is divided into five main sections. The first section examines study response rates, comparing those from the electoral rolls sample who were contacted and those who could not be contacted. The second examines the demographic profiles and questionnaire responses of *MESAM4 participants*, which are compared with information from *questionnaire only participants* and *national sleep survey participants*[12]. The third section examines the additional data collected from participants who had a MESAM4 study, such as body mass index and neck circumference. The fourth section specifically focuses on the objective sleep data collected. Unadjusted prevalence and adjusted prevalence rates are presented for obstructive sleep apnoea (OSA). The final section assesses the validity of self-reported snoring frequency and observed apnoeas.

4.2 Methods

4.2.1 Measures

Details regarding the questions and the nature of objective sleep data (MESAM4) collected in the community sample are outlined in Chapter 3 (Methods).

4.2.2 Statistical analyses

To provide an overview of each variable, descriptive statistics are presented in this chapter for all data, summarised by ethnicity and sex. Age was not considered in these analyses as it was controlled for by the age stratification of the sample.

Population prevalence estimates

In order to adjust for the possible effect on disease rates of different age structures (Borman 1992), prevalence estimates for OSA were calculated by weighting the sample

[12] To avoid confusion in this chapter, individuals who agreed to an overnight sleep study are referred to as '*MESAM4 participants*', and individuals who only answered the questionnaire are referred to as '*questionnaire only participants*'.

prevalences by the 1996 New Zealand census population proportions in the Wellington region in 10-year age groups from 30-60 years, for each sex and ethnic group.

Logistic regression analyses

Logistic regression analyses were used to identify independent predictors of OSA. Three logistic regression models were run at three thresholds of RDIa and RDIc (≥ 5, ≥ 10, ≥ 15) as follows:

Model 1 : ethnicity, sex and age.
Model 1a: ethnicity, sex, age, body mass index (BMI), and other variables (see Table 3.4)
Model 1b: ethnicity, sex, age, neck circumference, and other variables (see Table 3.4)

Collinearity

Prior to entry into the logistic regression models, variables were assessed for collinearity using a correlation matrix. Where collinearity occurs, there are significant interrelationships ($r>0.70$) between predictor variables, which may cause inflation of the variance of the parameter estimates (Tabachnick and Fidell, 1996). Although a strong correlation between neck circumference and BMI was not found in the community sample, other studies have shown a very strong correlation between the two (Ben-Noun et al. 2001, Hoffstein and Mateika 1992), therefore separate models were created for each of these variables.

Models were also scanned for inordinately large parameter estimates or standard errors, which generally indicated too many empty cells for a particular variable. For this reason, of the co-morbid conditions only asthma and hypertension were included in multivariate analyses.

4.3 Response Rates

Of the 1200 people selected from the electoral roll, 786 (66%) were successfully contacted by phone. Of those who were contacted, 364 (Māori=169, non-Māori=195) agreed to overnight sleep monitoring in their homes. A further 341 (Māori=137, non-Māori=204) who declined overnight sleep monitoring, answered the sleep questionnaire over the phone. Eighty-one people (Māori=42, non-Māori=39) refused to participate in the study (Table 4.1).

Table 4.1 Breakdown of community responses

Response Description	Māori n (%)	non-Māori n (%)	Total n (%)
MESAM4 sleep study	169 (14.08%)	195 (16.25)	364 (30.33%)
Answered questionnaire only	137 (11.42)	204 (17.00%)	341 (28.42%)
Refused	42 (3.50%)	39 (3.25%)	81 (6.75%)
Unable to contact	198 (16.50%)	127 (10.58%)	325 (27.08%)
Return to senders (RTS)	35 (2.92%)	22 (1.83%)	57 (4.75%)
Deceased/overseas/moved out of town	19 (1.58%)	13 (1.08%)	32 (2.67%)
Total	600 (50%)	600 (50%)	1200 (100%)

Table 4.2 and Table 4.3 respectively summarise the demographic breakdown of those who agreed to an overnight sleep study (*MESAM4 participants*) and those who answered the questionnaire only (*questionnaire only participants*).

Table 4.2 MESAM4 responders, by ethnicity, sex and age group

Age group (years)	Māori		non-Māori		Total n (%)
	Men n (%)	Women n (%)	Men n (%)	Women n (%)	
30-39	20 (5.49%)	26 (7.14%)	26 (7.14%)	26 (7.14%)	96 (26.37%)
40-49	28 (7.69%)	29 (7.96%)	39 (10.71%)	39 (10.71%)	129 (35.44%)
50-59	39 (10.71%)	41 (11.26%)	34 (9.34%)	34 (9.34%)	139 (38.19%)
Total	87 (23.90%)	96 (26.37%)	96 (26.37%)	99 (27.20%)	364 (100%)

Table 4.3 Questionnaire only responders, by ethnicity, sex and age group

Age group (years)	Māori		non-Māori		Total
	Men	Women	Men	Women	
30-39	18 (5.28%)	21 (6.16%)	21 (6.16%)	24 (7.04%)	84 (24.63%)
40-49	23 (6.74%)	4 (1.17%)	22 (6.45%)	46 (13.48%)	115 (33.72%)
50-59	21 (6.16%)	30 (8.80%)	41 (12.02%)	50 (14.66%)	142 (41.64%)
Total	62 (18.18%)	75 (21.99%)	84 (24.63%)	120 (35.19%)	341 (100%)

Response rates (RR) were calculated by descent, as only descent not ethnicity of non-responders was available from electoral roll information. A number of different methods of calculating response rates were explored (Table 4.4).

Table 4.4 Response rate equations and calculations

	Equation	Calculation	RR (%)
RR_1	$\frac{\text{number of people who agreed to sleep study}}{\text{number in original sample}}$	364/1200	30.33
RR_2	$\frac{\text{number of people who agreed to sleep study}}{\text{number in original sample-(deceased/overseas/moved out of town)}}$	364/1168	31.12
RR_3	$\frac{\text{number of people who agreed to sleep study}}{\text{(1170-RTS)}}$	364/1111	32.76
RR_4	$\frac{\text{number of people who agreed to sleep study}}{\text{number of people contacted}}$	364/786	46.31
RR_5	$\frac{\text{total number of people who answered the questionnaire}}{\text{number in original sample}}$	705/1200	58.75
RR_6	$\frac{\text{total number of people who answered the questionnaire}}{\text{number in original sample- (deceased/overseas/moved out of town)}}$	705/1170	60.20
RR_7	$\frac{\text{total number of people who answered the questionnaire}}{\text{(1170-RTS)}}$	705/1113	63.34
RR_8	$\frac{\text{total number of people who answered the questionnaire}}{\text{number of people contacted}}$	705/786	89.70

If the initial electoral roll sample is considered, the response rate for agreeing to an overnight sleep study is 30.33% (RR_1). However to be included in this study, participants had to be currently living in the Wellington area. During the course of recruitment, 32 people were found to be living outside the Wellington area or were deceased. Excluding these people from the denominator increases the response rate to 31.12% (RR_2). Similarly, if return to senders (RTS) (n=57) are excluded, the response rate increases further to 32.76% (RR_3). From a less conservative perspective, if only

people contacted are considered, the rate further increases to 46.31% (RR_4). Alternatively, if all questionnaire responses are considered, the response rate is further increased to 58.7% (RR_5). Furthermore, if only people who were contacted are considered, the response rate for all questionnaire data collected is 89.70% (RR_8).

Differences between *questionnaire only participants* and *MESAM4 participants* in their responses to the questionnaires are addressed in subsequent sections of this chapter. However no differences in the age or socioeconomic deprivation distributions were found between those who agreed to a MESAM4 study and those who answered the questionnaire only or refused both, and furthermore no differences were found in the odds of responding (agreeing to an overnight sleep study) by ethnicity or sex.

In terms of questionnaire data, given the high response rate amongst those who were contacted, response analyses focused predominately on differences between those who were contacted versus those who were not. The electoral roll information, descent, age and socioeconomic profiles were used to assess potential biases in the sample that were contacted.

Table 4.5 demonstrates that more non-Māori than Māori could be contacted in each age group, with a significant trend for an increasing chance of being contacted for non-Māori with increasing age, but not for Māori.

Table 4.5 People who were contacted, by ethnicity and age group

	Māori			**non-Māori**		
Age group (yrs)	**Contacted (n)**	**Total**	**Contacted (%)**	**Contacted (n)**	**Total**	**Contacted (%)**
30-39	113	193	59%	129	192	67%
40-49	117	192	61%	148	195	76%
50-59	118	196	60%	161	200	81%
Total	**348**	**581**	**60%**	**438**	**587**	**75%**
p-value for trend*			**0.7404**			**0.0025**

*Cochran-Armitage test for trend

Socioeconomic deprivation (NZDep96) profiles were also analysed to examine the possible influence of deprivation on the likelihood of being contacted. Those who were contacted were significantly less deprived than those who were not contacted (χ=82.91, p<0.0001). Given the known differences in deprivation profiles between Māori and non-Māori, these samples were tested separately. Trends (Cochran-Armitage test for trend) were calculated for Māori and non-Māori within each decile. For both Māori and

non-Māori, significant trends ($p<0.0001$) indicated that chances of being contacted decreased with increasing deprivation. This is clearly illustrated in Figure 4.1.

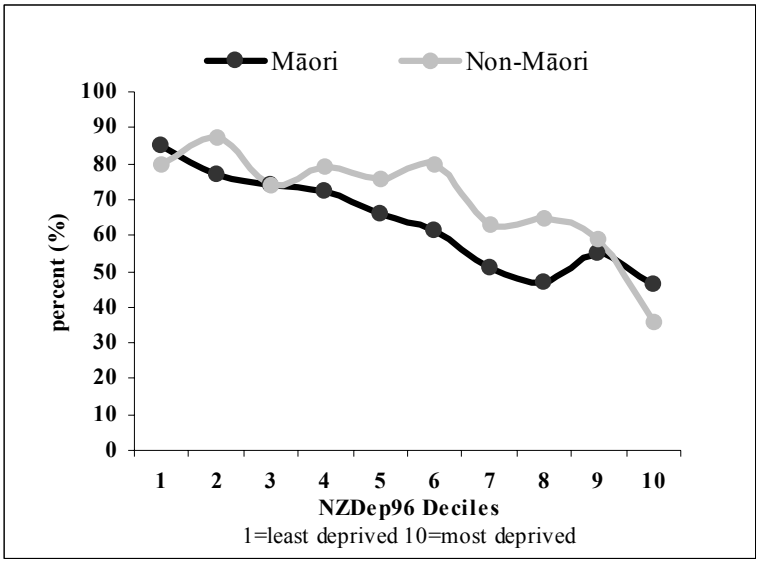


Figure 4.1 Percentage of Māori and non-Māori contacted, by NZDep96

Logistic regression was used to test whether the differences in being contacted between Māori and non-Māori were therefore primarily due to the NZDep96 profiles of the two groups and the contact bias demonstrated by the level of deprivation (Table 4.6).

Table 4.6 Odds of being contacted

	Variable	Odds ratio	95% CI	p-value
Model 1*	Non-Māori vs. Māori	1.97	1.53-2.53	<0.0001
Model 2*	Non-Māori vs Māori	1.30	0.99-1.70	0.0641
	NZDep96	0.83	0.80-0.87	<0.0001

* 1113 observations

Model 1 shows that the odds of being contacted were significantly higher for non-Māori compared with Māori. However when deprivation was added to the model (Model 2), the difference between Māori and non-Māori was no longer significant. This suggests that differences in NZDep96 profiles accounted for the majority of the difference between Māori and non-Māori being contacted.

To investigate possible sampling biases, deprivation distributions in the electoral roll sample (n=1200) were compared with distributions in the Wellington region adult population (Figure 4.2).

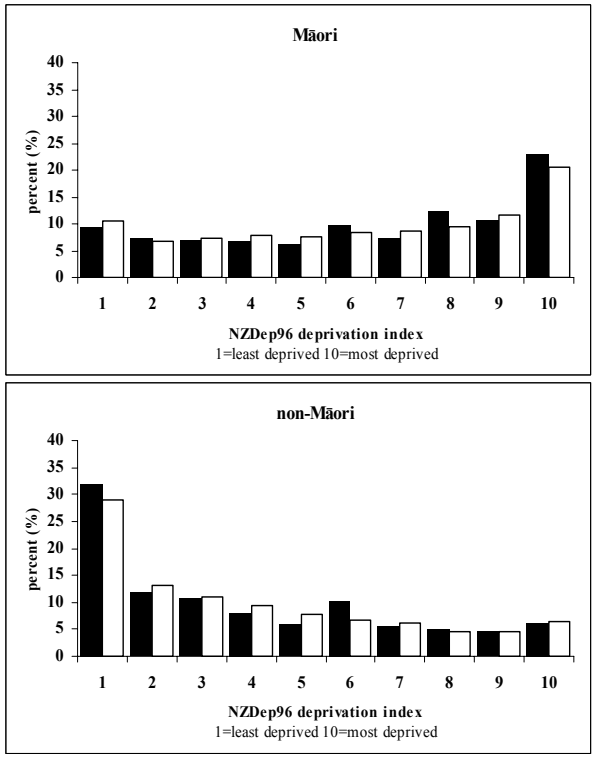


Figure 4.2 NZDep96 profiles: Initial sample vs. Wellington Adults

(Source: Crampton et al. 2000a)

The socioeconomic deprivation profiles of the electoral roll sample closely resembled those of the Wellington population, indicating that the initial study sample draw was representative of the Wellington adult population.

4.4 MESAM4 Analytical Sample

Six participants (3 Māori and 3 non-Māori) were removed from further analyses due to excessive artefact in their respective MESAM4 sleep data, which left data from 358 participants available for final analysis. A breakdown of the analytical sample by ethnicity and sex is presented in Table 4.7.

Table 4.7 Analytical sample, by ethnicity, sex and age group

	Mean age (yrs)	Age group (yrs)			Total
		30-39	40-49	50-59	
Māori men	45.37	23	33	25	**81**
Māori women	47.14	19	28	38	**85**
Non-Māori men	46.86	26	28	41	**95**
Non-Māori women	46.05	25	38	34	**97**
Total	**46.37**	**93**	**127**	**138**	**358**

Non-Māori men and women comprised the largest portion of the sample, at 27% respectively, followed by Māori women (24%), and Māori men (23%). The male to female ratio was even (49% men, 51% women). Age distributions did not differ significantly between the four groups (χ^2=2.47, DF=3, p=0.4815), or between sex within ethnicity or ethnicity within sex. However examination by deprivation profiles highlights clear differences between Māori and non-Māori (Figure 4.3).

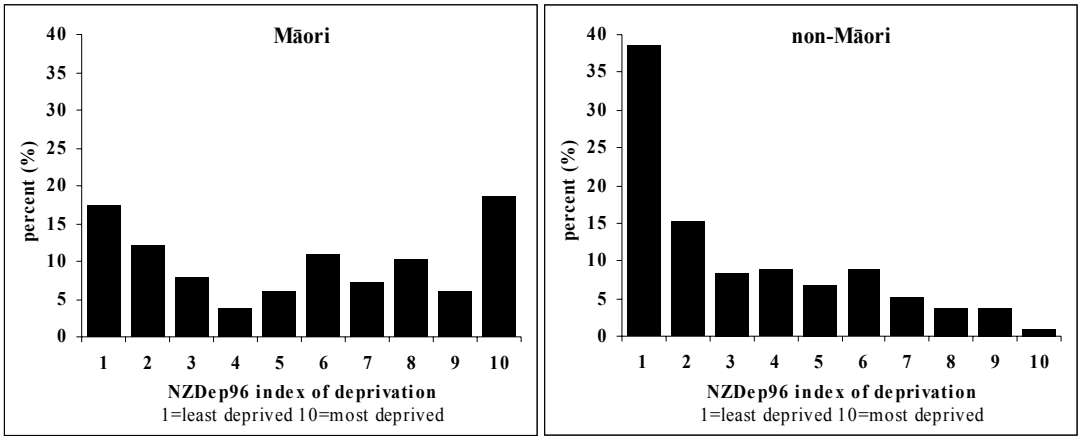


Figure 4.3 NZDep96 profiles of MESAM4 participants, by ethnicity

To situate *MESAM4 participants* relative to the general population, deprivation profiles were compared with those found in the national sleep survey (Figure 4.4).

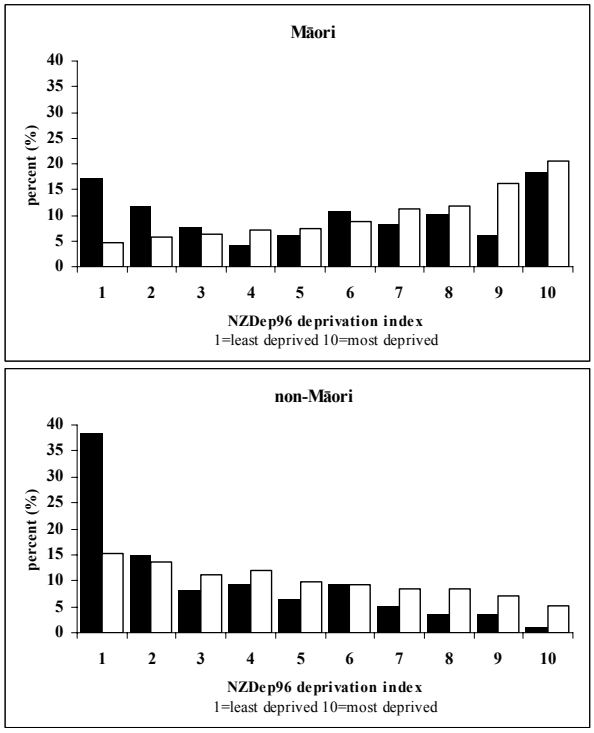


Figure 4.4 NZDep96 profiles: MESAM4 participants vs. National sleep survey

(Source: Harris 2003)

Both Māori and non-Māori *MESAM4 participants* were less deprived than Māori and non-Māori national sleep survey responders.

The deprivation profiles of *MESAM4 participants* were also compared with adults in the Wellington region (Figure 4.5).

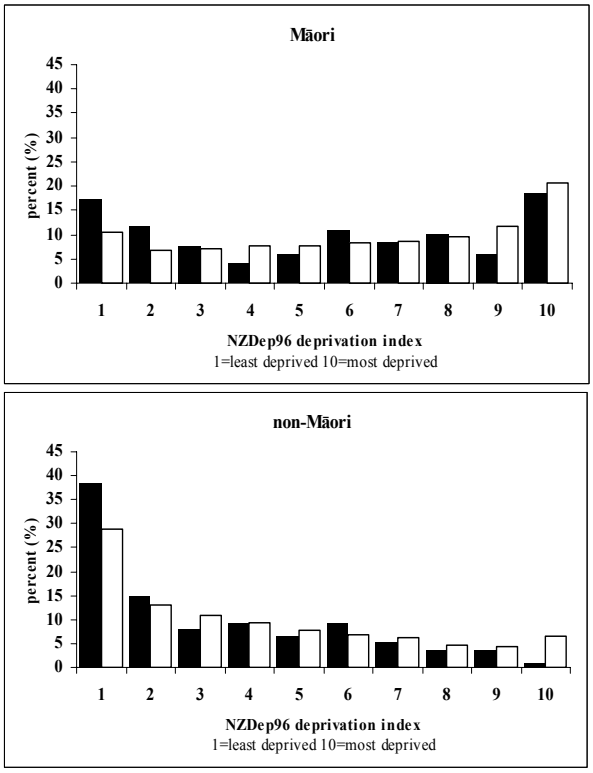


Figure 4.5 NZDep96 profiles: MESAM4 participants vs. Wellington region adults

(Source: Crampton et al. 2000a)

Both Māori and non-Māori participants were overrepresented in the less deprived deciles and underrepresented in the most deprived deciles.

These analyses suggest that the deprivation bias seen in the analytical sample relative to the general population was not only due to the bias of being able to contact individuals who were less deprived, but also to the particular socioeconomic profile of the Wellington adult population.

4.4.1 General sleep variables

Getting enough sleep

Participants were asked how often they think they get enough sleep, with the options of *never*, *rarely*, *often* or *always*. Their responses are displayed in Figure 4.6.

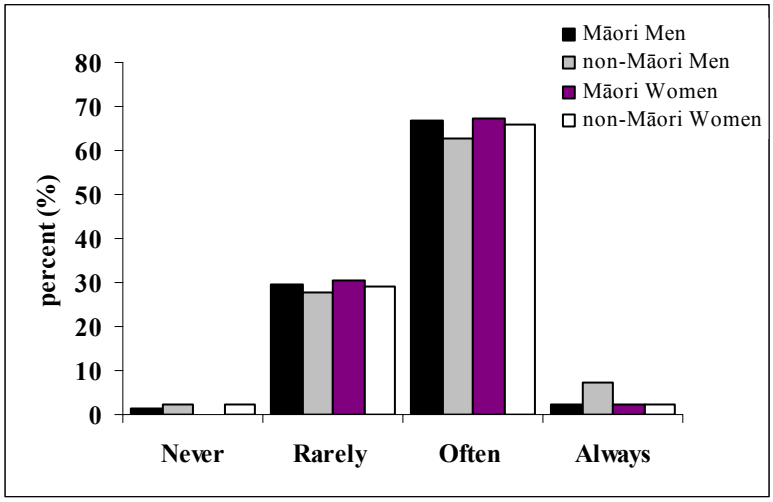


Figure 4.6 MESAM4 participants: How often do you think you get enough sleep?

Responses were similarly distributed across groups, with the majority of participants indicating that they *often* get enough sleep (57.26%).

As a measure of self-perceived chronic sleep restriction, *never* and *rarely* categories were collapsed and proportions calculated by ethnicity and sex (Table 4.8).

Table 4.8 MESAM4 participants vs. Questionnaire only and National survey: Never/rarely get enough sleep, by ethnicity and sex

	Māori		non-Māori	
	Men	Women	Men	Women
MESAM4	30.49 (20.80-41.64)	29.89 (20.33-39.33)	29.17 (20.54-40.65)	32.32 (23.27-42.47)
Questionnaire only	41.49 (29.51-55.15)	30.67 (20.53-42.38)	28.57 (44.85-70.49)	25.00 (17.55-33.73)
National survey	37.92 (34.80-39.84)	37.05 (34.76-39.39)	36.42 (34.13-38.76)	35.37 (33.28-37.50)

Thirty-one percent of *MESAM4 participants* (n=110) reported *never* or *rarely* getting enough sleep. Chi-squared comparisons indicated no significant differences between Māori and non-Māori, men and women. Furthermore, no significant differences between men and women within ethnic groups were found. The proportions of

MESAM4 participants who reported chronic sleep restriction were not significantly different from those for the questionnaire only or national survey respondents.

Wake feeling refreshed

Participants were asked how often they wake feeling refreshed, with the options of *never*, *rarely*, *often*, or *always* (Figure 4.7).

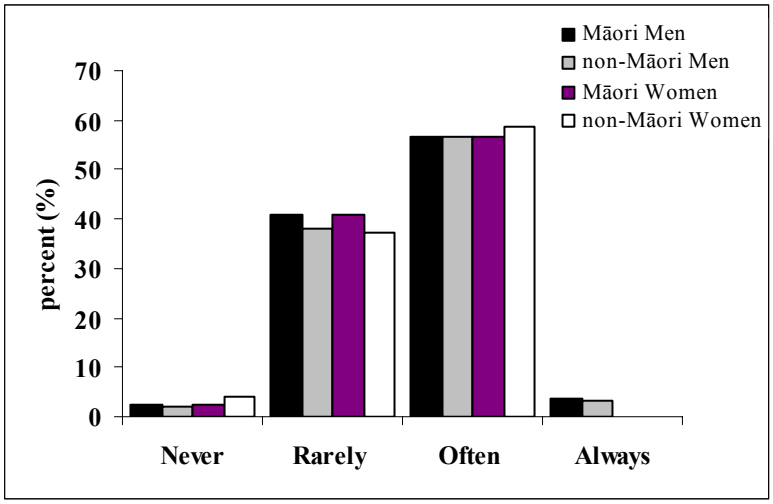


Figure 4.7 MESAM4 participants: How often do you wake feeling refreshed?

Responses were equally distributed across groups, with the majority of participants indicating that they 'often' wake feeling refreshed (65.36%).

Again, *never* and *rarely* categories were collapsed as a measure of self-perceived poor quality sleep. In total, 41% of *MESAM4 participants* (n=147) reported *never* or *rarely* waking feeling refreshed.

Table 4.9 MESAM4 participants vs. Questionnaire only and National survey: Never/rarely wake feeling refreshed?

	Māori		non-Māori	
	Men	**Women**	**Men**	**Women**
MESAM4	43.90 (32.24-54.69)	41.38 (30.01-50.56)	39.58 (29.52-51.20)	**42.42 (31.34-51.69)**
Questionnaire only	37.10 (25.16-50.31)	32.00 (21.69-43.78)	28.57 (19.24-39.47)	**28.33* (20.49-37.28)**
National survey	43.51 (40.98-46.14)	44.21 (41.84-46.59)	45.80 (43.41-48.20)	43.48 (41.30-45.68)

* $p<0.05$

No significant differences were found between Māori and non-Māori, men and women *MESAM4 participants*. Also no significant differences were found between men and women within ethnic groups.

All group proportions were similar to those found in the national survey. Amongst non-Māori women, those who agreed to a MESAM4 study were more likely to report unrefreshing sleep (42.42% vs. 28.33%, OR=1.50, p=0.03) than those who answered the questionnaire only (Table 4.9).

Average hours sleep in 24 hours

The average duration of sleep reported by participants was 7.41 hours per day (SD 1.42 hours). A summary of data by ethnicity and sex is presented in Table 4.10.

Table 4.10 MESAM4 participants: Usual hours sleep in 24 hours, by ethnicity and sex

	Median (hrs)	Interquartile range (hrs)
Māori men	7.5	7.0-8.0
Māori women	7.5	6.5-8.0
Non-Māori men	7.5	7.0-8.0
Non-Māori women	7.5	7.0-8.0
Total	7.5	6.5-8.0

The grouped data were not quite normally distributed (Figure 4.8) therefore tests for differences in the medians between groups were calculated. Amongst men, there were no significant differences found between Māori and non-Māori (p=0.5168). Differences were found however between Māori and non-Māori women (p=0.0072). Within each ethnic group there were no significant differences by sex (Māori, p=0.0620, non-Māori, p=0.1627).

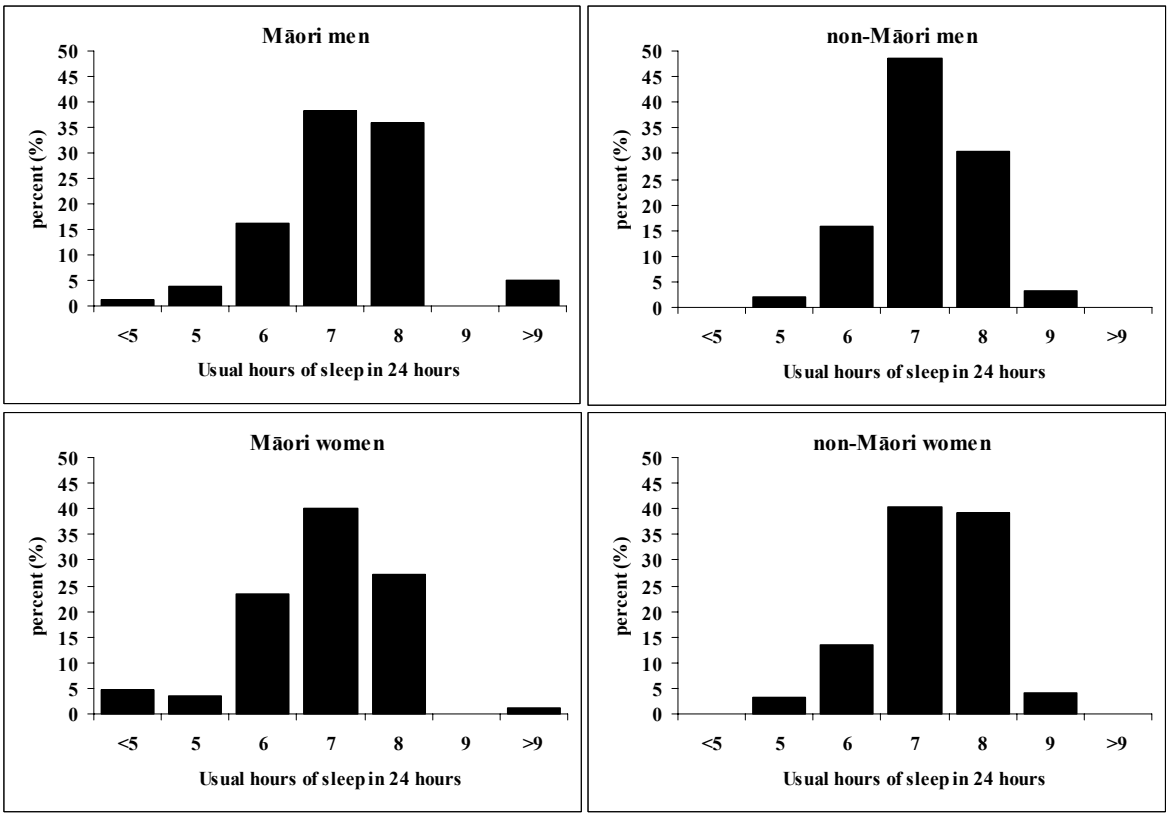


Figure 4.8 MESAM4 participants: Reported usual hours sleep in 24 hours, by ethnicity and sex

When compared with national survey responders and questionnaire only responders, no differences between median average hours sleep were found between groups (Table 4.11).

Table 4.11 MESAM4 participants vs. Questionnaire only and National survey: Median usual hours sleep, by ethnicity and sex

	Māori		non-Māori	
	Men	**Women**	**Men**	**Women**
MESAM4	**7.5 (7.0-8.0)**	**7.5 (6.5-8.0)**	**7.5 (7.0-8.0)**	**7.5 (7.0-8.0)**
Questionnaire only	7.5 (7.0-7.5)	7.5 (6.5-8.0)	7.5 (7.0-8.0)	7.5 (7.0-8.0)
National survey	7.0 (6.5-8.0)	7.5 (6.5-8.0)	7.0 (6.5-8.0)	7.5 (7.0-8.0)

4.4.2 OSAS symptoms

Subjective snoring

Participants were asked how often they snored, with the options of *never*, *rarely*, *often* or *always*. Responses to this question are shown in Figure 4.9.

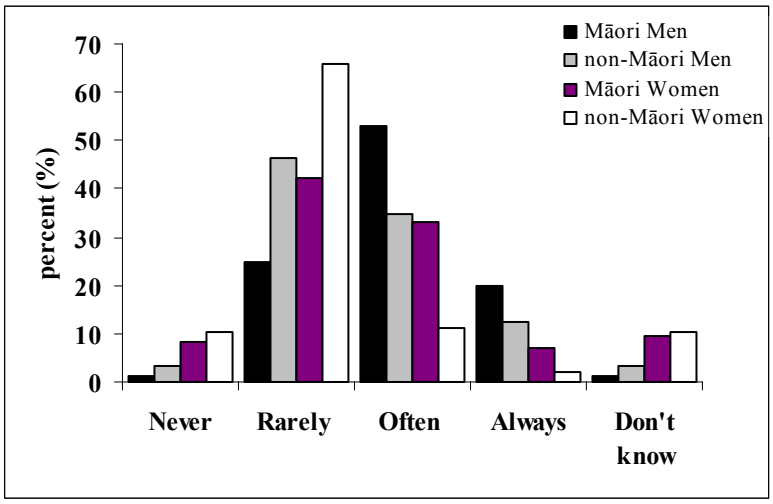


Figure 4.9 MESAM4 participants: How often do you snore?

Although *don't know* was not a valid option, 22 participants wrote it as their response to this particular question. Although differences were found between groups for reporting snoring *always* in the national survey, no differences were found in this study (Table 4.12).

Table 4.12 MESAM4 participants vs. Questionnaire only and National survey: 'Always' snore, by ethnicity and sex

	Māori		non-Māori	
	Men	**Women**	**Men**	**Women**
MESAM4	**20.00 (11.89-30.44)**	**7.79 (2.91-16.19)**	**13.04 (6.93-21.68)**	**2.30 (0.28-7.97)**
Questionnaire only	22.58 (12.93-34.97)	5.88 (1.63-14.38)	13.58 (6.98-23.00)	2.68 (0.56-7.63)
National survey	16.82 (14.92-18.86)	7.76 (6.52-9.15)	10.83 (9.39-12.42)	4.18 (3.35-5.16)

Differences were however detected when *often* and *always* categories were collapsed (Table 4.13).

Table 4.13 MESAM4 participants: Often/Always snore, by ethnicity and sex

	Māori (%)	95% CI	non-Māori (%)	95% CI	Ratio	95% CI	p-value
Men	73.75	62.72-82.96	48.91	38.34-59.56	1.51	1.18-1.93	**0.0009**
Women	44.16	32.84-55.93	14.94	8.20-24.20	2.96	1.69-5.18	**<0.0001**

Within Māori and non-Māori groups, men were significantly more likely to report habitual snoring than women (p<0.001 respectively).

Objective snoring

An overall snore percentage was calculated from scored MESAM4 studies, which indicated the percentage of the night spent snoring. Distributions of the data are illustrated in Figure 4.10 and summarised in Table 4.14.

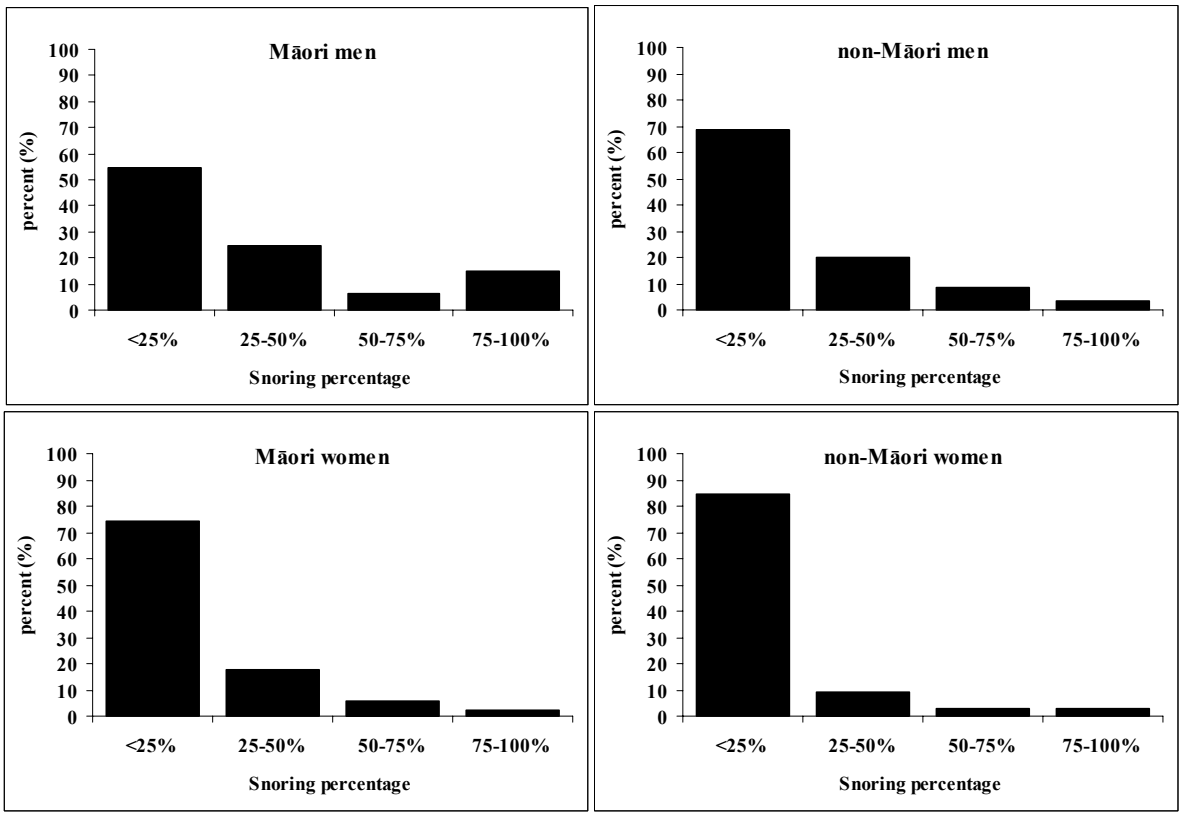


Figure 4.10 Distribution of the percentage of night spent snoring, by ethnicity and sex

As data were not normally distributed, non-parametric tests were conducted to test the difference in medians between groups. For both men and women, Māori spent a higher proportion of the night snoring than non-Māori (Men p=0.0030, Women p<0.0001

respectively). Within ethnic groups, men had significantly greater median time spent snoring than women (Māori p<0.0001, non-Māori p=0.0007) (Table 4.14).

Table 4.14 Percentage of the night spent snoring, by ethnicity and sex

	Māori (IQR)	non-Māori (IQR)	p-value
Men	23.48 (11.75-43.80)	12.06 (1.64-34.58)	0.0030
Women	9.48 (1.69-9.48)	2.47 (0.00-16.08)	<0.0001
p-value	<0.0001	0.0007	

Observed apnoeas

Participants were asked if anyone had ever told them that they stop breathing sometimes during sleep, with the options of *yes* or *no*. The proportion of participants who said that they had been told they stop breathing is illustrated in Figure 4.11. In total, 46 participants (10.27%) reported witnessed apnoeas.

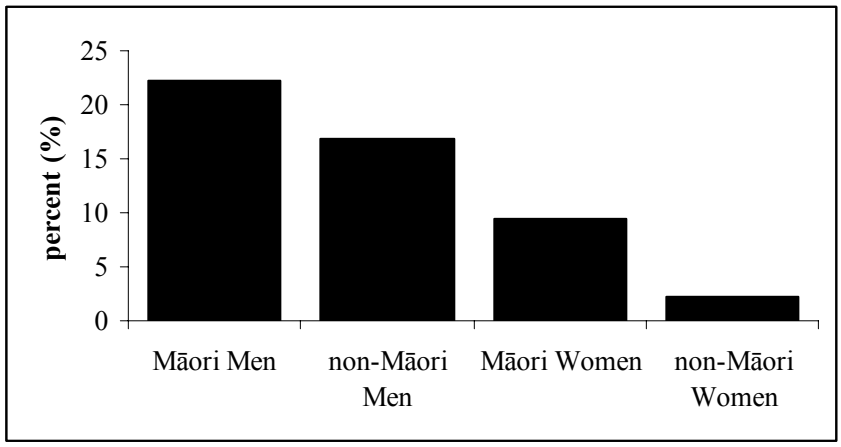


Figure 4.11 MESAM4 participants: Distribution of observed apnoeas, by ethnicity and sex

Differences between Māori and non-Māori, men and women were not statistically significant (Men p=0.37, Women p=0.26). However sex differences were found within ethnic groups (Māori p=0.0003, non-Māori p=0.0232) (Table 4.15).

When compared to the national survey, no differences were found. Although there were larger proportions of MESAM4 responders than questionnaire only responders who reported witness apnoeas, the only significant difference found was for Māori men. Māori men who answered the questionnaire only were significantly less likely to report witnessed apnoeas than those who agreed to a MESAM4 study.

Table 4.15 MESAM4 participants vs. Questionnaire only and National survey: Observed apnoeas, by ethnicity and sex

	Māori		non-Māori	
	Men	**Women**	**Men**	**Women**
MESAM4	**22.22 (13.73-32.83)**	9.41 (4.15-17.71)	16.84 (9.94-25.91)	4.12 (1.13-10.22)
Questionnaire only	**8.06* (2.67-17.83)**	4.00 (0.83-11.25)	10.71 (5.02-19.37)	3.33 (0.92-8.31)
National survey	31.50 (9.11-33.96)	12.13 (10.79-13.96)	19.40 (17.55-21.36)	6.27 (5.25-7.43)

* $p<0.05$

Daytime sleepiness

Sleepiness was assessed using the Epworth sleepiness scale (0-24). Distributions of scores for men and women, by ethnicity are shown in Figure 4.12.

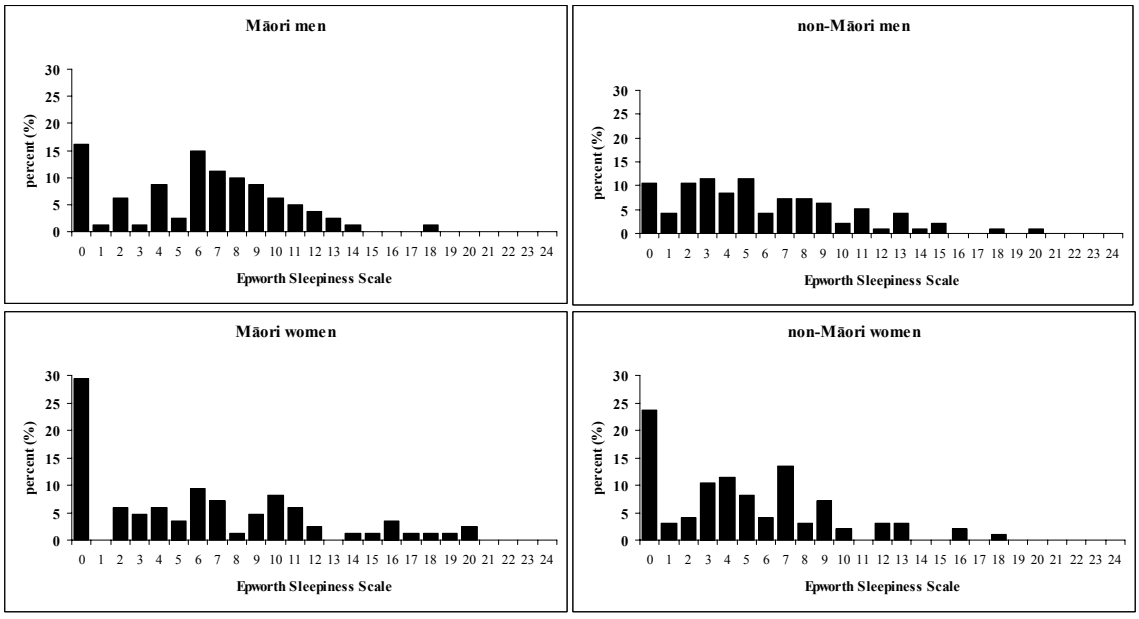


Figure 4.12 MESAM4 participants: Distribution of ESS score, by ethnicity and sex

Table 4.16 presents the proportion of people with scores indicating excessive daytime sleepiness (ESS>10), compared with national survey and questionnaire only responders. No differences were found between Māori men and non-Māori men in reporting excessive daytime sleepiness. Similarly no differences were found between Māori and non-Māori women. Sex differences were found for non-Māori only. No significant

differences were found between *MESAM4 participants* and the *national survey participants* or *questionnaire only participants*.

Table 4.16 MESAM4 participants vs. Questionnaire only and National survey: excessive daytime sleepiness (ESS>10)

	Māori		non-Māori	
	Men	**Women**	**Men**	**Women**
MESAM4	**13.58 (6.98-23.00)**	**20.00 (12.10-30.08)**	**15.79 (9.12-24.70)**	**9.28 (4.33-16.88)**
Questionnaire only	9.68 (3.64-19.88)	5.33 (1.47-13.10)	11.90 (5.86-20.81)	5.00 (1.86-10.57)
National Survey	26.46 (4.15-28.87)	23.35 (21.29-25.50)	16.58 (14.82-18.45)	12.15 (10.73-13.68)

4.4.3 OSAS risk factors

Body Mass Index (BMI)

BMI was calculated from height and weight measurements (BMI= (kg/m²), as measured by the researcher on the night of the sleep study. BMI ranged from 18.54 to 48.07 (Median=27.96, IQR=25.21-30.58). A summary of data by ethnicity and sex are presented in Table 4.17, and BMI distributions are illustrated in Figure 4.13.

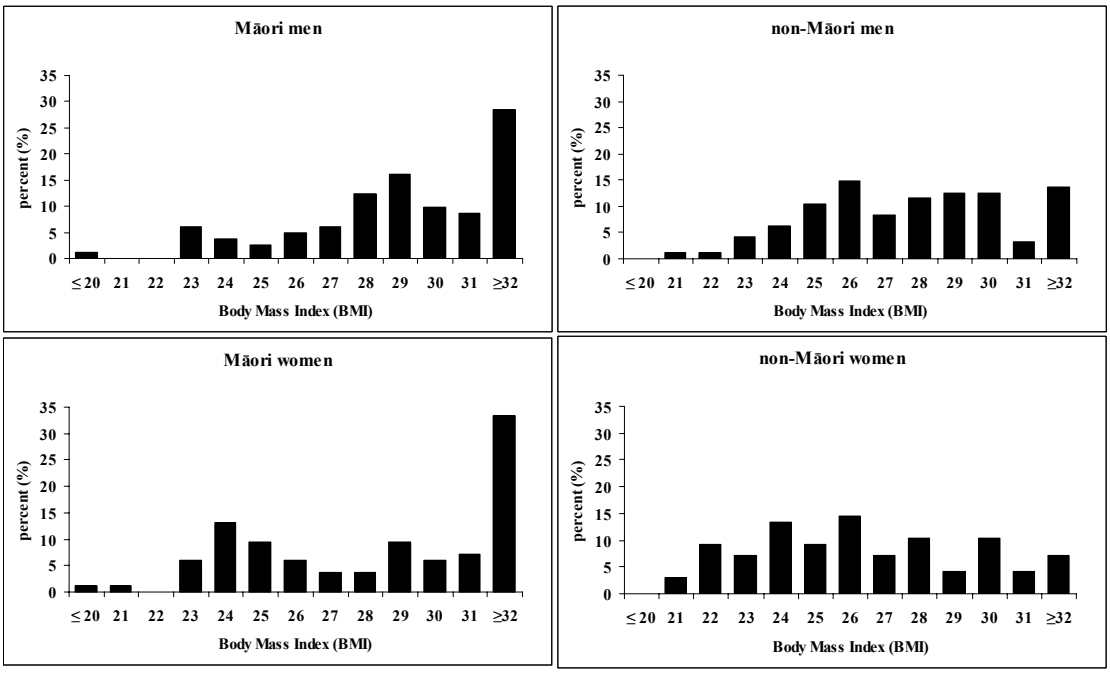


Figure 4.13 MESAM4 participants: Distribution of BMI, by ethnicity and sex

As data were not normally distributed, differences between and within groups were tested using the Wilcoxon test ranked sum test (Table 4.17).

Table 4.17 MESAM4 participants: Median BMI, by ethnicity and sex

	Māori (IQR)	non-Māori (IQR)	p-value
Men	29.21 (27.53-31.77)	27.65 (24.90-32.98)	0.0023
Women	29.14 (24.90-32.98)	26.09 (23.77-28.55)	0.0001
p-value	0.4526	0.0016	

Amongst men, there were significant differences found between Māori and non-Māori (p=0.0023). Similarly, between Māori and non-Māori women, significant differences were found (p=0.0001). Within ethnic groups, no differences were found between Māori men and women (p=0.4526). Among non-Māori, men had a significantly higher BMI than women (p=0.0016). BMI was also examined categorically. Tests between groups are presented in Table 4.18 and Table 4.19.

Table 4.18 MESAM4 participants: Overweight, by ethnicity and sex

	Māori (%)	95% CI	non-Māori (%)	95% CI	Ratio	95% CI	p-value
Men	61.73	50.26-72.31	58.94	48.38-68.94	1.04	0.82-1.33	0.8437
Women	34.12	24.18-45.20	46.39	36.20-56.81	0.73	0.51-1.06	0.4072

No differences were found between Māori and non-Māori, men and women for being overweight. Within ethnic group, men were significantly more likely to be classified as overweight than women.

Table 4.19 MESAM4 participants: Obese, by ethnicity and sex

	Māori (%)	95% CI	non-Māori (%)	95% CI	Ratio	95% CI	p-value
Men	20.99	12.73-31.46	23.16	8.30-23.49	0.91	0.52-1.56	0.7297
Women	23.63	21.05-41.53	17.53	3.63-15.61	1.74	1.02-2.99	**0.0385**

Overall, 23% (23.63% women, 22.16% men) of participants were classified as obese. For men, no differences were found between Māori and non-Māori for the likelihood of being obese. For women, Māori were more likely to be obese (23.63% vs. 17.53%, OR

1.74, p=0.0385). Sex differences within ethnic groups were only significant for non-Māori, where men were more likely to be obese than women.

Neck circumference

Neck circumference was measured by the researcher to the nearest 0.5 cm at the level of the Adam's apple (cricothyroid membrane). Distributions are presented by ethnicity and sex in Figure 4.14.

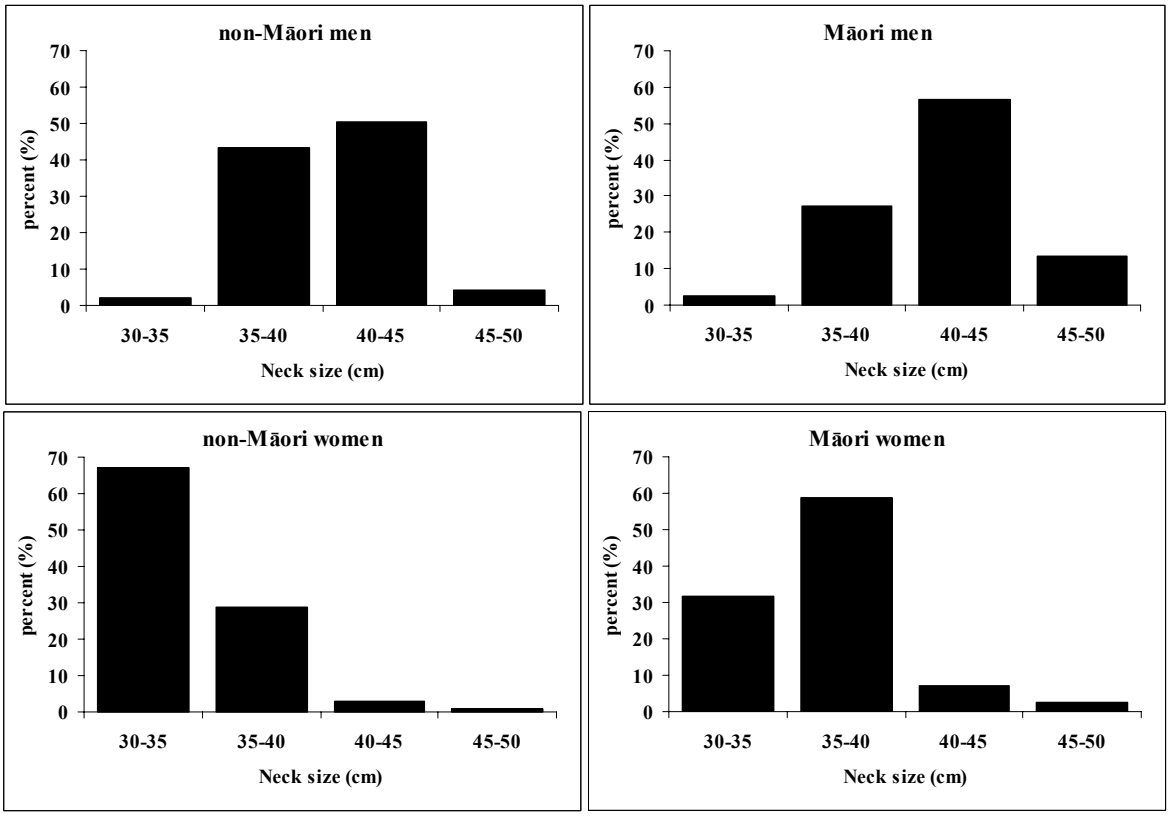


Figure 4.14 MESAM4 participants: Neck circumference distributions, by ethnicity and sex

The data were not normally distributed. Tests for differences in the medians between groups were therefore conducted (Table 4.20). Māori had significantly larger necks than non-Māori (Men p=0.0279, Women p<0.0001). Within each ethnic group, men had larger necks than women (Māori p<0.0001, non-Māori p<0.0001).

Table 4.20 MESAM4 participants: Median neck circumference, by ethnicity and sex

	Māori (cm) (IQR)	non-Māori (cm) (IQR)	p-value
Men	40.00 (39.00-42.00)	41.00 (38.00-42.00)	0.0279
Women	35.50 (34.00-37.50)	33.50 (32.50-35.50)	<0.0001
p-value	<0.0001	<0.0001	

Comparisons could not be made with *questionnaire only participants*, because often neck measurements were not provided, as participants did not have immediate access to a tape measure. This was despite paper tape measures being provided in the study packs, along with a reminder to keep them close to the telephone.

In the national sleep survey (Harris 2003), participants were required to measure their necks with the same paper tape measure. Therefore to validate this method of collecting neck measurements, *MESAM4 participants* were also asked to measure their necks with the same paper tape measure (without instruction from the researcher). These measurements were then compared to measurements obtained by the researchers. The distribution of the measurement differences between participants and researchers are displayed in Figure 4.15.

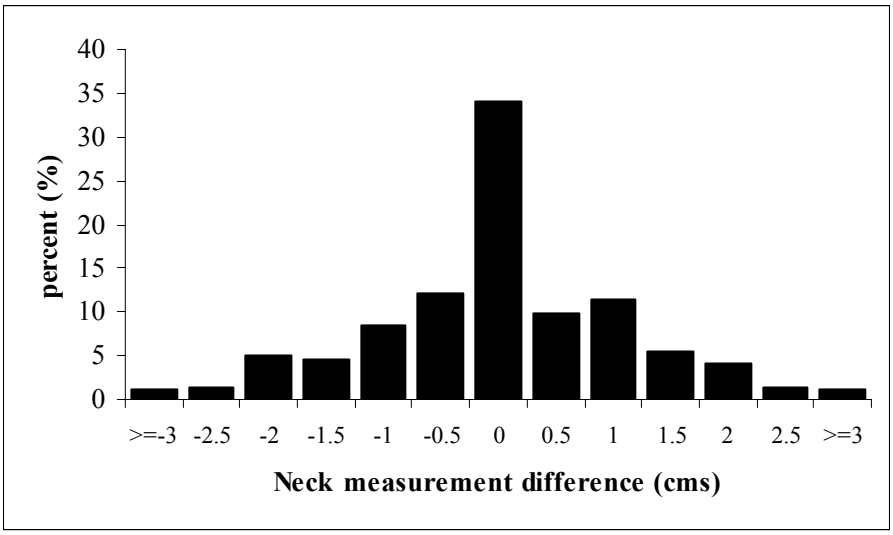


Figure 4.15 MESAM4 participants: Difference between participants and researcher neck measurements

Thirty five percent of participants accurately measured their neck circumference (i.e., no difference between participant and researchers measurements), with the majority (76%) of participants within ± 1 cm of the researchers' measurement. These findings suggest that the method used to collect neck circumference in the national sleep survey was valid and replicable. Neck measurements in this study were therefore compared with those found in the national survey (Table 4.21).

Table 4.21 MESAM4 participants vs. National Survey: Mean neck size, by ethnicity and sex

	Māori		non-Māori	
	Men	**Women**	**Men**	**Women**
MESAM4	40.96 (40.32-41.60)	35.86 (35.33-36.40)	39.91 (39.36-40.46)	34.18 (33.69-34.67)
National Survey	42.18 (41.96-42.41)	36.39 (36.19-36.58)	40.27 (40.11-40.43)	34.36 (34.32-34.60)

No significant differences were found in mean neck measurements between *MESAM4 participants* and *national survey participants*.

Co-morbid disease

Participants were asked whether at the time of the study they were having any treatment for the following conditions: asthma, high blood pressure, heart trouble, diabetes, stroke, thyroid problem, psychological problem, or sleeping problems (Table 4.22).

Table 4.22 MESAM4 participants: Number of people receiving treatment for medical conditions by ethnicity

Medical condition	Māori	non-Māori	Total
Asthma	22	15	37
Hypertension	16	11	27
Heart Trouble	5	5	10
Diabetes	5	0	5
Stroke	5	0	5
Thyroid problem	2	1	3
Psychological problem	2	4	6
Sleep problem	0	5	5
Total	57	41	98

In total, 21% of participants reported having current treatment for at least one or more medical conditions. There were only a handful of people who reported at least two medical conditions (Figure 4.16). Asthma (10.34%) and hypertension (7.54%) were most frequently reported.

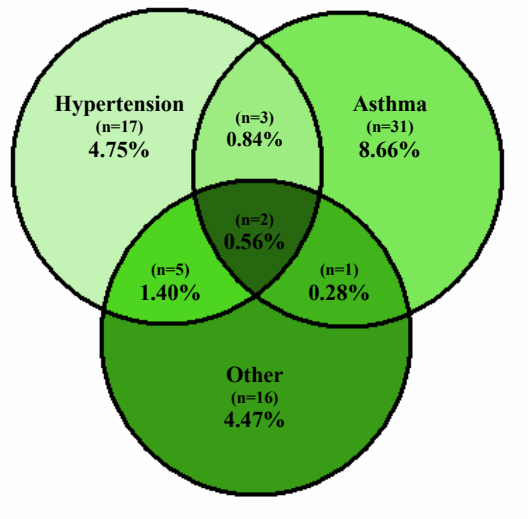


Figure 4.16 MESAM4 participants: Receiving treatment for asthma, hypertension and other medical conditions

Smoking status

In regards to smoking, participants were asked to describe themselves as one of the following: a regular smoker, an occasional smoker, an ex-smoker, or a non-smoker (Figure 4.17).

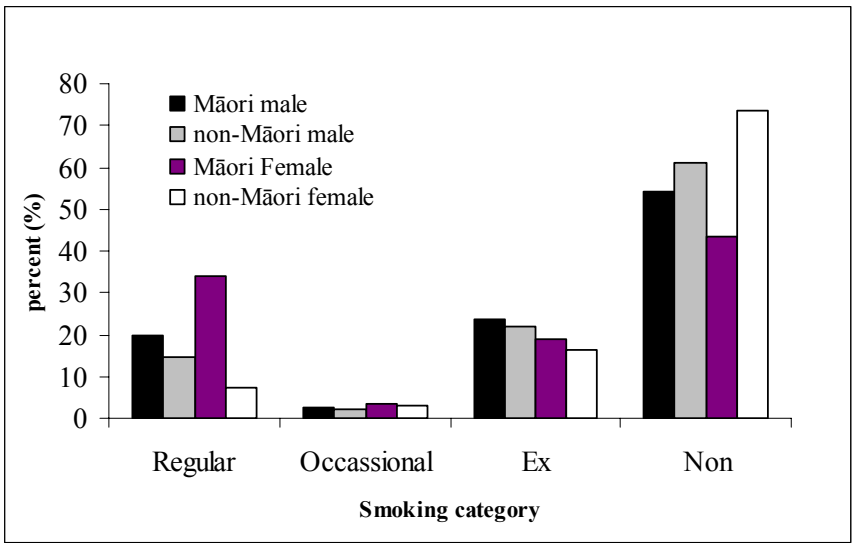


Figure 4.17 MESAM4 participants: Cigarette smoking, by ethnicity and sex

Proportions were calculated for people who said they were regular smokers. As shown in Table 4.23, no significant differences were found between Māori and non-Māori men. Among women, Māori were three times more likely than non-Māori to report regular smoking ($p<0.0001$).

Table 4.23 MESAM4 participants: Regular smokers, by ethnicity and sex

	Māori (%)	95% CI	non-Māori (%)	95% CI	Ratio	95% CI	p-value
Men	22.22	13.73-32.83	16.84	9.94-25.90	1.32	0.72-2.42	0.37
Women	37.65	27.36-48.82	10.31	5.06-18.14	3.65	1.91-6.98	**<0.0001**

Alcohol consumption

Two questions regarding alcohol consumption were asked to obtain a picture of the different drinking patterns reported among Māori and non-Māori.

To capture these patterns of drinking, the first alcohol question asked how often the person drinks, and the second questions asked the number of drinks consumed on a typical drinking occasion. Responses to these questions are presented by ethnicity and sex in Figure 4.18 and Figure 4.19.

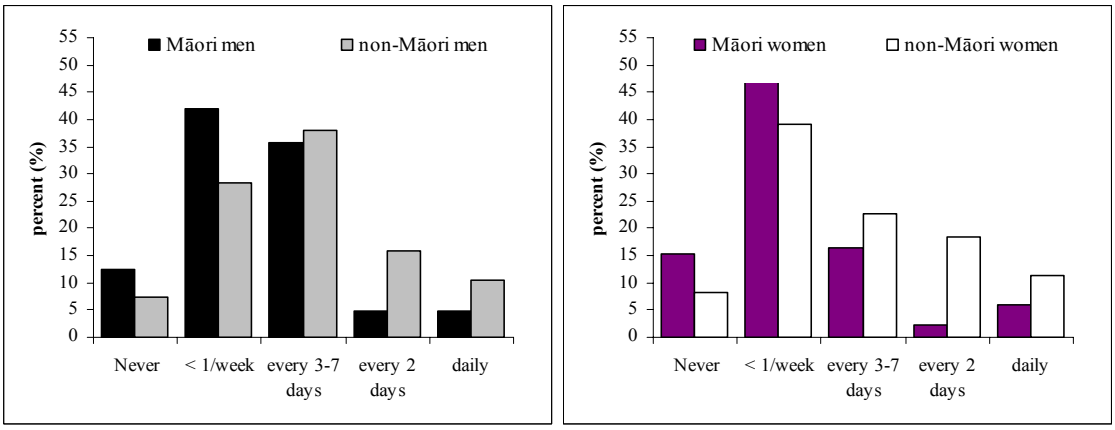


Figure 4.18 MESAM4 participants: Frequency of alcohol consumption, by ethnicity and sex

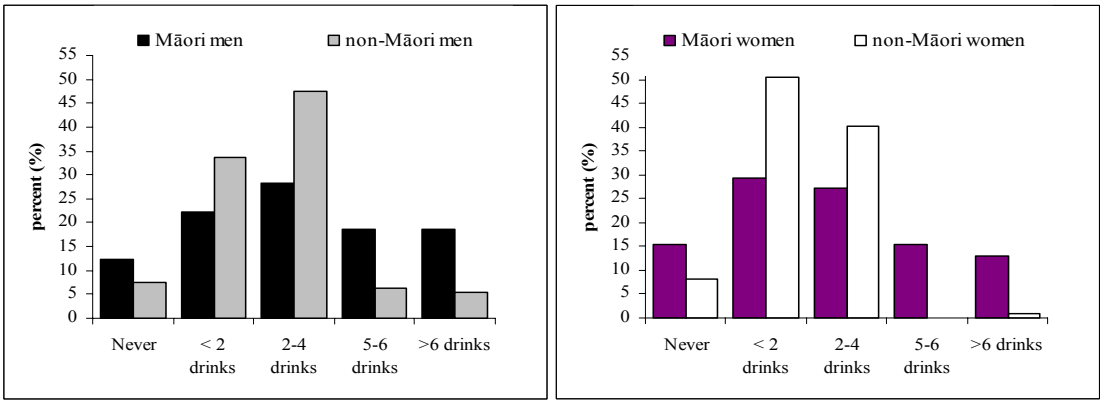


Figure 4.19 MESAM4 participants: Amount of alcohol consumed on a typical drinking occasion, by ethnicity and sex

The distribution of results indicates that non-Māori men and women drink alcohol more frequently than Māori men and women. However, Māori men and women drink more on a typical occasion than non-Māori men and women.

To test for significant differences in the drinking patterns by ethnicity and sex, the results were grouped to reflect different frequency of drinking and amount consumed (Table 4.24 and Table 4.25).

Table 4.24 MESAM4 participants: Alcohol frequency (at least once a week), by ethnicity and sex

	Māori (%)	95% CI	non-Māori (%)	95% CI	Ratio	95% CI	p-value
Men	45.68	34.56-57.13	64.21	53.72-73.79	0.71	0.54-0.94	**0.01**
Women	24.71	15.99-35.25	52.58	42.18-62.81	0.47	0.31-0.71	**0.0001**

Table 4.25 MESAM4 participants: Alcohol consumption (≥ 5 drinks on typical occasion), by ethnicity and sex

	Māori (%)	95% CI	non-Māori (%)	95% CI	Ratio	95% CI	p-value
Men	37.04	26.56-48.49	11.58	5.92-19.77	3.20	1.71-5.97	**<0.0001**
Women	28.24	19.00-39.04	1.03	0.03-5.61	27.39	3.79-198.18	**<0.0001**

Both non-Māori men and women were significantly more likely to drink alcohol at least once a week than Māori men and women. However, on a typical drinking occasion, Māori men and women were significantly more likely to consume 5 or more drinks than non-Māori men and women.

4.4.4 Other variables

Community Services Card (CSC)

Participants were asked if they were eligible for a community services card with the options of *yes*, *no* or *don't know*. The number of responses for each option is presented by ethnicity and sex in Table 4.26.

Table 4.26 MESAM4 participants: Are you eligible for a community services card?

	Yes	No	Don't know
Māori men	9	68	4
non-Māori men	4	88	3
Māori women	13	69	3
non-Māori women	5	88	4
Total	31	313	14

For those who were eligible for a CSC among *MESAM4 participants*, no differences were found between Māori and non-Māori men (OR 2.64, 95 CI 0.84-8.25, p=0.08). For women, Māori were three times more likely to be eligible for a community services card than non-Māori (OR 2.97, 95% CI 1.10-7.98, p=0.02). Within ethnic groups, no differences were found between men and women.

Table 4.27 MESAM4 participants vs. Questionnaire only and National survey: CSC Eligibility, by ethnicity and sex

	Māori		non-Māori	
	Men	**Women**	**Men**	**Women**
MESAM4	**12.20 (6.01-21.29)**	**14.94 (8.20-24.20)**	**4.17 (1.15-10.33)**	**5.05 (1.66-11.40)**
Questionnaire only	17.74 (9.20-29.53)	10.67 (4.72-19.94)	7.14 (2.67-14.90)	9.17 (4.67-15.81)
National survey	**33.84 (31-34.36.33)****	**47.13 (44.75-49.52)****	**20.36 (18.47-22.36)****	**26.18 (24.31-28.20)****

**p<0.01

Although CSC eligibility mirrored patterns seen in the national survey, eligibility amongst participants in this study was significantly less than those reported in the national survey across all groups. No differences were found between *MESAM4 participants* and people who only answered the questionnaire (Table 4.27).

4.5 MESAM4 Data

Subjective sleep rating

To assess the quality of participants' sleep whilst wearing the MESAM4 equipment, the morning after their sleep study, each participant was asked to rate their sleep on a five-point scale (1=*much worse*, 3=*typical*, 5=*much better*) compared to a *normal nights sleep* (Figure 4.20)

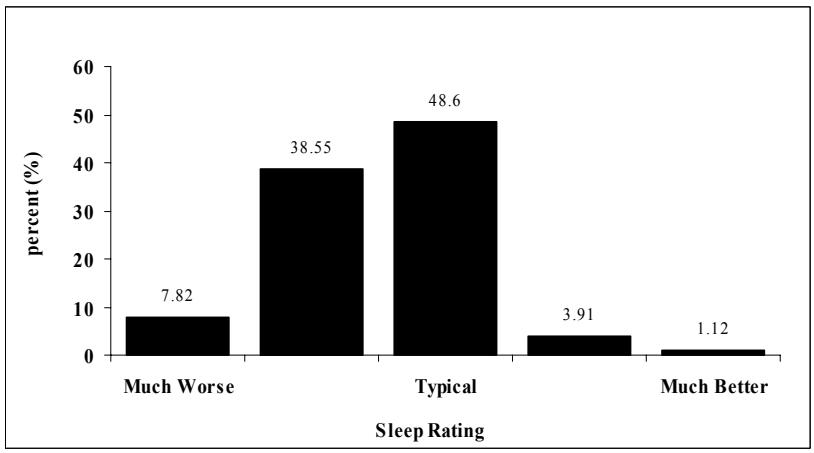


Figure 4.20 Participants rating of study sleep compared to a normal nights sleep

Nearly half (48.60%) of the participants rated their sleep as being *typical* compared to a normal nights sleep. A few (7.82%) considered their study sleep to be much worse than normal. Surprisingly, there were some (1.12%) who considered their sleep to be much better.

Total Sleep Time (TST)

Total sleep time was calculated by summing the number of 5 minute epochs where the participant was assessed (by the scorer) to be sleeping. Distributions are presented in Figure 4.21.

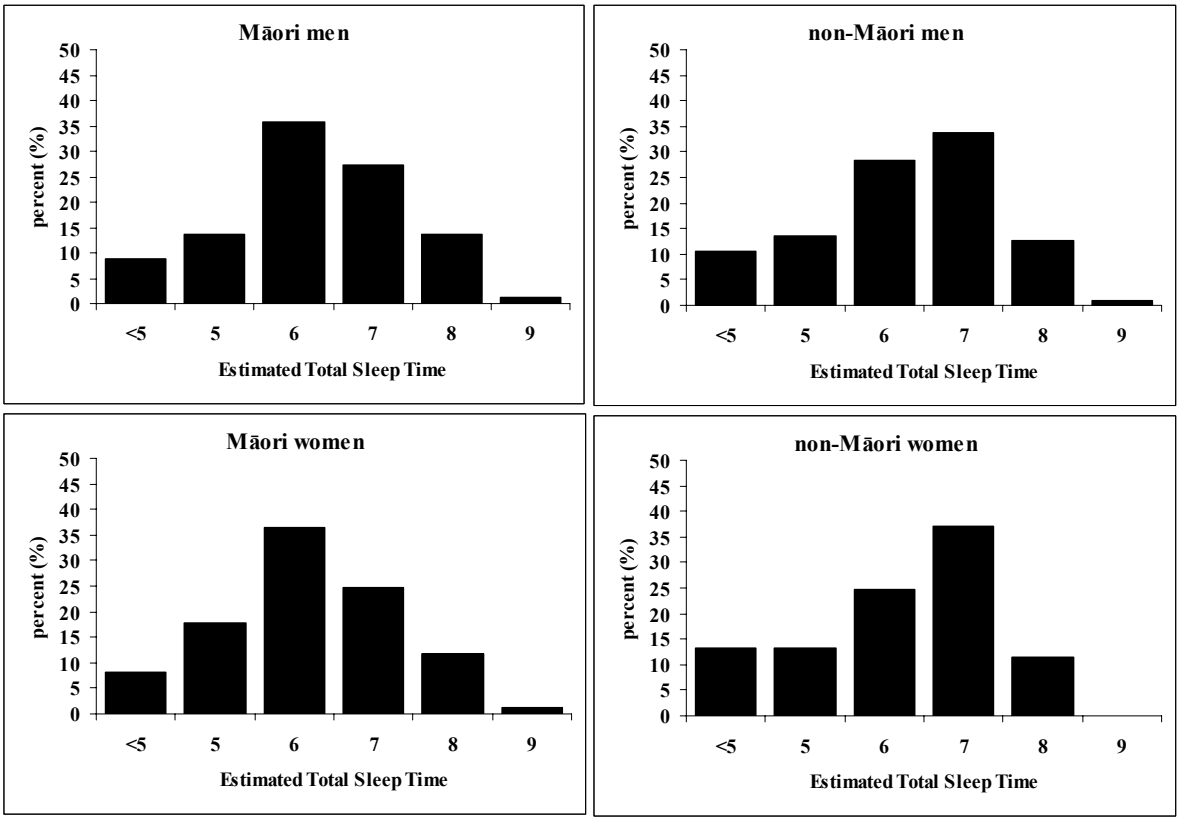


Figure 4.21 Distribution of estimated Total Sleep Time (TST)

For all *MESAM4 participants*, the median sleep duration while wearing the sleep equipment was 6.46 hours (IQR=5.67-7.17). The data were not normally distributed, therefore the Wilcoxon ranked sum test was used to test for differences in the medians between groups. No differences were found between Māori and non-Māori, men and women (Men p=0.9468, Women p=0.5137). Similarly, no sex differences were found within ethnic groups (Māori p=0.3542, non-Māori p=0.7281).

Table 4.28 Estimated total sleep time, by ethnicity and sex

	Median (hrs)	Interquartile range (hrs)
Māori men	6.50	5.75-7.17
Māori women	6.30	5.50-7.08
Non-Māori men	6.50	5.67-7.33
Non-Māori women	6.50	5.33-7.17
Total	**6.46**	**5.70-7.20**

Obstructive Sleep Apnoea (OSA)

As outlined in the Chapter 3, two respiratory indices were calculated to estimate OSA from the MESAM4 traces, RDIa and RDIc. Figure 4.22 presents the distribution of RDI scores for each group. Unadjusted proportions for each index at varying thresholds, and tests for differences between groups are presented in Table 4.29 to Table 4.34.

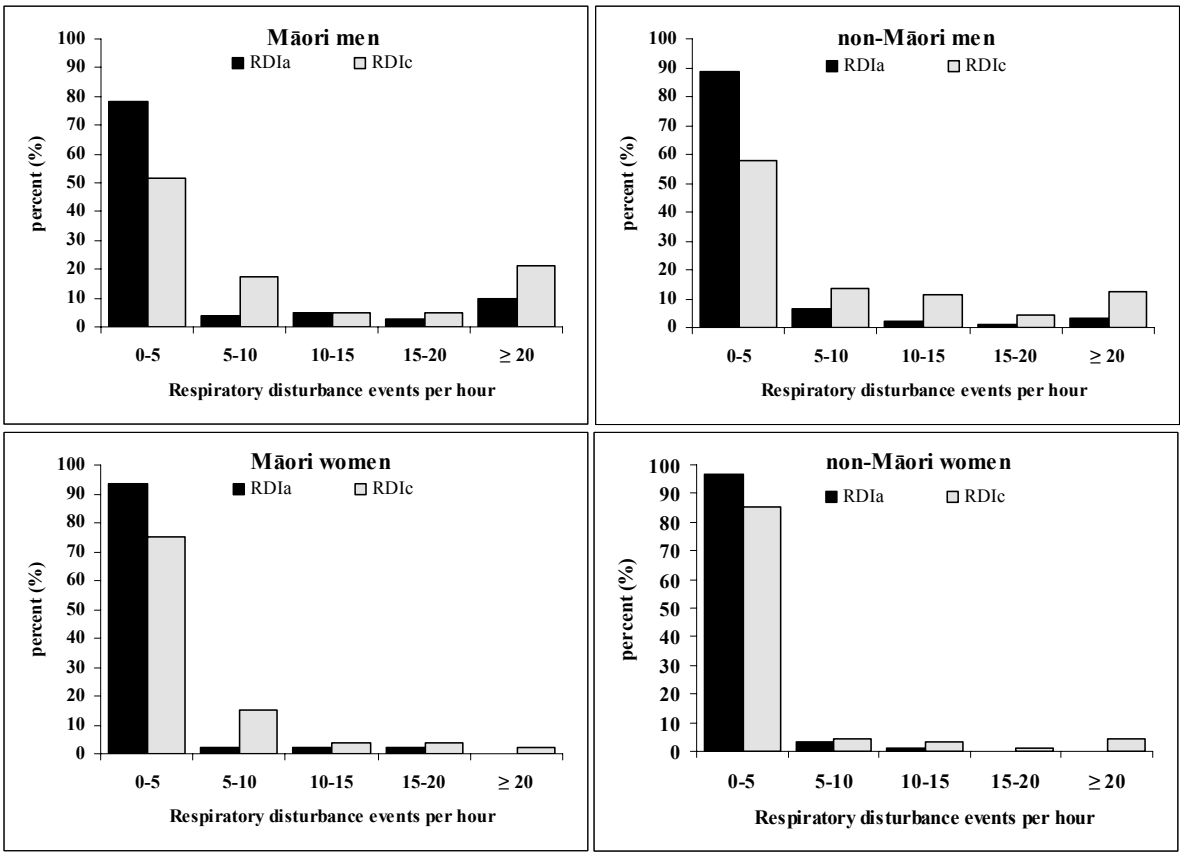


Figure 4.22 RDI distributions, by ethnicity and sex

The distributions are clearly skewed towards the normal end of the spectrum, with more OSA apparent in men than women. The distributions also highlight the difference between the two indices calculated. With the addition of scored hypopnoeas (RDIc), the prevalence and severity of respiratory disturbance is markedly elevated in each group.

Table 4.29 RDIa ≥ 5

	n	%	95% CI	Ratio	p-value
Overall	39	10.89	7.86-14.59		
Men	**29**	**16.48**	**11.32-22.80**	**3.00**	**0.0009**
Women	**10**	**5.49**	**2.67-9.87**		
Māori	23	13.86	8.99-20.06	1.66	0.0945
non-Māori	16	8.33	4.84-13.18		
Māori men	17	20.99	12.73-31.46	1.66	0.1364
non-Māori men	12	12.63	6.70-21.03		
Māori women	6	7.06	2.63-14.73	1.71	0.386
non-Māori women	4	4.12	1.13-10.22		

Table 4.30 RDIa ≥ 10

	n	%	95% CI	Ratio	p-value
Overall	25	6.98	4.57-10.14		
Men	**20**	**11.36**	**7.08-17.00**	**4.13**	**0.0014**
Women	**5**	**2.75**	**0.90-6.29**		
Māori	**18**	**10.84**	**6.55-16.60**	**2.97**	**0.0077**
Non-Māori	**7**	**3.65**	**1.48-7.37**		
Māori men	**14**	**17.28**	**9.78-27.30**	**2.73**	**0.0223**
non-Māori men	**6**	**6.32**	**2.35-13.24**		
Māori women	4	4.71	1.30-11.61	4.57	Null
non-Māori women	1	1.03	0.26-5.61		

Table 4.31 RDIa ≥ 15

	n	%	95% CI	Ratio	p-value
Overall	16	4.47	2.56-7.16		
Men	**14**	**7.95**	**4.42-12.99**	**7.23**	**0.0017**
Women	**2**	**1.10**	**0.13-3.91**		
Māori	**12**	**7.23**	**3.79-12.29**	**3.48**	**0.0188**
non-Māori	**4**	**2.08**	**0.57-5.25**		
Māori men	10	12.35	6.08-21.53	2.93	0.0468
non-Māori men	4	4.21	1.16-10.43		
Māori women	2	2.35	0.29-8.24	0.00	Null
non-Māori women	0	0.00	0.00		

Table 4.32 RDIc ≥ 5

	n	%	95% CI	Ratio	p-value
Overall	114	31.84	27.05-36.95		
Men	**79**	**44.89**	**37.40-52.55**	**2.33**	**<0.0001**
Women	**35**	**19.23**	**13.78-25.72**		
Māori	60	36.14	28.84-43.95	1.28	0.104
non-Māori	54	28.13	21.89-35.05		
Māori men	39	48.15	36.90-59.53	1.14	0.422
non-Māori men	40	42.11	32.04-52.67		
Māori women	21	24.71	15.99-35.25	1.71	0.079
non-Māori women	14	14.42	8.12-23.02		

Table 4.33 RDIc ≥ 10

	n	%	95% CI	Ratio	p-value
Overall	68	18.99	15.06-23.49		
Men	**52**	**29.55**	**22.92-36.88**	**3.36**	**<0.0001**
Women	**16**	**8.79**	**5.11-13.88**		
Māori	33	19.88	14.10-26.77	1.09	0.691
non-Māori	35	18.23	13.04-24.43		
Māori men	25	30.86	21.07-42.11	1.09	0.723
non-Māori men	27	28.42	19.64-38.60		
Māori women	8	9.41	4.15-17.71	1.14	0.782
non-Māori women	8	8.24	3.63-15.61		

Table 4.34 RDIc ≥ 15

	n	%	95% CI	Ratio	p-value
Overall	47	13.13	9.81-17.07		
Men	**37**	**21.02**	**15.26-27.79**	**3.83**	**<0.0001**
Women	**10**	**5.49**	**2.67-9.87**		
Māori	26	15.66	10.49-22.10	1.43	0.1868
non-Māori	21	10.94	6.90-16.23		
Māori men	21	25.93	16.82-36.86	1.54	0.1405
non-Māori men	16	16.84	9.94-25.91		
Māori women	5	5.88	1.93-13.20	1.14	0.830
non-Māori women	5	5.15	1.69-11.62		

Depending on the definition of OSA used, the overall prevalence estimates ranged from 4.47% to 31.84%. Using the most conservative index, RDIa, prevalence ranged from 4.47% to 10.89%. For men and women, based on RDIa, prevalence estimates ranged from 7.95% to 16.48% and 1.10%-5.49% respectively.

The results indicate that men were significantly more likely than women to have OSA. Maori men had the highest prevalence of OSA, followed by non-Māori men, Māori women and finally non-Māori women. Based on RDIa (≥ 10, ≥ 15) Māori were significantly more likely than non-Māori to have OSA.

4.6 Predictors of OSA

This section presents predictors of OSA identified by multiple logistic regression at different thresholds of RDIa and RDIc (≥ 5, ≥ 10, ≥ 15). Table 4.35 presents the relationships between ethnicity, sex, age and respiratory disturbance. After controlling for age and sex, ethnicity is only predictive of RDIa≥ 10 and ≥ 15. Māori were three times more likely to have 10 or more respiratory events per hour, and fours times more likely to have 15 or more respiratory events per hour.

Table 4.35 OSA – Logistic regression model 1

Variable	RDIa≥ 5	RDIa≥ 10	RDIa≥ 15	RDIc≥ 5	RDIc≥ 10	RDIc≥ 15
Ethnicity (Māori vs. non-Māori)	1.88 (0.94-3.75)	**3.48 (1.39-8.71)****	**4.26 (1.31-13.90)****	1.53 (0.96-2.44)	1.16 (0.67-2.00)	1.61 (0.85-3.04)
Sex (men vs. women)	**3.55 (1.66-7.58)****	**4.88 (1.77-13.49)****	**8.83 (1.94-40.15)****	**3.54 (2.19-5.71)****	**4.43 (2.41-8.16)****	**4.73 (2.26-9.91)****
Age (yearly increase)	1.04 (0.99-1.08)	1.03 (0.98-1.08)	1.07 (1.00-1.14)	1.02 (1.00-1.05)	1.02 (0.99-1.06)	1.03 (0.99-1.07)

** p< 0.01

Table 4.36 and Table 4.37 display two models; one including BMI and one including neck circumference, along with a number of other variables. A total of 335 participants provided sufficient data to be included in these models. .

Table 4.36 OSA – Logistic regression model 1a (BMI)

Variable	RDIa			RDIc		
	≥5	≥10	≥15	≥5	≥10	≥15
Ethnicity (Māori vs. non-Māori)	0.84 (0.31-2.24)	1.63 (0.48-5.55)	3.19 (0.56-18.05)	0.98 (0.52-1.84)	0.55 (0.25-1.18)	0.69 (0.27-1.76)
Sex (men vs. women)	**4.44 (1.57-12.57) ****	**6.65 (1.66-26.68)****	**12.25 (1.70-88.34)****	**3.84 (2.07-7.13)****	**4.42 (2.04-9.58)****	**5.24 (1.87-14.68)****
Age (yearly increase)	1.05 (0.99-1.11)	1.06 (0.98-1.13)	**1.11 (1.01-1.22)***	1.02 (0.98-1.05)	1.03 (0.99 -1.08)	1.02 (0.97-1.08)
NZDep96 (deciles)	1.00 (0.87-1.15)	1.04 (0.88-1.22)	1.04 (0.85-1.28)	0.98 (0.89-1.08)	1.04 (0.93-1.17)	1.02 (0.89-1.16)
CSC (eligible vs. other)	1.47 (0.71-3.04)	2.2 (1.00-4.90)	2.37 (0.86-6.53)	0.50 (0.25-1.00)	0.91 (0.45-1.83)	1.25 (0.57-2.74)
BMI (increasing)	**1.19 (1.08-1.32) ****	**1.21 (1.08-1.37)****	**1.17 (1.03-1.34)***	**1.21 (1.12-1.30)****	**1.22 (1.12-1.32)****	**1.29 (1.16-1.44)****
Wake feeling refreshed (never/rarely vs. often /always)	0.46 (0.15-1.36)	0.40 (0.11-1.44)	1.16 (0.20-6.63)	0.82 (0.38-1.75)	0.50 (0.21-1.22)	0.38 (0.13-1.11)
Enough sleep (never/rarely vs. often/always)	2.36 (0.72-7.72)	3.78 (0.92-15.57)	1.45 (0.25-8.39)	1.55 (0.69-3.50)	2.08 (0.80-5.39)	4.12 (1.23-13.82)
Observed apnoeas (apnoea vs. not)	**4.20 (1.69-10.433) ****	1.56 (0.48-5.05)	**4.49 (1.07-18.82)***	**4.71 (2.03-10.95)****	**4.19 (1.86--9.43)****	**5.73 (2.32-14.17)****
Excessive daytime sleepiness (ESS>10 vs. ESS ≤ 10)	2.18 (0.81-5.83)	1.54 (0.46-5.20)	0.79 (0.15-4.23)	**3.25 (1.51-7.03)****	1.61 (0.68-3.81)	1.02 (0.35-2.96)
Snore (always vs. never/rarely/often)	1.91 (0.71-5.17)	2.42 (0.78-7.51)	0.66 (0.14-3.04)	1.38 (0.57-3.32)	1.57 (0.63-3.87)	2.05 (0.75-5.55)
Asthma (curr. treatment vs. no/don't know)	1.79 (0.49-56.51)	0.67 (0.10-4.71)	1.25 0.18-8.92)	2.01 (0.81-5.02)	1.58 (0.56-4.46)	1.25 (0.34-4.60)
Hypertension (treatment vs. no/don't know)	0.60 (0.124-2.92)	0.57 (0.09-3.65)	0.82 (0.11-6.04)	0.62 (0.19-2.05)	0.39 (0.09-1.60)	0.60 (0.12-2.90)
Smoking (Regular vs. occasional/ex-smoker/non-smoker)	1.62 (0.59-4.50)	1.66 (0.49-5.68)	0.85 (0.16-4.54)	1.70 (0.84-3.41)	1.39 (0.61-3.16)	1.05 (0.37-2.96)
Alcohol (exceed rec. vs. non-drinker)	1.78 (0.57-5.51)	1.49 (0.41-5.48)	1.28 (0.22-7.37)	0.92 (0.38-2.18)	1.06 (0.39-2.88)	1.59 (0.51-5.01)

*p<0.05 **p<0.01

Table 4.37 OSA – Logistic regression model 1b (Neck circumference)

Variable	RDIa			RDIc		
	≥5	≥10	≥15	≥5	≥10	≥15
Ethnicity (Māori vs. non-Māori)	1.07 (0.41-2.80)	2.02 (0.62-6.60)	4.10 (0.75-22.32)	1.16 (0.63-2.14)	0.70 (0.33-1.47)	1.03 (0.42-2.53)
Sex (men vs. women)	1.12 (0.30-4.17)	1.84 (0.35-9.65)	4.12 (0.48-34.90)	1.03 (0.45-2.33)	0.92 (0.34-2.48)	0.71 (0.21-2.45)
Age (yearly increase)	1.05 (0.99-1.11)	1.06 (0.99-1.13)	**1.11 (1.02-1.22)***	1.01 (0.98-1.05)	1.03 (0.99-1.07)	1.03 (0.98-1.09)
NZDep96 (deciles)	1.01 (0.88-1.16)	1.05 (0.90-1.23)	1.07 (0.88-1.30)	0.98 (0.89-1.08)	1.04 (0.93-1.17)	1.01 (0.89-1.15)
CSC (eligible vs. else)	1.32 (0.64-2.74)	1.96 (0.89-4.29)	2.12 (0.79-5.71)	**0.47 (0.23-0.94)***	0.82 (0.41-1.65)	1.09 (0.49-2.44)
Neck circumference (increasing)	**1.28 (1.08-1.52) ****	**1.23 (1.01-1.51)***	1.19 (0.95-1.50)	**1.29 (1.14-1.45)****	**1.34 (1.17-1.53)****	**1.42 (1.20-1.67)****
Wake feeling refreshed (never/rarely vs. often /always)	0.43 (0.14-1.29)	0.41 (0.12-1.45)	1.04 (0.19-5.74)	0.82 (0.39-1.74)	0.48 (0.20-1.17)	0.38 (0.13-1.08)
Enough sleep (never/rarely vs. often/always)	2.21 (0.68-7.21)	3.14 (0.79-12.54)	1.30 (0.23-7.41)	1.33 (0.61-2.92)	1.82 (0.71-4.68)	**3.40 (1.04-11.12)***
Observed apnoeas (apnoea vs. not)	**4.92 (2.01-12.05)****	2.21 (0.73-6.67)	**6.32 (1.62-24.67)****	**5.35 (2.33-12.28)****	**4.82 (2.16-10.77)****	**6.87 (2.84-16.63)****
Excessive daytime sleepiness (ESS>10 vs. ESS ≤ 10)	2.00 (0.76-5.31)	1.47 (0.45-4.79)	0.69 (0.13-3.53)	**3.17 (1.48-6.80)****	1.54 (0.66-3.60)	1.01 (0.36-2.83)
Snore (always vs. never/rarely/often)	1.58 (0.58-4.30)	1.97 (0.64-6.04)	0.54 (0.12-2.47)	1.17 (0.49-2.83)	1.29 (0.52-3.19)	1.53 (0.57-4.14)
Asthma (curr. treatment vs. no/don't know)	1.57 (0.44-5.65)	0.72 (0.12-4.26)	1.22 (0.19-7.71)	1.90 (0.76-4.76)	1.44 (0.50-4.13)	1.05 (0.28-3.87)
Hypertension (treatment vs. no/don't know)	0.78 (0.17-3.59)	0.88 (0.15-5.10)	1.15 (0.17-8.00)	0.78 (0.25-2.41)	0.50 (0.13-1.95)	0.83 (0.18-3.72)
Smoking (Regular vs. occasional/ex-smoker/non-smoker)	1.31 (0.49-3.52)	1.29 (0.40-4.18)	0.72 (0.14-3.68)	1.41 (0.71-2.80)	1.12 (0.49-2.52)	0.78 (0.29-2.14)
Alcohol (exceed rec. vs. non-drinker)	1.65 (0.53-5.13)	1.54 (0.43-5.51)	1.31 (0.24-6.99)	0.84 (0.36-1.96)	0.96 (0.36-2.58)	1.44 (0.46-4.50)

*p<0.05 **p<0.01

For both models, after controlling for a number of other variables, ethnicity was no longer a predictive factor at any of the thresholds. Age was only predictive at RDIa ≥15. For Model 1a (BMI), sex was the most consistent predictor at each severity threshold. Men were 4 times more likely than women to have five or more respiratory disturbances per hour. BMI (increasing) was also a consistent predictor at each threshold. Reported observed apnoeas was a significant predictor at each threshold except RDIa≥ 10. In addition, daytime sleepiness predicted an RDIc≥ 5. For Model 1b (Neck circumference), neck circumference was the most consistent predictor followed by reported apnoeas. In contrast to Mode1 1a, sex did not predict respiratory disturbance at any threshold, probably because of sex differences in neck size.

4.7 Population Prevalence Estimates

In this section, general population estimates are presented for OSA. RDIa is presented rather than RDIc, as it is the more conservative measure of the two. As mentioned previously, to estimate population prevalences, data were weighted by the population proportions of age, sex and ethnicity in the 30-60 year age group in the Wellington region, using the 1996 census information. Weighting by adjusting for the population age structures of each group changed the unadjusted proportions only slightly. It also did not change any of the tests of significance between the groups and ratios (Table 4.38).

Table 4.38 Population prevalence estimates for OSA, by ethnicity and sex

OSA definition		Māori (%) (95% CI)	non–Māori (%) (95% CI)	Relative Risk (95% CI)	p-value
RDIa≥ 5	**Men**	21.98 (10.30-33.66)	11.37 (4.49-18.26)	1.93 (0.86-4.32)	0.125
	Women	6.28 (-0.104-12.67)	3.02 (0.04-6.00)	2.08 (0.50-8.57)	0.364
	Total	13.88 (7.34-20.43)	7.14 (3.42-10.86)	1.94 (1.21-3.11)	0.079
RDIa≥10	**Men**	16.69 (6.84-26.54)	5.85 (0.80-10.89)	2.85 (1.00-8.12)	0.055
	Women	5.40 (0.87-2.68)	0.91 (-0.87-2.68)	5.93 (0.61-58.24)	0.176
	Total	10.87 (5.11-16.63)	3.34 (0.70-5.99)	3.25 (1.91-5.52)	**0.02**
RDIa≥15	**Men**	11.86 (3.50-20.21)	3.04 (-0.14-6.21)	3.90 (1.11-13.77)	0.053
	Women	1.54 (-0.79-3.87)	0	0	Null
	Total	6.54 (2.32-10.76)	1.50 (-0.07-3.06)	4.36 (2.89-8.32)	**0.03**

Respiratory disturbance scores were also examined in conjunction with criteria for excessive daytime sleepiness as measured by an ESS>10, which is required for a diagnosis of obstructive sleep apnoea syndrome (OSAS) (American Academy of Sleep Medicine Task Force. 1999). With the addition of daytime sleepiness criteria, no differences were found between the groups. The insignificant differences between groups is most likely due to the small numbers in each group, however, most of the trends are in the expected direction (Table 4.39).

Table 4.39 OSAS prevalence estimates, by ethnicity and sex

OSAS definition		Māori (%) (95% CI)	non–Māori (%) (95% CI)	RR (95% CI)	p-value
RDIa≥ 5 + ESS>10	Men	8.01 (1.92-14.09)	12.11 (4.32-19.90)	0.66 (0.24-1.79)	0.416
	Women	8.48 (1.81-15.15)	2.37 (-1.17-5.91)	3.58 (0.66-19.38)	0.113
	Total	3.14 (0.43-5.84)	2.36 (-0.03-4.74)	1.33 (0.35-5.02	0.74
RDIa≥10 + ESS>10	Men	6.07 (0.31-11.53)	7.46 (1.29-13.63)	0.82 (0.24-2.76)	0.741
	Women	1.98 (-0.50-4.46)	0.71 (-0.68-2.09)	2.81 (0.27-28.77)	0.378
	Total	1.64 (-0.01-3.28)	1.39 (-0.55-3.32)	1.18 (0.21-6.59)	0.846
RDIa≥15 + ESS>10	Men	4.37 (-0.55-9.28)	4.05 (-0.56-8.67)	1.08 (0.22-5.34)	0.928
	Women	1.98 (-0.50-4.46)	0.71 (-0.68-2.09)	2.81 (0.27-28.77)	0.378
	Total	1.28 (-0.21-2.77/0	0	0	Null

4.8 Validation of Self-Reported Snoring and Observed Apnoeas

Snoring

To assess the validity of the snoring question (How often do you snore?), comparisons were made between reported snoring frequency and the actual percentage of the night spent snoring as measured by the MESAM4 (Figure 4.23). Responses from those participants who reported *don't know* to this question are also examined (Figure 4.24).

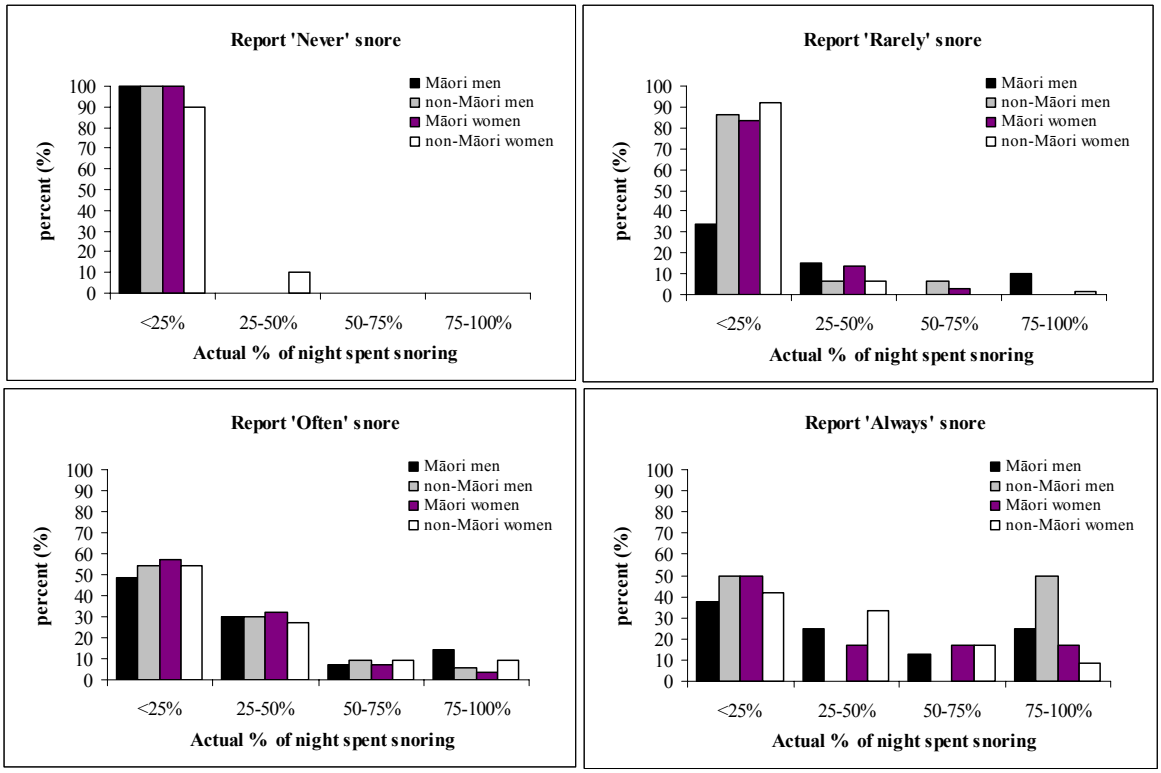


Figure 4.23 Reported snoring vs. actual snoring

In total, 126 participants reported that they *often* snore and only 25 reported that they *always* snore. When compared to the actual percentage of the night spent snoring, the general patterns of distributions indicate that to some degree the subjective snore ratings captured objective measures of snoring. Reporting snoring *always* had a higher degree of discrimination for increased actual snoring compared to snoring often, especially for non-Māori men. Among those participants (n=185) who reported *never* snoring, the majority snored for less than 25% of the night.

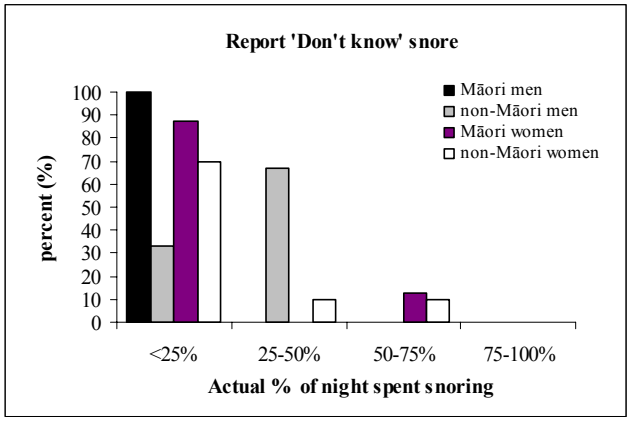


Figure 4.24 Reporting 'don't know' to snore question vs. actual snoring

The majority of participants who responded *don't know* to the snoring question (n=22) snored for less than 25% of the night. However there were a small portion of women, both Māori and non-Māori, who actually snored for 50-70% of the recording night.

Observed apnoeas

To assess the validity of the question regarding observed apnoeas (Has anyone ever told you that you sometimes stop breathing during sleep?), reported apnoeas were compared with actual apnoeas as defined by RDIa≥ 5 (Figure 4.25).

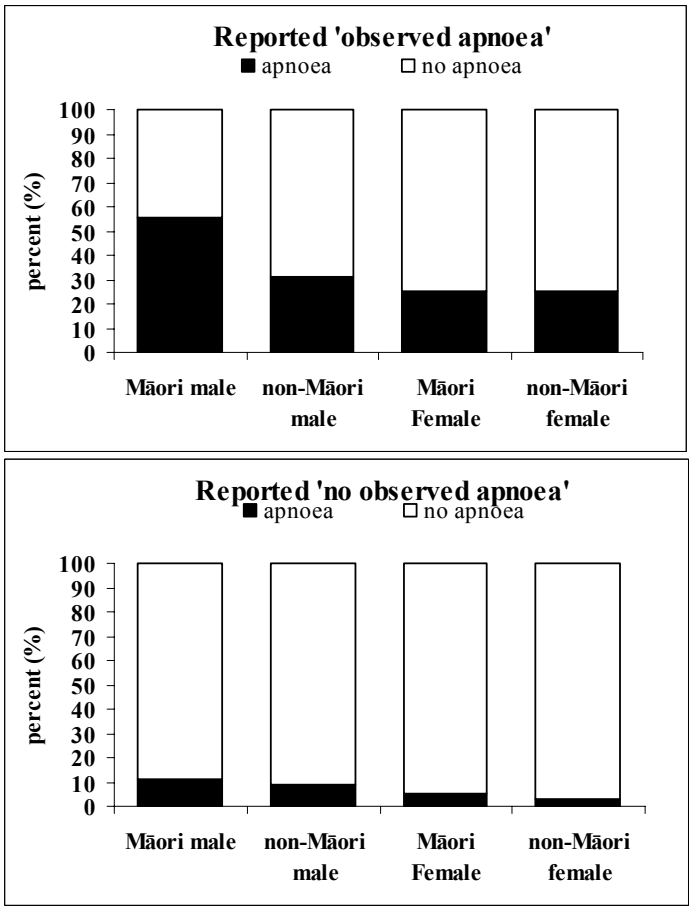


Figure 4.25 Reported vs. actual apnoeas

Of the 49 participants who reported observed apnoeas, only 39% had actual apnoeas. Results indicate that Māori men were more accurate than the others at reporting apnoeas, where 56% of reported apnoeas were confirmed by objective measures. Conversely, of the 312 participants who reported no observed apnoeas, 93% were correct. Across groups, the distribution was similar. Overall, the reporting of no observed apnoeas was more accurate than the reporting of observed apnoea across all groups.

CHAPTER 5
THE CLINICAL SAMPLE

5.1 Introduction

The first section of this chapter examines the demographic profiles of patients along with their questionnaire responses. The second section examines the additional data collected from clinic participants, such as body mass index and neck circumference. The third section, examines the objective sleep data. The final section assesses the validity of self-reported snoring frequency and observed apnoeas.

5.2 Method

5.2.1 Measures

The details regarding the questionnaire and objective sleep measures (polysomnography) utilised in the clinic are outlined in the Chapter 3 (Methods).

5.2.2 Statistical analyses

Descriptive statistics are presented in this chapter for all data, by ethnicity and sex, to summarise the general characteristics of each variable. Age is not included in the analyses as the number of participants in some groups is too small for a valid analysis, in particular Māori women (n=15).

Logistic regression analyses

Logistic regression was used to investigate independent predictors of OSA. As in Chapter 4 (The Community Sample) three logistic regression models were run at three varying thresholds of RDIa and RDIc ($\geq 5, \geq 10, \geq 15$), as outlined below:

Model 1 : ethnicity, sex and age.
Model 1a: ethnicity, sex, age, BMI, and other variables (see Table 3.4)
Model 1b: ethnicity, sex, age, neck circumference, and other variables (see Table 3.4)

Collinearity

Prior to entry into the logistic regression models, variables were assessed for collinearity using a correlation matrix. Although a strong correlation between neck circumference and BMI was not found in the clinical sample, it is well established that the two are strongly correlated (Ben-Noun et al. 2001, Hoffstein and Mateika 1992).

Therefore separate models were developed including each of these variables. Collinearity was not found among any of the other variables.

For each analysis, if there is no mention of missing data or outliers, it can be assumed that responses from all participants are included (n=510).

5.3 Sample for Analysis

The clinical sample consisted of consecutive patients referred to the sleep clinic (Wellsleep) in Wellington for suspected OSA from August 1999 to May 2001. All patients (n=666) who were asked to take part in the study consented to participate. Of these, data from 125 were excluded from analyses as they did not meet the inclusion criteria in Table 5.1. Data from a further 31 participants were excluded due to insufficient polysomnographic data[13], which meant data from 510 participants were available for these analyses, of which 70% (n=355) of studies were carried out in the clinic and the rest as home studies. A breakdown of the sample by ethnicity, age group, and sex is presented in Table 5.2.

Table 5.1 Inclusion criteria

Inclusion criteria
1. aged between 30-60 years
2. Referred for diagnostic study for suspected OSA
3. Minimum of 3 hours of sleep during diagnostic study*

* Studies with only 1-3 hours of sleep were reviewed by the respiratory physician, and were included if deemed representative of the patient's typical sleep. Studies less than 1 hour were excluded.

Table 5.2 Clinical analytical sample by age group, ethnicity and sex

	Mean age (yrs)	Age group (yrs)			Total
		30-39	40-49	50-59	
Māori men	44.57	17 (3.33%)	23 (4.5%)	17 (3.33%)	57 (11.18%)
Māori women	46.54	3 (0.59%)	7 (1.37%)	5 (0.98%)	15 (2.94%)
Non-Māori men	47.60	68 (13.33%)	114 (22.35%)	150 (29.41)	332 (65.10%)
Non-Māori women	48.98	18 (3.53%)	30 (5.88%)	58 (11.37%)	106 (20.78%)
Total	47.52	106 (20.78%)	174 (34.12)	230 (45.10%)	510 (100%)

[13] Insufficient data was due to one of the following: 1) study could not be located; or 2) insufficient total sleep time

The male to female ratio was approximately 3:1. The overall mean age of participants was 48 years (SD=8.21, Range=30.86). There were no differences in age distribution between the four groups (χ^2=7.70, DF=3, p=0.0528, Kruskal-Wallis test). There were only a small number of Māori in this sample (n=72), which equates to a Māori to non-Māori ratio of approximately 6:1. Figure 5.1 shows the breakdown of the clinical sample by ethnicity.

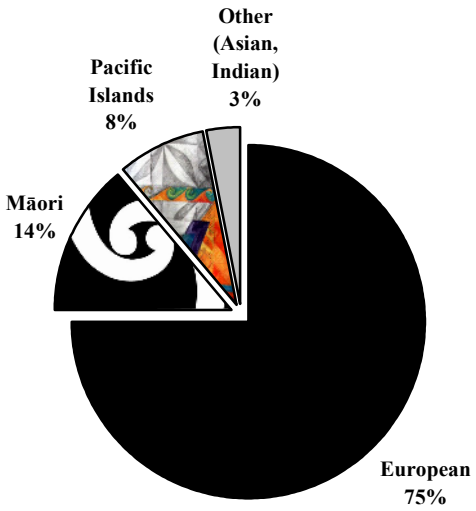


Figure 5.1 Ethnicity of clinical participants

The ethnic make-up of the clinical sample roughly represents the ethnic profile in the Wellington region for this age group (SNZ 1997a).

The majority of patients were publicly funded from the Wellington region (82% Māori, 51% non-Māori). The next largest group were privately funded participants, who were predominately non-Māori. A small percentage of participants were from out of town, and publicly funded by the health providers in their respective regions (Mid Central, Nelson-Marlborough, Hawkes Bay) (Figure 5.2).

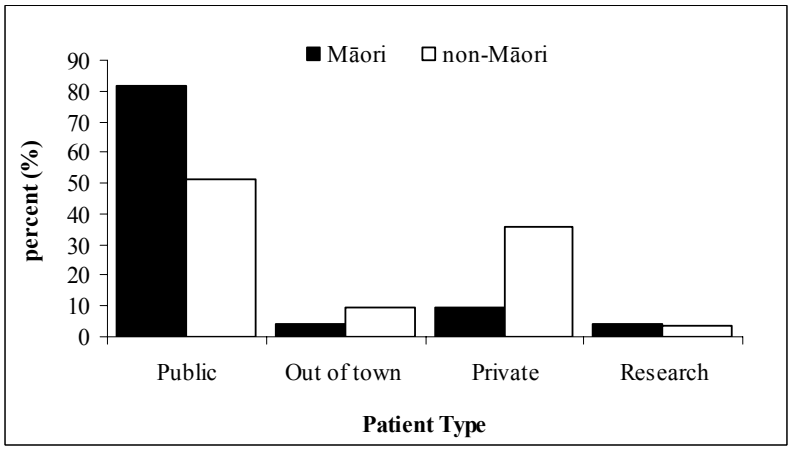


Figure 5.2 Clinical patient type

5.3.1 General sleep variables

Getting enough sleep

Participants were asked how often they think they get enough sleep, with the options of *never*, *rarely*, *often* or *always*. The majority of participants reported that they *rarely* get enough sleep. Overall, 68% of participants reported that they *never* or *rarely* get enough sleep (Figure 5.3).

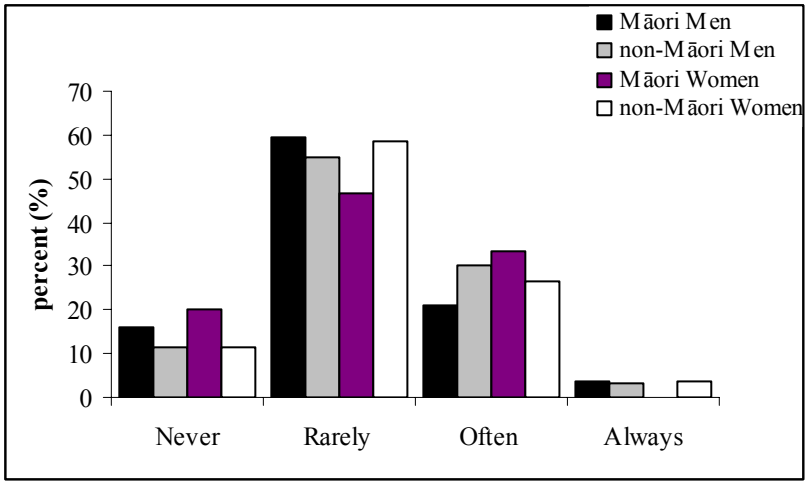


Figure 5.3 How often do you think you get enough sleep?

As a measure of self-perceived chronic sleep deprivation, *never* and *rarely* categories were grouped together and proportions calculated for Māori and non-Māori, men and women to examine differences by ethnicity and sex. Chi-squared comparisons indicated no significant differences between Māori and non-Māori, men and women.

Similarly, no differences between men and women were found within ethnic groups (Table 5.3).

Table 5.3 Never/Rarely get enough sleep, by ethnicity and sex

	Māori (%)	95% CI	non-Māori (%)	95% CI	Ratio	95% CI	p-value
Men	75.44	62.24-85.87	66.57	61.21-71.62	1.13	0.96-1.34	0.19
Women	66.67	38.38-88.18	69.81	60.13-78.35	0.96	0.65-1.40	0.80

Wake feeling refreshed

Participants were asked how often they wake feeling refreshed, again with the options of *never*, *rarely*, *often*, or *always*. Responses were distributed similarly between groups, with the majority (80%) of participants reporting *never* or *rarely* waking feeling refreshed (Figure 5.4).

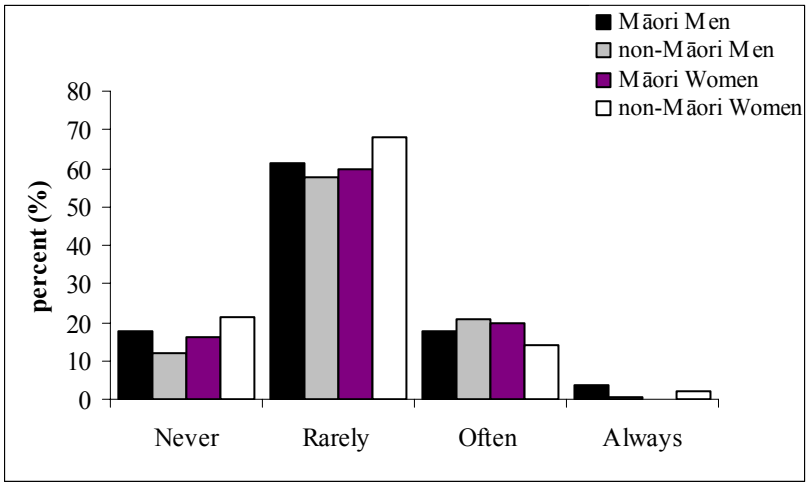


Figure 5.4 How often do you wake feeling refreshed?

As a measure of poor quality sleep, *never* and *rarely* categories were collapsed. No significant differences were found between Māori and non-Māori, men and women. Similarly, within ethnic groups no differences were found between men and women (Table 5.4).

Table 5.4 Never/Rarely wake feeling refreshed, by ethnicity and sex

	Māori (%)	95% CI	non-Māori (%)	95% CI	Ratio	95% CI	p-value
Men	78.95	66.11-88.62	78.92	74.13-83.18	1.00	0.87-1.16	0.10
Women	80.00	75.57-90.37	83.96	75.57-90.37	0.95	0.73-1.24	0.69

Average hours sleep in 24 hours

The average duration of usual sleep reported by participants was 7.25 hours per day (SD=1.92 hours). Distributions are illustrated in Figure 5.5 and a summary of data is presented in Table 5.5.

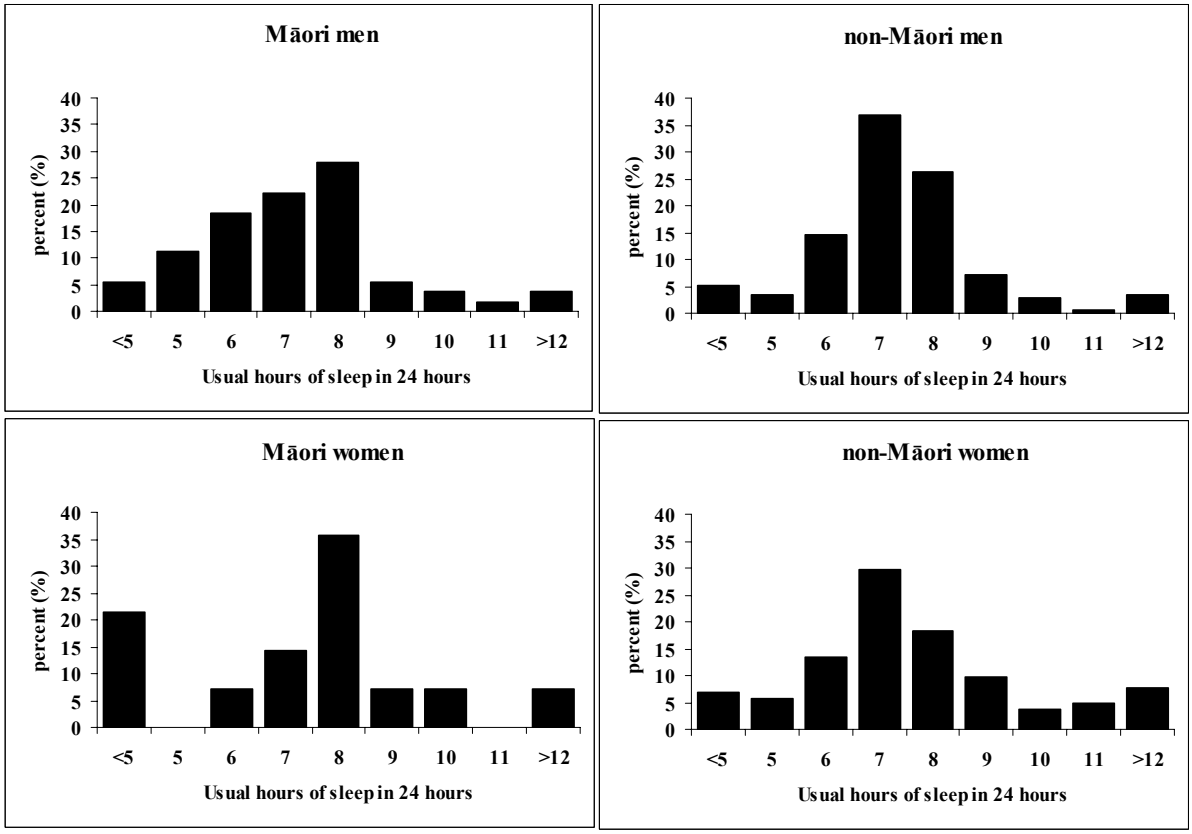


Figure 5.5 Distribution of reported average hours of sleep, by ethnicity and sex

Table 5.5 Usual hours sleep in 24 hours, by ethnicity and sex

	n	Median (hrs)	Interquartile range (hrs)
Māori men	54	7.00	6.0-8.0
Māori women	14	8.00	6.0-8.0
Non-Māori men	328	7.00	6.5-8.0
Non-Māori women	104	7.00	6.25-8.50
Total	500	7.00	6.3-8.0

Ten outliers were excluded from this particular analysis because they stated that they got *zero* hours sleep on average in 24 hours. As data were not normally distributed, tests for differences in the medians between groups were calculated. Amongst men, no significant differences were found between Māori and non-Maori (p=0.2067). Similarly

there were no differences between Māori and non-Māori females (p=0.9590). Within ethnic groups, there were also no significant differences found between men and women (Māori p=0.4768, non-Māori p=0.2343).

5.3.2 OSAS symptoms

Subjective snoring

Participants were asked how often they snored, with the options of *never*, *rarely*, *often* or *always*. Responses were similarly distributed between groups, with the vast majority reporting that they *often* or *always* snore (Figure 5.6).

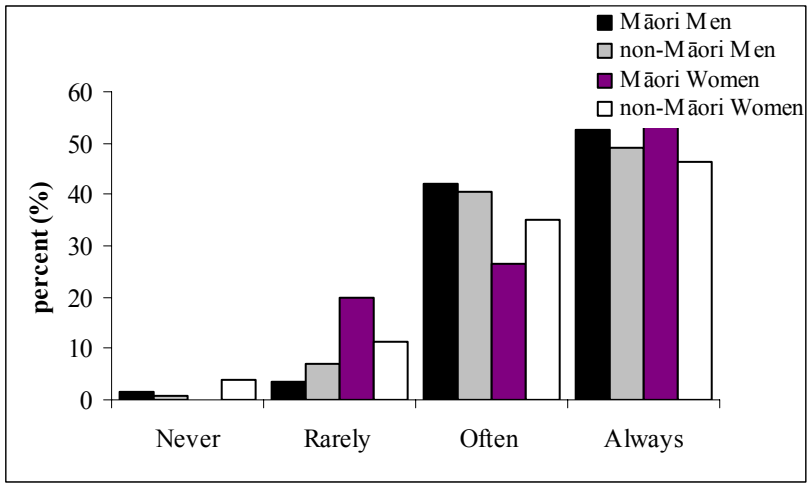


Figure 5.6 How often do you snore?

Although *don't know* was not a valid response, 12 patients responded in this manner. As a measure of habitual snoring, the *always* category was tested alone, then in conjunction with the *often* category. Using chi-square comparisons no significant ethnic or sex differences were found between the different groupings of the responses on the frequency of snoring (Table 5.6 and Table 5.7).

Table 5.6 Always snore, by ethnicity and sex

	Māori (%)	95% CI	non-Māori (%)	95% CI	Ratio	95% CI	p-value
Men	52.63	38.97-66.02	50.31	44.73-55.88	1.05	0.80-1.37	0.7463
Women	53.33	26.59-78.73	48.04	38.04-58.16	1.11	0.66-1.86	0.7017

Table 5.7 Often/Always snore, by ethnicity and sex

	Māori (%)	95% CI	Non-Māori (%)	95% CI	Ratio	95% CI	p-value
Men	94.74	85.38-98.90	91.98	88.46-94.69	1.03	0.96-1.10	0.4684
Women	80.00	51.91-95.67	84.31	75.78-90.76	0.95	0.73-1.24	0.6723

Objective snoring

An overall snore percentage for each patient was calculated from their sleep studies. Snoring was only measured in 355 participants (70%), as this was not a standard measured at the clinic. The majority of these patients snored for less than half the night (Figure 5.7).

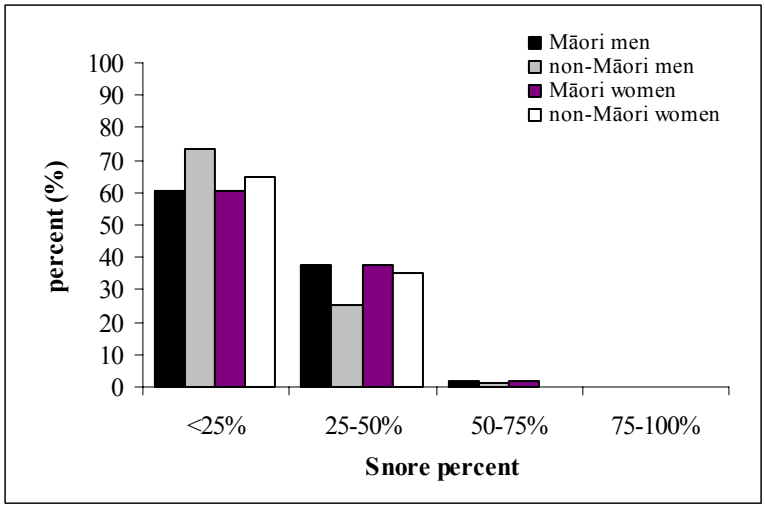


Figure 5.7 Percentage of night spent snoring, by ethnicity and sex

As data were not normally distributed, non-parametric tests were conducted to test the difference in the median percentage of sleep time spent snoring. The only difference found between groups, was between Māori and non-Māori men, where Māori men spent a higher proportion of the night snoring than non-Māori (Table 5.8)

Table 5.8 Percentage of the night spent snoring, by ethnicity and sex

	Māori (IQR)	non-Māori (IQR)	p-value
Men	19.40 (10.50-32.50)	13.80 (5.6-25.6)	0.0321
Women	19.65 (3.1-31.30)	16.20 (5.7-37.50)	0.8792
p-value	0.7033	0.5198	

Observed apnoeas

Participants were asked if anyone had ever told them that they stop breathing sometimes during sleep. As illustrated in Figure 5.8, Māori men reported the highest proportion of observed apnoeas, followed by non-Māori men, Māori women, and non-Māori women.

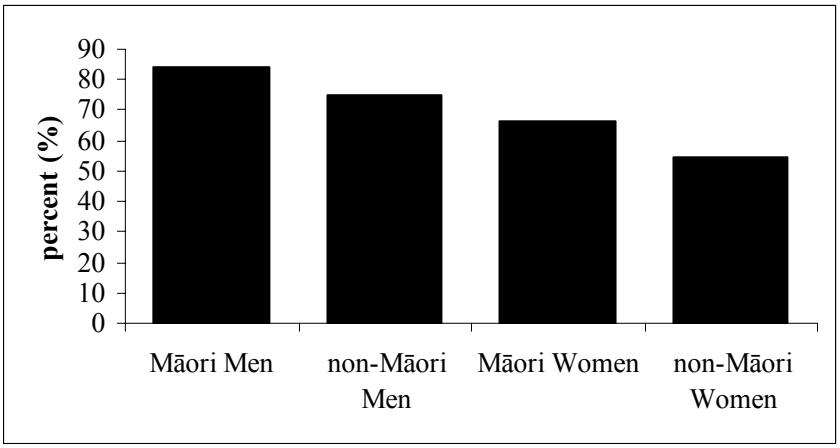


Figure 5.8 Distribution of observed apnoeas, by ethnicity and sex

Differences in reported apnoeas were not however statistically different between Māori and non-Māori, men (p=0.3700) and women (p=0.2600). Within ethnic groups, differences between men and women were significant for non-Māori, but not for Māori (Table 5.9).

Table 5.9 Observed apnoeas, by ethnicity and sex

	Māori (%)	95% CI	non-Māori (%)	95% CI	Ratio	95% CI	p-value
Men	84.21	72.13-92.52	74.70	69.66-79.29	1.13	0.99-1.28	0.1198
Women	66.67	38.38-88.18	54.72	44.75-64.41	1.22	0.82-1.81	0.3826

Daytime sleepiness

The Epworth Sleepiness Scale (ESS) was used to assess excessive daytime sleepiness. The distributions of ESS scores for Māori and non-Māori, men and women are presented in Figure 5.9.

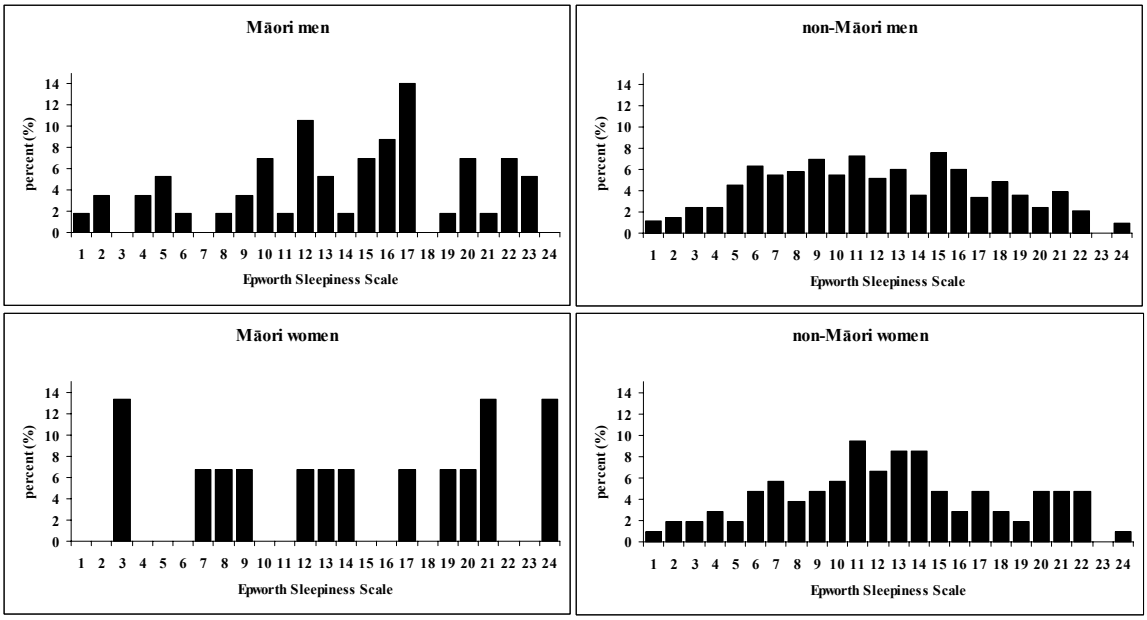


Figure 5.9 Distribution of ESS scores by ethnicity and sex

As there are only a few Māori men and women in the sample, the distributions are somewhat fragmented. In comparison, the distributions for non-Māori men and women are clearer and are similar in shape.

An ESS score greater than 10 was used as a marker of excessive daytime sleepiness (EDS) and differences between Māori and non-Māori men and women were tested. For men, Māori were more likely to report excessive daytime sleepiness (72% vs. 57%, Ratio=1.27, 95% CI 1.05-1.53, p=0.0301). No differences were found between Māori and non-Māori women. Within ethnic groups, no sex differences were found (Table 5.10).

Table 5.10 Excessive Daytime Sleepiness (EDS), by ethnicity and sex

	Māori (%)	95% CI	non-Māori (%)	95% CI	Ratio	95% CI	p-value
Men	71.93	58.46-83.03	56.63	51.11-62.03	1.27	1.05-1.53	**0.0301**
Women	66.67	38.38-88.18	65.09	55.22-74.10	1.02	0.70-1.50	0.9047

5.3.3 OSAS risk factors

Body Mass Index (BMI)

Height and weight measurements were recorded from participants' clinical notes. As expected, the distributions of BMI indicate extreme skewing towards higher values (Figure 5.10).

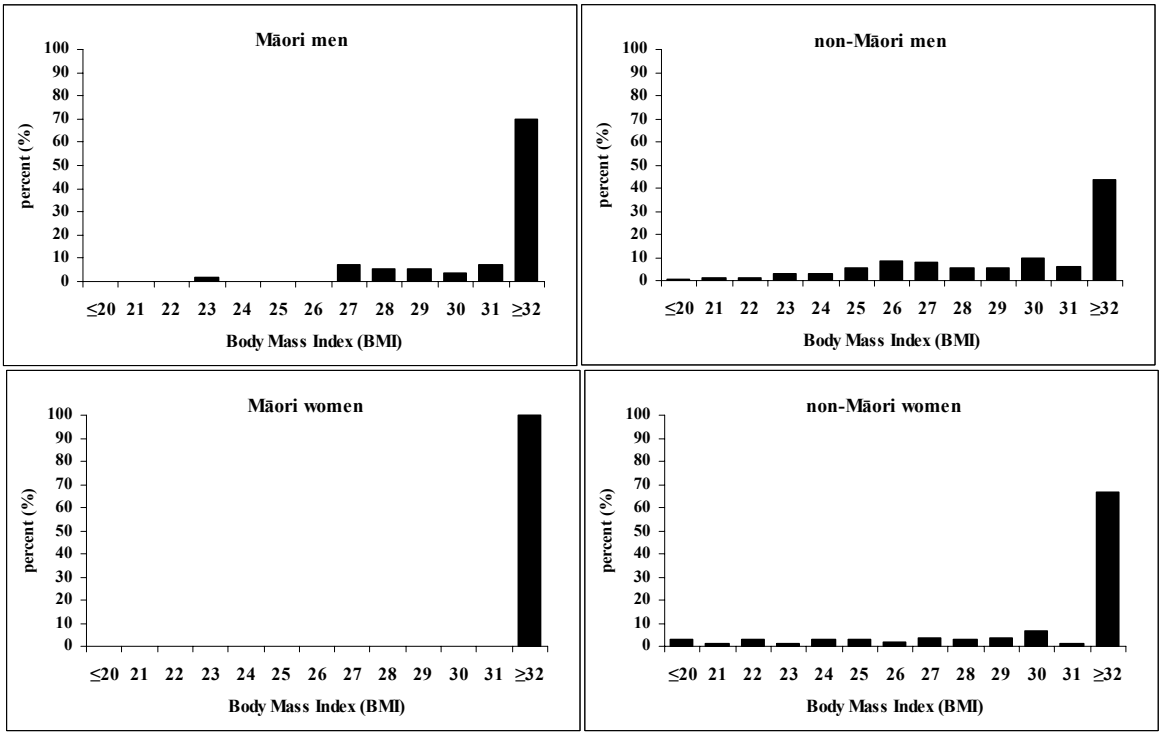


Figure 5.10 BMI distributions, by ethnicity and sex

The skewing was more prominent for Māori men and women. In particular, all Māori women had a BMI ≥ 32. As data were clearly not normally distributed, differences within and between groups were tested using the Wilcoxon ranked sum test (Table 5.11).

Table 5.11 Median BMI, by ethnicity and sex

	Māori (IQR)	non-Māori (IQR)	p-value
Men	36.33 (30.61-41.34)	30.43 (26.90-35.70)	<0.0001
Women	45.47 (36.20-51.90)	36.26 (29.41-41.14)	0.0027
p-value	0.0038	<0.0001	

Māori had a significantly higher median BMI than non-Māori amongst both men and women. Within ethnic groups, women had a significantly higher BMI than men. BMI

was also examined categorically as obese (Māori BMI > 32kg/m^2 and non-Māori BMI > 30kg/m^2) (Table 5.12).

Table 5.12 Obesity, by ethnicity and sex

	Māori (%)	95% CI	non-Māori (%)	95% CI	Ratio	95% CI	p-value
Men	66.67	52.94-78.60	52.41	46.89-57.89	1.27	1.03-1.57	0.046
Women	93.33	68.05-99.83	69.81	60.13-78.35	1.34	1.11-1.61	0.0558

Amongst men, Māori were more likely to be obese than non-Māori. However no differences were found between Māori and non-Māori women for the likelihood of being obese.

Neck circumference

Neck circumference distributions as measured by the sleep technician are presented in Figure 5.11.

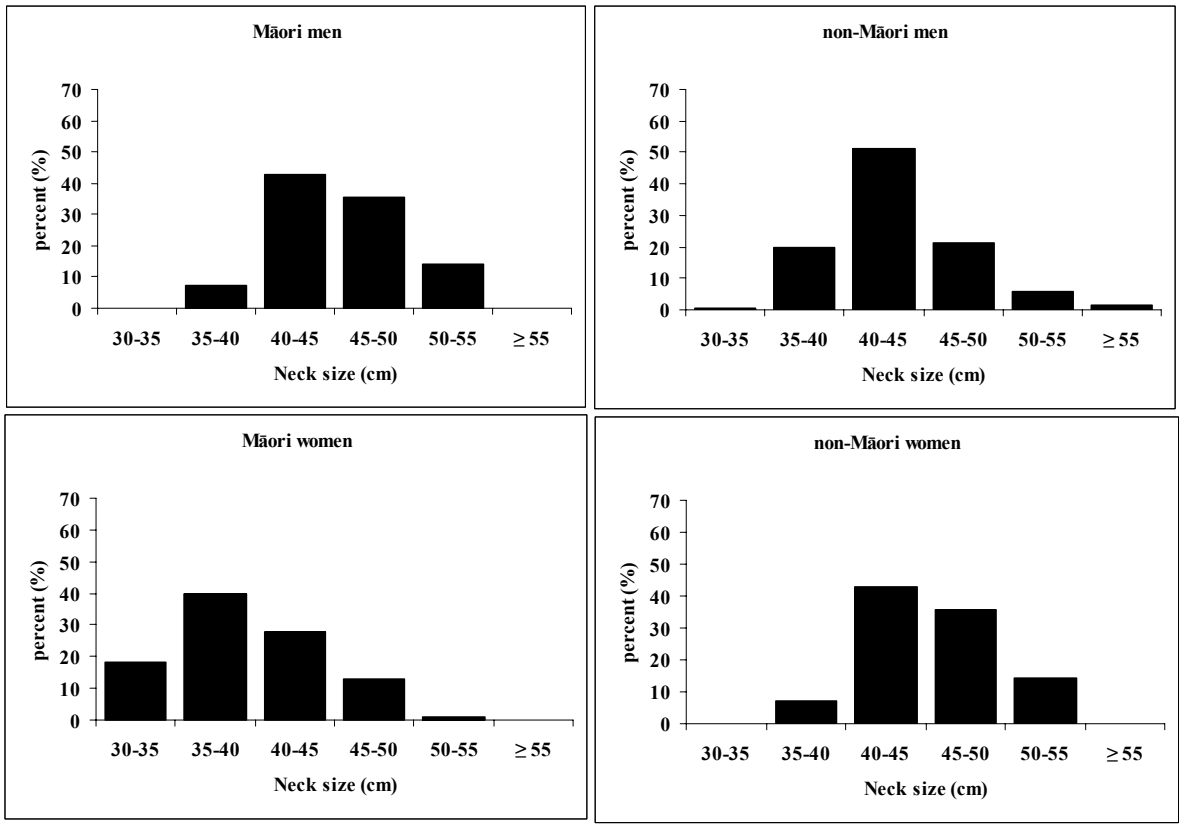


Figure 5.11 Distribution of neck circumference, by ethnicity and sex

The median neck size for Māori men was significantly larger than non-Māori men. Similarly, Māori women had significantly larger median neck size than and non-Māori women (Table 5.13).

Table 5.13 Median neck circumference, by ethnicity and sex

	n	Median	IQR
Māori men	56	44.75	42.75-47.50
Māori women	316	42.50	40.00-45.00
non-Māori men	14	42.00	40.00-47.00
non-Māori women	100	38.75	35.50-43.00
Total	486	42.00	39.50-45.00

Co-morbid disease

Participants were asked whether at the time of the study they were having treatment for any of the following conditions: asthma, high blood pressure, heart trouble, diabetes, stroke, thyroid problems, psychological problems or sleeping problems. Two hundred and ninety four patients were receiving treatment at the time of the study for at least one or more conditions (Table 5.14). Overall, the most commonly reported conditions were hypertension (27.25%) and asthma (20 %) (Figure 5.12).

Table 5.14 Currently receiving treatment for medical conditions, by ethnicity and sex

Condition	Māori		non-Māori		Total
	Men	Women	Men	Women	
Asthma	14	6	52	30	102
Hypertension	14	6	34	85	139
Heart Trouble	8	6	45	14	73
Diabetes	7	4	22	12	45
Stroke	0	0	9	1	10
Thyroid problem	1	0	4	8	13
Psych. problem	5	1	27	20	53
Sleep problem	4	1	23	7	35
Total	72	30	267	126	470

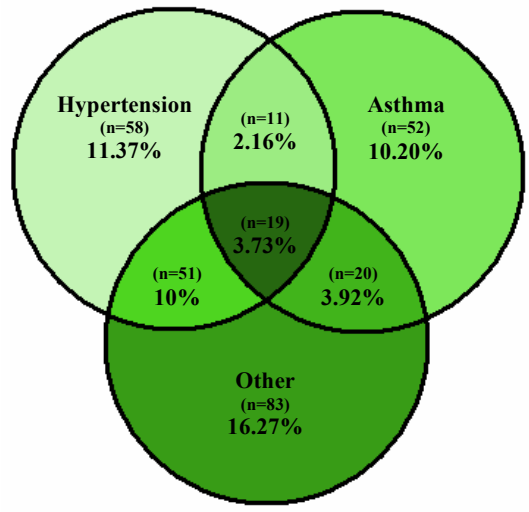


Figure 5.12 Patients receiving treatment for asthma, hypertension and other conditions

Smoking status

Participants were asked to describe themselves as one of the following: *regular smoker* (I smoke one or more cigarettes per day), *occasional smoker* (I do not smoke every day), *ex-smoker* (I use to smoke but not any more), or a *non-smoker* (I have never smoked regularly) (Figure 5.13).

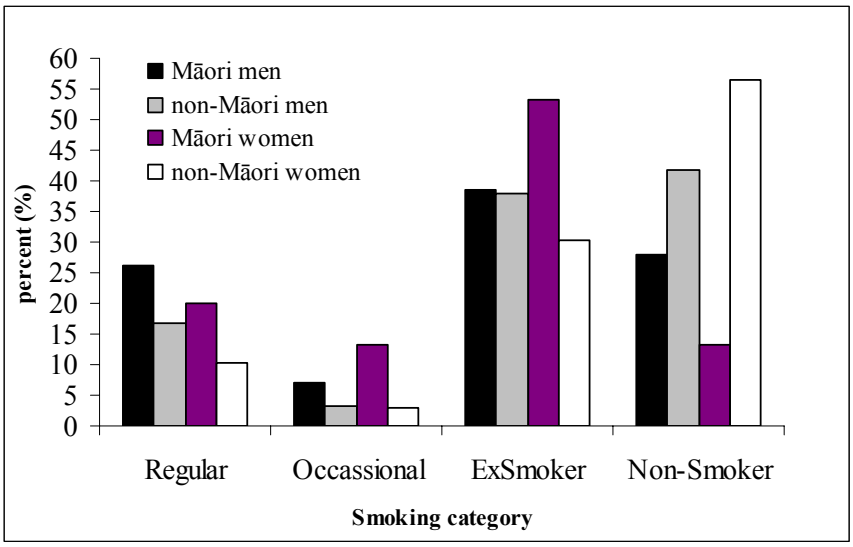


Figure 5.13 Cigarette smoking, by ethnicity and sex

The majority of this sample was comprised of *ex-smokers* and *non-smokers.* Māori men were more likely than non-Māori men to be current (*regular*) smokers. Similarly, Māori women were more likely than non-Māori women to be current smokers. Within ethnic groups, no differences were found between men and women (Table 5.14).

Table 5.15 Current smokers, by ethnicity and sex

	Māori (%)	95% CI	Non-Māori (%)	95% CI	Ratio	95% CI	p-value
Men	33.33	21.40-47.07	20.18	16.00-24.91	1.65	1.08-2.53	**0.0271**
Women	33.33	11.82-61.62	13.21	7.41-21.17	2.52	1.06-6.00	**0.0449**

Alcohol consumption

To obtain a picture of the different patterns of drinking reported among Māori and non-Māori participants, two questions were asked regarding alcohol consumption. The first question asked how often participants drink alcohol (Figure 5.14), and the second question asked how many drinks they would normally consume on a typical drinking occasion (Figure 5.15).

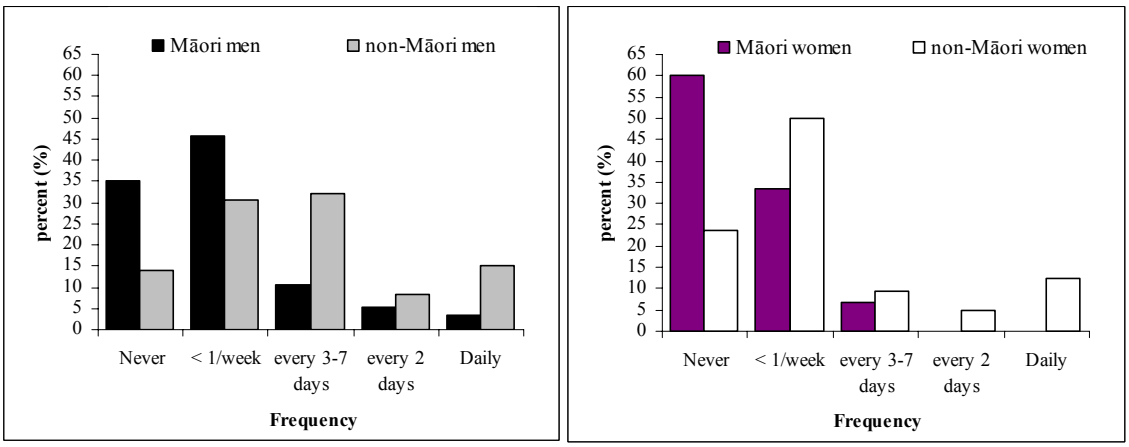


Figure 5.14 Frequency of alcohol consumption, by ethnicity and sex

Figure 5.15 Amount of alcohol consumed on a typical drinking occasion, by ethnicity and sex

The graphs indicate that Māori men and women drank alcohol less frequently than non-Māori men and women. However on a typical drinking occasion Māori men and women consumed more alcohol than non-Māori on a typical drinking occasion.

To test for differences in the drinking patterns for each group, responses were grouped to reflect different frequencies of drinking and amounts consumed (Table 5.16 and Table 5.17).

Table 5.16 Alcohol frequency (at least once a week), by ethnicity and sex

	Māori (%)	95% CI	Non-Māori (%)	95% CI	Ratio	95% CI	p-value
Men	19.30	10.05-31.91	55.42	49.90-60.85	0.35	0.20-0.60	**<0.0001**
Women	6.67	0.17-31.95	26.42	18.32-35.87	0.25	0.04-1.72	0.0935

Table 5.17 Alcohol consumption (≥ 5 drinks on typical occasion), by ethnicity and sex

	Māori (%)	95% CI	non-Māori (%)	95% CI	Ratio	95% CI	p-value
Men	36.84	24.45-50.66	20.48	16.27-25.23	1.80	1.21-2.69	**0.0066**
Women	13.33	1.66-40.46	8.49	3.96-15.51	1.57	0.37-6.59	0.5414

For men, Māori were significantly less likely to consume alcohol at least once a week. But on a typical drinking occasion, Māori men were more likely to consume a greater quantity of alcohol than non-Māori men. No differences were found between Māori and non-Māori women for either frequency of consumption (p=0.0935) or amount consumed (p=0.5414). Within ethnic groups, no differences were found between men and women.

5.3.4 Other variables

Community Services Card (CSC)

As a crude measure of socio-economic deprivation, participants were asked if they were eligible for a community services card (CSC) with the options of *yes*, *no* or *don't know*. The numbers of responses in each option are presented in Table 5.18 and proportions of eligible cardholders are presented in Figure 5.16.

Table 5.18 Are you eligible for a community services card?

	Yes	No	Don't know
Māori men	22	30	5
non-Māori men	73	224	35
Māori women	11	4	0
non-Māori women	37	64	5
Total	143	322	45

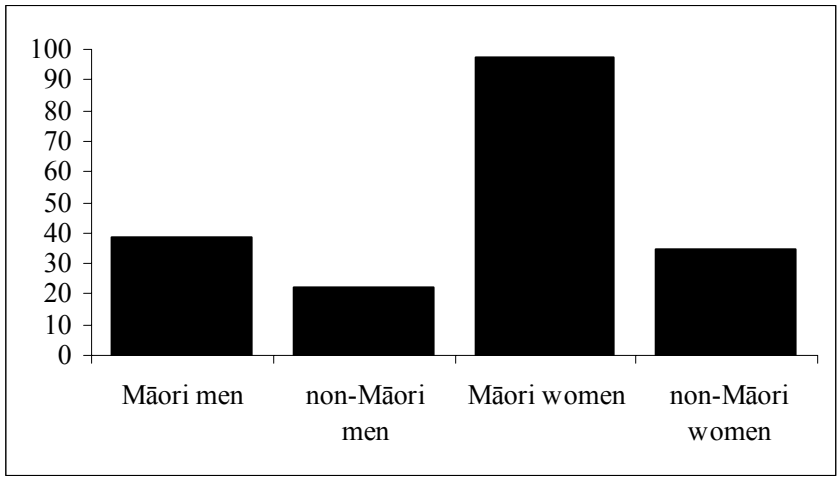


Figure 5.16 Percentage of participants eligible for CSC

Māori women made up the greatest proportion of eligible cardholders, followed by Māori men. When proportions were tested for statistical difference, both Māori men and women were twice as likely to be eligible for a CSC than non-Māori. There were however no differences between men and women within ethnic groups (Table 5.19).

Table 5.19 CSC eligibility, by ethnicity and sex

	Māori (%)	95% CI	non-Māori (%)	95% CI	Ratio	95% CI	p-value
Men	38.60	26.00-52.43	21.99	17.65-26.83	1.76	1.19-2.58	**0.007**
Women	73.33	44.90-92.21	34.91	25.90-44.78	2.10	1.41-3.12	**0.004**

5.4 Polysomnography Data

Total sleep time

The distributions of total sleep time (TST) are illustrated in Figure 5.17. The high percentage of people with less than five hours sleep reflects patients who had split-night studies[14]. In patients with severe apnoea, a reliable assessment of the respiratory disturbance index is possible with a partial night study. Only data from the diagnostic half of their study were used for these analyses.

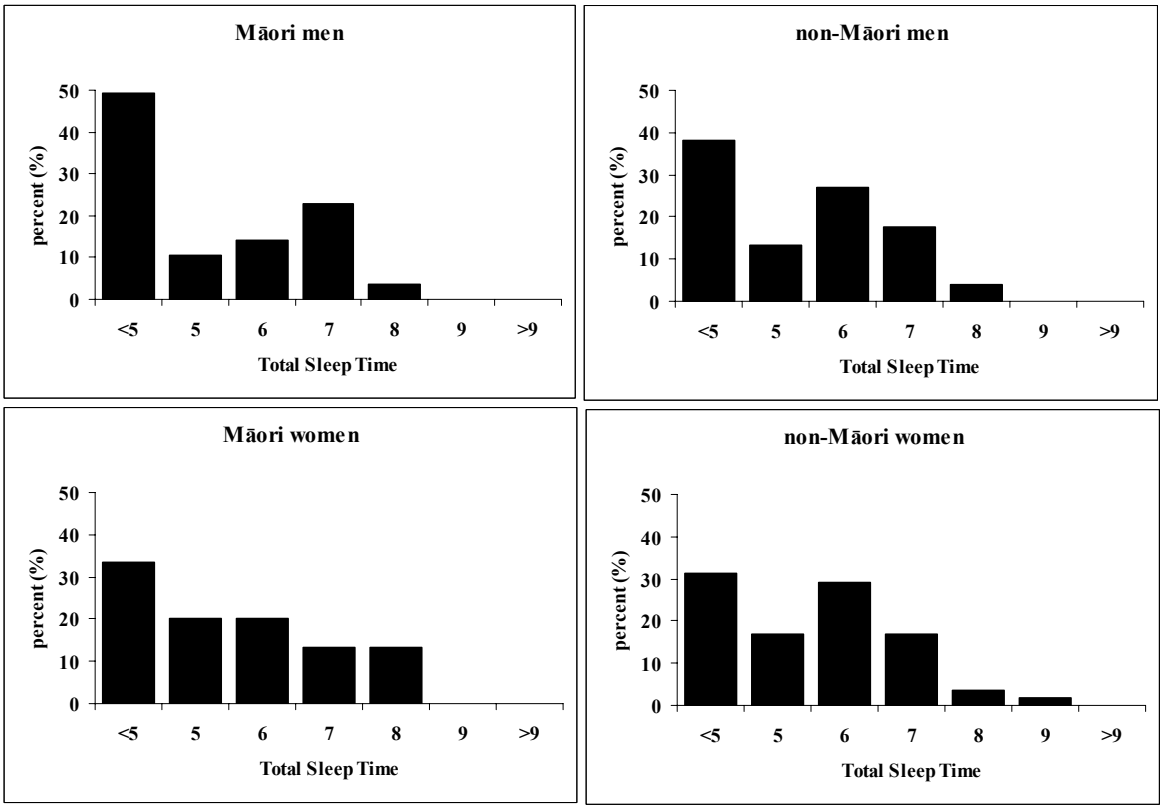


Figure 5.17 Distribution of Total Sleep Time (TST)

The median hours of sleep attained during diagnostic studies was 5.49 hours (IQR=3.50-6.52). The data were not normally distributed therefore the Wilcoxon ranked sum test was used to test for differences in the medians between groups. No differences were found between Māori and non-Māori, men and women. Similarly, no differences were found within ethnic groups (Table 5.20)

[14] Split night studies utilise the first 2 or 3 hours for evaluating the presence of sleep apnoea and the second half to titrate and adjust CPAP treatment.

Table 5.20 Total sleep time (TST), by ethnicity and sex

	Māori (IQR)	non-Māori (IQR)	p-value
Men	4.66 (2.78-6.65)	5.52 (3.36-6.49)	0.2178
Women	5.36 (3.84-7.13)	5.63 (4.38-6.51)	0.9311
p-value	0.2121	0.1983	

Obstructive Sleep Apnoea (OSA)

For comparability with community MESAM4 sleep studies, equivalent respiratory disturbance indices were extracted from the polysomnographic data. Distributions for RDIa and RDIc are presented in Figure 5.18.

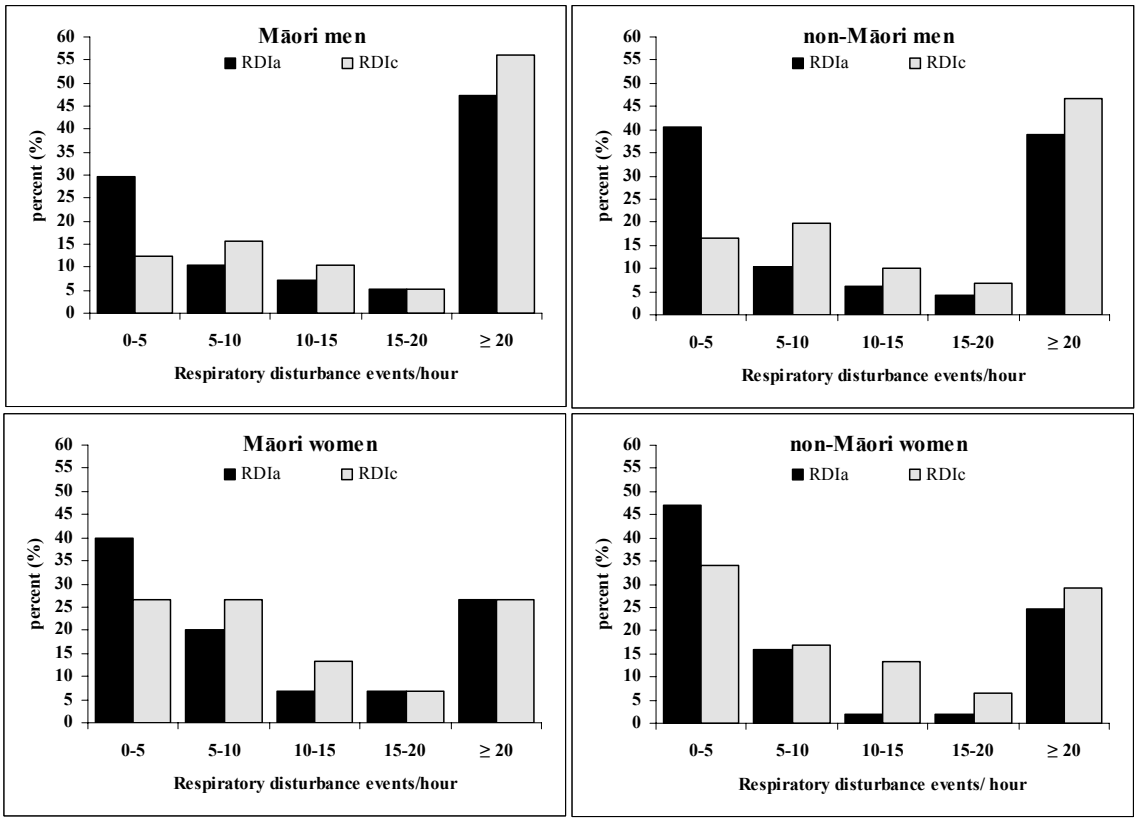


Figure 5.18 RDI distributions, by ethnicity and sex

Compared with women, distributions for men were more skewed towards higher RDI scores. As the data were not normally distributed, tests between the medians scores were conducted between groups. For both RDIa and RDIc, no significant differences were found between Māori and non-Māori, men and women. However, within ethnic groups, men had more severe OSA than women (Table 5.21 and Table 5.22).

Table 5.21 Median RDIa, by ethnicity and sex

	Māori (IQR)	non-Māori (IQR)	p-value
Men	15.82 (4.63-57.94)	9.33 (1.99-41.67)	0.09
Women	6.73 (1.73-27.75)	5.21 (0.45-17.27)	0.9154
p-value	0.005	<0.0001	

Table 5.22 Median RDIc, by ethnicity and sex

	Māori (IQR)	non-Māori (IQR)	p-value
Men	27.70 (8.2-59.2)	16.40 (7.1-49.60)	0.0547
Women	9.10 (3.6-22.8)	9.65 (2.30-26.70)	0.6537
p-value	0.0490	0.001	

Unadjusted proportions for each index at varying thresholds, and tests for differences between the groups are presented in Table 5.23 to Table 5.28.

Table 5.23 RDIa ≥ 5

	No.	%	95% CI	Ratio	p-value
Overall	302	59.22	54.81-63.52		
Men	237	53.72	44.43-62.83	1.10	0.5621
Women	65	60.93	55.88-65.80		
Māori	49	68.06	56.01-78.56	1.17	0.5621
non-Māori	253	57.76	52.98-62.44		
Māori men	40	70.18	56.60-81.57	1.13	0.5499
non-Māori men	197	59.34	53.84-64.67		
Māori women	9	60.00	32.29-83.66	1.21	0.6986
non-Māori women	56	52.83	42.89-62.60		

Table 5.24 RDIa ≥ 10

	n	%	95% CI	Ratio	p-value
Overall	242	47.45	43.05-51.89		
Men	197	50.64	45.56-55.72	1.26	0.2278
Women	45	37.19	28.58-46.44		
Māori	40	55.56	43.37-67.28	1.08	0.7715
non-Māori	202	46.12	41.38-50.91		
Māori men	34	59.65	45.82-72.44	1.16	0.4933
non-Māori men	163	49.10	43.60-56.61		
Māori women	6	40.00	16.34-67.71	1.00	0.9932
non-Māori women	39	36.80	42.89-62.60		

Table 5.25 RDIa ≥ 15

	n	%	95% CI	Ratio	p-value
Overall	206	40.39	36.10-44.79		
Men	173	44.47	39.47-49.57	1.47	0.0619
Women	33	27.27	19.58-36.12		
Māori	35	48.61	36.65-60.69	1.13	0.6922
non-Māori	171	39.04	34.45-43.79		
Māori men	30	52.63	38.97-66.02	1.10	0.6783
non-Māori men	143	43.07	37.68-48.59		
Māori women	5	33.33	11.82-61.62	1.18	0.7990
non-Māori women	28	26.42	18.33-35.87		

Table 5.26 RDIc ≥ 5

	n	%	95% CI	Ratio	p-value
Overall	408	80.00	76.26-83.39		
Men	327	84.06	80.04-87.56	1.25	0.1217
Women	81	66.94	57.81-75.22		
Māori	61	84.72	74.31-92.12	1.05	0.832
non-Māori	347	79.22	75.12-82.93		
Māori men	50	87.72	76.32-94.92	1.03	0.8586
non-Māori men	277	83.43	78.99-87.27		
Māori women	11	73.33	44.90-92.21	1.07	0.8716
non-Māori women	70	66.04	56.20-74.96		

Table 5.27 RDIc ≥ 10

	n	%	95% CI	Ratio	p-value
Overall	311	60.98	56.50-65.24		
Men	252	64.78	59.81-69.53	1.18	0.3116
Women	59	48.76	39.57-58.01		
Māori	48	66.67	54.57-77.34	1.09	0.7402
non-Māori	263	60.05	55.29-64.67		
Māori men	41	71.93	58.46-83.03	1.11	0.5991
non-Māori men	211	63.55	58.12-68.74		
Māori women	7	46.67	21.27-73.41	1.08	0.8857
non-Māori women	52	49.06	39.22-58.95		

Table 5.28 RDIc ≥ 15

	n	%	95% CI	Ratio	p-value
Overall	256	50.20	45.77-54.62		
Men	43	54.76	49.66-59.78	1.43	0.0496
Women	213	35.54	22.05-44.75		
Māori	40	55.56	43.37-67.28	1.03	0.9053
non-Māori	216	49.32	44.54-54.10		
Māori men	35	61.40	47.58-74.01	1.12	0.5800
non-Māori men	178	53.61	48.09-59.08		
Māori women	5	33.33	11.82-61.62	0.92	0.8843
non-Māori women	38	35.85	26.77-45.75		

Overall, the prevalence of OSA in the clinical sample ranged from 40.39% to 80.00% depending on the threshold used to define OSA. For men, the prevalence ranged from 44.47% to 84.06%, and for women, the prevalence ranged from 27.27% to 66.94%. As this sample was specifically referred for suspected OSAS, it is not surprising that few statistical differences were found in the prevalence and severity of OSA between Māori and non-Māori, men and women. However, differences between men and women were trending towards significance as OSA severity criteria increased (RDIa and RDIc≥ 15).

As mentioned previously, the definition of obstructive sleep apnoea syndrome (OSAS) used in this study was the presence of significant respiratory disturbance, in conjunction with the presence of excessive daytime sleepiness, which was defined as an ESS>10. Unadjusted proportions for (OSAS) at varying thresholds and tests for differences between groups are presented in Table 5.29 and Table 5.30.

Table 5.29 OSAS (RDIa + ESS >10) estimates, by ethnicity and sex

Variable		Māori (%) (95% CI)	non – Māori (%) (95% CI)	Relative Risk (95% CI)	p-value
RDI≥ 5 + ESS>10	Men	52.63 (38.97-66.02)	38.55 (33.29-44.02)	1.21 (0.78-1.86)	0.4226
	Women	40.00 (16.34-67.71)	34.91 (25.90-44.76)	1.05 (0.35-3.17)	0.9275
RDI≥ 10 + ESS>10	Men	49.12 (35.63-62.71)	33.13 (28.09-38.48)	1.30 (0.82-2.04)	0.3010
	Women	26.67 (7.79-55.10)	26.84 (16.69-33.84)	1.14 (0.31-4.21)	0.8473
RDI≥ 15 + ESS>10	Men	45.61 (32.36-59.34)	29.52 (24.66-34.74)	1.73 (1.08-2.78)	0.0562
	Women	20.00 (4.33-48.09)	18.87 (11.92-27.63)	1.25 (0.29-5.50)	0.7846

Table 5.30 OSAS (RDIc + ESS >10) estimates, by ethnicity and sex

Variable		Māori (%) (95% CI)	non – Māori (%) (95% CI)	Relative Risk (95% CI)	p-value
RDI≥ 5 + ESS>10	Men	63.16 (49.35-75.55)	50.30 (44.79-55.81)	1.17 (0.79-1.74)	0.4453
	Women	46.67 (21.27-73.41)	44.34 (34.69-54.31)	0.92 (0.33-2.54)	0.862
RDI≥ 10 + ESS>10	Men	54.39 (40.66-67.65)	41.57 (36.21-47.07)	1.21 (0.79-1.86)	0.4029
	Women	26.67 (7.79-55.10)	33.96 (25.05-43.80)	0.89 (0.24-3.22)	0.8583
RDI ≥15 + ESS>10	Men	49.12 (35.63-62.71)	36.45 (31.26-41.88)	1.19 (0.76-1.86)	0.4794
	Women	20.00 (4.33-48.09)	23.58 (15.88-32.82)	1.07 (0.25-4.63)	0.9277

No differences were found between Māori and non-Māori, men and women for the prevalence of OSAS, however most trends are in the direction of Māori having more prevalent OSAS.

5.5 Predictors of OSA

This section presents predictors of OSA identified by logistic regression analyses at varying thresholds of RDIa and RDIc (≥ 5,≥ 10,≥ 15). Table 5.31 presents the relationships between ethnicity, sex, age and OSA.

Table 5.31 OSA – Logistic regression model 1

Variable	RDIa ≥ 5	RDIa ≥ 10	RDIa ≥ 15	RDIc ≥ 5	RDIc ≥ 10	RDIc ≥ 15
Ethnicity (Māori vs. non-Māori)	1.70 (0.99-2.92)	1.53 (0.92-2.56)	1.54 (0.92-2.57)	1.48 (0.74-2.96)	1.40 (0.82-2.39)	1.33 (0.93-2.75)
Sex (men vs. women)	1.40 (0.92-2.13)	**1.78 (1.17-2.72)****	**2.19 (1.40-3.44)****	**2.66 (1.66-4.25)****	**2.00 (1.32-3.03)****	**2.25 (1.47-3.45)****
Age (yearly increase)	**1.03 (1.01-1.06)****	1.02 (1.00-1.04)	1.02 (1.00-1.04)	1.19 (0.90-1.58)	1.02 (1.00-1.05)	1.02 (1.00-1.04)

** p< 0.01

After controlling for sex and age, ethnicity was not predictive of OSA. Sex was the most consistent predictor, with men 2-3 times more likely than women to suffer from OSA.

Additional variables relating to OSA were then added to the model. As mentioned previously, two separate models were constructed - one with BMI (Model 1a) and the other with neck circumference (Model 1b) (Table 5.32 and Table 5.33 respectively).

Table 5.32 OSA – Logistic regression model 1a (BMI)

Variable	RDIa			RDIc		
	≥ 5	≥ 10	≥ 15	≥ 5	≥ 10	≥ 15
Ethnicity (Māori vs. non-Māori)	0.56 (0.35-1.19)	0.73 (0.45-1.18)	0.75 (0.45-1.24)	0.70 (0.46-1.07)	0.64 (0.41-0.98)	0.75 (0.48-1.18)
Sex (men vs. women)	**2.57 (1.44-4.61)****	**3.88 (2.30-6.51)****	**4.93 (2.80-8.67)****	**3.71 (2.50-5.52)****	**3.62 (2.34-5.58)****	**3.87 (2.42-6.21)****
Age (yearly increase)	**1.05 (1.02-1.07)****	**1.04 (1.01-1.07)****	**1.04 (1.01-1.07)****	**1.02 (1.00-1.04)***	**1.03 (1.01-1.06)****	**1.03 (1.01-1.06)****
CSC (eligible vs. other)	1.42 (0.87-2.33)	**1.78 (1.09-2.89)***	1.59 (0.97-2.60)	0.99 (0.59-1.68)	1.13 (0.71-1.82)	1.14 (0.71-1.83)
BMI (increasing)	**1.17 (1.12-1.21)****	**1.14 (1.10-1.18)****	**1.13 (1.10-1.17)****	**1.12 (1.08-1.15)****	**1.13 (1.10-1.17)****	**1.12 (1.09-1.16)****
Wake feeling refreshed (never/rarely vs. often /always)	0.75 (0.44-1.30)	0.65 (0.37-1.16)	0.94 (0.52-1.71)	0.80 (0.48-1.31)	0.70 (0.42-1.17)	0.63 (0.37-1.08)
Enough sleep (never/rarely vs. often/always)	1.32 (0.79-2.19)	1.31 (0.78-2.200	0.90 (0.53-1.54)	1.38 (0.84-2.25)	**1.73 (1.06-2.81)****	1.43 (0.81-2.72)
Observed apnoeas (apnoea vs. not)	**2.55 (1.53-4.26)****	**3.75 (2.40-5.86)****	**3.97 (2.43-6.41)****	**3.39 (2.26-5.07)****	**3.83 (2.59-5.65)****	**4.62 (3.05-7.00)****
Excessive daytime sleepiness (ESS>10 vs. ESS ≤ 10)	1.75 (0.96-2.62)	1.75 (0.94-2.66)	1.68 (1.08-2.62)	2.12 (1.41-3.19)	**1.79 (1.21-2.64)***	**1.63 (1.09-2.43)***
Snore (always vs. never/rarely/often)	**2.57 (1.61-4.08)****	**2.80 (1.87-4.23)****	**2.65 (1.73-4.04)****	**2.38 (1.53-3.07)****	**2.08 (1.41-3.07)****	**1.95 (1.32-2.88)****
Asthma (curr. treatment vs. no/don't know)	**0.34 (0.19-0.60)****	**0.29 (0.16-0.52)****	**0.32 (0.17-0.58)****	**0.48 (0.26-0.87)****	**0.46 (0.28-0.75)****	**0.32 (0.19-0.55)****
Hypertension (treatment vs. no/don't know)	1.61 (0.98-2.64)	1.63 (0.83-2.65)	1.91 (0.96-3.10)	1.44 (0.83-2.50)	1.14 (0.71-1.85)	1.32 (0.82-2.13)
Smoking (Regular vs. occasional/ex-smoker/non-smoker)	0.95 (0.59-1.54)	0.85 (0.52-1.39)	0.88 (0.53-1.44)	1.19 (0.74-1.92)	0.92 (0.59-1.46)	0.88 (0.56-1.40)
Alcohol (exceed rec. vs. non-drinker)	1.15 (0.65-2.02)	1.17 (0.66-2.06)	1.15 (0.64-2.05)	0.93 (0.52-1.64)	0.77 (0.45-1.32)	1.25 (0.73-2.16)

*p<0.05 **p<0.01

Table 5.33 OSA – Logistic regression model 1b (Neck)

Variable	RDIa			RDIc		
	≥ 5	≥ 10	≥ 15	≥5	≥ 10	≥ 15
Ethnicity (Māori vs. non-Māori)	0.74 (0.48-1.13)	0.89 (0.57-1.40)	0.89 (0.55-1.44)	0.80 (0.53-1.19)	0.77 (0.51-1.15)	0.88 (0.57-1.35)
Sex (men vs. women)	1.51 (0.99-2.30)	0.80 (0.46-1.63)	0.58 (0.34-1.03)	1.76 (0.97 -3.03)	1.00 (0.58-1.72)	1.04 (0.61-1.79)
Age (yearly increase)	**1.04 (1.01-1.06)****	**1.03 (1.01-1.06)****	**1.03 (1.00-1.05)****	**1.05 (1.00-1.08)****	**1.04 (1.00-1.05)****	**1.03 (1.00-1.05)***
CSC (eligible vs. other)	1.37 (0.96-1.94)	1.42 (1.00-2.00)*	1.16 (0.82-1.64)	1.25 (0.76-2.05)	1.49 (1.35-2.00)*	1.49 (0.95-2.33)
Neck circumference (increasing)	**1.25 (1.17-1.33)****	**1.26 (1.18-1.34)****	**1.32 (1.24-1.42)****	**1.25 (1.17-1.33)****	**1.26 (1.18-1.34)****	**1.23 (1.15-1.30)****
Wake feeling refreshed (never/rarely vs. often /always)	0.83 (0.51-1.36)	0.72 (0.42-1.21)	0.99 (0.57-1.72)	0.89 (0.55-1.43)	0.78 (0.48-1.25)	0.70 (0.42-1.15)
Enough sleep (never/rarely vs. often/always)	1.16 (0.73-1.84)	1.21 (0.75-1.95)	0.89 (0.54-1.48)	0.17 (0.74-1.87)	1.49 (0.94-2.34)	1.51 (0.94-2.40)
Observed apnoeas (apnoea vs. not)	**3.90 (2.65-5.75)****	**4.10 (2.69-6.27)****	**4.24 (2.67-6.74)****	**3.86 (2.60-5.72)****	**4.18 (2.88-6.06)****	**3.17 (1.78-5.64)****
Excessive daytime sleepiness (ESS>10 vs. ESS ≤ 10)	1.42 (0.90-2.84)	1.56 (0.97-2.50)	1.50 (0.92-2.46)	1.47 (0.88-2.45)	**1.82 (1.1-2.86)****	**1.79 (1.22-2.64)***
Snore (always vs. never/rarely/often)	**3.11 (2.12-4.56)****	**3.14 (2.13-4.63)****	**2.88 (1.93- 4.31)****	**2.92 (1.89-4.52)****	**2.51 (1.71-3.67)****	**2.25 (1.54-3.29)****
Asthma (curr. treatment vs. no/don't know)	**0.36 (0.21-0.64)****	**0.37 (0.22-0.64)****	**0.40 (0.22-0.70)****	**0.25 (0.14-0.47)****	**0.58 (0.36-0.94)****	**0.24 (0.12-0.46)****
Hypertension (treatment vs. no/don't know)	0.61 (0.37-0.99)	1.25 (0.74-2.11)	1.31 (0.77-2.23)	1.39 (0.71-2.72)	1.01 (0.58-1.73)	1.08 (0.64-1.81)
Smoking (Regular vs. occasional/ex-smoker/non-smoker)	0.90 (0.51-1.59)	0.92 (0.58-1.46)	0.94 (0.59-1.51)	1.19 (0.75-1.90)	0.95 (0.61-1.48)	0.92 (0.59-1.44)
Alcohol (exceed rec. vs. non-drinker)	0.99 (0.64-1.56)	1.31 (0.76-2.26)	1.29 (0.74-2.25)	1.10 (0.63-1.91)	0.91 (0.54-1.54)	1.42 (0.83-2.41)

*p<0.05 **p<0.01

BMI and neck circumference were consistent predictors in their respective models. Sex remained a significant predictive variable, but only for the model that included BMI (Model 1a), with men 2-4 times more likely to have OSA. Significant predictors were very similar between the two models, with only slight variations amongst some of the predictive variables. Observed apnoeas and self-reported snoring were also consistent predictors in each model. Asthma had a consistent inverse relationship over both models. Excessive daytime sleepiness was only significant in Model 1b for RDIc≥ 10 and 15.

5.6 Validation of Self-Reported Snoring and Observed Apnoeas

Snoring

As a crude validation of self-reported snoring (How often do you snore?), comparisons were made with the actual percentage of the night spent snoring as measured by the microphone taped over the trachea (Figure 5.19).

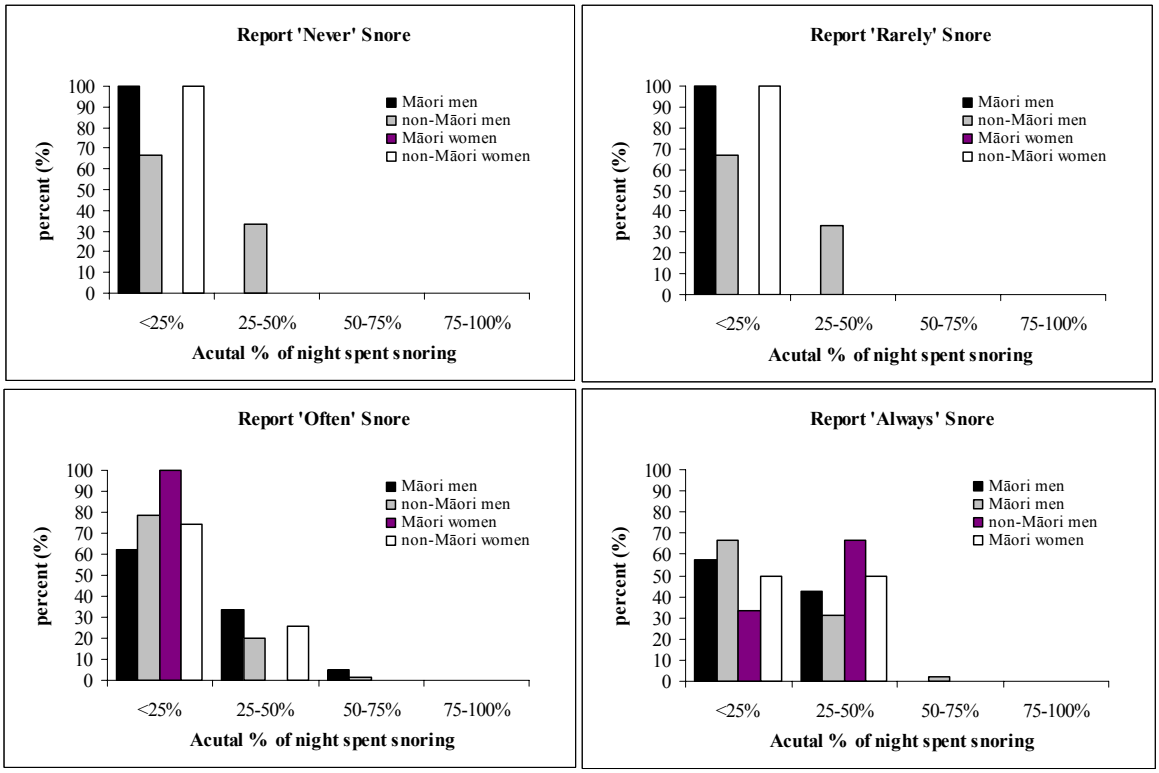


Figure 5.19 Clinical participants: Reported snoring vs. actual snoring

Of the participants with objective snoring data, 316 reported that they *often* or *always* snore. Of these, 31% snored for at least 25% of the night and only 1% snored for at least 50% of the night. Among those participants (n=30) who reported that they *never* or *rarely* snore, only 17% snored for at least 25% of the night. In general the subjective reports of snoring were in the same direction as objective snoring measure.

Observed apnoea

To assess the validity of the question regarding apnoeic events (Has anyone ever told you that you stop breathing sometimes during sleep?) responses to this question were compared with recorded apnoeas as defined by RDIa≥ 5 (Figure 5.20).

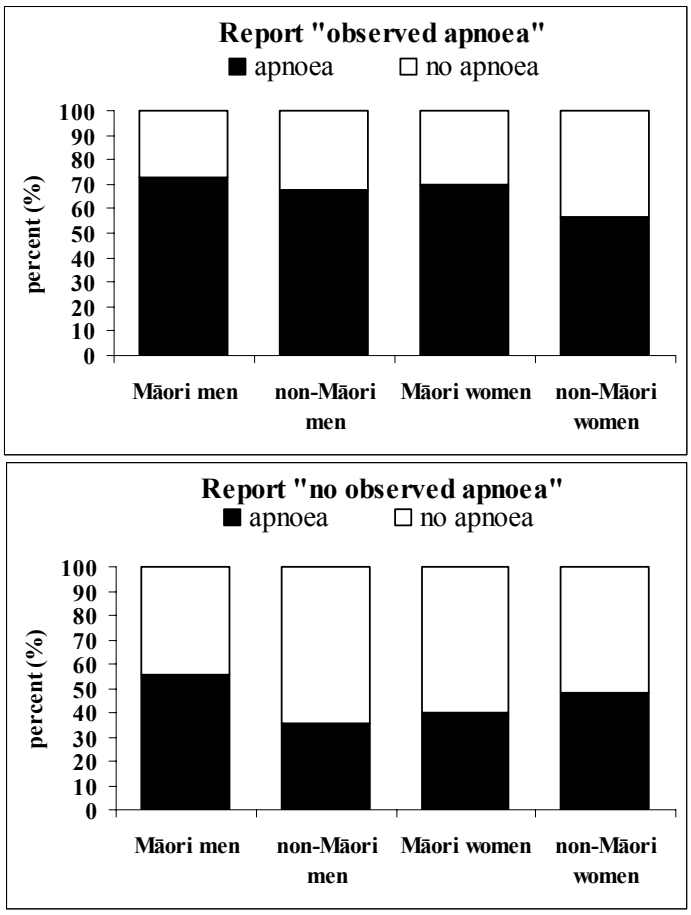


Figure 5.20 Reported vs. recorded apnoeas

The results indicated that this question has similar accuracy across groups. Of those who reported observed apnoeas (n=364), 66% had actual apnoeic events. In contrast, of those who reported no apnoea (n=146), only 41% had actual apnoeic events. The general trend of results is in the expected direction therefore the subjective measure of observed apnoeas showed some discriminatory ability.

CHAPTER 6
DEVELOPING A SCREENING TOOL

6.1 Introduction

The main focus of this chapter is on the development of a mathematic model to predict OSA using data combined from the clinic and community samples. This chapter is divided into three main sections. The first section examines the demographic profiles and objective sleep data of the combined sample. The second aims to find the best fitting and most parsimonious models to describe the relationship between OSA and a set of predictor variables. The final section evaluates the performance of each model, with a close examination of the nature of misclassified results (false negatives and false positives).

6.2 Method

6.2.1 Measures

The details regarding the questionnaire and objective sleep measures utilised in the clinic and community samples are outlined in Chapter 3 (Methods).

6.2.2 Statistical analyses

To evaluate the feasibility of combining the two sets of data, the Breslow-Day chi-squared test was used to test the homogeneity of odds ratios between OSA and the potential predictive variables between the clinic and community data. Where non-significance ($p>0.05$) was found, homogeneity of the odds ratios was assumed.

Multiple logistic regression modelling was used as the primary method of analysis. This allowed prediction of OSA as a discrete outcome (present/absent) from a combination of variables. Prior to entry into the models, variables were examined for collinearity using a correlation matrix and the Pearson correlation coefficient. Results showed that body mass index and neck circumference measurement were strongly correlated ($n=862$, $r=0.70$, $p<0.0001$) therefore separate models were fitted for each (Figure 6.1).

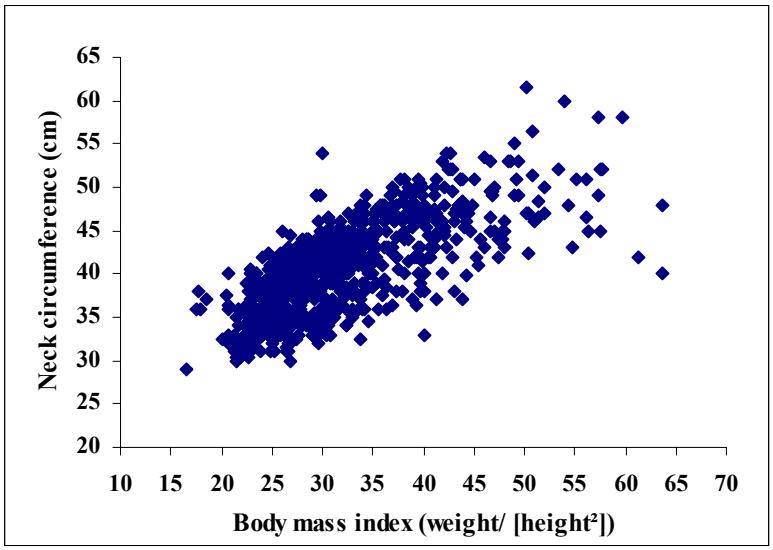


Figure 6.1 Body mass index (kg/m^2) vs. neck circumference (cm)

A set of demographic and questionnaire variables were evaluated individually and in combination for their associations with OSA at varying levels of the respiratory disturbance index (RDI) using a combination of forward selection and backward elimination (*stepwise selection*) to determine the best possible models.

Model development and evaluation

In the development of multivariate models a *significant p-value* approach was adopted whereby a statistical significance of $p < 0.05$ was used for entry or retention of predictors in each model. To assess and compare the fit and performance of each model, a number of different model statistics were examined (- 2 Log L, Likelihood ratio, Wald test, Hosmer-Lemeshow test, Pearson chi-square test, Deviance test, concordance and discordance, and the area under the curve (AUC)).

Concordance and discordance values along with discrimination were used to evaluate the ability of the model to predict the outcome. The higher the concordance, and lower the discordance, the greater the ability of the model to predict the outcome.

Discrimination was defined as the ability of the equation to distinguish high-risk subjects from low-risk subjects, and is quantified by the area under the receiver operating characteristic (ROC) curve (AUC). The greater the area under an ROC curve,

the greater the efficacy of the model. As a general rule, an acceptable level of discrimination is an AUC ≥ 70 (Hosmer and Lemeshow 2002).

Final models

For the final models selected, pretest probabilities of OSA are presented, which provides the necessary context for the models. The likelihood that a patient whose test is positive actually has the disease is higher if the pretest probability is higher. Conversely, if the pre-test probability is low and the test result is negative, the posttest probability is lower (Flemons and Whitelaw 2002).

The logistic regression coefficients are used from the final models to estimate a participant's likelihood of OSA, expressed as a probability ranging from 0 to 1, using the following equation:

$$P = \frac{\exp(\beta_0 + \beta_1 x_1 + \ldots \beta_1 x_1)}{1 + \exp(\beta 0 + \beta_1 x_1 + \ldots \beta_1 x_1)}$$

P is the probability of the condition being true, *exp* is the exponential function, β_0 is the intercept, $\beta_1 x_1$ is the coefficient for explanatory variable i, and x1…..i is the value of the explanatory variable (Hosmer and Lemeshow 2002).

Classification tables are calculated and presented to provide information about the frequency with which observations were correctly and incorrectly classified as events or non-events for the full range of probability cut-off points, along with sensitivity, specificity and rate of false positive and negative predictions. As the same data used to fit the models were also used to test the predictive accuracy of each model, the classification tables were adjusted for bias using the *jackknifing* procedure. This procedure omits each observation one by one from the model when it (the observation) is being classified (SAS Institute 1995).

To select the optimal probability cut-off point to define a positive prediction for each model (with OSA/without OSA), receiver operator characteristic (ROC) curves[15] were constructed for each model, along with plots of the sensitivity and specificity.

[15] ROC curves were developed using Microsoft Excel add-on software called Analyses-It.

6.3 Combining Samples

As the community (n=358) and clinical (n=510) samples were derived from two distinctive populations, tests for homogeneity were carried out to assess the feasibility of combining the two sets of data. A number of pertinent variables were chosen for testing (Table 6.1).

Table 6.1 Community and Clinic: Tests for homogeneity

Variable		Community		Clinic		p-value
		% (95% CI)	OR (95% CI)	% (95% CI)	OR (95% CI)	
Wake feeling refreshed (never/rarely)	RDIa≥ 5	14.29 (9.07-21.01)	1.79 (0.92-3.49)	57.84 (52.89-62.69)	0.75 (0.48-1.18)	0.03
	RDIa≥ 10	8.84 (4.79-14.65)	1.61 (0.71-3.63)	47.55 (42.61-52.52)	1.02 (0.66-0.58)	0.3316
	RDIa≥ 15	5.44 (2.38-10.44)	1.46 (0.54-3.98)	40.20 (35.40-45.13)	0.96 (0.62-1.49)	0.4516
Enough sleep (never/rarely)	RDIa≥ 5	10.00 (5.10-17.19)	0.87 (0.42-1.82)	57.18 (51.80-62.45)	0.77 (0.52-1.12)	0.7636
	RDIa≥ 10	5.45 (2.0-11.50)	0.69 (0.27-1.78)	46.55 (41.22-51.95)	0.89 (0.61-1.30)	0.6237
	RDIa≥ 15	3.64 (1.00-9.04)	0.74 (0.23-2.34)	40.52 (35.32-45.88)	1.02 (0.70-1.49)	0.6061
Snore (often/always)	RDIa≥ 5	30.56 (16.35-48.11)	4.8 (2.13-10.98)	72.00 (66.00-77.46)	2.79 (1.92-4.04)	0.2237
	RDIa≥ 10	22.22 (10.12-39.15)	5.43 (2.12-13.92)	61.60 (55.26-67.66)	3.02 (2.10-4.35)	0.2491
	RDIa≥ 15	8.33 (1.75-22.47)	2.18 (0.59-8.13)	54.40 (48.00-60.69)	3.09 (2.13-4.49)	0.6144
Snore (always)	RDIa≥ 5	18.54 (12.69-25.67)	5.04 (2.22-11.42)	63.78 (59.15-68.22)	5.28 (2.67-10.44)	0.9301
	RDIa≥ 10	12.58 (7.75-18.95)	6.51 (2.17-19.59)	51.33 (46.64-56.04)	4.57 (2.16-9.66)	0.6015
	RDIa≥ 15	7.95 (4.17-13.47)	5.24 (1.45-18.92)	43.78 (39.14-48.50)	3.89 (1.78-8.50)	0.6986
Observed apnoea	RDIa≥ 5	39.13 (25.08-54.63)	8.91 (4.25-18.66)	66.48 (61.38-731.32)	2.84 (1.92-4.22)	0.0062
	RDIa≥ 10	21.74 (10.95-36.36)	5.5 (2.30-13.15)	56.59 (51.33-61.75)	3.98 (2.59-6.12)	0.51
	RDIa≥ 15	17.39 (7.82-31.42)	8.00 (2.84-22.55)	48.90 (43.66-54.17)	4.03 (2.54-6.39)	0.23
Excessive daytime sleepiness (ESS >10)	RDIa≥ 5	21.51 (11.06-34.70)	2.66 (1.23-5.76)	65.26 (59.65-70.57)	1.88 (1.31-2.70)	0.4192
	RDIa≥ 10	11.54 (4.54-23.44)	1.97 (0.75-5.19)	54.55 (48.80-60.20)	2.08 (1.44-2.99)	0.9213
	RDIa≥ 15	5.77 (12.06-15.95)	1.38 (0.38-5.02)	47.40 (41.71-53.14)	2.13 (1.47-3.12)	0.523
Smokers (regular)	RDIa≥ 5	13.16 (6.46-22.87)	1.32 (0.61-2.85)	62.86 (52.88-72.09)	1.21 (0.78-1.89)	0.8476
	RDIa≥ 10	9.21 (3.78-18.06)	1.49 (0.60-3.71)	51.43 (41.47-61.30)	1.22 (0.80-1.88)	0.7018
	RDIa≥ 15	3.95 (0.82-11.12)	0.85 (0.24-3.06)	45.71 (35.96-55.72)	1.32 (0.85-2.03)	0.5244
CSC eligibility	RDIa≥ 5	22.58 (9.59-41.10)	2.69 (1.07-6.73)	67.13 (58.79-74.75)	1.60 (1.06-2.39)	0.3033
	RDIa≥ 10	16.13 (5.45-33.73)	2.95 (1.02-8.51)	57.34 (48.84-65.57)	1.74 (1.18-2.57)	0.3526
	RDIa≥ 15	9.68 (2.04-25.75)	2.59 (0.70-9.63)	48.95 (40.51-57.44)	1.63 (1.10-2.41)	0.5039

Of the eight variables examined, community and clinic participants differed significantly on two. In particular, RDIa≥ 5 was significantly more common in community participants who reported that they *never/rarely* wake feeling refreshed compared to clinic patients who reported similarly. Furthermore, community patients who reported observed apnoeas had a higher risk of having an RDIa≥ 5 than participants from the clinical sample who reported observed apnoeas.

6.4 Combined Sample Characteristics

Although demographic and objective sleep variables have been previously presented for community and clinical participants in respective chapters, combined sample characteristics are presented in this chapter to provide a picture of potential biases inherent in the combined data.

Table 6.2 Combined sample, by ethnicity, sex, and age group

	Mean age (yrs)	Age group (yrs)			Total n (%)
		30-39 n (%)	40-49 n (%)	50-60 n (%)	
Māori men	45.04	40 (4.61%)	56 (6.45%)	42 (4.84%)	138 (15.90%)
Māori women	47.05	22 (2.53%)	35 (4.03)	43 (4.95)	100 (11.52%)
Non-Māori men	47.44	94 (10.83%)	142 (16.36)	191 (22.00)	427 (49.19%)
Non-Māori women	47.58	43 (4.95%)	68 (7.83%)	92 (10.60%)	203 (23.39%)
Total	47.04	199 (22.93%)	301 (34.68%)	368 (42.40%)	868 (100%)

The overall mean age of the combined sample was 47 years (SD=8.28, Range=30-62), with a large portion of participants in the 50-60 year age group. Across the four groups, age distributions differed significantly (χ^2=9.53, DF=3, p=0.0230). Amongst men, Māori were on average younger than non-Māori (p=0.0036). No differences were found between Māori and non-Māori women, or between men and women within ethnicity. Non-Māori men comprised the largest portion of the sample, followed by non-Māori women, Māori men and Māori women. The male to female ratio was approximately 2:1 (Table 6.2).

As can be seen in Figure 6.2, a wide-range of OSA severity was displayed. RDI scores ranged from 0 through to 194 events per hour (Community range=0-64, Clinic range=0-194).

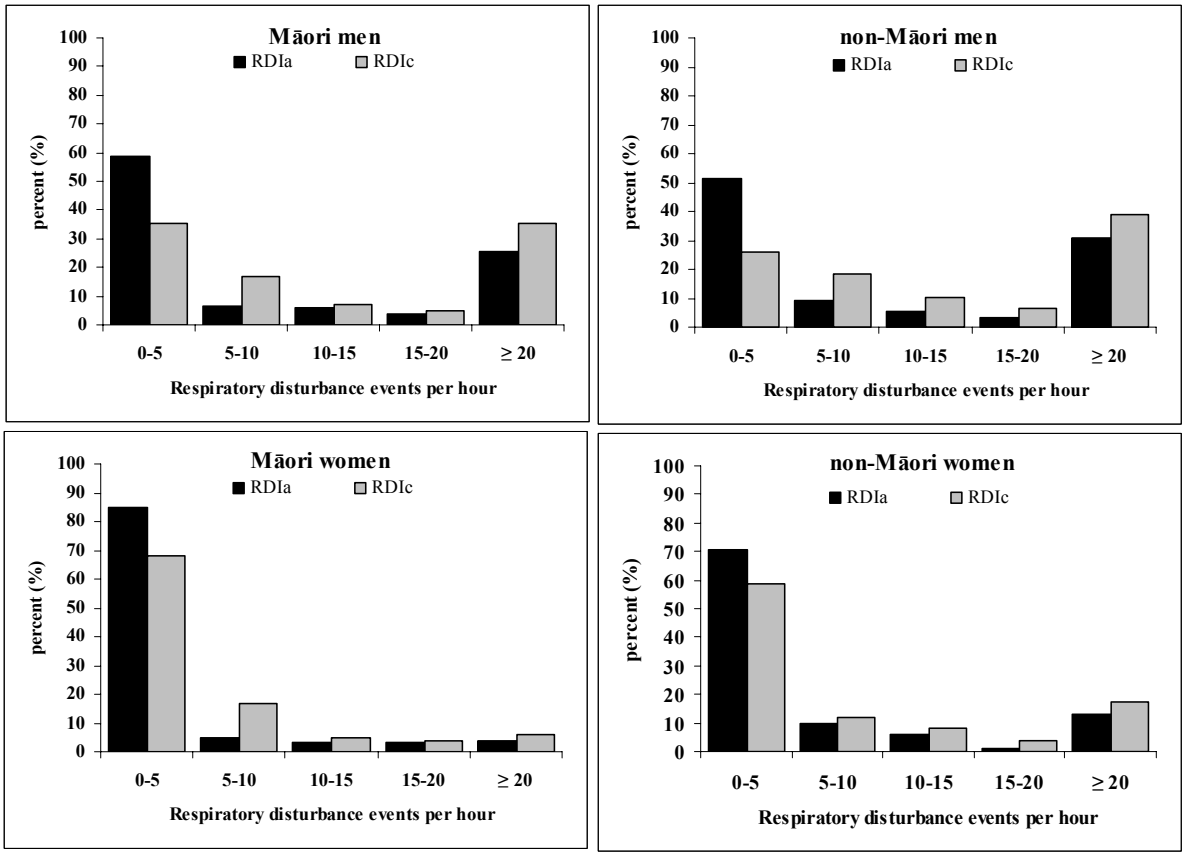


Figure 6.2 RDI distributions, by ethnicity and sex

The graphs clearly demonstrate differences between men and women. However, the differences between Māori and non-Māori are less pronounced than expected, which is probably due to the structure of the sample. The differences between RDIa and RDIc are also less pronounced in this combined dataset compared to the community sample, most likely because RDIc was a more reliable measure in the clinical sample, particularly in terms of the identification of hyponeic events.

For both RDI scores, no significant differences were found in median scores between Māori and non-Māori women (RDIa p=0.1035, RDIc p=0.3035). Amongst men, non-Māori men had significantly higher median RDIa scores than Māori men (p=0.0161). However for RDIc scores, no significant differences were found (p=0.0953). For both Māori and non-Māori, men had significantly higher median RDI scores than women ($p<0.0001$).

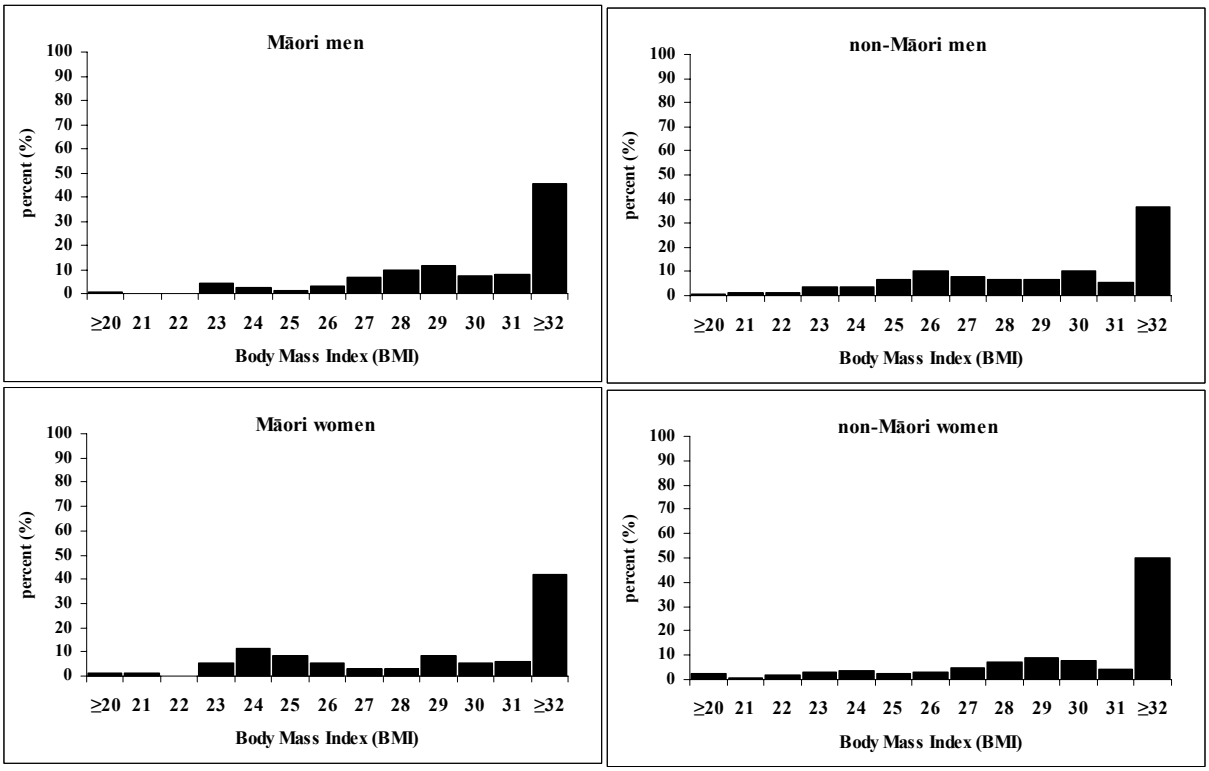


Figure 6.3 BMI distributions, by ethnicity and sex

Amongst men, Māori had higher median BMI than non-Māori (p=0.0006). However no differences were found between Māori and non-Māori women (p=0.6049). Within ethnic groups, Māori men had a higher median BMI than Māori women (p=0.0344). In contrast, no differences were detected between non-Māori men and women (p=0.3677) (Figure 6.3).

6.5 Univariate Predictors of OSA

In order to decide which variables should be considered for multivariate analyses, demographic and questionnaire variables were first tested individually for their ability to predict RDIa at different thresholds. These variables have been described previously in Chapter 3 (Methods). The results of the univariate models for RDIa are presented in Table 6.3. As similar results were found for RDIc, these results are presented in Appendix 12. Of the two measures of socio-economic deprivation, only CSC could be tested in the models, as NZDep96 was not available for clinical participants. However this was not seen as a problem given that the screening tool is intended primarily for use in a primary care setting where NZDep96 information is not readily available.

Table 6.3 Possible univariate predictors of OSA

Variable	Description	RDIa ≥ 5			RDIa ≥ 10			RDIa ≥ 15		
		OR	95% CI	p-value	OR	95% CI	p-value	OR	95% CI	p-value
Ethnicity	Māori vs. non-Māori	0.59	0.43-0.81	0.001	0.65	0.47-0.92	0.0141	0.64	0.45-0.93	0.0182
Sex	men vs. women	2.69	1.97-3.66	<0.0001	3.13	2.21-4.40	<0.0001	3.76	2.54-5.57	<0.0001
Age 1	yearly increments	1.03	1.01-1.05	0.0005	1.02	1.00-1.04	0.0144	1.02	1.01-1.04	0.0149
Age 2	10 year increments	1.31	1.10-1.56	0.0026	1.2	1.00-1.44	NS	1.19	0.98-1.45	NS
CSC eligibility	yes vs. no	2.71	1.93-3.81	<0.0001	2.79	1.98-3.93	<0.0001	2.58	1.82-3.67	<0.0001
BMI 1	Increasing	1.19	1.16-1.23	<0.0001	1.16	1.13-1.19	<0.0001	1.15	1.13-1.18	<0.0001
BMI 2	overweight vs. ideal	2.30	1.42-3.72	0.0008	2.57	1.55-5.30	0.0008	2.94	1.49-5.82	0.0019
	obese vs. ideal	12.51	7.84-19.96	<0.0001	16.00	8.95-28.70	<0.0001	14.43	7.59-27.45	<0.0001
Neck 1	cm increments	1.37	1.31-1.44	<0.0001	1.39	1.32-1.46	<0.0001	1.41	1.33-1.48	<0.0001
Neck 2	> national av. vs. < national av.	7.41	5.27-10.40	<0.0001	7.65	5.17-11.29	<0.0001	6.85	4.50-10.42	<0.0001
ESS 1	>10 vs. ≤10	4.25	3.18-5.68	<0.0001	4.23	3.11-5.75	<0.0001	4.27	3.09-5.92	<0.0001
ESS 2	11-15 vs. ≤10	3.16	2.24-4.46	<0.0001	3.11	2.16-4.47	<0.0001	2.91	1.97-4.30	<0.0001
	16+ vs. ≤10	6.16	4.20-9.02	<0.0001	6.05	4.13-8.86	<0.0001	6.51	4.39-9.65	<0.0001
Snore 1	always vs. never/rarely/often	5.69	4.17-7.77	<0.0001	5.85	4.26-20.02	<0.0001	5.53	3.97-7.71	<0.0001
Snore 2	often/always vs. never/rare	10.82	6.67-17.56	<0.0001	11.2	6.27-20.05	<0.0001	9.97	5.32-18.67	<0.0001
Snore 3	rarely vs. never	0.38	0.13-1.14	NS	0.45	0.12-1.73	NS	0.35	0.08-1.42	NS
	often vs. never	3.08	1.14-8.27	0.02	3.31	0.98-11.21	NS	2.43	0.71-8.27	NS
	always vs. never	9.05	3.57-26.09	<0.0001	11.3	3.35-38.27	<0.0001	8.2	2.43-27.60	0.0067
	don't know vs. never	1.03	0.28-3.30	NS	1.57	0.24-5.65	NS	0.54	0.08-3.49	NS
Observed apnoea	yes vs. no	7.98	5.84-10.92	<0.0001	8.8	6.20-12.48	<0.0001	9.64	6.51-14.26	<0.0001
Wake feeling refreshed 1	never/rarely vs. often/always	1.78	1.35-2.35	<0.0001	1.80	1.34-2.42	<0.0001	1.98	1.44-2.71	<0.0001
Wake feeling refreshed 2	never vs. always	1.39	0.40-4.82	NS	2.07	0.52-8.20	NS	1.65	0.41-6.56	NS
	rarely vs. always	0.95	0.29-3.17	NS	1.47	0.38-5.16	NS	1.09	0.28-4.17	NS
	often vs. always	0.43	0.13-1.43	NS	0.62	0.16-2.41	NS	0.50	0.13-1.92	NS

Table 6.3 Possible univariate predictors of OSA (cont…)

Variable	Description	RDIa ≥ 5			RDIa ≥ 10			RDIa ≥ 15		
		OR	95% CI	p-value	OR	95% CI	p-value	OR	95% CI	p-value
Getting enough sleep 1	never/rarely vs. often/always	1.78	1.35-2.35	<0.0001	1.80	1.34-2.42	<0.0001	1.98	1.44-2.71	<0.0001
Getting enough sleep 2	never vs. always	1.56	0.65-3.79	NS	1.02	0.43-2.41	NS	1.11	0.47-2.63	NS
	rarely vs. always	0.55	0.26-1.17	NS	0.44	0.21-0.93	0.0317	0.43	0.21-0.96	0.0386
	often vs. always	0.33	0.16-0.70	<0.0001	0.25	0.12-0.52	0.0003	0.23	0.11-0.49	0.0003
Asthma	yes vs. no/don't know	1.02	0.70-1.18	NS	0.76	0.50-1.14	NS	0.70	0.45-1.10	NS
Hypertension	yes vs. no/don't know	3.42	2.41-4.86	<0.0001	3.12	2.20-4.42	<0.0001	3.35	2.35-4.79	<0.0001
Heart Trouble	yes vs. no/don't know	4.14	2.53-6.76	<0.0001	3.77	2.37-6.00	<0.0001	3.67	2.31-5.82	<0.0001
Diabetes	yes vs. no/don't know	4.35	2.31-8.19	<0.0001	4.41	2.44-8.07	<0.0001	3.46	1.94-6.17	<0.0001
Stroke	yes vs. no/don't know	1.56	0.45-5.43	NS	2.29	0.67-7.97	NS	2.97	0.85-1.37	NS
Thyroid problem	yes vs. no/don't know	4.35	2.31-8.19	NS	1.53	0.62-8.07	NS	0.51	0.15-1.76	NS
Psychological problem	yes vs. no/don't know	2.33	1.36-4.01	0.0022	2.24	1.31-3.84	0.0032	2.20	1.27-3.80	0.0048
Sleep problem	yes vs. no/don't know	0.99	0.52-1.89	NS	0.93	0.47-1.86	NS	0.82	0.38-1.74	NS
Smoking 1	regular/occasional vs. other	1.15	0.80-1.67	NS	1.16	0.82-1.64	NS	1.13	0.81-1.57	NS
Smoking 2	regular vs. other	1.11	0.75-1.65	NS	1.07	0.73-1.56	NS	1.10	0.77-1.57	NS
Smoking 3	regular vs. non-smoker	1.46	1.00-2.14	NS	1.45	0.96-2.18	NS	1.42	0.92-2.17	NS
	occasional vs. non-smoker	1.60	0.76-3.40	NS	2.07	0.97-4.44	NS	1.63	0.72-3.68	NS
	ex-smoker vs. non-smoker	2.00	1.46-2.75	<0.0001	2.01	1.44-2.80	<0.0001	1.75	1.23-2.48	0.0018
Alcohol 1	exceed rec. limits vs. non-drinkers	1.38	0.93-2.03	NS	1.43	0.96-2.14	NS	1.37	0.89-2.08	NS
Alcohol 2	daily vs. non-drinkers	0.69	0.40-1.18	NS	0.58	0.31-1.06	NS	0.76	0.42-1.40	NS
Alcohol 3	moderate vs. non-drinkers	0.55	0.41-0.73	<0.0001	0.53	0.39-0.71	<0.0001	0.52	0.38-0.71	<0.0001

As expected, most variables were significantly associated with each threshold of RDI. Ethnicity was found to be a significant predictive variable, but results are contrary to those found in the community sample and clinic samples separately, where Māori were more likely to have OSA at a univariate level. The reversal in the direction of the odds ratios, indicating that non-Māori are more likely to have OSA is a result of merging such different samples together. The new distribution of ethnicity and OSA prevalence in the combined sample gives an odds ratio which is very different from those found in each respective sample. For this reason, ethnicity was not considered in the multivariate models. Men were 3-4 times more likely to have OSA than women. Age as a yearly increase (*Age 1*) was a better predictor than age defined in 10 year increments (*Age 2*). Those participants who were eligible for a community services card (CSC) had an increased risk of having OSA.

Both measures of body mass index (BMI) were significant. Similarly for neck circumference, *Neck 1* and *Neck 2* were significantly associated with OSA. Excessive daytime sleepiness (EDS) classified as ESS>10 (*ESS 1*) was a strong explanatory variable. It was also strong when it was classified in 3 categories with ESS≤ 10 as the reference group (*ESS 2*). For self-reported snoring, the two dichotomous classifications (*Snore 1* and *Snore 2*) were significantly associated with OSA. Classified in 4 categories (*Snore 3*), snoring was not consistently associated with OSA. Participants who reported receiving treatment for hypertension, diabetes, stroke and psychological problems were more likely to have OSA than those who were not receiving treatment for these conditions. Conversely, receiving treatment for asthma, stroke or thyroid problems showed no consistent relationship with RDI. Smoking was in no way predictive of OSA, except where ex-smokers were two times more likely than non-smokers to have an RDI≥ 5 (*Smoke 3*). Similarly, alcohol consumption had limited predictive ability, except where moderate drinkers were less likely than non-drinkers to have OSA (*Alcohol 3*). For the general sleep variables, getting enough sleep and waking feeling refreshed had better predictive ability as dichotomous variables than as variables in four categories.

6.6 Multivariate Predictors of OSA

To account for the relative importance of various other factors, combinations of predictive variables were tested collectively. Decisions regarding the combination of variables to be entered in each model were in part informed from the results of the univariate models and multivariate models in community and clinical samples respectively, and also from other similar studies (Flemons et al.1994, Maislin et al. 1995). The overall goal of these analyses was to maintain simplicity and predictive accuracy. For the initial models (full models), a number of variables were simultaneously entered. These are presented in Table 6.4 and Table 6.5.

Table 6.4 Model 1: BMI and other variables (n=829)

Variable	RDIa			RDIc		
	≥ 5	≥ 10	≥ 15	≥ 5	≥ 10	≥ 15
Sex (men vs. women)	**3.34 (2.06-5.41)****	**4.67 (2.74-7.96) ****	**6.16 (3.42-11.09) ****	**4.04 (2.66-6.04) ****	**3.86 (2.49-5.99) ****	**4.41 (2.73-7.14) ****
Age (yearly increase)	**1.06 (1.03-1.08)****	**1.05 (1.01-1.07) ****	**1.05 (1.02-1.08) ****	**1.02 (1.00-1.05)***	**1.04 (1.01-1.06) ****	**1.04 (1.01-1.06) ****
CSC (eligible vs. else)	1.09 (0.66-1.78)	1.23 (0.76-1.98)	1.11 (0.68-1.83)	0.80 (0.46-1.37)	0.91 (0.57-1.47)	0.85 (0.53-1.36)
BMI (continuous)	**1.17 (1.13-1.21)****	**1.15 (1.11-1.19) ****	**1.15 (1.12-1.19) ****	**1.14 (1.10-1.19) ****	**1.14 (1.10-1.18) ****	**1.13 (1.10-1.17) ****
Wake feeling refreshed (never/rarely vs. often/always)	**1.37 (0.81-2.32)**	**1.50 (0.86-2.63)**	**1.03 (0.56-1.88)**	**1.34 (0.82-2.19)**	1.42 (0.86-2.34)	1.55 (0.91-2.65)
Get enough sleep (never/rarely vs. often always)	**0.64 (0.39-1.06)**	**0.65 (0.39-1.08)**	**1.00 (0.58-1.72)**	**0.69 (0.42-1.11)**	**0.51 (0.32-0.83)***	**0.53 (0.32-0.87)***
Epworth Scale (ESS>10 vs. ESS≤ 10)	**1.70 (1.15-2.51)****	**1.68 (1.12-2.51) ****	**1.63 (1.07-2.51) ****	**2.05 (1.37-3.06) ****	**1.65 (1.14-2.40) ****	**1.51 (1.03-2.22) ****
Snore (always vs. often/rarely/never)	**2.73 (1.82-4.08)****	**2.91 (1.94-4.38) ****	**2.85 (1.85-4.38) ****	**2.28 (1.47-3.56) ****	**2.02 (1.37-2.97) ****	**1.98 (1.34-2.91) ****
Observed apnoeas (apnoea vs. not)	**3.88 (2.57-5.86)****	**4.05 (2.60-6.30) ****	**4.25 (2.61-6.91) ****	**3.62 (2.42-5.42) ****	**3.95 (2.70-5.78) ****	**4.83 (3.21-7.25) ****
Hypertension (curr. treatment vs. not)	1.45 (0.87-2.42)	1.40 (0.85-2.30)	1.63 (0.98-2.70)	1.23 (0.70-2.17)	1.19 (0.72-1.94)	1.31 (0.80-2.13)
Heart (curr. treatment vs. not)	1.39 (0.71-2.73)	1.31 (0.68-2.52)	1.32 (0.68-2.56)	0.92 (0.44-1.91)	0.80 (0.42-1.50)	0.74 (0.39-1.40)
Diabetes (curr. treatment vs. not)	0.74 (0.31-1.73)	0.97 (0.44-2.13)	0.78 (0.36-1.71)	1.40 (0.47-4.23)	0.76 (0.34-1.69)	0.88 (0.41-1.90)
Stroke (curr. treatment vs. not)	0.48 (0.10-2.48)	0.91 (0.18-4.66)	1.27 (0.25-6.48)	1.15 (0.14-9.28)	0.70 (0.15-3.20)	1.11 (0.24-5.13)
Psychological (curr. treatment vs. not)	1.76 (0.87-3.57)	1.73 (0.88-3.26)	2.24 (1.09-4.59)	1.11 (0.52-2.40)	1.37 (0.70-2.71)	1.92 (0.97-3.78)

Table 6.5 Model 2: Neck circumference and other variables (n=810)

Variable	RDIa			RDIc		
	≥ 5	≥ 10	≥ 15	≥ 5	≥ 10	≥ 15
Sex (men vs. women)	0.82 (0.51-1.33)	1.21 (0.73-2.03)	1.33 (0.76-2.33)	1.48 (0.94-2.32)	1.20 (0.76-1.88)	1.48 (0.94-2.32)
Age (yearly increase)	**1.05 (1.02-1.08)****	**1.04 (1.02-1.07)****	**1.05 (1.02-1.08)****	1.02 (1.00-1.04)	**1.04 (1.1-1.06)****	**1.02 (1.01-1.04)****
CSC (eligible vs. else)	1.24 (0.76-2.01)	1.33 (0.82-2.15)	1.15 (0.70-1.90)	0.97 (0.57-1.64)	1.01 (0.63-1.63)	0.97 (0.57-1.64)
Neck circumference (continuous)	**1.26 (1.19-1.33)****	**1.24 (1.18-1.31)****	**1.30 (1.23-1.38)****	**1.19 (1.13-1.26)****	**1.22 (1.16-1.29)****	**1.19 (1.13-1.26)****
Wake feeling refreshed (never/rarely vs. often/always)	1.62 (0.96-2.73)	**1.82 (1.04-3.18)***	1.21 (0.66-2.21)	1.50 (0.92-2.42)	**1.66 (1.01-2.73)***	**1.85 (1.08-3.16)***
Get enough sleep (never/rarely vs. often always)	0.61 (0.37-1.01)	0.61 (2.50-6.06)	0.94 (0.55-1.63)	0.72 (0.44-1.16)	**0.52 (0.32-0.84)****	**0.52 (0.31-0.86)***
Epworth Scale (ESS>10 vs. ESS ≤10)	**1.65 (1.12-2.41)****	**1.62 (1.09-2.41)***	**1.55 (1.01-2.37)***	**1.99 (1.34-2.97)****	**1.63 (1.13-2.36)****	**1.99 (1.34-2.97)****
Snore (always vs. often/rarely/never)	**2.42 (1.63-3.60)****	**2.59 (1.73-3.88)****	**2.54 (1.65-3.91)****	**2.09 (1.35-3.24)****	**1.86 (1.26-2.73)****	**2.09 (1.65-3.24)****
Observed apnoeas (apnoea vs. not)	**3.46 (2.33-5.15)****	**3.62 (2.35-5.57)****	**3.76 (2.32-6.09)****	**3.36 (2.26-4.97)****	**3.63 (2.50-5.28)****	**3.36 (2.26-4.97)****
Hypertension (curr. treatment vs. not)	1.47 (0.86-2.44)	1.35 (0.82-2.22)	1.43 (0.86-2.39)	1.42 (0.81-2.50)	1.16 (0.71-1.91)	1.42 (0.81-2.50)
Heart (curr. treatment vs. not)	1.34 (0.69-2.61)	1.26 (0.66-2.42)	1.25 (0.64-2.44)	0.89 (0.43-1.84)	0.78 (0.41-1.47)	0.89 (0.43-1.84)
Diabetes (curr. treatment vs. not)	0.80 (0.35-1.85)	0.98 (0.45-2.15)	0.74 (0.34-1.62)	1.55 (0.52-4.63)	0.79 (0.36-1.75)	1.55 (0.52-4.63)
Stroke (curr. treatment vs. not)	0.36 (0.07-1.70)	0.69 (0.14-3.34)	0.98 (0.20-4.84)	0.75 (0.11-5.06)	0.53 (0.12-2.34)	0.75 (0.11-5.06)
Psychological (curr. treatment vs. not)	1.85 (0.92-3.73)	2.09 (1.05-4.15)	2.15 (0.99-4.55)	1.29 (0.58-2.83)	1.44 (0.73-2.86)	1.29 (0.58-2.83)

Model 1 and Model 2 displayed similar predictive variables across each RDI threshold, however in Model 2, after controlling for neck circumference, sex was no longer a predictive variable. Across both models, age was a consistent predictor. Being eligible for a community services card (CSC) was no longer a significant predictor.

Neck circumference and body mass index (BMI) were consistent predictors in their respective models. The general sleep variables, *wake feeling refreshed* and *getting enough sleep* had little predictive value. In the instances where *getting enough sleep* were significant, the results were counterintuitive (getting enough sleep more often was negatively associated with OSA). The reporting of observed apnoeas had good predictive power. Of the co-morbid medical conditions, receiving treatment for hypertension, heart condition, diabetes, stroke or psychological problems were not independent predictors of OSA. When tested against a constant-only model, each model was statistically reliable (Likelihood Ratio Test: Model 1, χ^2=441.95 DF=14, p=<0.0001 - Model 2, χ^2=425.55, DF=14, p=<0.0001).

Interactions

The full models were also tested for interactions between the predictive variables. In particular, differences in the relationships of possible predictive variables and outcome variables between men and women were tested. The only significant interaction found was between sex and body mass index (χ^2=6.49, DF=1, p=0.0108) indicating that the slope for BMI was slightly different within each sex. To further investigate this interaction, the association between BMI and having OSA was examined separately for men and women. No differences were found for increasing BMI (Men vs. women, OR=0.25, 95% CI 0.03-2.32, p=0.22137) or decreasing BMI (Men vs. women, OR=0.23, 95% CI 0.02-2.26, p=0.20586). There was however a differential effect of increasing BMI between men (Increasing BMI vs. decreasing BMI, OR=1.2, 95% CI 1.15-1.24, p<0.0001) and women (Increasing BMI vs. decreasing BMI, OR=1.1, 95% CI 1.04-1.15, p<0.0001), where increasing BMI was a slightly stronger predictor in men than in women.

Reduced models

Following the full models, reduced models were fitted comprising different combinations of variables. The statistics for each model are summarised for each RDIa threshold in Table 6.6 to Table 6.8 along with the respective full models (as presented above). Since there were few differences in predictive variables between models for RDIa and RDIc, the model statistics for RDIc have been appended (Appendix 12).

Table 6.6 RDIa≥ 5: Summary of fitted models

Model	Model predictors*	Model Statistics§†									
		-2 log L	Likelihood	Wald	DF	H&L	Pearson	Deviance	Con	Dis	AUC
1	sex, age1, CSC, BMI1, ESS1, snore1, apnoea, refreshed1, enough1, hypertension, heart, diabetes stroke, psych.	673.17	441.95	214.87	14	5.41	1.03	0.83	88.90	11.00	0.89
1a	sex, age1, BMI1, ESS1, snore1, apnoea	685.22	430.96	213.03	6	7.34	1.08	0.83	88.50	11.40	0.89
1b	sex, age1, BMI2, ESS1, snore1, apnoea	740.02	382.88	218.53	6	7.74	1.14	0.90	86.90	13.00	0.87
1c	sex, age1, BMI1, ESS1, snore1, apnoea, hypertension	682.21	433.96	215.78	7	7.91	1.06	0.83	88.60	11.30	0.89
1d	sex, age1, BMI1, BMI1xSex, ESS1, snore1, apnoea	677.71	438.47	218.24	7	14.64	0.94	0.83	88.80	11.10	0.89
2	sex, age1, CSC, Neck1, ESS1, snore1, apnoea, refreshed1, enough1, hypertension, heart, diabetes stroke, psych.	671.56	416.89	212.75	14	3.45	0.94	0.85	88.50	11.40	0.89
2a	age, neck1, ESS1, snore1, breath	688.03	401.42	208.20	5	11.96	0.99	0.86	87.80	12.10	0.88
2b	age, neck2, ESS1, snore1, breath	758.32	312.53	191.23	5	7.00	0.92	0.97	84.10	15.70	0.84
2c	sex, age1, neck1, ESS1, snore1, apnoea, hypertension	685.69	403.78	210.44	6	9.33	0.97	0.85	87.90	12.00	0.88

*Predictors in grey indicate non-significance ($p>0.05$)

§ Model statistics in grey indicate the tests did not achieve goodness of fit significance

† -Model statistics names in full: -2 Log L model fit statistics, Likelihood ratio test, Wald test, Hosmer-Lemeshow test, Pearson Chi-Square and Deviance Goodness of fit statistics, % Concordant, % Discordant, Area Under the Curve (AUC)

Table 6.7 RDIa≥ 10: Summary of fitted models

Model	Model predictors*	Model Statistics§†									
		-2 log L	Likelihood	Wald	DF	H&L	Pearson	Deviance	% Con	% Dis	AUC
1	sex, age1, CSC, BMI1, ESS1, snore1, apnoea, refreshed1, enough1, hypertension, heart, diabetes stroke, psych.	633.52	396.96	191.13	14	4.34	0.96	0.78	88.90	10.90	0.89
1a	sex, age1, BMI1, ESS1, snore1, apnoea	646.53	384.71	190.18	6	12.43	0.94	0.79	88.50	11.40	0.89
1b	sex, age1, BMI2, ESS1, snore1, apnoea	670.09	368.04	199.48	6	6.04	1.00	0.81	87.70	12.20	0.88
1c	sex, age1, BMI1, ESS1, snore1, apnoea, hypertension	643.74	387.76	192.48	7	5.31	0.78	0.93	88.60	11.30	0.89
1d	sex, age1, BMI1, BMI1xSex, ESS1, snore1, apnoea	627.89	403.34	198.86	7	12.28	1.19	0.76	89.30	10.60	0.89
2	sex, age1, CSC, Neck1, ESS1, snore1, apnoea, refreshed1, enough1, hypertension, heart, diabetes stroke, psych.	613.30	390.33	189.46	14	3.85	0.90	0.77	88.90	11.00	0.89
2a	age, neck1, ESS1, snore1, breath	625.07	379.31	189.13	5	13.50	0.89	0.78	88.40	11.50	0.89
2b	age, neck2, ESS1, snore1, breath	719.46	279.95	170.77	5	5.75	0.90	0.92	84.00	15.90	0.84
2c	sex, age1, BMI1, ESS1, snore1, apnoea, hypertension	624.39	379.99	190.37	6	13.54	0.89	0.99			

*Predictors in grey indicate non-significance (p>0.05)

§ Model statistics in grey indicate the tests did not achieve goodness of fit significance

† -Model statistics names in full: -2 Log L model fit statistics, Likelihood ratio test, Wald test, Hosmer-Lemeshow test, Pearson Chi-Square and Deviance Goodness of fit statistics, % Concordant, % Discordant, Area Under the Curve (AUC)

Table 6.8 RDIa≥ 15: Summary of fitted models

Model	Model predictors*	Model Statistics§†									
		-2 log L	Likelihood	Wald	DF	H&L	Pearson	Deviance	Con	Dis	AUC
1	sex, age1, CSC, BMI1, ESS1, snore1, apnoea, refreshed1, enough1, hypertension, heart, diabetes stroke, psych.	585.71	366.85	172.57	14	3.65	0.88	0.72	89.30	10.60	0.89
1a	sex, age1, BMI1, ESS1, snore1, apnoea	598.24	354.93	171.19	6	4.32	0.89	0.73	89.00	11.00	0.89
1b	sex, age1, BMI2, ESS1, snore1, apnoea	646.41	313.93	175.58	6	7.49	0.99	0.78	86.80	13.00	0.87
1c	sex, age1, BMI1, ESS1, snore1, apnoea, hypertension	592.92	360.25	174.02	7	2.05	0.87	0.72	89.00	10.90	0.89
1d	sex, age1, BMI1, BMI1xSex, ESS1, snore1, apnoea	590.92	362.25	178.02	7	10.10	0.93	0.72	89.20	10.70	0.89
2	sex, age1, CSC, Neck1, ESS1, snore1, apnoea, refreshed1, enough1, hypertension, heart, diabetes stroke, psych.	568.05	360.54	171.94	14	4.56	0.86	0.72	89.10	10.80	0.89
2a	age, neck1, ESS1, snore1, breath	577.80	351.38	171.50	5	5.22	0.86	0.72	88.70	11.20	0.89
2b	age, neck2, ESS1, snore1, breath	687.00	242.47	149.30	5	5.07	0.89	0.88	83.30	16.60	0.83
2c	sex, age1, BMI1, ESS1, snore1, apnoea, hypertension	576.18	353.01	173.89	6	3.00	0.86	0.72	88.70	11.10	0.89

*Predictors in grey indicate non-significance (p>0.05)

§ Model statistics in grey indicate the tests did not achieve goodness of fit significance

† -Model statistics names in full: -2 Log L model fit statistics, Likelihood ratio test, Wald test, Hosmer-Lemeshow test, Pearson Chi-Square and Deviance Goodness of fit statistics, % Concordant, % Discordant, Area Under the Curve (AUC)

Overall, as indicated by the Likelihood ratio tests and Wald test statistics, each model performed significantly better than respective constant only models. Similarly model fit statistics indicated that the majority of the models at each threshold satisfactorily fitted the data. These findings would tend to indicate that each model was able to reliably distinguish between participants with and without OSA. Model discrimination was examined by the area under the curve (AUC). All AUCs were in an acceptable range, with Model 2c yielding the lowest AUC.

Models 1a and 2a

The reduced nested models, which omitted insignificant predictors from the full models, were not reliably different from the full models (Model 1 and 2 respectively). Similarly the predictive power was no better. However, given the importance of simplicity for the proposed use of the predictive tool, these models were still seen as better than the full models. For RDIa≥ 15, the overall significance of Model 1a by the likelihood ratio test was 354.93 ($p < 0.0001$) with 6 DF, with 89% concordant pairs and 11% discordant pairs. The Hosmer-Lemeshow goodness of fit test was 4.32 ($p = 0.73$) with 8 DF. For Model 1a, the AUC was 0.89 (95% CI 0.86-0.91, $p<0.0001$), which was the same as Model 2a (AUC=0.89, 95% CI 0.85-0.91, $p<0.0001$). These measurements would tend to indicate that these models provide "good" diagnostic tests. There were no significant differences between AUC's of the two models. Overall, these models satisfied model fit statistics and provided good discrimination between those with OSA and those without OSA.

Model 1b and 2b

The next set of models tested BMI and neck circumference respectively as categorical variables rather than continuous variables. These models did not improve on the previous models and were slightly inferior in terms of model fit statistics and discrimination.

Model 1c and 2c

In these particular models, based on previous predictive tools (Flemons et al. 1994), hypertension was included in the models even though it was not significant in the full models. For Model 1c, hypertension was only significant at the RDIa≥ 15 threshold, whereas it was not significant at any threshold in Model 2c.

Model 1d

In this model, the interaction term between sex and BMI was included. With the addition of this interaction, sex was no longer a reliable predictor. This would tend to indicate that the sex variable to some degree accounted for the different affect of BMI within sex. Therefore the addition of this interaction only complicated the model without improving its accuracy.

Best models

Based on the results presented above, taking into consideration the significant p-value approach, model fit statistics, predictive power and simplicity, it was decided that for the purpose of this thesis, Model 1a and 2a were the most parsimonious and efficient models for the prediction of OSA. These two models are therefore evaluated in the remainder of this chapter. Pre-test probabilities of disease are important when evaluating the utility of the screening tool in different populations, these are presented in Table 6.9 and Table 6.10.

Table 6.9 Model 1a: Pre-test probability

Model 1a	RDIa						RDIc					
	≥ 5		≥ 10		≥ 15		≥ 5		≥ 10		≥ 15	
	n	%	n	%	n	%	n	%	n	%	n	%
Total (n=829)	332	40.05	260	31.36	217	26.18	501	60.43	368	44.39	297	35.83
Māori	71	31.14	57	25.00	47	20.61	117	51.32	79	34.65	65	28.51
non-Māori	261	43.43	203	33.78	170	28.29	384	63.89	289	48.09	232	38.60
Men	260	47.27	212	38.55	182	33.09	393	71.45	296	53.82	245	44.55
Women	72	25.81	48	17.20	35	12.54	108	38.71	72	25.81	52	18.64
Māori men	57	41.61	48	35.04	40	29.20	88	64.23	65	47.45	55	40.15
non-Māori men	203	49.15	164	39.71	142	34.38	305	73.85	231	55.93	190	46.00
Māori women	14	15.38	9	9.89	7	7.69	29	31.87	14	15.38	10	10.99
non-Māori women	58	30.85	39	20.74	28	14.89	79	42.02	58	30.85	42	22.34

Table 6.10 Model 2a: Pre-test probability

Model 2a	RDIa						RDIc					
	≥ 5		≥ 10		≥ 15		≥ 5		≥ 10		≥ 15	
	n	%	n	%	n	%	n	%	n	%	n	%
Total (n=810)	323	39.88	252	31.11	211	26.05	490	60.62	361	44.7	290	35.93
Māori	70	30.84	57	25.11	47	20.70	116	51.10	78	34.36	65	28.63
non-Māori	253	43.40	195	33.45	164	28.13	374	64.15	283	48.54	255	43.74
Men	255	47.57	207	38.62	178	33.21	385	71.83	293	54.66	241	44.96
Women	68	24.82	45	16.42	33	12.04	105	38.32	68	24.82	49	17.88
Māori men	57	41.91	48	35.29	40	29.41	88	64.71	65	47.79	55	40.44
non-Māori men	198	49.50	159	39.75	138	34.50	297	74.25	228	57.00	186	46.50
Māori women	13	14.29	9	9.89	7	7.69	28	30.77	13	14.29	10	10.99
non-Māori women	55	30.05	36	19.67	26	14.21	77	42.08	55	30.05	39	21.31

6.7 Estimated Probability of OSA

Parameters for Model 1a and 2a are presented in Table 6.11 and Table 6.12 for RDIa ≥ 15 only. This threshold is a commonly used to indicate moderate OSA or sleep disordered breathing (SDB). Model parameters for RDIa ≥ 5 and 10 are presented in Appendix 12. Figure 6.4 illustrates how the predicted probabilities are estimated using the model parameters presented below in a mathematic equation.

Table 6.11 Model 1a (RDIa≥ 15): Model parameters

Explanatory variable	DF	Estimate (β)	Standard Error	Chi-square	p-value
Intercept	1	-11.36	1.0519	111.574	<0.0001
Sex	1	1.7292	0.2919	35.0929	<0.0001
Age	1	0.0509	0.0131	15.0047	0.0001
Body mass index (BMI)	1	0.1469	0.0159	85.3918	<0.0001
Observed apnoea	1	1.4861	0.2443	37.004	<0.0001
Excessive daytime sleepiness	1	0.5356	0.2157	6.167	0.013
Habitual Snoring	1	1.0188	0.2142	22.6305	<0.0001

Table 6.12 Model 2a (RDIa≥ 15): Model parameters

Explanatory variable	DF	Estimate (β)	Standard Error	Chi-square	p-value
Intercept	1	-16.47	1.4736	124.959	<0.0001
Age	1	0.0408	0.0133	9.4566	0.0021
Neck circumference	1	0.2853	0.0287	98.5707	<0.0001
Observed apnoea	1	1.3946	0.243	32.9427	<0.0001
Excessive daytime sleepiness	1	0.4977	0.2183	5.1996	0.0226
Habitual Snoring	1	0.8261	0.2184	14.3114	0.0002

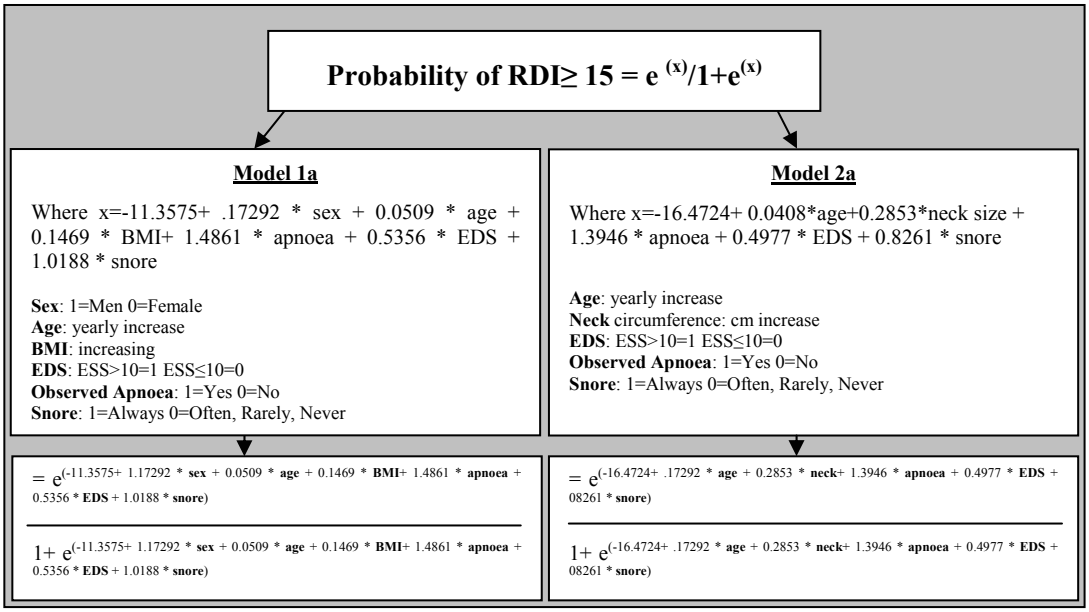


Figure 6.4 Diagram of the application of the probability equation to each model

6.8 Evaluation of the Prediction Models

To evaluate the performance of each model, classification tables are presented at 0.05 increments of the calculated probability (Table 6.13 and Table 6.14 respectively).

Table 6.13 Classification table for Model 1a (BMI) for estimating the probability of an individual having an RDI≥ 15

Prob level	Correct		Incorrect		%				
	Event	non-Event	Event	Non-Event	Correct	Sensitivity	Specificity	False positive	False negative
0.00	217.00	0.00	612.00	0.00	26.20	100.00	0.00	73.80	.
0.05	214.00	269.00	343.00	3.00	58.30	98.60	44.00	61.60	1.10
0.10	205.00	359.00	253.00	12.00	68.00	94.50	58.70	55.20	3.20
0.15	196.00	402.00	210.00	21.00	72.10	90.30	65.70	51.70	5.00
0.20	190.00	443.00	169.00	27.00	76.40	87.60	72.40	47.10	5.70
0.25	183.00	476.00	136.00	34.00	79.50	84.30	77.80	42.60	6.70
0.30	170.00	495.00	117.00	47.00	80.20	78.30	80.90	40.80	8.70
0.35	163.00	517.00	95.00	54.00	82.00	75.10	84.50	36.80	9.50
0.40	156.00	530.00	82.00	61.00	82.80	71.90	86.60	34.50	10.30
0.45	139.00	546.00	66.00	78.00	82.60	64.10	89.20	32.20	12.50
0.50	126.00	556.00	56.00	91.00	82.30	58.10	90.80	30.80	14.10
0.55	111.00	567.00	45.00	106.00	81.80	51.20	92.60	28.80	15.80
0.60	102.00	576.00	36.00	115.00	81.80	47.00	94.10	26.10	16.60
0.65	87.00	588.00	24.00	130.00	81.40	40.10	96.10	21.60	18.10
0.70	74.00	598.00	14.00	143.00	81.10	34.10	97.70	15.90	19.30
0.75	59.00	602.00	10.00	158.00	79.70	27.20	98.40	14.50	20.80
0.80	50.00	603.00	9.00	167.00	78.80	23.00	98.50	15.30	21.70
0.85	37.00	606.00	6.00	180.00	77.60	17.10	99.00	14.00	22.90
0.90	20.00	608.00	4.00	197.00	75.80	9.20	99.30	16.70	24.50
0.95	10.00	611.00	1.00	207.00	74.90	4.60	99.80	9.10	25.30
1.00	0.00	612.00	0.00	217.00	73.80	0.00	100.00	.	26.20

Table 6.14 Classification table for Model 2a (Neck) equation for estimating the probability of an RDI≥ 15

Prob level	Correct		Incorrect		%				
	Event	non-Event	Event	Non-Event	Correct	Sensitivity	Specificity	False positive	False negative
0.00	211	0	599	0	26.00	100.00	0.00	74.00	.
0.05	207	263	336	4	58.00	98.10	43.90	61.90	1.50
0.10	197	358	241	14	68.50	93.40	59.80	55.00	3.80
0.15	186	403	196	25	72.70	88.20	67.30	51.30	5.80
0.20	181	434	165	30	75.90	85.80	72.50	47.70	6.50
0.25	177	462	137	34	78.90	83.90	77.10	43.60	6.90
0.30	169	488	111	42	81.10	80.10	81.50	39.60	7.90
0.35	158	503	96	53	81.60	74.90	84.00	37.80	9.50
0.40	144	520	79	67	82.00	68.20	86.80	35.40	11.40
0.45	131	531	68	80	81.70	62.10	88.60	34.20	13.10
0.50	122	540	59	89	81.70	57.80	90.20	32.60	14.10
0.55	111	554	45	100	82.10	52.60	92.50	28.80	15.30
0.60	98	566	33	113	82.00	46.40	94.50	25.20	16.60
0.65	87	575	24	124	81.70	41.20	96.00	21.60	17.70
0.70	75	584	15	136	81.40	35.50	97.50	16.70	18.90
0.75	65	589	10	146	80.70	30.80	98.30	13.30	19.90
0.80	53	596	3	158	80.10	25.10	99.50	5.40	21.00
0.85	41	598	1	170	78.90	19.40	99.80	2.40	22.00
0.90	24	598	1	187	76.80	11.40	99.80	4.00	23.80
0.95	7	599	0	204	74.80	3.30	100.00	0.00	25.40
1.00	0	599	0	211	74.00	0.00	100.00	.	26.00

Both classification tables present very similar summaries, which is not surprising given the similarity of variables in the two models. The tables illustrate how the selection of different probability cut-off points affects the accuracy of each model. A move towards a less rigorous criterion of positivity improves the tests sensitivity, increasing the number of participants with OSA who are correctly identified, while reducing specificity, thus increasing the number of non-OSA participants who test positive.

6.8.1 Optimal cut-off point

As results of the mathematical models are expressed in terms of the probability of a particular outcome, the probability cut-off chosen for assignment of OSA (with/without) is critical in evaluating the success of each model.

The decision regarding the optimal cut-off point took into account the relative importance of identifying people with OSA and excluding those without OSA. Sensitivity and specificity plots were examined (Figure 6.5), along with ROC curves (Figure 6.6), and likelihood ratios (Table 6.15 and Table 6.16). However it should be noted that the choice of the cut-off point can be varied according to the intended use of the information.

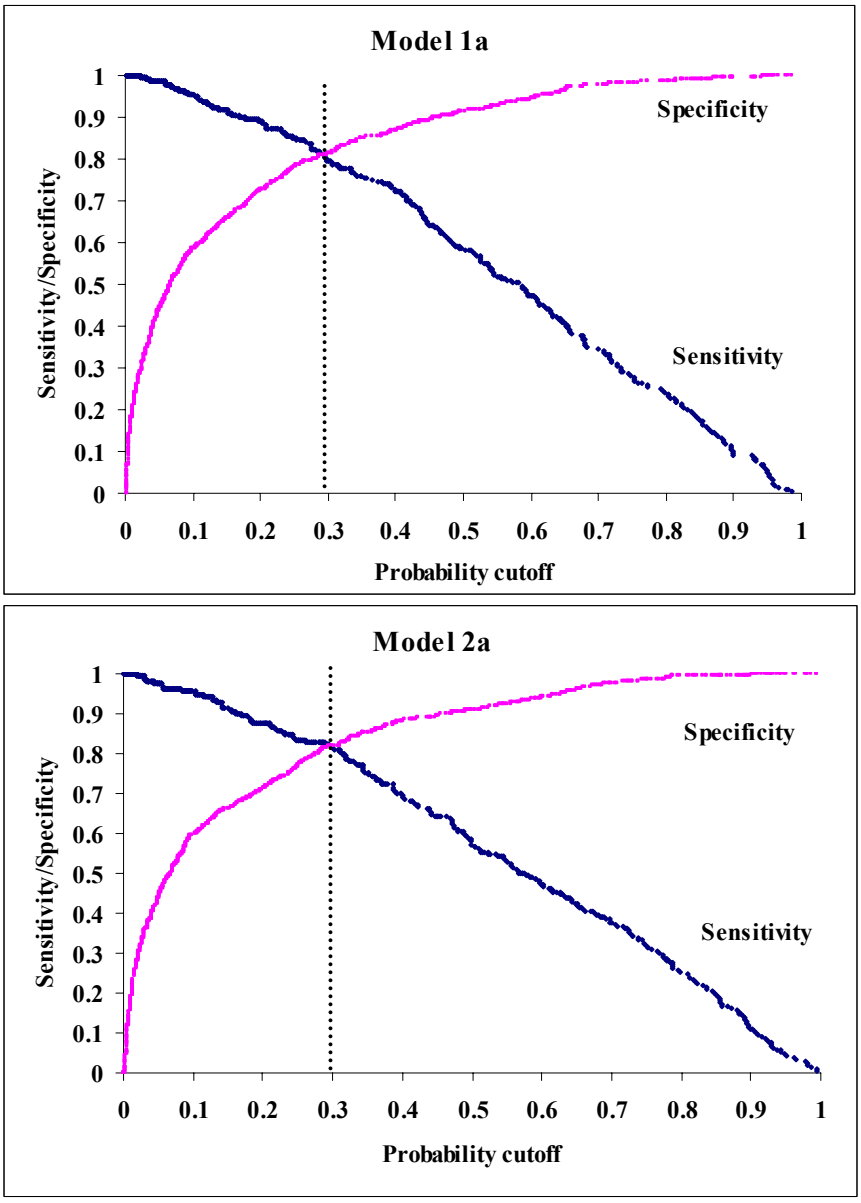
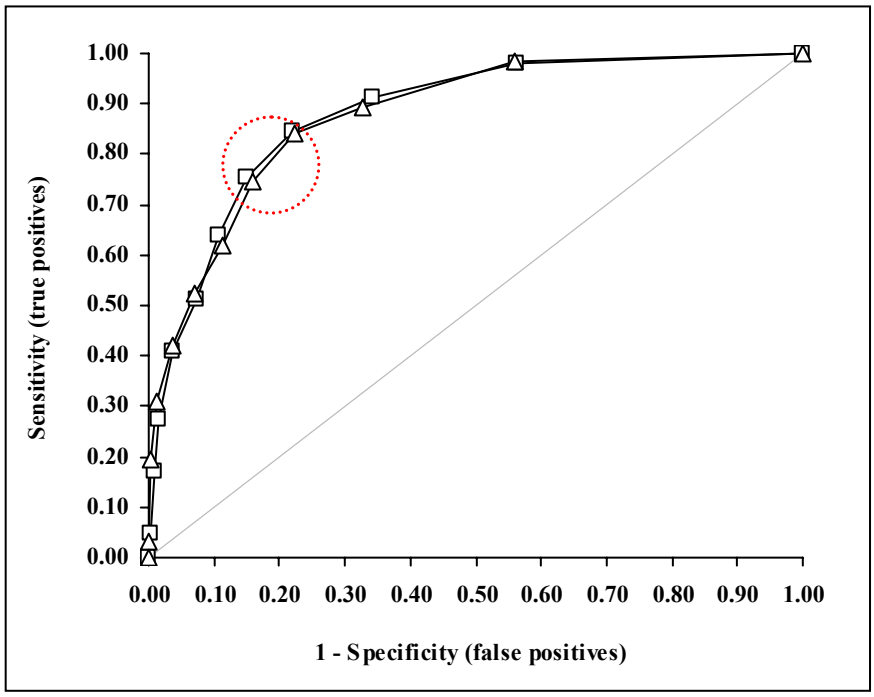


Figure 6.5 Model 1a and 2a: Plot of sensitivity and specificity versus all possible cut-points

For both models, sensitivity and specificity curves crossed at the probability cut-point of 0.30 indicating that sensitivity and specificity are maximised at this particular cut-point.



□=Model 1a Δ=Model 2a

Figure 6.6 ROC curves for Model 1a and 2a

For ROC curves, generally the best cut-point is at or near the shoulder of the ROC curve where substantial gains can be made in sensitivity with only moderate reductions in specificity. For these models, the optimal cut-points fall approximately between the calculated probabilities of 0.30-0.40 (as indicated in the graph with the dashed circle).

Table 6.15 Likelihood ratios for Model 1a

Predicted Probability	OSA (RDI≥ 15)		no-OSA (RDI<15)		Likelihood Ratio
	No. Patients	%	No. Patients	%	
0.00-0.10	12	0.06	359	0.59	0.09
0.10-0.20	15	0.07	84	0.14	0.50
0.20-0.30	20	0.09	52	0.09	1.08
0.30-0.40	14	0.06	35	0.06	1.13
0.40-0.50	30	0.14	26	0.04	3.25
0.50-0.60	24	0.11	20	0.03	3.38
0.60-0.70	28	0.13	22	0.04	3.58
0.70-0.80	24	0.11	5	0.01	13.52
0.80-0.90	30	0.14	5	0.01	16.89
0.90-1.00	20	0.09	4	0.01	14.08
Total	217		612		

Table 6.16 Likelihood ratios for Model 2b

Predicted Probability	OSA (RDI≥ 15)		no- OSA (RDI<15)		Likelihood Ratio
	No. Patients	%	No. Patients	%	
0.00-0.10	14	0.07	358	0.60	0.11
0.10-0.20	16	0.08	76	0.13	0.60
0.20-0.30	12	0.06	54	0.09	0.63
0.30-0.40	25	0.12	32	0.05	2.22
0.40-0.50	22	0.10	20	0.03	3.12
0.50-0.60	24	0.11	26	0.04	2.62
0.60-0.70	23	0.11	18	0.03	3.63
0.70-0.80	22	0.10	12	0.02	5.20
0.80-0.90	29	0.14	2	0.00	41.16
0.90-1.00	24	0.11	1	0.00	68.13
Total	211		599		

For Model 1a, the likelihood ratio increased significantly when it reached a probability of 0.40, whereas for Model 2a the likelihood increased significantly at a probability cut-off point of 0.30, whereby participants whose calculated probability scores were between 0.30-0.40 were twice as likely as those with lower probabilities to have an RDI ≥ 15. The results of these analyses tend to indicate the optimal cut-points of these models are somewhere between 0.30-0.40. Based on the likelihood ratio results, it was decided that the optimal cut-point for Model 1a would be 0.40, and the optimal cut-point for Model 2a would be 0.30.

Using these cut-points to indicate a negative and positive screen, 2x2 contingency tables were constructed for each model and various summary statistics were calculated (Table 6.17 through to Table 6.20).

Table 6.17 Model 1a: 2x2

Model 1a	Disease RDI ≥ 15	Not disease RDI < 15
Screen positive (Prob ≥ 0.40)	A 156	B 81
Screen negative (Prob <0.40)	C 61	D 531

Table 6.18 Model 1a: diagnostic summary

Statistic	Equation	Proportion	Confidence Intervals	
		Estimate	Lower	Upper
Sensitivity	a/(a+c)	0.72	0.66	0.78
Specificity	d/(b+d)	0.87	0.84	0.89
Likelihood ratio +	a/(a+c)/(b/b+d))	5.43	4.36	6.76
Likelihood ratio -	c/(a+c)/(d/b+d))	0.32	0.26	0.40
False positive rate	b/(b+d)	0.13	0.11	0.16
False negative rate	c/(a+c)	0.28	0.22	0.32
Prob of disease	(a+c)/(a+b+c+d)	0.26	0.23	0.29
PPV	a/(a+b)	0.66	0.60	0.72
p(pos test wrong)	b/(a+b)	0.34	0.28	0.40
NPV	d/(c+d)	0.90	0.87	0.92
p(neg test wrong)	c/(c+d)	0.10	0.08	0.13
p(test positive)	(a+b)/(a+b+c+d)	0.29	0.26	0.32
p(test negative)	(c+d)/(a+b+c+d)	0.71	0.68	0.74
Overall accuracy**	(a+d)/(a+b+c+d)	0.83	0.80	0.85

Model 1a correctly classified 82.50% of participants, with a sensitivity of 71.90%, specificity of 86.60%, positive predictive value (PPV) of 65.55% and a negative predictive value (NPV) of 89.68%.

Table 6.19 Model 2a: 2x2

Model 1a	Disease RDI ≥ 15	Not disease RDI < 15
Screen positive (Prob ≥ 0.30)	A 169	B 112
Screen negative (Prob <0.30)	C 42	D 487

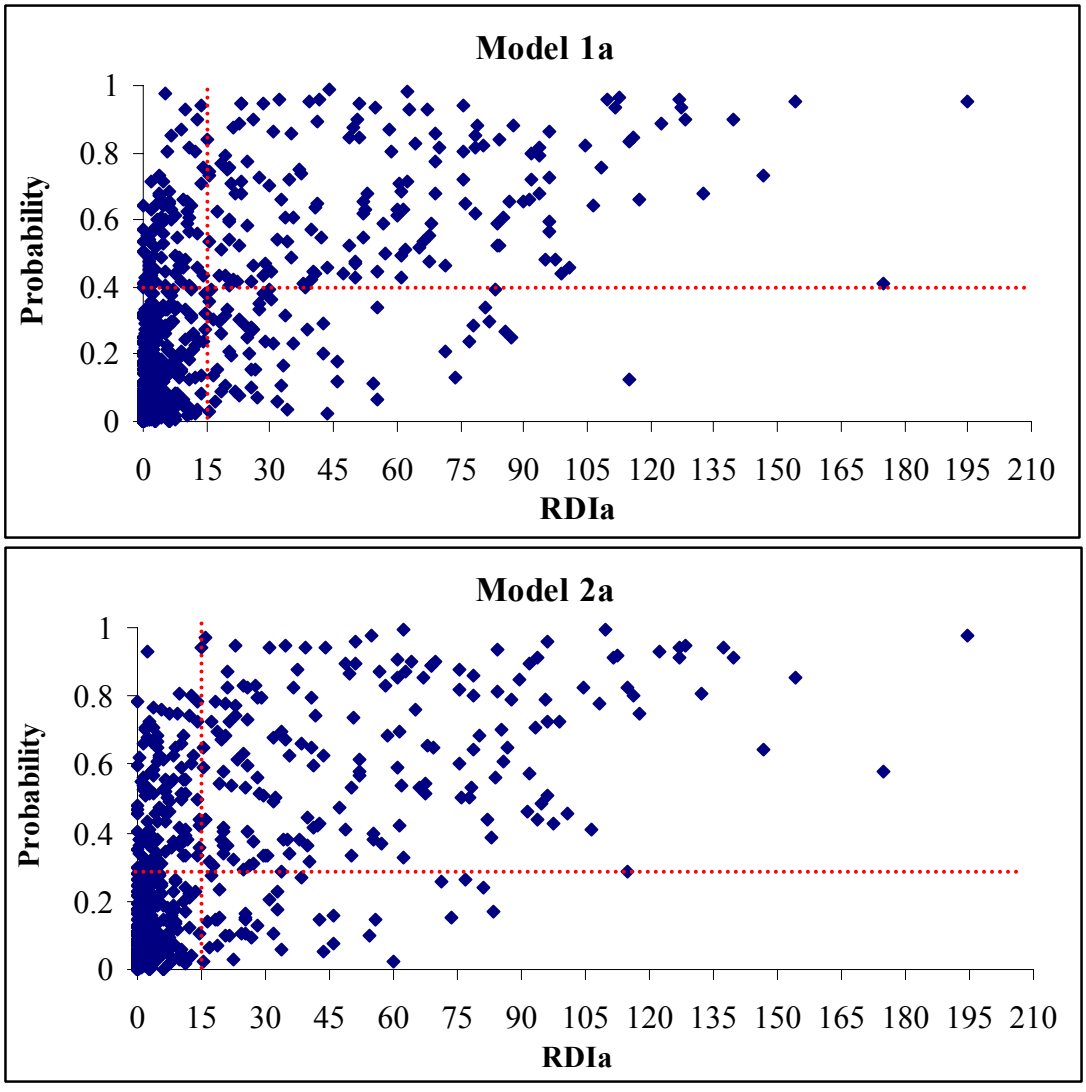
Table 6.20 Model 2a: diagnostic summary

Statistic	Equation	Proportion	Confidence Interval	
		Estimate	Lower	Upper
Sensitivity	a/(a+c)	0.80	0.75	0.85
Specificity	d/(b+d)	0.81	0.78	0.85
Likelihood ratio +	a/(a+c)/(b/b+d))	4.32	3.61	5.18
Likelihood ratio -	c/(a+c)/(d/b+d))	0.24	0.19	0.32
False positive rate	b/(b+d)	0.19	0.15	0.22
False negative rate	c/(a+c)	0.20	0.15	0.23
Prob of disease	(a+c)/(a+b+c+d)	0.26	0.23	0.29
PPV	a/(a+b)	0.60	0.55	0.66
p(pos test wrong)	b/(a+b)	0.40	0.34	0.45
NPV	d/(c+d)	0.92	0.90	0.94
p(neg test wrong)	c/(c+d)	0.08	0.06	0.10
p(test positive)	(a+b)/(a+b+c+d)	0.35	0.31	0.38
p(test negative)	(c+d)/(a+b+c+d)	0.65	0.62	0.69
Overall accuracy**	(a+d)/(a+b+c+d)	0.81	0.78	0.84

Similarly, Model 2a correctly classified 81.10%, with a sensitivity of 80.10%, specificity of 81.50%, PPV of 60.36% and a NPV of 92.06%.

6.8.2 False positives and false negatives

To further explore the performance of each model based on the optimal cut-points (Model 1a=0.40 & Model 2a=0.30) decided above, the characteristics of false positives and negatives were examined for each model. Figure 6.7 presents RDIa distributions in conjunction with respective predicted probabilities for participants. Table 6.21 provides a summary of RDIa for misclassified results, and Figure 6.8 illustrates the distribution of misclassified results amongst Māori and non-Māori, men and women.



The red dashed lines represent RDIa threshold of ≥ 15 and optimal probability cut-point for each model

False positives	True positives
True negatives	False negatives

Figure 6.7 Model 1a and 2a: RDI by probability

For Model 1a, 142 participants were misclassified (false positives=81, false negatives=61). For Model 2a, 154 participants were misclassified (false positives=112, false negatives=42). Overall the false negative and positive rates between the two models were very similar. In an ideal model, with 100 percent accuracy, all participants would be plotted both below the probability cut-point and to the left of the RDIa threshold, or above the probability and to the right of the RDIa threshold.

Table 6.21 RDIa for false negatives and positives, by sample and model

		False positives		False negatives	
		Clinic	**Community**	**Clinic**	**Community**
Model 1a	**Median**	5.04	2.94	30.48	25.33
	IQR	2.20-9.95	0.98-7.73	21.223-50.11	18.62-25.50
	Range	0-14.10	0-14.33	10.14-114.75	15.43-31.91
	n	69	13	52	9
Model 2a	**Median**	5.03	3.04	32.62	25.33
	IQR	2.21-10.00	0.98-8.28	21.68-46.04	18.62-25.50
	Range	0-14.72	0-14.33	10.41-114.75	15.42-60.00
	n	92	19	33	9

As expected, median RDIa scores for false positives were low. In false negatives, RDIa scores were alarmingly high, especially in the clinic. Of concern was one false negative outlier in the clinic who had an RDIa of 114.75. This particular patient was a *non-Māori women aged 39 years with an ESS score of 10, neck circumference of 46cm, and a BMI of 43.50 kg/m^2, reported snoring always, and did not report observed apnoeas.* Her calculated predicted probability using Model 1a was 0.12, and 0.29 using Model 2a.

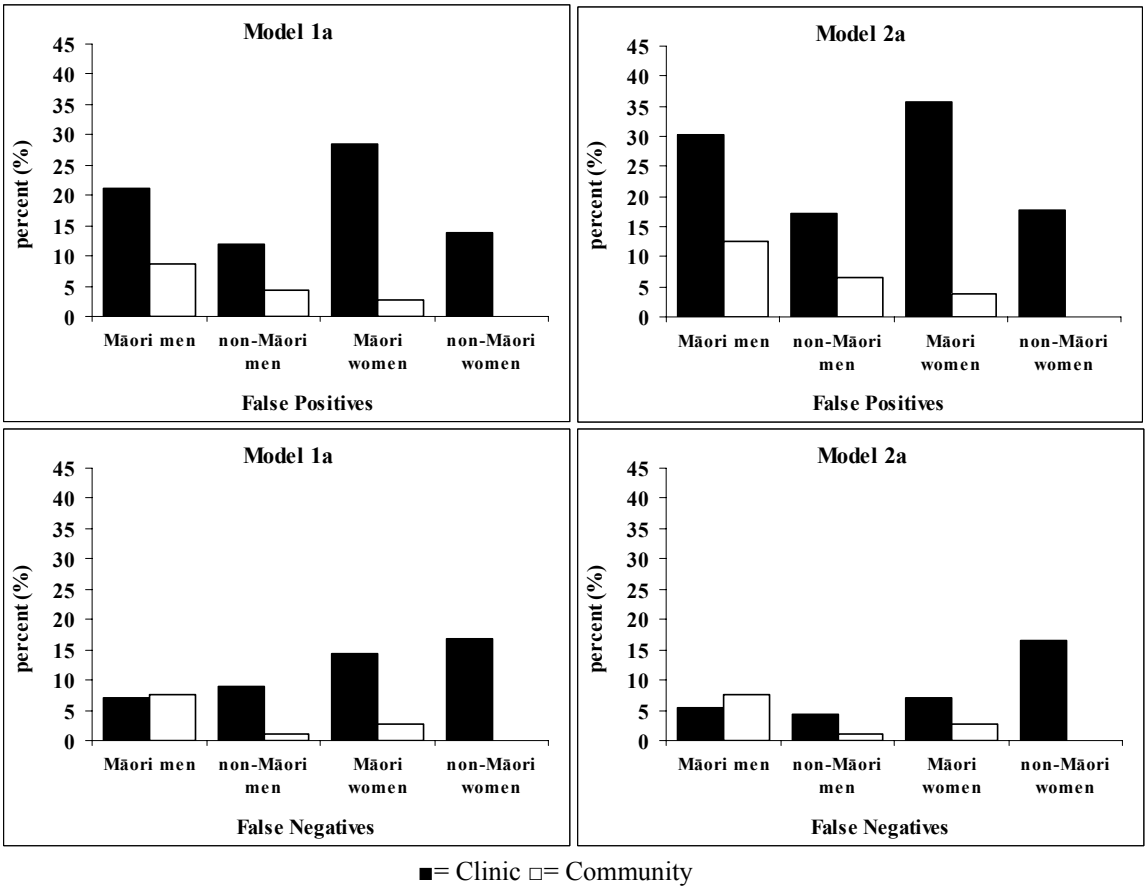


Figure 6.8 False positives and false negatives for each model, by sample, ethnicity, and sex

For both models, the highest rates of false positive and negative results were amongst clinic patients. Māori men and women had the highest rates of false positive results, and false negative results were highest amongst women in general. However, given that there was only a small number of Māori women in each model, the reliability of these rates is questionable. In the community, false negative and positive rates were highest amongst Māori men. In contrast, no non-Māori women in the community were misclassified.

Predictor variables

To further examine the characteristics of the misclassified participants, predictor variables were examined to identify any predictors that may have had a disproportionate affect on misclassified results.

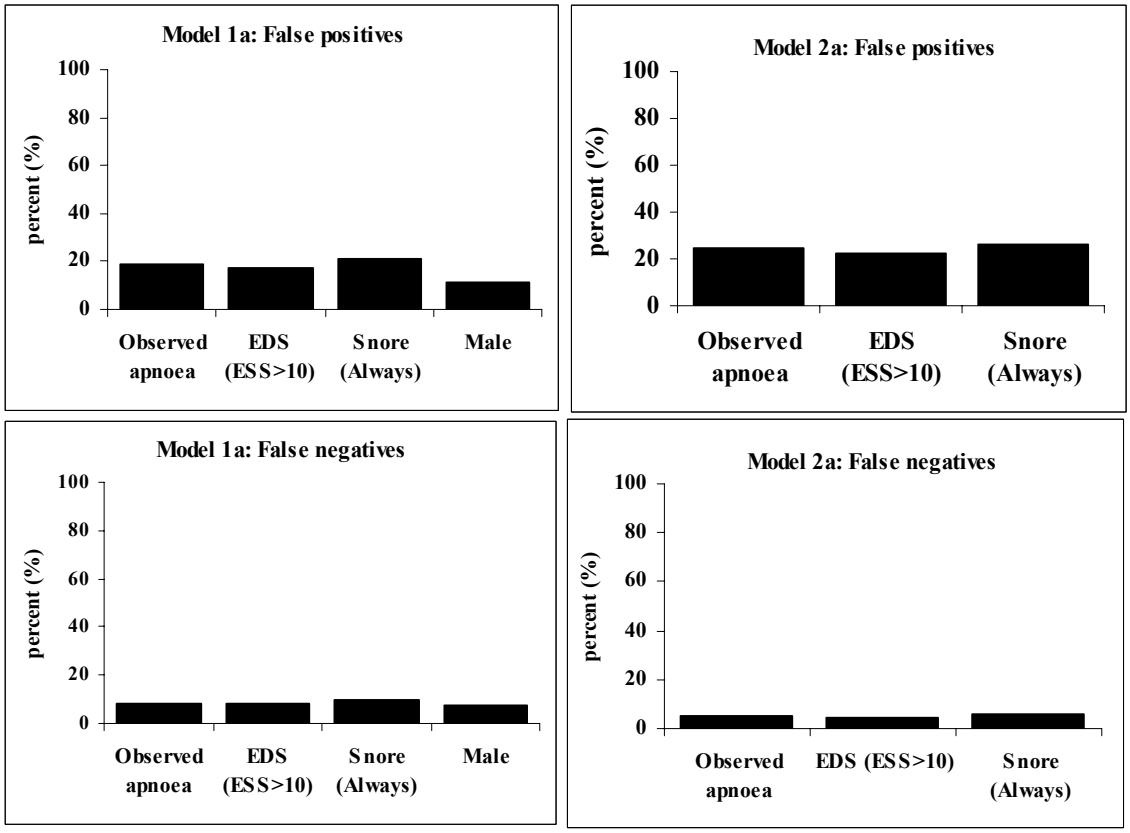


Figure 6.9 Proportion of false positives and negatives for each predictor, by model

The rates of false positive and negative results across predictors were similar across each model. These results suggest that no one particular predictor variable contributed more to misclassification, but rather a combination of predictors.

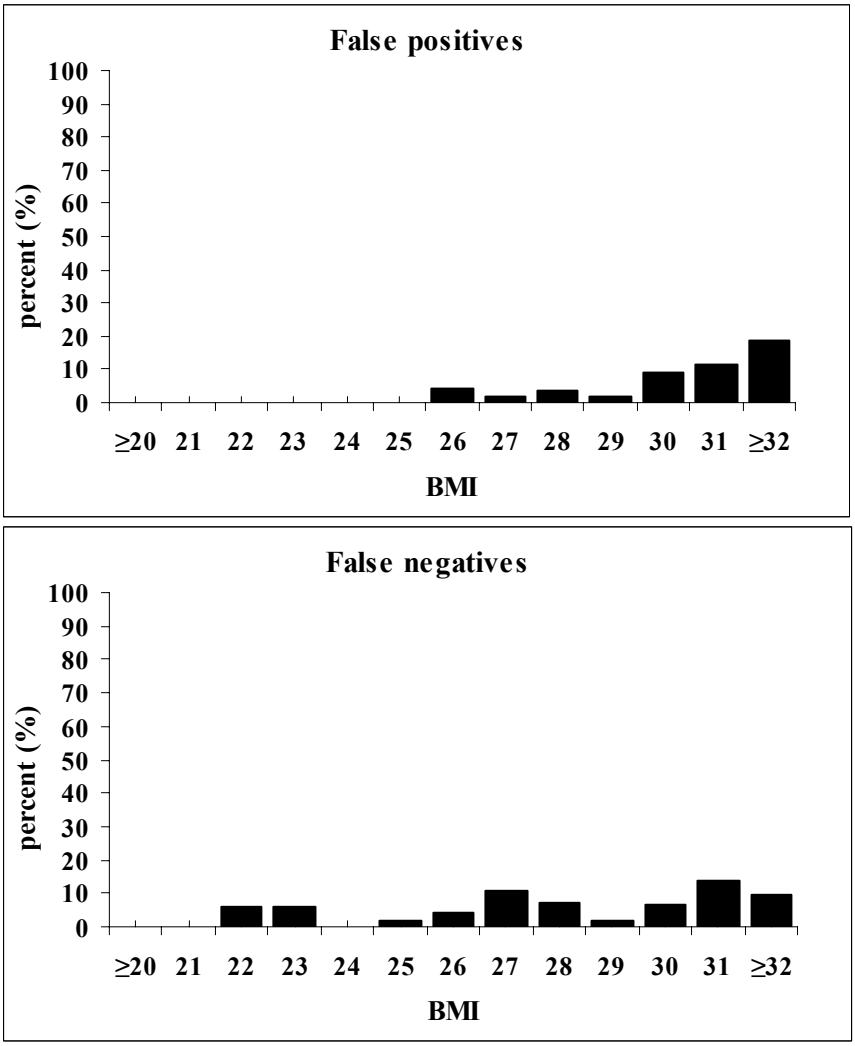


Figure 6.10 Model 1a: Proportion of false negatives and positives, by BMI

False positives tended to have higher levels of BMI, that is, no less than 26 kg/m^2, with a high percentage with BMI greater or equal to 32 kg/m^2. In contrast, participants with false negative results more frequently presented with BMIs less than 32 kg/m^2. The median BMI for false positive results was significantly higher than those with false negative results (34.87 kg/m^2, IQR=32.04-42.56 vs. 31.77 kg/m^2, IQR=28.37-37.35 kg/m^2, p=0.0004) (Figure 6.10).

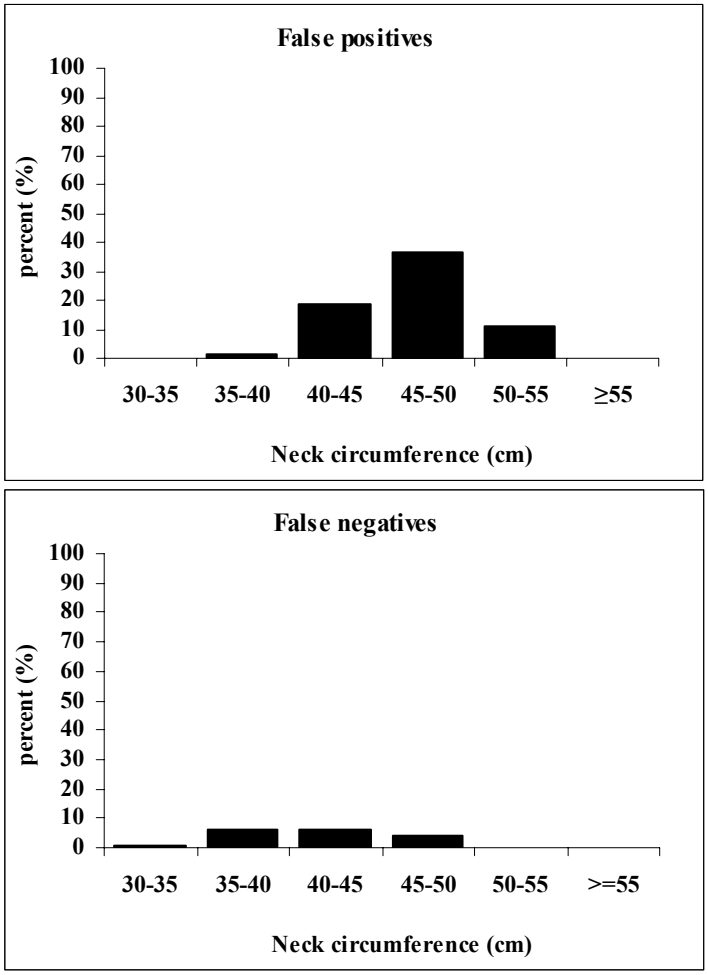


Figure 6.11 Model 2a: Proportion of false negatives and positives, by neck circumference

For Model 2a, false positives were most prevalent amongst those who had neck circumference measurements within the 40-55 cm range. False negatives were higher amongst those whose neck circumference ranged from 35-40 cm. People with false positive results had a higher median neck size (44cm, IQR 41-46 cm) compared with false negatives (41cm, IQR 41-42 cm, $p<0.0001$) (Figure 6.11).

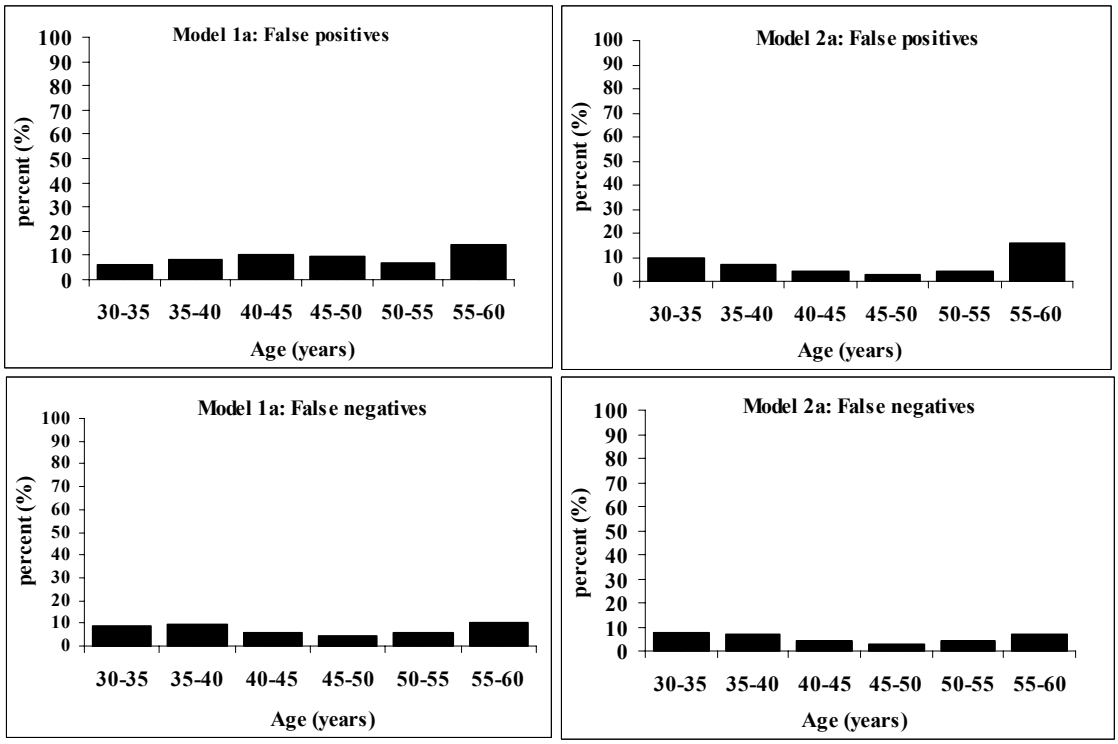


Figure 6.12 Misclassified participants in each age-group, by model

False positive and negative results were fairly evenly distributed across the age groups. However there was a slightly higher prevalence of misclassification among the 55-60 age groups relative to other age groups (Figure 6.12).

CHAPTER 7
DISCUSSION

The main goals of this study were to estimate the prevalence of obstructive sleep apnoea (OSA) in a randomly selected community sample, and to develop a clinical screening tool for OSAS. This chapter compiles and explains the results, and discusses the implications and recommendations of this study. In general, results are discussed within a disparities and Treaty of Waitangi analytical framework. Similarly, implications and recommendations focus on addressing Māori and non-Māori disparities, with the overall aim of contributing positively to Māori health development.

The chapter is divided into three main sections. The first section provides a summary and explanation of results. The second section considers the strengths and weaknesses of this study, which provides the essential context for consideration of the findings. The final section discusses the implications and recommendations from this study along with the further research needs that it highlights.

7.1 Summary and Explanation of Results

Results are discussed in three main sections. The first section presents univariate results for both the community and clinical samples. The second section presents OSA and OSAS prevalence estimates for each sample, and the final section presents the multivariate results from each sample respectively followed by the results of the development of the prediction models from the combined sample. Where possible the findings of the present study are compared with other comparable studies. However it should be noted that these comparisons are limited for a number of reasons. These include differences in study populations, differences in measurement of variables and differences in analytical techniques used.

7.1.1 Univariate results

In order to provide an overview of participant characteristics and a description of variables used in the development of the prediction models, the univariate results for the community and clinical samples are presented simultaneously under the following categories: general sleep, OSAS symptoms, OSAS risk factors and other variables (including co-morbid diseases). Table 7.1 presents a summary of total population

univariate results alongside results from the national sleep survey (Harris 2003) as a reference for the reader.

Table 7.1 Summary of univariate results

	Clinic sample n=510	Community sample* n=358	National sleep survey* (Harris 2003) n=6928
Average sleep duration (hr)	7.35	7.25	7.39
never/rarely get enough sleep (%) (95% CI)	68 (63.60-71.89)	31 (24.16-39.98)	37.30 (35.84-38.72)
never/rarely wake refreshed (%) (95% CI)	80 (76.26-83.39)	41 (33.80-52.34)	46.20 (44.70-47.65)
always snore (%) (95% CI)	50 (44.60-53.45)	7.51 (3.94-11.07)	7.54 (6.80-8.30)
observed apnoeas (%) (95% CI)	71 (66.83-74.89)	10.27 (6.16-14.38)	13.11 (12.15-14.10)
ESS>10 (%) (95% CI)	72 (56.00-64.70)	14.00 (8.69-19.65)	14.90 (13.83-15.89)
current smokers (%) (95% CI)	20.59 (17.53-24.77)	16.48 (11.01-21.95)	20.40 (15.98-24.80)
eligible for CSC (%) (95% CI)	28.04 (24.18-32.16)	5.65 (2.39-8.90)	15.79 (11.83-19.74)
average neck circumference (cm)	42.00	37.46	37.41
obese (%)	53.73	19.82	N/A

* Population (age, gender, ethnicity) adjusted results

General sleep variables

For average reported hours of sleep, no significant differences were found between Māori men and non-Māori men in the clinical or community samples. However, in the community sample, Māori women reported significantly less hours of sleep on average than non-Māori women (p=0.0072). Within ethnic groups, no differences were found between men and women in either sample. In the national sleep survey however, differences were detected between men and women within ethnic groups (Harris 2003).

Overall, the average reported sleep durations in the clinical and community samples were only slightly lower than national population estimates (Harris 2003), and are consistent with other studies, where the average length of sleep is reported to be between 7 and 8 hours (Partinen and Hublin 2000).

There were relatively high reports of inadequate sleep from both the community and clinical samples, as measured by the questions *how often do you get enough sleep* and *how often do you wake feeling refreshed*. As expected, reports of inadequate sleep were particularly high in the clinical sample. The estimates in the community sample were comparable to those from the national sleep survey (Harris 2003). For both variables, in

each sample, no differences were found between Māori and non-Māori within sex, or between women within each ethnic group. This is consistent with the national sleep survey results (Harris 2003).

OSAS symptoms

In contrast to the national sleep survey (Harris 2003), no differences were detected in either the clinical or community samples, between Māori and non-Māori, men or women in reporting snoring *always*. In the community sample however, differences were detected when snoring was categorised as *often/always*. These subjective reports of snoring concur with the objective measures of snoring recorded by the MESAM4, which showed that Māori men spent on average a higher proportion of the night snoring than non-Māori men (23.48% vs. 12.06%, p=0.003). Similar results were found between Māori and non-Māori women (9.48% vs. 2.47%, p<0.0001). Overall, 21% (95% CI 12.73-31.46) of Māori men and 12% (95% CI 6.92-19.77) of non-Māori men spent at least 50% of the night snoring. For women, 8% (95% CI 3.38-16.23) of Māori and 6% (95% CI 1.30-21.98) of non-Māori spent at least 50% of the night snoring.

Interestingly, the objective snoring estimates for Māori men are more similar to those reported in the Australian population study of men (Bearpark et al.1995) than those of non-Māori men. Twenty two percent (95% CI 17.4-26.9) of Australian men were reported to snore for at least 50% of the night. In the national sleep survey (Harris 2003) however, the prevalence of self-reported snoring among non-Māori corresponded more closely to other studies of predominately White populations, whereas self-reported snoring among Māori was significantly higher. This suggests that the objective snoring estimates for both Māori and non-Māori may be underestimated in the present study.

In the clinical sample, Māori men and Māori women spent the highest proportion of the night snoring, but because the objective snoring measures were different in the clinical and community samples, the two cannot be directly compared. Overall, the results in the community and clinical samples indicate that snoring is very common, particularly in Māori and in men.

While the reporting of observed apnoeas was more common in the clinical sample, the distribution of responses between the two samples was similar, and consistent with distributions reported in the national sleep survey. Māori men reported the highest proportion of observed apnoeas, followed by non-Māori men, Māori women and non-

Māori women. Across the community and clinical samples, differences were only detected between men and women in the community sample. In comparison, differences were detected between Māori and non-Māori, men and women in the national sleep survey (2003).

The Epworth Sleepiness Scale (ESS) was used to assess excessive daytime sleepiness (EDS), which was defined as a score greater than 10. Overall, the prevalence of EDS among community participants was similar to the national sleep survey (14% and 15% respectively). However no differences were detected between Māori and non-Māori, men and women. In the clinical sample, differences were found between Māori and non-Māori men.

OSAS risk factors

Among community participants, body mass index (BMI) ranged from 18.54 to 48.07 kg/m^2 (Median=27.69 kg/m^2). Māori men had significantly higher BMI than non-Māori men (29.21 kg/m^2 vs. 27.65 kg/m^2, p=0.0023). Similarly, Māori women had significantly higher BMI than non-Māori women (29.14 kg/m^2 vs. 26.09 kg/m^2, p<0.0001). Overall, 23% of participants (22.16% men and 23.63% women) were obese. Although Māori displayed higher median BMI than non-Māori, differences in the prevalence of obesity were only found between Māori and non-Māori women, which is consistent with known ethnic differences in BMI (Russel et al. 1999, Swinburn et al. 1999). On the other hand, there was no difference in obesity between Māori and non-Māori men. In the national nutrition survey (Russel et al. 1999), which obtained data from 4636 New Zealanders aged at least 15 years (of which 15% were Māori), Māori (27% males, 27.9% females) were significantly more likely to be classified as obese than 'NZ Europeans' and 'others' (12.6% males, 16.7% females). Given the known association between socioeconomic gradients and obesity (Howden-Chapman and Tobias 2000, Bovet et al. 2002), the lack of difference between Māori and non-Māori in the present study may be in part be due to the fact that the Wellington population is less deprived than the New Zealand population in general (Crampton et al. 2000).

In the clinical sample, the overall median BMI was 32.31 kg/m^2 (Range=16.50-63.66 kg/m^2), with 60% of all patients classified as obese. The median BMI for Māori men was significantly higher than for non-Māori men (36.33 kg/m^2 vs. 30.43 kg/m^2, p<0.0001). Similarly, the median BMI for Māori women was significantly higher than for non-Māori women (45.47 kg/m^2 vs. 36.26 kg/m^2, p=0.0027). These results are

congruent with the higher BMI among Māori compared with Europeans reported in other clinical populations (Baldwin et al. 1998, Coltman et al. 2000). Not surprisingly, obesity levels were high across all groups in the clinical sample. Although differences were not detected between groups, the skewed distribution of both Māori men and women towards the higher levels of BMI is of some concern. Māori women in particular had strikingly high BMI (Median=45.47 kg/m^2, Range 34.41-57.67 kg/m^2) compared to all other groups, and all but one patient was classified as obese. These results may indicate potential issues in health services access for Māori who are not extremely obese, particularly Māori women.

In both the community and clinical samples, the median neck circumference for Māori men was significantly larger than non-Māori men, and similarly Māori women had a larger median neck size than non-Māori women. These findings are consistent with findings in other New Zealand clinical studies (Baldwin et al. 1998, Coltman et al. 2000), and are consistent with the national sleep survey results (Harris 2003).

Markedly different patterns of alcohol usage were found between Māori and non-Māori in both samples. Non-Māori tended to drink alcohol more often than Māori, while on a typical drinking occasion Māori drank more than non-Māori. These results concur with the largest published analysis of alcohol consumption in New Zealand for Māori, which combined five large New Zealand surveys (including the national sleep survey). Data included 44830 people in total, of which 6926 were Māori (Bramely et al. 2003).

Smoking rates in the community were considerably less than those reported in the national survey (Harris 2003), and less than those found in the 1996 census (SNZ 1997a, 1997b). It is well established that Māori men and women have significantly higher rates of smoking than non-Māori men and women (Te Puni Kōkiri 2000, Ministry of Health 2003a). Results were, however, mixed in the present study. Māori women were more likely to be current smokers than non-Māori women in the community sample, but not in the clinical sample. In contrast, Māori men were more likely to be current smokers than non-Māori men in the clinical sample, but this was not found in the community sample. The lack of difference between Māori and non-Māori men in the community sample may reflect the relative lack of deprivation among participants in the present study. Increasing socioeconomic deprivation has been shown to be associated with increased smoking, which therefore contributes to the socioeconomically based inequalities in health (Crampton et al. 2000, Te Puni Kōkiri

2000, Ministry of Health 2002c). However, the insignificant difference between Māori and non-Māori women in the clinical study is more likely attributable to a lack of statistical power, as Māori women were significantly underrepresented.

Other variables

Medical conditions were more frequently reported in the clinical sample. However, in both samples, asthma and hypertension were most commonly reported, both of which are known to affect Māori disproportionately (Pōmare et al. 1995, Ellison-Loschmann and Pearce 2000). Eligibility for a community services card (CSC) was also used as a crude measure of socioeconomic position. Māori women in the community sample were three times more likely to be eligible for a CSC than non-Māori women, but no differences were found for men. Again, this probably reflects the lower levels of deprivation observed in the community sample compared to the national population. In the clinical sample, Māori men and women were significantly more likely than non-Māori men and women to be eligible for a CSC, which may reflect the higher proportion of non-Māori participants seen as privately funded patients. This highlights another issue in the accessibility of sleep services to Māori, which is discussed later in Section 7.5

7.1.2 Unadjusted OSA and OSAS prevalence in the clinical sample

The overall prevalence of OSA in the clinical sample ranged from 40.39% to 80.00% depending on the definition (RDIa or RDIc) and threshold (≥ 5, ≥ 10, ≥ 15) used. For men, the prevalence ranged from 44.47% to 84.06%, and for women, the prevalence ranged from 27.27% to 66.94%. With the addition of excessive daytime sleepiness (ESS >10) as minimum criteria for OSAS, the prevalence for Māori men ranged from 45.61%-63.16% compared with 29.52%-50.30% in non-Māori men. For Māori women, the prevalence ranged from 20%-46.70% compared with 18.87%-44.34% in non-Māori women.

Although no significant differences were found between groups, the trends indicate more prevalent and severe OSAS in Māori. The lack of differences found between groups is not surprising, given that the clinical participants were specifically referred for suspected OSAS. However, it is also likely that the differences did not reach significance because of the small number of Māori in the sample, particularly Māori women.

7.1.3 General population prevalence estimates

General population OSA prevalence estimates were calculated in the community sample for OSA as defined by the most conservative measure, RDIa, and for OSAS with the addition of daytime sleepiness criteria (ESS >10). Weighting by the population age structures for Māori and non-Māori caused little change in the unadjusted prevalence estimates, and all significant and non-significant differences between groups were unchanged.

The major findings in the community sample were the significant differences found in OSA prevalence between Māori and non-Māori. Overall, Māori were 3.5 times more likely than non-Māori to have an RDIa≥ 15. Within sex, Māori men were three times more likely than non-Māori men to have an RDIa≥ 10 and RDIa≥ 15. These results support clinical observations that suggest a higher prevalence amongst Māori (Frith and Cant 1985, Baldwin et al. 1998). They also support the findings in the national sleep survey (Harris 2003), which showed significant differences in self-reported OSAS symptoms and risk factors between Māori and non-Māori. These ethnic disparities are also consistent with overseas studies, which show that minority ethnic groups are more likely to have SDB than Caucasians (Ancoli-Israel et al. 1995, Kripke et al. 1997, Redline et al. 1997, Young et al. 2002b).

As the present study utilised the same objective sleep measures (MESAM4) as those used in the Australian population study of 294 men (Bearpark et al. 1995), the data for men can be directly compared. The age range in the present study is also comparable with the benchmark Wisconsin sleep cohort study (Young et al. 1993) of 602 randomly selected men and women, who were monitored using overnight polysomnography. Table 7.2 presents the prevalence estimates of Māori and non-Māori in the present study along with the estimates from Bearpark et al. (1995) and Young et al. (1993).

Table 7.2 Comparison of prevalence studies

First author (Year)	N	Age range (Years)	Women			Men		
			AHI/RDI			AHI/RDI		
			≥ 5	≥ 10	≥ 15	≥ 5	≥ 10	≥ 15
Young et al. (1993)	626	30-60	9 (5.6-12)	5 (2.4-7.8)	4 (1.5-6.6)	24 (19-28)	15 (12-19)	9.1 (6.4-11)
Bearpark et al. (1995)	309	40-65	N/A	N/A	N/A	26 (21-31)	10 (7-13)	Not given
Mihaere et al. (2003) Māori	166	30-60	6.28 (-0.1-12.7)	5.40 (0.9-2.7)	1.54 (-0.8-3.9)	21.98 (10.3-33.7)	16.69 (6.8-26.5)	11.86 (3.5-20.2)
Mihaere et al. (2003) non-Māori	192	30-60	3.02 (0-6.)	0.91 (0-6.2)	0	11.37 (4.49-18.26)	5.85 (0.8-10.9)	3.04 (1.1-13.8)

For Māori men the prevalence estimates of OSA at all thresholds are more similar to those reported in the Australian (Bearpark et al. 1995) and Wisconsin sleep study (Young et al. 1993) studies than for non-Māori men, whose estimates are considerably lower. Similarly, rates for Māori women are more consistent with rates reported in the Wisconsin study (Young et al. 1993) compared to non-Māori women. Based on findings from the national sleep survey (Harris 2003), which show that the prevalence of OSAS symptoms in non-Māori are generally more comparable to those in a number of other studies, which consisted of predominately White populations, while the prevalence estimates among Māori were significantly higher, it would be expected that the same pattern would be seen in the present study. This would tend to suggest that the estimates for Māori and non-Māori, men and women reported in the present study are an underestimate. The likely reason for this discrepancy may be explained by the response bias towards less deprived participants in the present study. Another possible reason is the conservative nature of the scoring in the current study, however this scoring method was similar to that of the Australian study, yet their prevalence estimates were similar to those in the Wisconsin study.

With the addition of excessive daytime sleepiness, OSAS (using RDI≥ 5) occurred in 8.01% (95% CI 1.92-14.09) of Māori men, 12.11% (95% CI 4.32-19.90) of non-Māori men, 8.48% (95% CI 1.81-15.15) of Māori women, and 2.36% (-0.03-4.74) of non-Māori women. While the estimates for men are slightly higher than the 4% reported in the Wisconsin sleep study (Young et al. 1993), the difference is probably not statistically significant given the wide 95% confidence intervals. This also applies to

the estimates for Māori and non-Māori women. The variation been studies may also be due to the variation in the criteria for hypersomnolence.

Among non-Māori, men were 3-4 times more likely than women to have OSA or OSAS. For Māori, the sex difference was considerably higher (3-7 fold). The differences in prevalence by sex for non-Māori are consistent with other studies that have reported that men are 2-3 times more likely to have OSAS than women (Strohl and Redline 1996, Partinen and Hublin 2000). In the national sleep survey, sex ratios of 2-3 times for men compared to women for snoring always and observed apnoeas, were found within both Māori and non-Māori groups (Harris 2003). The increased risk displayed among Māori men in the present study may therefore reflect a response bias.

The present study was not powerful enough to consistently detect differences between Māori and non-Māori within each sex, as the number of cases became too small, especially with the addition of excessive daytime sleepiness (ESS > 10). However most trends were in the expected direction (i.e., higher prevalence among Māori and men).

7.1.4 Multivariate predictors of OSA

In the clinical and community samples, potential OSA predictors were tested at three thresholds (≥ 5,≥ 10,≥ 15) for both RDI measures (RDIa and RDIc). For comparability, identical models were run in each sample with the exception of the socioeconomic variable, NZDep96, which was only available for community participants. The initial models (Model 1 and 2) examined the impact of demographic variables (ethnicity, sex, age) on the prevalence of OSA. To examine whether demographic predictors remained significant after controlling for other variables, a number of other models were run in each sample. Due to potential collinearity issues with body habitus variables, two separate models were developed. One model included BMI and other variables (Model 1a), and the other included neck circumference and other variables (Model 1b).

Model 1 – Ethnicity, sex, age

In the community sample, after controlling for sex and age, ethnicity was still a marker of risk for OSA (RDIa≥ 10 and RDIa≥ 15). Māori were 3.5 times (95% CI 1.39-8.71) more likely to have RDIa≥ 10, and 4 times (95% CI 1.31-13.90) more likely to have an RDIa≥ 15 than non-Māori. Sex was also a consistent predictor, with men 4-9 times more likely to have OSA than women. Increasing age however, was not found to be a

significant risk factor. Ethnicity was not a marker for OSA defined by RDIc at any threshold. This is most likely due to the inaccuracy of the measurement of hypopnoeas within the RDIc score.

These results can be compared with results from the Sleep Heart Health Study (Young et al. 2002b). Compared to White Americans, Native Americans were more likely to have an AHI ≥ 15 (OR 1.70, 95% CI 1.37-2.11), although no difference was found between Whites Americans and African-Americans (OR 1.23 95% CI 0.97-1.60) the trends are in the expected direction. The Sleep Heart Health Study also found that men had 2.7 times the odds of having an AHI ≥ 15, which is considerably less than the rate reported in the present study. Furthermore, for every 10-year increment in age, the risk of having an AHI ≥ 15 increased by 24%, however the age range (40-98 years) was much wider than in the present study.

In the clinical sample, ethnicity was not a risk marker for OSA, which is not surprising given the lack of differences found between Māori and non-Māori in the univariate analyses. However the insignificant differences, as mentioned previously, are most likely attributable to the disproportionately small number of Māori patients. Overall, men were 2-3 times more likely than women to have OSA. Age was found to only be associated with an increased risk of having an RDIa ≥ 5 (3% increase in risk for every additional year).

Models 1a and 1b

The major finding in these models was that ethnicity was no longer a significant risk factor after controlling for a number of other established risk factors. This finding is consistent with other studies (Baldwin et al. 1995, Young 2002b), and indicates that differences between Māori and non-Māori are largely due to factors other than ethnicity per se. Some studies however have found race to be a risk factor for SDB (RDI ≥ 30) independent of other factors including age, sex and obesity (Ancoli-Israel et al.1995, Redline et al. 1997). However, the disproportionate numbers of non-White participants included in these studies do not allow enough statistical power to adequately inform ethnic inequalities.

Neck circumference and BMI were consistent predictors in their respective models. Some studies have found neck circumference to be a stronger predictor than BMI (Stradling and Crosby 1991, Hoffstein and Mateika 1992, Flemons et al. 1994, Davies

et al. 2002). While other studies suggest that waist circumference is a better predictor than either BMI or neck circumference in men (Grunstein et al. 1993, Deegan and McNicholas 1996), while the opposite is true for women (Deegan and McNicholas 1996). The variation of findings across these studies may relate to the fact that measures of body habitus are correlated, some highly so. Differences in findings may therefore reflect varying degrees of measurement accuracy or perhaps statistical problems with the variables being strongly interrelated, especially in smaller samples

A number of other studies have reported male sex as an independent predictor of OSA (Viner et al. 1991, Hoffstein and Szalai 1993, Young et al. 1993, Ancoli-Israel et al. 1995, Maislin et al. 1995, Bixler et al. 2001, Young et al. 2002b), which is consistent with the models that included BMI in the present study. However, male sex was not a significant predictor after controlling for neck circumference. Dixon et al. (2003) reported similar results to these, where neck circumference was shown to explain the same amount of variance as BMI and sex collectively. Since BMI and neck circumference measurements provide quite different information on obesity. The later is a general description of the degree of central obesity, which is more common in obese men than women and is one of the main pathogenic determinants of OSA. This may in part explain why obese males are more likely to have OSA than obese females.

Reporting of observed apnoeas was consistently found to be a significant independent predictor of OSA, which is consistent with findings from a number of other studies (Kapuniai et al. 1988, Crocker et al. 1991, Hoffstein and Szalai 1993, Kump et al. 1994, Pillar et al. 1994, Young et al. 2002b, Dixon et al. 2003).

The general sleep variables, (never/rarely getting enough sleep or wake feeling refreshed) were not significant risk factors. While these features are common amongst OSA patients, they are also very common in non-OSA individuals and therefore are not very useful in screening patients. Other studies have also failed to find any significant independent relationship with similar variables (Stradling and Crosby 1991, Hoffstein and Mateika 1992, Hoffstein and Szalai 1993, Flemons et al. 1994).

In the community sample, socioeconomic position as measured by NZDep96 was not an independent risk factor for OSA. In the national sleep survey (Harris 2003) however, an independent relationship was found between OSAS symptoms (excessive daytime sleepiness and observed apnoeas) and deprivation. The inability to find a significant

relationship in the present study most likely reflects the different deprivation profiles compared to that of the general population, and/or the lack of statistical power in the community sample due to the small number of people with OSA.

In the clinical sample, more significant predictors were found, probably because of the increased number of positive cases compared to the community sample, which would obviously increase the statistical power. In addition to significant relationships found for the community sample, the clinical sample showed significant odds ratios for habitual snoring (defined by snoring always) and excessive daytime sleepiness (EDS, defined by ESS>10). A number of other studies have found self-reported snoring to be an independent predictor of OSA (Viner et al. 1991, Kump et al. 1994, Flemons et al. 1994, Maislin et al. 1995, Ip et al. 2001, Young et al. 2002b). In regards to daytime sleepiness, other studies have demonstrated limited predictive ability, if any (Kapuniai et al. 1988, Kump et al. 1994, Crocker et al. 1990).

Interestingly in the clinical sample, participants who reported receiving current treatment for asthma showed a decreased likelihood for OSA. This may be because some symptoms for nocturnal asthma are similar to those for OSA (Douglas 2002). However, this relationship was no longer significant when clinical and community data were combined.

7.1.5 Development of the prediction models for OSA

It has been said that successful modelling is part science, part statistical methods and part experience and common sense (Homser and Lemeshow 2002). One of the goals of this study was to develop a questionnaire-based screening tool for OSA. To do this, data were combined from community and clinical participants and a range of variables were tested in a series of models. The goal of modelling was to find the most efficient and parsimonious multivariate models that were able to adequately predict the probability of an individual having OSA.

Univariate modelling

Initial modelling examined the univariate relationships between potential predictors and OSA. The majority of variables tested were significant and consistently so across different levels of OSA severity.

Smoking was not identified as a significant risk factor for OSA, despite a number of other studies having found a significant relationship between the two (Bloom et al. 1988, Wetter et al. 1994, Bearpark et al. 1995). The present study did however find that ex-smokers were more likely than non-smokers to have OSA, which suggests that smoking may have some long-term effects on OSA. In contrast, Wetter and colleagues (1994) found that former smokers were not significantly more likely to have OSA (AHI $\geq$ 5) than those who had never smoked. Other research indicates a dose–effect between smoking and SBD (Kauffman et al. 1989). However, smoking amount (i.e., light or heavy) was not measured in the current study.

Of the three alcohol variables tested, only one was significant, but it was not in the expected direction - moderate drinkers were less likely to have OSA than non-drinkers. This finding is probably not due to alcohol consumption per se. It is possible that moderate drinkers as a group had lower prevalence of other OSA risk factors. This interpretation is supported by the finding that alcohol consumption was not a significant independent predictor in the multivariate models run in the community and clinical samples respectively, and is consistent with other studies (Bearpark et al. 1995, Olson et al. 1995, Hui et al. 1999).

Of the co-morbid diseases, people who reported current treatment for hypertension, diabetes, stroke and psychological problems were more likely to have OSA than those who were not receiving treatment for these conditions. These findings provide support for the possible contribution of higher rates of co-morbid disease to the greater severity of SDB (Ancoli-Israel et al. 1995). However when added to multivariate models, the relationships were no longer significant. A number of other studies have found hypertension to be a significant independent risk factor for OSA (Crocker et al. 1991, Flemons et al. 1994, Duran et al. 2001).

Multivariate modelling

Two multivariate models were initially constructed (full models), which were predominately informed by the univariate results. One model included BMI and a number of other variables, and the other included neck circumference along with a number of other variables. Across models, the consistent predictors included male sex, increasing age, increasing BMI, increasing neck circumference, excessive daytime sleepiness (ESS>10), snoring (always), and observed apnoeas. These were comparable to the independent risk factors found in the separate models run in the community and

clinical samples, with the exception of age, which was not previously found to be an independent predictor.

Models were tested for interactions, but only between men and women, as the disproportionately low numbers of Māori, did not permit tests between Māori and non-Māori. In the national sleep survey (Harris 2003), a significant interaction was found between ethnicity and smoking in the prediction of observed apnoeas. Among non-smokers, Māori were significantly more likely to report observed apnoeas than non-Māori (OR 1.68, 95% CI 1.39-2.04, $p<0.0001$), after controlling for other factors. However, among smokers there was no significant difference in the reporting of observed apnoeas between Māori and non-Māori, after controlling for other factors. These findings show that the difference between Māori and non-Māori in reporting observed apnoeas was dependent on their smoking status, which may have implications for the results of the present study.

A significant interaction was found between sex and BMI, which indicated that increasing BMI was a slightly stronger risk factor for men than for women. It was however decided that this interaction would not be included in the final model because it complicated the model without significantly improving it.

Best multivariate models for screening

Using backward and forward selection, a number of models were tested and two models (Models 1a and 2a) were seen as superior in simplicity and accuracy. The predictors retained in these models included male sex, increasing age, increasing BMI, increasing neck circumference, excessive daytime sleepiness (ESS>10), snoring (always), and observed apnoeas.

Based on the model parameters for moderate-severe OSA (RDIa≥ 15) (American Academy of Sleep Medicine 1999), the probability of OSA was calculated for each participant in the combined sample, and an optimal probability cut-point for each model was chosen based on a number of evaluations (ROC curves, specificity and sensitivity plots and likelihood ratios). A probability cut-off point of 0.40 was selected for Model 1a (BMI), and a cut-point of 0.30 was selected for Model 2a (neck circumference).

Using these cut-points, Model 1a correctly classified 82.50% of participants, with a sensitivity of 71.90% (95% CI 75-85%), specificity of 86.60%, positive predictive value

(PPV) of 65.55%, and a negative predictive value (NPV) of 89.68%. Similarly, Model 2a correctly classified 81.10% (95% CI 78.42-83.81%), with a sensitivity of 80.10% (95% CI 74.71-85.48%), specificity of 81.50% (95% CI 78.36-84.58), PPV of 60.36% (95% CI 54.63-66.09%), and a NPV of 92.06% (95% CI 89.78-94.38).

The area under the curves (AUC) for Model 1a and Model 2a were 0.89 respectively, indicating that the models had very good discriminatory power and are equally good, which is not surprising given that neck circumference has equivalent predictive ability to BMI and sex combined (Dixon et al. 2003). The post-test probabilities given a positive screening test (or PPV) for each model were substantially higher than the pre-test probability (prevalence) of RDI $\geq$ 15, which indicates that the models provided additional information beyond what would be expected from the prevalence alone. Furthermore, the 18-fold difference between the positive and negative likelihood ratios for both models indicates that they provide good diagnostic information. Thus these models are potentially clinically useful. Table 7.3 summarises predictive models from other studies.

Table 7.3 Summary of OSA clinical prediction models

First author and date of study	Sites	Type	OSA diagnostic criterion	Equipment	Sample size	Patients with OSA	Predictive Variables	AUC	Sensitivity	Specificity
Kapuniai et al. (1988)	Honolulu	Clinic and Volunteers	AHI ≥ 10	PSG	53		Observed apnoea, snoring		78%	67%
Crocker et al. (1990)	Newcastle, Australia	Clinic	AHI ≥ 15	Partial PSG	100	27%	Age, witnessed apnoeas, BMI, hypertension		79%	50%
Viner et al. (1991)	Toronto, Canada	Clinic	AHI ≥ 10	PSG	410	46%	Body mass index, snoring, age, sex	0.77	94%	28%
Hoffstein and Szalai (1993)					594	46%	BMI, age, gender, observed apnoea, pharayngeal examination			
Flemons et al. (1994)	Calgary, Alberta	Sleep clinic	AHI ≥ 10	PSG	180	46%	Neck circumference, hypertension, snoring, gasping/choking		81%*	17%†
Kump et al. (1994)		Community		Portable Monitor (Eden Tec)	465		self-reported snoring intensity, observed choking, falling asleep while driving, gender, BMI	0.87		
Maislin et al. (1995)	Philidelphia, Pittsburg, Baltimore	Sleep clinic (but not referred)	RDI > 10	PSG	427	60%	Snoring, gasping/snorting, observed apnoeas, BMI, age, sex	0.79	88%	55%

*Positive predictive value; † Negative predictive value

Other predictive models display reasonably high sensitivities (minimize false negatives) but the specificities tended to be low (increase the number of false positives). Thus they can be useful in excluding the diagnosis and detecting the majority of patients with sleep apnoea, but many patients without sleep apnoea will also be detected (false positives). Despite these flaws, a number of these models have also been found to be superior to the subjective impressions of physicians (Croker et al. 1990, Viner et al. 1991, Hoffstein and Szalai 1993, Flemons et al. 1994). Therefore it would be expected that the models developed in the present study would also be superior to subjective impressions of physicians and thus would be very useful in a primary care setting.

Misclassified results

An analysis of the misclassified cases showed that the majority were from the clinical sample. The higher rate of misclassification in the clinical sample may reflect the 'pre-screened' nature of this sample, which meant that the predictive ability of clinical features was reduced. Of concern however was the high rate of false negatives amongst women in general, which suggests that perhaps models should be developed separately for men and women. This is also suggested by the results of Rowley et al. (2000), who prospectively tested the models of Crocker et al. (1990), Viner et al. (1991), Flemons et al. (1994) and Maislin et al. (1995) in an independent groups of patients (n=370) referred for suspected OSA. All four models performed better for men, with AUCs ranging from 0.71-0.80 for men compared to 0.61-0.65 for women.

Analyses of the false negatives in the present study also showed that there were a significant number of patients who had relatively low BMI values. This highlights the fact that obesity is only one risk factor of OSA. Therefore the relative weighting of BMI in Model 1a may cause a significant number of OSAS cases to be overlooked if this model was used as a screening tool.

7.2 Study Strengths

The strengths of this study are discussed in relation to the prevalence estimates from the community sample and the development of the clinical screening tool respectively, along with the general strengths of the study.

7.2.1 OSAS prevalence estimates

Previous estimates of OSAS prevalence in New Zealand have been confined to clinical populations (Frith and Cant 1985, Baldwin et al.1998). The current study provides the first objective prevalence estimates of OSAS in a community sample. Although this study was confined to Wellington residents, it has an advantage over a number of other studies from other countries, which are restricted to specific subgroups of the population, such as males (Bearpark et al. 1995) and state employees (Young et al. 1993).

Achieving near equal numbers of Māori and non-Māori participants, provided equal explanatory power for each group ensuring that the study provided information that was at least as reliable for Māori as it was for non-Māori. In particular, it allowed accurate estimates of OSA prevalence for both Māori and non-Māori, and provided enough statistical power to inform Māori and non-Māori disparities.

The clinical screening tool

Although a number of other studies have developed clinic prediction tools (Kapuniai et al. 1988, Crocker et al. 1990, Viner et al. 1991, Flemons et al. 1994), most have been limited to relatively small clinic sample of predominately White populations with symptoms suggestive of OSAS, which essentially limits their clinical utility in a primary care setting. The present study is the first to combine a large clinical sample and a relatively large community-based comparison group.

While it may not have been ideal joining such disparate samples together, increased statistical power was achieved without increasing the cost of the study. However this was offset by the inability to produce information for Māori to at least the same depth and breadth as obtained for non-Māori. The combined sample comprised predominately non-Māori participants, therefore the prediction models may not adequately reflect the profile of risk factors for Māori. It is not recommended however that referral to specialist sleep services are based solely on the results of the screening tool, but rather they are considered in a wider context of population prevalences and disparities between Māori and non-Māori.

7.2.2 General

This thesis presents a thorough description of the study population, which will assist readers in deciding whether the results are generalisable to their own patient population or general population. Although the response rate was not ideal, the study design allowed a comprehensive assessment of potential sources of bias inherent in the data.

Grounding the present study within a Kaupapa Māori Research framework makes this study very rare in the field of health research in New Zealand and overseas with other indigenous populations. Many previous studies in New Zealand have failed to adequately explain health disparities between Māori and non-Māori, and in particular Māori needs (Reid et al. 2000, Thomas 2001). Such inadequacies increase the risk of policies and interventions being based on non-Māori needs, which may play a role in increasing disparities.

The combined results of this study and the national sleep survey (Harris 2003) will provide valuable information with which to assess the public health impact of OSAS and to plan for population health care needs of all New Zealanders.

7.3 Study Limitations

In most research, inaccuracies in the collection of data are inevitable. Although it is sometimes difficult to determine the precise impact that such bias may have on the end results, it is important to attempt to identify the magnitude as well as the direction of the bias for any estimate (Hennekens and Buring 1987).

This section identifies the major sources of potential error for the estimation of OSAS prevalence, and for the prediction models respectively, and discusses how they were either minimised or how they may affect the results of the study.

7.3.1 Limitations of the OSAS prevalence estimates

Response bias

Overall, the response rate ranged from 30.33-89.70%, depending on how response was defined. The most conservative response rate was 46%, which reflects the number of people who agreed to a MESAM4 sleep study (n=364), with respect to the total number of people who were contacted (n=786) as the denominator. Although there is no official

standard for a minimum acceptable response rate, 70% is often used as a benchmark in epidemiological studies.

The low response rate increased the potential for response bias in this study - people who responded may have differed significantly from those who did not in the variables of interest. This creates the potential for under or overestimation of OSA. The potential for response bias was further enhanced by not being able to contact a large portion of the electoral roll sample (n=325), as this required a valid telephone number.

Overall, significantly more non-Māori than Māori could be contacted by phone, to seek their participation in the study (16.50% vs. 10.58%, $p<0.0001$). This is not surprising given that a higher proportion of Māori than non-Māori report not having a telephone (SNZ 1997a, SNZ 1997b). However this difference was no longer significant after controlling for deprivation. For both Māori and non-Māori, the chances of being contacted declined similarly with increasing deprivation. This relationship was expected, as not having a telephone is one of the criteria for deprivation in NZDep96. NZDep96 also takes into consideration people not living in their own home as a measure of deprivation. This particular factor would have also affected our ability to contact people, as these people are likely to move from house to house more frequently, which would increase the chance of their electoral roll information being out of date. The comparison of deprivation profiles of MESAM4 participants with adults in the Wellington region indicated that both Māori and non-Māori MESAM4 participants were slightly overrepresented in the less deprived deciles and underrepresented in the most deprived deciles.

Increasing deprivation on NZDep96 is associated with a number of adverse health outcomes (Salmond et al. 1999, Howden-Chapman and Tobias 2000), which are also associated with OSAS, such as obesity, smoking, hypertension, and other cardiovascular diseases. Thus the contact bias reflected in the deprivation profiles of MESAM4 participants is likely to underestimate the prevalence of OSAS for both Māori and non-Māori. Given the known differences in deprivation profiles of Māori and non-Māori, the bias may have greater impact on prevalence estimates for Māori than for non-Māori, which would lead to an underestimation of any disparities between Māori and non-Māori. Others have also noted that, for Māori, it is often the non-

participating part of the population that is exposed to the highest risks of disadvantage (Ajwani et al. 2003).

For non-Māori only, a trend was found between increasing age and being contacted. Evidence suggests that in this particular age range, there is a relationship between increasing age and increased prevalence of OSA (Young et al. 2002b). This is further supported in the results of the present study, where increasing age was a significant predictor of OSA after controlling for a number of other confounding variables in the combined sample. Given that the contact gradients were significant for non-Māori, but not for Māori, this contact bias may cause an overestimation of OSA prevalence in non-Māori, which again would lead to an underestimation of disparity between Māori and non-Māori.

Of the 54% (n=422) who did not have an overnight study, 81% (n=341) answered the questionnaire over the phone, which allowed thorough assessment of potential biases in questionnaire responses of those who did agree to a MESAM4 sleep study.

No demographic differences were found between those who agreed to a MESAM4 sleep study and those who only answered the questionnaire. In terms of questionnaire responses, non-Māori women who had a MESAM4 sleep study were more likely to report *never/rarely* waking refreshed than those who answered the questionnaire (42.42% vs. 28.33%, $p<0.01$). Māori men who had a MESAM4 sleep study were more likely to report observed apnoeas than those who only answered the questionnaire (22.22% vs. 8.06%, $p<0.01$). These discrepancies suggest that non-Māori women and Māori men who had perceived sleep difficulties were more likely to agree to a sleep study, and therefore the prevalence of OSAS may be overestimated for these two groups. However, when compared with the national sleep survey (Harris 2003), no differences were found, suggesting that perhaps non-Māori women and Māori men who only answered the questionnaire tended to under report sleep difficulties.

Neck circumference measurements unfortunately could not be compared between MESAM4 participants and those who only answered the questionnaire, as only a few people in the latter group had access to a tape measure when the researcher called them, despite being provided with a tape measure in the study packs sent to them.

Measurement error

The classification of ethnicity in this study has the potential to introduce a number of potential measurement errors. The Māori versus non-Māori comparison adopted in the present study is rather simplistic. The inclusion of all other ethnic groups, apart from Māori, into the non-Māori group may be problematic, particularly with regard to the Pacific Island ethnic groups, unless they also identify as Māori. Clinical reports show a disproportionate number of both Māori and Pacific peoples with more severe and prevalent OSAS (Baldwin et al. 1998, Middleton et al. 2002). It is therefore likely that the prevalence among Pacific peoples more closely aligns with Māori, rather than other ethnic groups. Therefore classification as non-Māori would lend to an overestimation of non-Māori OSAS prevalence, and in turn an underestimation of disparities between Māori and non-Māori. However this was not an issue in the present study, as there were only a small number (n=3) of community participants who self-identified as belong to the Pacific ethnic group, therefore the non-Māori results do reflect predominately a Pākehā reality. Relatively small numbers of Pacific peoples were also seen in the national sleep survey (Harris 2003) (n=121). The electoral roll unfortunately does not permit identification of Pacific peoples for sampling.

The analytical approach taken also assumes that Māori can be regarded as a homogenous group. Māori are, however, as diverse and complex as other sections of the population, even though they may have certain characteristics in common (Durie 1998).

Over the four census periods from 1981 to 1996, there have been significant changes in the New Zealand census question relating to ethnicity. Accompanying the changes in the census question, there have been marked changes in the proportion of Māori and NZ European groups calculated from responses. Problems have been noted with the ethnicity question in the 1996 census, in the wording and the offering of more categories. This essentially led to more people identifying with multiple ethnic groups, with responses more likely to be based on ancestry than ethnicity (Thomas 2001, Robson and Reid 2001). If Māori ethnicity is more strongly associated with OSAS than Māori ancestry, the use of the 1996 question in the present study may lead to an underestimation of OSAS prevalence for the Māori Ethnic Group (MEG) as defined in

this study, and an overestimation for non-Māori, again underestimating the disparities between these groups.

It is also important to consider that the different methods of collecting the questionnaires may have compromised the comparability of responses, if responses to the questions differed according to whether they were answered by phone or answered in the presence of a researcher. In the piloting of the sleep questionnaire (Harris 2003), no systematic differences were found between questionnaire responses when participants answered by phone and mail, about 5 weeks apart. However this is not strictly comparable to the situation in the present study.

In total, six participants (3 Māori, 3 non-Māori) were removed from analysis due to excessive artefact in their respective MESAM4 studies. This meant that only data from 358 participants (166 Māori, 192 non-Māori) were available for final analyses. Given the small proportion of participants excluded, it is likely that their exclusion would have had minimal affect on the study findings.

Instrumental error

The imprecision of the measuring equipment may also contribute an important source of bias. The diagnostic accuracy of the MESAM4 equipment found in the validation study (Appendix 3) reached a sensitivity of 83% and a specificity of 100% for detecting OSA (RDIa $\geq$ 5) against gold standard polysomnography. However, given the small number of participants (n=13) these results are somewhat limited.

Furthermore, problems were found with the position sensor, and therefore supine sleep could not be controlled for. A supine sleeping position has not only been shown to increase the frequency of apnoeas and hypopnoeas (Nakano et al. 2003), but also increase the severity of the events (Oksenberg et al. 2000). Therefore increased supine sleep would lead to an overestimation of the severity of OSA. It is possible that wearing the sleep monitoring equipment encouraged people to sleep on their backs, however others have reported that the MESAM4 equipment had no major effect on time spent lying in the supine position (Bearpark et al. 1995). In light of these issues, to some degree, using the MESAM4 was a compromise, but full polysomnography would have been too expensive and the extra time required to set up equipment may have dramatically reduced the participation rate.

The definition of the RDI also significantly affects the identification of OSA (Redline et al. 2000). To address this issue, a variety of RDI thresholds were used in the present study, and population prevalence estimates were based on the most conservative definition (RDIa), which has been validated in this study and another study (Bearpark et al. 1995). Given the method of calculating sleep time, the scoring method would tend to err on the side of underestimation rather than overestimation.

The single night of recording in the present study is also limited, as there is clear evidence for night-to-night variability, particularly in mild-moderate OSAS patients (Lord et al. 1991, Le Bon et al. 2000, Bittencourt et al. 2001). A number of factors can influence the variability in OSAS severity, including posture, sleep state, nasal patency, and alcohol levels (Bassiri and Guilleminault 2000). However, due to time and money constraints in the present study, it was not possible to record participants on more than one night, so this issue could not be addressed.

Furthermore, although studies were conducted at home, they could have been susceptible to a *first night effect*, which is the effect of the environment and sleep recording equipment on the quality of the participant's sleep during the first night of recording (Agnew et al. 1996). To assess this potential issue in the community sample, participants were asked to rate their study sleep compared to a *normal night's* sleep. The majority of participants rated their sleep as either *typical* or just below *typical*, which would suggest that a *first night effect* had little impact on the results of the present study. However these ratings may have been subject to bias, in that participants may have reported positively to please the researcher.

Generalisability

The specific age range of 30-60 years used in this study is based on overseas research, where prevalence has been shown to be high in this age range (Young et al. 1993, Bearpark et al. 1995). The findings of these studies however may not reflect the reality in New Zealand, in particular for the Māori population. Furthermore, the findings are not generalisable to other age groups.

To assess the generalisability of the MESAM4 data, deprivation profiles and questionnaire responses were compared with those in the national sleep survey (Harris 2003). The results showed that MESAM4 participants were significantly less deprived

than national sleep survey responders, which reflects the deprivation profiles in the Wellington population. One in four people in the Wellington region live in the least deprived deciles, which is strikingly high when compared to other areas in New Zealand, with the exception of Auckland (Crampton et al. 2000a). The only differences found between responses to the questionnaire were in the eligibility for a community services card (CSC). MESAM4 participants were significantly less likely to be eligible for a CSC, which is consistent with the difference seen in deprivation profiles. Given the profound differences in deprivation profiles between MESAM4 participants and national sleep survey participants, it is somewhat surprising that they did not differ more in questionnaire responses.

Because the primary focus of the analysis was Māori and non-Māori comparisons, the generalisability of prevalence estimates to other ethnic groups is not known. Furthermore, the fluidity and dynamic nature of the concept of ethnicity means that the results of this study should not be generalised across time, generations, or populations with different histories (Senior and Bhopal 1999).

7.3.2 Limitations of the prediction models

Measurement error

The dichotomising of OSA with the screening tool is somewhat problematic, since obstructive events per se are not necessarily a disease. The disease OSAS usually presents as somnolence, fatigue and difficulty concentrating, which is also subject to individual variation. Furthermore there is a potential hazard in using a cut-off point developed for one population in another. Rowley and colleagues (2000) attempted to prospectively validate four models (Crocker et al. 1990, Viner et al. 1991, Flemons et al. 1994, Maislin et al. 1995) in a sleep clinic population using the original probability cut-offs from each study. The sensitivities ranged from 76%-96% for distinguishing between patients with or without an AHI $\geq$ 10, while the specificities were low, ranging from 13%-54%. There were a number of reasons given as to why these models were not accurate in that particular sample. Firstly participants in the test sample were extremely obese, and there was a relatively higher proportion of females (50%) than what would be expected at a sleep clinic. This study highlights the importance of validating the models in other populations. For this reason, it is recommended that the models developed in the present study be used in conjunction with clinical judgement,

whereby the clinician determines the threshold that seems appropriate for each patient based on case presentation.

The method used to evaluate the screening tool is limited, as it was not evaluated in an independent group of participants and therefore may inflate the accuracy of the models. However to minimise this bias, the statistical technique of *jackknifing* was used (SAS Institute 1995), which omitted each of the observations from the predictive model while it was being classified. Ideally, with a bigger sample size, the data could have been split in half, and the screening tool developed on one half and tested on the other.

Tests of homogeneity were used to test the validity of joining the community and clinical samples together. However given the smaller numbers in the community sample, and the wider confidence intervals, it is possible that differences in the two samples could not be adequately detected.

The statistical process involved in the development of the screening tool is also subject to potential errors. In this study, univariate models were initially run, followed by stepwise selection, with both backward elimination and forward selection. One problem with basing the multivariate models on the univariate analyses is that it ignores the possibility that a collection of variables, each of which is weakly associated with the outcome, can become an important predictor when taken together (Hosmer and Lemeshow 2002). However findings of other similar studies were taken into consideration, therefore if a variable was not significant in the univariate models, it was still considered for selection in the multivariate model if other studies identified it as a significant predictor (e.g., hypertension).

Instrumental bias

The equipment used to measure height and weight in the community and clinical sample was not identical. In some instances, self-report was used in the clinical sample, which may be subject to gender specific biases. It has been shown that males tend to overestimate their weight and women tend to underestimate their weight (Villanueva 2001). The lack of standardisation of height and weight measurements across the two samples has the potential to cause some imprecision in the predictive ability of BMI.

Information bias

One major source of information bias is the self-report questions used in the prediction models. Reliability of self-report data is of particular concern for behaviour that occurs during sleep, such as snoring and apnoeas. There is also evidence that suggests that men may tend to over report their snoring (Wiggins et al. 1990, Young et al. 2002a). Therefore, to address the possibility of information bias, self-reported measures of snoring and observed apnoeas were compared with objective measures.

In the community sample, when compared to the actual percentage of the night spent snoring, subjective snoring captured objective snoring to some extent. Reporting snoring *always* had a higher degree of discrimination for increased actual snoring compared to reporting snoring *often*, especially for non-Māori men. For those who reported snoring *often* or *always*, 76% snored for at least 10% of the night. However, only 20% snored for at least 50% of the night. For those participants who reported *never* or *rarely* snoring, the majority snored for less than 25% of the night. Similar relationships were seen in the clinical sample. For those who reported snoring *often* or *always* 31% snored for at least 25% of the night. On the other hand, of those who reported that they *never* or *rarely* snore, 17% snored for at least 25% of the night.

Of the 49 participants who reported observed apnoeas in the community sample, 39% had actual apnoeas (RDIa ≥ 5). Māori men were more accurate than others at reporting apnoeas, 56% of those who reported apnoeas had apnoeas detected by the MESAM4. Conversely, of the 312 participants who reported no observed apnoeas, 93% were correct. Across groups, the distribution was similar. Overall, the reporting of no observed apnoeas was more accurate than the reporting of observed apnoeas in the community sample. In the clinical sample, 66% (n=364) of those who reported observed apnoeas had actual events. In contrast, only 41% of those who reported no apnoea had actual apnoeic events. Thus the subjective measure of observed apnoeas had some, but less discriminatory ability in this population compared to the community sample. Although the relationships between the subjective and objective measures are in the expected direction, these results are limited as they are restricted to only one night of recording.

In comparison, the Australian population study (Bearpark et al. 1995) found greater discrepancies between reported and recorded snoring. Of the 48 participants who reported *never* or *hardly ever* snoring, only 29% (n=14) did not snore. The remaining

71% snored for at least half the night. On the other hand, of the 29 who reported always snoring, 14% (n=4) did not snore on the study night. Among those participants who reported at least some witnessed apnoeas (n=76), only 27 (35%) had RDI ≥ 5.

Although *don't know* was not a valid answer for the question pertaining to snoring it was a consideration given that the risk factors for those who report *don't know* have been shown to be similar to those for frequent snoring and included, male sex, higher BMI, smoking and use of sinus medication (Bliwise et al. 1999). In the present study, the majority of participants (16/22) who responded in this manner snored for less than 25% of the night. However, there were a small portion of women, both Māori and non-Māori who actually snored for 50-70% of the night, which may lend support to the suggestion that women tend to underreport snoring (Larsson et al. 2003). Future studies should consider the *don't know* response to questions about snoring as a response of potential interest.

Generalisability

The screening tool is expected to be more reliable for the specific age range (30-60 years) used in this study. With regard to ethnicity, given that the non-Māori ethnic group included a mix of different ethnicities, but was mostly comprised of New Zealanders of European descent, it is possible that other ethnic groups may not be accurately screened with the proposed tool.

The predictive characteristics of OSAS may also vary from one population to another, as do the distributions of other clinical characteristics including co-morbid conditions, age and ethnicity. These differences may affect the sensitivity and specificity of the screening tool for different populations. However, since the models were based on both clinical and community data it would be expected that the characteristic of patients may be closely aligned with those in a primary care setting, especially considering that the majority of clinical sample were referred from primary care. It has been suggested that using models such as the one developed in the present study in an independent setting, the intercept of the logistic regression may be removed, to provide a unit less measure of relative OSA risk (Maislin et al. 1995).

7.4 Summary of Key Findings

The results of this study show that for 30-60 year olds, OSA is highly prevalent in New Zealand, and that the prevalence among Māori is significantly greater. This is not surprising given the national sleep survey results (Harris 2003), which show that population prevalence estimates of OSAS symptoms and risk factors are significantly higher among Māori compared with non-Māori for both men and women.

The higher risk of OSA among Māori was shown to be attributable to well-recognised risk factors such as BMI, male sex and neck circumference, rather than ethnicity per se. However, whether the independent predictors of OSA were different for Māori and non-Māori, could not be determined in the present study due to the small number of OSA cases in the community sample.

The multivariate predictive tools provided reliable estimates of *a priori* probability of OSA, with sensitivities ranging from 71.90%-80.10%, specificities ranging from 81.50-86.60%, PPV 65.60-60.36%, and NPV 89.68-92.06%. These results indicate that these tools may have good clinical utility as screening tests, and have the potential to increase case recognition in a primary care setting. However, the generalisability and external validity of these models have yet to be tested in an independent setting.

7.5 Implications and Recommendations

A number of implications can be taken from this study, relating specifically to the recognition, diagnosis and management of OSAS in New Zealand, and to Māori health development and the elimination of disparities.

The findings of this study highlight that OSA and OSAS are common problems in New Zealand. Therefore, identification of patients with this condition is an important public health issue. However, one major factor hindering the recognition of OSA as a serious health problem in New Zealand is the lack of education among both the general public and health care professionals. It is anticipated that the results of this study may lead to an increased awareness of OSAS in New Zealand.

The ethnic prevalence differences for OSA found in this study, suggest that Māori are significantly more likely to suffer from OSA than non-Māori. With regard to ethnic inequalities, the disparities between Māori and non-Māori are of particular concern, not only in terms of increased needs, but also because they breach Māori rights under the Treaty of Waitangi. In order to improve the overall health of New Zealanders, particular attention must be given to those with the poorest health. For that reason, focusing on eliminating Māori health inequalities will benefit not only Māori, but also all New Zealanders. In general, policies and interventions are likely to be more appropriate to the task of reducing inequalities in health if they are underpinned by the principles of the Treaty. This recognises that all New Zealanders should have equitable access to health services.

Specialist sleep services

The rapid pace of advances in sleep disorders medicine has lead to the development of treatment services in many countries being driven primarily by perceived business opportunities, rather than the distribution of need in the community. New Zealand has lagged behind in the provision of services, but the epidemiological ground work in the present study and the national sleep study (Harris 2003) provides a unique opportunity to take an evidence-based approach to the development of services targeted to reach those most in need. As well as contributing to the health of all New Zealanders, this work provides a model for other countries.

Current specialist sleep services are unable to meet population health care needs (Neill et al. 2000). The findings of the present study and the national sleep survey (Harris 2003) provide firm evidence of the need for an increase in the number of sleep services. However, the uneven distribution of disease shown in both the clinical and community samples, in particular the disparities shown between Māori and non-Māori, should be an important consideration in effective and efficient planning for such services.

In the present study, the distribution of patients seen at the clinic did not reflect the distribution of disease shown in the community sample. The findings in the community sample indicate that the risk of OSA for Māori compared to non-Māori was approximately 3-4:1. Taking into account the ethnic profile of the Wellington region for persons aged 30-60 years (~14% Māori, 86% non-Māori & ~50% female, 50% male) and the estimated OSA prevalence indicated in the current study, the ratio of Māori to non-Māori men in the clinic sample should be approximately 2:1. The ratio however is 1:6 in favour of non- Māori men. This pattern of discrepancy is also similar for Māori and non-Māori women. These discrepancies suggest, that, for Māori there are significant barriers in accessing specialist sleep services.

Approximately half of the specialist services currently available are privately funded, requiring patients to pay a substantial amount or to have medical insurance, which are both likely to be significant barriers for Māori, as Māori are disproportionately represented among the most deprived sectors of New Zealand society (Crampton et al. 2000b) and are less likely to have medical insurance than non-Māori (Te Puni Kōkiri 2000). This is reflected in the clinical sample, where Māori patients were less likely to access the clinic privately. The current mix of public and private services may also contribute to increasing disparities in OSAS, especially if services are generally more accessible to non-Māori.

Other studies also suggest that Māori are experiencing barriers to specialist sleep care, with reports of more severe OSAS among Māori seen at sleep clinics (Frith and Cant 1985, Baldwin et al. 1998). In general, it has been shown that Māori receive less primary health care than might be expected and that Māori are also referred less often for specialist services (Baxter 2002). It is imperative that these issues are addressed as part of the efforts to eliminate disparities in the management of OSAS.

Given the higher need for sleep services among Māori, increased public funding is required. However, increased publicly funded services may paradoxically increase ethnic disparities whereby non-Māori, who are currently accessing private services, may change to publicly funded services if they become more readily available, serving only to shift costs without addressing disparities (Harris 2003). Therefore service development must ensure that Māori needs are met in order to address both population needs and inequalities. It has been suggested that achievement of a Māori quota may prevent further disparities (Harris 2003).

Although polysomnography is the gold standard for diagnosis, given the findings of the current study, and the inability of current services to adequately meet public demand, alternative diagnostic strategies should be evaluated. At present smaller centres in New Zealand do undertake more limited diagnostic studies. However there is debate in the literature about the use of such studies and their limitations (Flemons et al. 2003).

Whilst a screening tool for OSAS may improve the appropriate referral of Māori to specialist sleep services, the quality of health services for Māori is still likely to be of concern. Quality of service relates to effectiveness, accessibility, appropriateness and safety. Often health services are delivered in an inappropriate manner that disregards Māori cultural values. Therefore cultural safety within a framework of biculturalism should be an important component of health services (Richardson 2004). Furthermore, the consideration of the wider context within which Māori suffer from OSAS and other health problems, including barriers to care and broader issues of disparities in the distribution of the determinants of health, are important in terms of effective delivery of services.

These issues highlight the need for services to monitor how well they are delivering services to Māori. A commitment to eliminating ethnic health disparities requires a commitment to improving ethnicity data quality, which is in line with strategies of the current government (Ministry of Health 2000, 2002a, 2002b).

Public health

Population based strategies are critically needed to decrease the high prevalence and associated morbidity of OSAS in the community. While it is important to tackle lifestyle behaviours (such as smoking, nutrition and physical activity), this must be only

one part of the overall strategy. A concerted effort to address ethnic inequalities in OSAS will mean shifting the focus away from individuals to the wider determinants of lifestyle risk behaviours and the social, political, economic, and physical environment in which people live (Te Puni Kōkiri 2000, Ministry of Health 2002b, Ajwani et al. 2003).

As is suggested by the results of the present study, the national sleep survey (Harris 2003) and other clinical studies (Baldwin et al. 1998), obesity is an important contributing factor in health disparities between Māori and non-Māori. Obesity is a growing problem in New Zealand, between 1989 and 1997, prevalence estimates of obesity increased from 11% to 17% (Russel et al. 1999, Ministry of Health 2003b, 2003c). The expected continued increase in obesity levels is likely to impact unfavourably on population OSAS prevalence (Ministry of Health 2003b, 2003c).

Clinical and public health strategies for reducing obesity will be effective approaches to the management of OSAS, however they need to be equally effective for Māori and non-Māori to prevent further disparities. Recently, the Ministry of Health launched the Oranga Kai–Oranga Pumau Strategy (Health Eating – Healthy Action), which aims to improve nutrition, increase physical activity and reduce obesity for all New Zealanders (Ministry of Health 2003b). In terms of Māori health, the strategy identifies pathways for Māori health progress, which include increased Māori participation and effective health and disability services for Māori. However, strategies such as these need to be carefully implemented so as not to be victim blaming or to unintentionally contribute to the widening of gaps between Māori and non-Māori.

Primary care setting

OSAS is a disorder with serious medical, socioeconomic and psychological consequences, yet most patients with OSAS remain undetected (Young et al. 1997). General Practitioners (GPs), as the source of most referrals, have a vital role in screening for these patients. It is hoped that the results of this study in general will lead to an increased awareness of OSAS among medical professionals in New Zealand. There is also a need for increased information regarding sleep disorders in general and available treatments, which will allow medical professionals to make informed decisions (Neill et al. 2002).

Essentially the decision faced by GPs is whether a particular patient has features suggestive of OSAS that warrant referral to a respiratory physician. The process of clinical decision-making is complex. However, being able to ascertain the patients' likelihood of OSA using a prediction model may assist in the decision to refer a patient or not. However, in isolation, the dichotomous nature of these models is not an optimal indicator of OSAS severity. Therefore the need for referral must take into account other important clinical features such as symptom severity, quality of life, and the presence or absence of co-morbid conditions, especially cardiovascular diseases. Obviously, for patients with severe symptoms there is a greater need for accuracy, therefore a lower threshold for referral may be needed. Clinical judgement is also needed to detect unusual cases, or to recognise causes for daytime sleepiness other than OSAS, such as narcolepsy, anaemia, and insufficient sleep.

Although ethnicity was not a predictive variable in the prediction models, ethnicity is still an important consideration, given the higher occurrence of OSA among Māori, especially Māori men. This information is important in targeted case-finding and may assist in reducing known barriers in accessibility of specialist services for Māori. Furthermore, the identification of Māori patients with OSAS may be particularly important in the management of co-morbid diseases that affect Māori disproportionately. These issues highlight the importance of collecting ethnicity data. It is important to note however, that ethnicity should be considered a marker of risk that potentially relates to differential experiences and exposures rather than an inherent problem or genetic predisposition with being Māori.

The risk for OSAS among women is also an important consideration, particularly for Māori women. The few Māori women seen in the clinical sample (n=15) suggests that they may have disproportionately more problems with access to care than any other group, even though they experience higher risk than their non-Māori counterparts.

Primary care is also an important setting for the management of obesity. However, as mentioned previously, it is important that the social and structural context of obesity is taken into account. For example, physical activity is influenced by environmental factors, and the choice of activity may be limited for those in lower socioeconomic groups (Te Puni Kōkiri 2000, Ministry of Health 2002b). Effective treatment of obesity is of undoubted importance in OSA, and if it can be achieved it can produce marked

improvement in patients (Smith et al. 1985, Peppard et al. 2000). However, focusing purely on weight loss is a rather ineffective approach, particularly in severe OSAS cases.

Research

Finally, this study along with other studies that have utilised KMR methodology (Harris 2003, Paine et al. in press), have implications for research in general. The principles used in these studies are not confined to KMR and can be applied to other research. The key features include centering Māori rather than non-Māori or the total population; equal explanatory power; equal analytical power; and the importance of collection and classification of ethnicity. These principles may also be relevant internationally for populations where indigenous or ethnic minorities suffer health disparities (Harris 2003)

7.6 Further Research

Application of a clinical screening tool in primary care is limited until the prediction models can be validated in this setting. This exercise will provide insights into which of the two models is more accurate in this population. A study is currently underway to prospectively validate the models in patients referred to the sleep specialist from primary care. The necessary variables to estimate the likelihood of OSA have been embedded into the referral forms required by GPs for referral of patients to the respiratory medicine clinic at Wellington Hospital. These models will also be prospectively evaluated alongside other prediction models (Croker et al. 1990, Viner et al. 1991, Flemons et al. 1994). Upon acceptable validation, a package will be developed for GPs, which will include simple software to calculate the likelihood of OSA for a given patient along with some guidelines for its use.

A key element of a screening tool is the ease of implementation. From a practical perspective, it is only of value if it is incorporated into routine clinical practice. Therefore in order to achieve widespread acceptability and usage, further work may be needed to further simplify the models, which will allow decisions to be made quickly without dependence on auxiliary devices, such as a computer. Flemons (2000) provides a good example of such a model.

More accurate estimates of OSA and OSAS prevalence for Māori and non-Māori will be gained from the combination of data from the present study with data from the

national sleep survey (Harris 2003). In particular, the multivariate predictive model that includes neck circumference (Model 2a) will be applied to the national sleep survey data (Harris 2003) to derive national estimates of OSA and OSAS. A project is currently being planned to refine prevalence estimates and undertake an analysis of the economic and social costs/burden of OSAS in New Zealand. This project will provide essential information for health policy makers to establish priorities and budgets, and for economic evaluation of treatment options. This work will also provide a model for other countries.

A review of international literature indicates that detection of OSAS remains low in primary care settings, even after prominent events, such as a car crash that resulted from falling asleep at the wheel (Kramer et al. 1999, Millman 1999, Netzer et al. 1999). Preliminary results from a small study in New Zealand also indicate a low rate of recognition of OSA amongst GPs (Dr Jai Sood 2004, pers. comm.). Accurate recognition of sleep problems is essential, not only for treating individuals suffering from them, but also for assessing the costs of sleep disorders to society, and for making decisions about the use of limited health care resources in their diagnosis and treatment. More research is needed to further assess the level of awareness and recognition of OSAS throughout primary care settings in New Zealand.

Qualitative research in the area of sleep is scant, but valuable information may be gained using qualitative methods in assessing the impact of OSAS on individuals and their families. This type of research will also be useful in evaluating the appropriateness and effectiveness of current sleep services for Māori. A qualitative study is currently underway to assess potential barriers to accessing sleep services among Māori and Pacific taxi drivers (Riz Firestone 2003, pers. comm.). This study will utilise one of the multivariate predictive models (Model 2a) developed in the present study as a means of identifying drivers at high risk of OSAS for allocation to focus groups.

Finally, this thesis along with the national sleep survey (Harris 2003) emphasises the need for increased Kaupapa Māori Research in health as a means of prioritising Māori needs and providing Māori specific information. These studies also highlight the importance of understanding the data quality issues in the classification of ethnicity for providing useful information to adequately inform health policy and interventions.

7.7 Conclusions

This thesis provides strong population based information on the prevalence of OSA, which indicates that OSA is a common problem in New Zealand. It also demonstrates another area of health where Māori are disproportionately affected. Together with findings from the national sleep survey (Harris 2003), important information is provided from which to adequately plan and provide appropriate services to reduce ethnic health disparities and meet the needs of the New Zealand population.

The multivariate predictive models provide a simple, reliable and accurate method that may be used in a primary care setting to identify individuals at low and high risk of OSA. However, these models still need to be validated in a primary care setting. More reliable and systematic identification of OSA will assist in the referral of patients to specialist services, thereby reducing pressure on available resources, and reducing costs associated with inappropriate referrals. Furthermore, better management of OSAS may also assist in the management of other adverse outcomes that affect Māori disproportionately.

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APPENDIX 1
COMMUNITY INSTRUCTIONS FOR THE NIGHT

INSTRUCTIONS FOR THE NIGHT

There are no personal risks involved in wearing the sleep equipment. The **green lights** on the recording box are testing lights; they will all go out after 10 minutes. The red light on your finger will also do out and re-appear at the set recording time.

The **marker button** we ask you to press is **not an on/off switch**. Please press the marker button when:

1. you decide to try and sleep at night
2. you wake during the night for longer than 10 minutes
3. you decide to try and go back to sleep again
4. and finally when you wake up in the morning

The recorder should always be carried in the bag over your shoulder and none of the cables sensors or recorder should be pulled or detached.

Do not open the battery container or pull the plug out of the recorder socket.

Please be careful with the recorder and cables.

When you get into bed place the recorder under your pillow.

Under no circumstances should the sensor cables or recorder come into contact with water.

If any of the leads come off during the night use the tape to stick them back on.

If anything comes unplugged during the night, please plug it back into the same coloured socket.

In the morning when you wake, you can carefully detach all equipment from your body – you do not need to unplug any of the connections.

In case of an emergency or if you are unsure about anything you can contact:

_____

APPENDIX 2
VALIDATION STUDY- MESAM4 VERSUS PSG

One major problem conducting an epidemiological study of obstructive sleep apnoea (OSA) is the high cost involved. The gold standard diagnostic test for obstructive sleep apnoea syndrome (OSAS) is standard polysomnography (PSG), which is costly in terms of both time and money. This report describes a study that aimed to assess the feasibility and reliability of the use of the MESAM4[16] sleep recording system in the collection of population prevalence data in a home setting.

The MESAM4 is a simple four-channelled digital system that records, heart rate, respiratory sounds, arterial oxygen saturation, and body position. Previous studies have evaluated the diagnostic validity of MESAM4 by way of simultaneous recordings with polysomnography equipment with varying scoring methods and results. The general consensus from these studies is that manual scoring of MESAM4 traces is superior to the automatic scoring available (Stoohs and Guilleminault, 1992, Roos et al. 1993, Bearkpark et al. 1995, Esnaola et al. 1996, Cirignotta et al. 2000).

Methods and Procedures

Sixteen participants with varying levels of OSAS were recruited from the sleep clinic (Wellsleep) in Wellington and participants from a community study, to simultaneously wear the MESAM4 and PSG equipment overnight.

Overnight polysomnographic data were collected for clinic patients using the Compumedics™ computerized system. Studies included both attended clinic and unattended home polysomnography.

Polysomnography consisted of electroencephalography (EEG); electrooculography (EOG); and chin electromyography (EMG) to identify sleep stages, electrocardiogram (ECG), thermistry and nasal prongs to measure nasal and oral airflow, oximetry to measure oxyhemoglobin saturation. Thoracic and abdominal bands were used to measure respiratory effort. A position sensor was attached to the thoracic band to

[16] The word MESAM4 is an acronym derived from the name "Madaus Elektronik Sleep Apnoea Monitor"; with the '4' indicating that it is a four-channel version of the device.

measure body position. Leg paddles were attached to each of the outer calve muscles to measure leg movements. This enabled screening of another sleep disorder known as periodic limb movements (PLM), which has some daytime symptoms in common with OSA (Guilleminault and Anagnos, 2000, Stoohs et al 2001). The MESAM4 was worn in a pouch with a shoulder strap while the subject was mobile and placed under their pillow whilst in bed.

Scoring Criteria

Polysomnography

Sleep stages were scored according to standard criteria (Rechschaffen and Kales 1968). Respiratory events were scored according to the American Sleep Disorders Association (1995) criteria. An apnoeic event was defined as the cessation of nasal and oral airflow for at least 10 seconds and a hypopnoea defined as at least 50% reduction of at least 2 out of 3 signals (airflow, thoracic, abdominal movements) for 10 seconds or more.

MESAM4

MESAM4 studies were printed out via a computer and blindly[17] scored by an experienced scorer in five-minute epochs. Epochs were excluded from further analyses if artefact was found in any of the four signals for more than half of the epoch. Artefact occurred for a variety of reasons, including movement, electrode displacement and technical failure. Sleep onset was determined by drop in mean heart rate (HR). If baseline HR changed abruptly (≥ 3bpm) and maintained the new rate, this was considered indicative of the person being awake, and was scored as awake if it persisted for more than half of an epoch.

Apnoeas were scored if there was an episode of oxygen desaturation of ≥ 4% from the preceding baseline in conjunction with (1) an increase in heart rate (HR) of at least 10 beats per minute, or (2) a burst of snoring associated with commencement and termination of a desaturation episode, or (3) both 1 and 2 (Bearpark et al. 1995). Hypopnoeas were scored if there was a peak increase in HR by at least 10 beats per minute above the preceding baseline, in addition to snoring (Penzel et al. 1990). In

[17] The scorer did not know the identity of the participant, or the results of respective polysomnographic recordings.

order to differentiate the two types of events they were scored using different symbols (↓=Apnoea □=Hypopnoea).

Two respiratory disturbance indices were calculated from the scored apnoeas and hypopnoea. RDIa was the total number of scored apnoeas divided by the estimated total sleep time in hours. RDIc was the total number of both apnoeas and hypopnoea divided by the estimated total sleep time in hours.

Analyses

The strength of association between polysomnographic and MESAM4 recordings, for estimated total sleep time, scored apnoeas and hypopnoeas, was examined using the Spearman's rank test. Agreement between scoring methods was assessed using Bland-Altman (1986) plots, along with the mean of the differences between the PSG and MESAM4 (instrument bias) and the limits of agreement (2SD of the mean of the differences).

Raw scored events rather than calculated respiratory indices were compared between the two systems in order to control for estimated total sleep time (which is not necessarily accurately scored in the MESAM4 recordings).

Results

Six participants were excluded from the study due to varying technical issues. The final sample consisted of 12 participants (10 Men, 2 Women) whose median age was 52 years (SD=11.31 years, Range=33-74 years) and median BMI was 30.58 kg/m^2 (SD=4.46 kg/m^2, Range=25.10-40.10 kg/m^2). Three of the 12 studies were conducted as home studies.

Total sleep time

MESAM4 estimated total sleep time (TST) was strongly correlated with PSG total sleep time (r_s =0.93, 95% CI 0.75 – 0.98, p<0.0001).

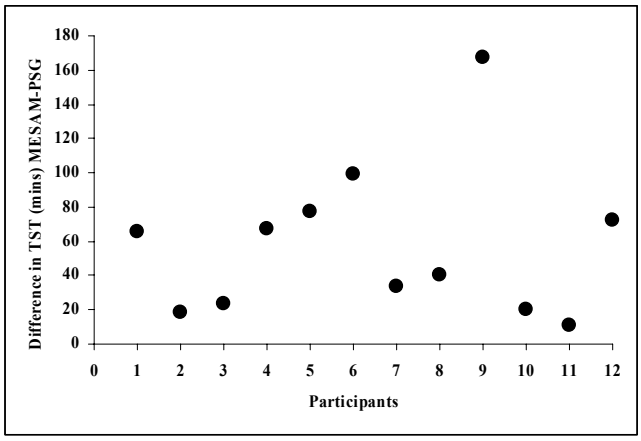

Figure 1. PSG versus MESAM4: Difference in Total Sleep Time (mins) for each participant

However as illustrated in Figure 1, TST was consistently overestimated by the MESAM4 for all participants (Median= 53mins, Range=10mins – 174mins).

Apnoeas and Hypopnoeas

The number of apnoeic events scored by the MESAM4 was strongly correlated with the PSG (r_s=0.98, 95% CI 0.94-1.00, p<0.0001). However in terms of agreement, the MESAM4 overestimated the number of apnoea in 83% of the participants, with a mean difference of 17.25 (95% CI-2.71-37.21) (Figure 2). The width of the limits of agreement (-44.32 to 78.81) indicates extensive variability between the two systems.

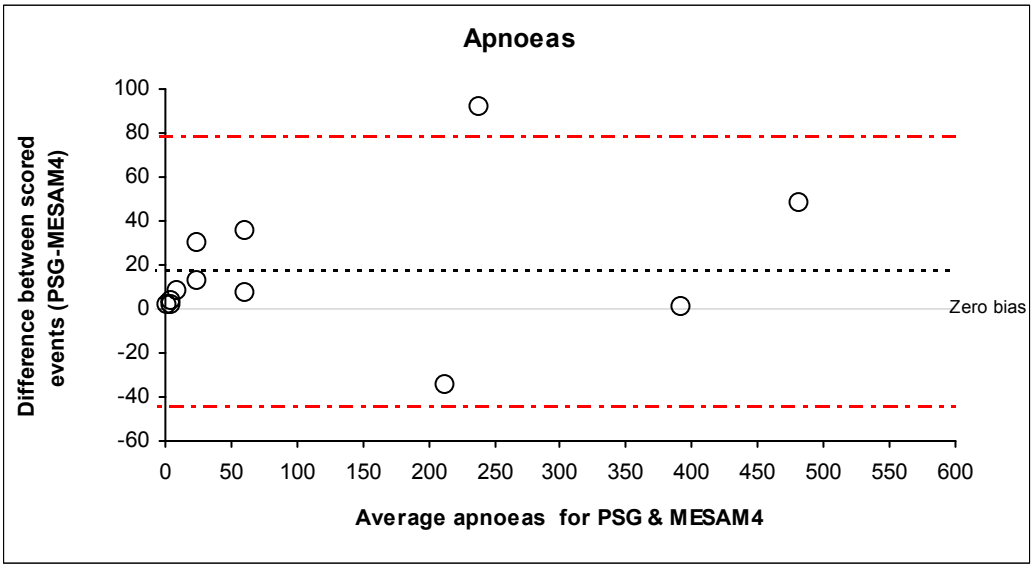

Figure 2 **Bland-Altman plot of illustrating the agreement between the MESAM4 and PSG (Dashed line indicates mean difference between the MESAM4 and PSG)**

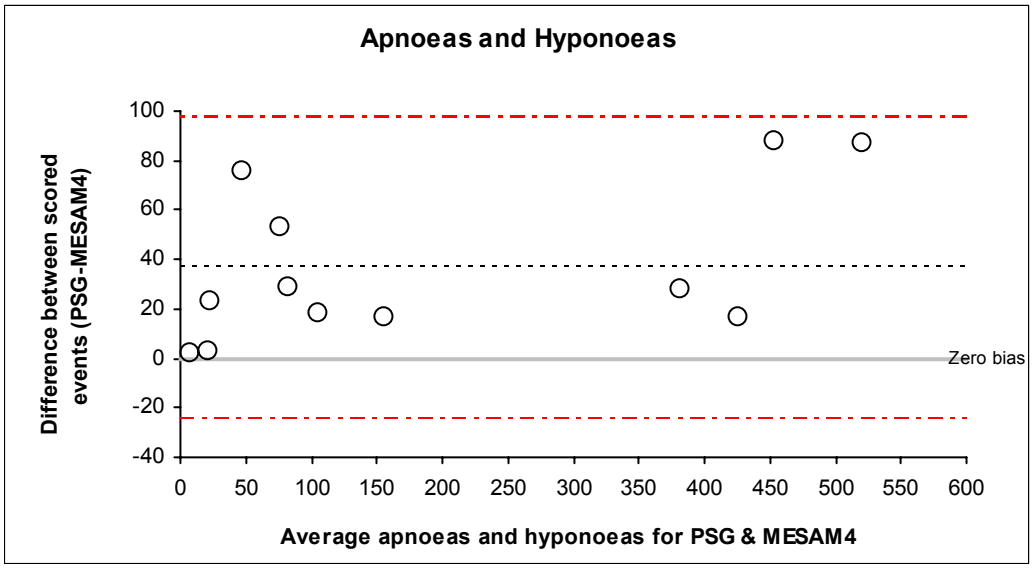

Figure 3 **Bland-Altman plot of the difference between scored apnoeas and hypopnoeas against the mean number of scored apnoea and hypopnoea. (Dashed line indicates mean difference between the MESAM4 and PSG)**

The total number of scored apnoeas and hypopnoeas correlated strongly with the PSG (r_s =0.99, 95% CI 0.96-1.00, p<0.0001) (Figure 3). However the limits of agreement were wider than scored apnoeas only, ranging from -24.55 to 98.05 (Mean Difference=36.75, 95% CI=16.88-56.62). Visual inspection of both Bland-Altman plots (Figure 2 and 3) does not indicate any systematic pattern in the disagreement between the two, which is most likely due to the small sample size.

Sensitivity and Specificity

In order to evaluate the discriminatory ability of the MESAM4, RDI thresholds of ≥ 5 and ≥ 10 were used to define abnormal respiratory events. Table 1 summarizes the validity of each index at each threshold. All participants who were classified abnormal via PSG were similarly classified with the MESAM4. Specificity was noticeably lower for RDIc. This was primarily due to one patient who was eventually diagnosed with severe periodic limb movements (PLMS). For RDIa≥ 10, respiratory disturbance was correctly classified in 5 of the 6 patients (PPV=0.83, 95% CI 0.36-0.99).

Table 1. Sensitivity, specificity, PPV, NPV of the MESAM4 system

	≥ 5		**≥ 10**	
	RDIa	**RDIc**	**RDIa**	**RDIc**
Sensitivity	100%	100%	100%	100%
Specificity	83%	25%	86%	67%
PPV	86%	73%	83%	89%
NPV	100%	100%	100%	100%

Summary

In line with previous validation studies, manual scoring of the MESAM4 correlated strongly with scoring of PSG. However agreement between the two systems varied significantly. Estimated total sleep times were consistently overestimated by the MESAM4 criteria, which highlight the need for additional information to assist the scorer in determining sleep onset and awakenings during the night. Accuracy could be improved by collecting subjective reports from participants regarding their sleep and wake times. The MESAM4 also has an event marker button where the participant can reference awakenings. This feature, however, was not utilised in this particular study.

The degree of disagreement between the two systems was more pronounced when hypopnoea events were compared. This would suggest that the criteria used for scoring hypopnoeas from the MESAM4 traces needs to be re-evaluated and validated. Despite this, the discriminatory ability of the MESAM4 system was sufficiently sensitive to allow detection of patients within the thresholds required for the prevalence estimates.

One major limitation of this study was the method used to compare the MESAM4 and PSG. Due to time constraints, comparisons were not conducted temporally; epoch-by-epoch, but rather only the total number of respiratory events for each study were

compared. Epoch-by-epoch comparison would have provided a clearer picture of the agreement between the two systems. The findings of this study may therefore underestimate the degree of disagreement between the MESAM4 and PSG. Furthermore, the small sample size, made it hard to explore systematic differences between the two systems.

Overall, the findings from this study suggest that the measurement of respiratory disturbance by the MESAM4 is valid and adequate for the assessment of sleep-related respiratory disturbances for use in epidemiological studies of general populations. Nevertheless, it is certainly not a replacement for polysomnography.

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APPENDIX 3
THE SLEEP QUESTIONNAIRE

APPENDIX 4
COMMUNITY SLEEPLOG

COMMUNITY SLEEPLOG

MESAM Number: _____ Setup Techs: _____

Recording Start Time: _____ pm

ID Number		Date					
Height	m	Weight	kg	BMI	Kg/m^2		
Neck Size	cm	BP (sys/dia)		Position	Time:		
Usual Bedtime	Hrs	Alcohol taken tonight (Y/N)		Is this usual? (Y/N)			
Usual Wake time	Hrs	Caffeine taken tonight (Y/N)		Is this usual? (Y/N)			
Current smoker? (Y/N)		Sedative medication (Y/N)		Given tonight? (Y/N)			
Medical & Medication – Comments							

MORNING QUESTIONNAIRE

1. What time did you go to sleep? _____
2. Did you awake during the night? Yes / No, if yes Time/s? _____ _____ _____
3. What time did you wake up in the morning? _____

   Estimate total sleep time: ____:_____ (hours: mins)
4. How would you rate your sleep last night, compared to a normal nights sleep for you?

   1 much worse 2 3 typical 4 5 much better
5. Did you have any difficulties with the sleep equipment?

   _____

   _____

APPENDIX 5
COMMUNITY COVERING LETTERS

19th April 2001

«ID»
«FIRSTNAME» «SURNAME1»
«FLAT»«HOUSENUM»«HOUSE_ALPHA» «STREET1»
«TOWN» «TOWNPOSTCODE»

Tēnā koe

We would like you to consider taking part in a study to find out how many people in New Zealand have problems breathing while they are asleep, including snoring and a problem known as Obstructive Sleep Apnoea (OSA). With this letter you will find an information sheet giving details of the study.

We are looking for people aged from 30 to 60 years, and living in the Wellington region. Your name has been randomly selected from the electoral roll.

We will be phoning you in the next couple of days, to see if you would like to be in the study and answer any questions that you might have about it. If you decide that you would like to be in the study, we will arrange with you one convenient evening, and two members of the research team will come to your home. They will give you a questionnaire to fill out, measure your blood pressure, weigh you, measure your height, and set up the sleep monitor. This will take approximately 30-40 minutes. In the morning you can take the equipment off yourself and they will come and collect it at a time that suits you.

Being in the study is entirely your own choice. It is important for you to know that your information will be kept completely confidential. If you do decide to be in the study, you can still choose to pull out at any time without having to give a reason. Whatever you choose, it will in no way affect your future health care. If your sleep recording suggests that you may have a problem with OSA, we will tell you, and your family doctor if you wish, and suggest where you can go for further evaluation and treatment.

This research is funded by the Health Research Council of New Zealand. Ethical approval has been given by the Wellington Ethics Committee. If you have any concerns or queries about the study, you may contact the committee: Wellington Ethics Committee, Wellington Hospital telephone 385-5999 ext 5185.

You can also call us with any concerns or queries about the study at the phone numbers given.

We have this phone number for contacting you (04) «Phone».
If this is not your phone number and you would like to be in the study, please contact me (Kara) on 918-6505 or email kmihaere@wnmeds.ac.nz.

Your participation in this study would be much appreciated.

Nāku noa

Kara Mihaere
Junior Research Fellow

Nb: Please leave this letter and tape measure (to measure your neck size) near the phone

19th April 2001

«ID»
«FIRSTNAME» «SURNAME1»
«FLAT»«HOUSENUM»«HOUSE_ALPHA» «STREET1»
«TOWN» «TOWNPOSTCODE»

Dear Sir/Madam

We would like you to consider taking part in a study to find out how many people in New Zealand have problems breathing while they are asleep, including snoring and a problem known as Obstructive Sleep Apnoea (OSA). With this letter you will find an information sheet giving details of the study.

We are looking for people aged from 30 to 60 years, and living in the Wellington region. Your name has been randomly selected from the electoral roll.

We will be phoning you in the next couple of days, to see if you would like to be in the study and answer any questions that you might have about it. If you decide that you would like to be in the study, we will arrange with you one convenient evening, and two members of the research team will come to your home. They will give you a questionnaire to fill out, measure your blood pressure, weigh you, measure your height, and set up the sleep monitor. This will take approximately 30-40 minutes. In the morning you can take the equipment off yourself and they will come and collect it at a time that suits you.

Being in the study is entirely your own choice. It is important for you to know that your information will be kept completely confidential. If you do decide to be in the study, you can still choose to pull out at any time without having to give a reason. Whatever you choose, it will in no way affect your future health care. If your sleep recording suggests that you may have a problem with OSA, we will tell you, and your family doctor if you wish, and suggest where you can go for further evaluation and treatment.

This research is funded by the Health Research Council of New Zealand. Ethical approval has been given by the Wellington Ethics Committee. If you have any concerns or queries about the study, you may contact the committee: Wellington Ethics Committee, Wellington Hospital telephone 385-5999 ext 5185.

You can also call us with any concerns or queries about the study at the phone numbers given.

We have this phone number for contacting you (04) «Phone».
If this is not your phone number and you would like to be in the study, please contact me (Kara) on 918-6505 or email kmihaere@wnmeds.ac.nz.

Your participation in this study would be much appreciated.
Yours sincerely

Kara Mihaere
Junior Research Fellow

Nb: Please leave this letter and tape measure (to measure your neck size) near the phone

APPENDIX 6
COMMUNITY INFORMATION SHEET

Department of Public Health
Wellington School of Medicine

Information Sheet

What is Obstructive Sleep Apnoea (OSA)?

If you have OSA, when you relax and fall asleep, your airway collapses and you cannot breathe. After a while (as much as several minutes) your brain wakes you up to make you breathe again. Usually you gasp or snore very loudly when this happens, but normally you wouldn't remember it in the morning. In severe cases, these breathing pauses can happen hundreds of times in the night.

People who have severe OSA are often very sleepy, because the quality of their sleep is disturbed. They wake up briefly throughout the night, but often don't remember it. Some research suggests that they might also be more likely to have other health problems, such as high blood pressure, heart attacks, or strokes. Being so sleepy, they may also have more accidents driving.

There are a variety of successful ways of treating OSA, and clinics that specialize in diagnosing and treating it.

What is this Study About?

At the moment, we have no idea how many people in New Zealand may have OSA, and compared to other countries; we have very few treatment clinics. There is some evidence that Māori and Pacific Island Peoples may be more affected than other New Zealanders. We need to know how many people are likely to be affected, and whether different groups may have special needs, so that we can argue for better treatment services for everybody.

Who is Being Asked to Participate?

Your name has been drawn at random from the Electoral Rolls for the Wellington Region, as part of a sample of 400 people aged 30-60 years.

What Happens if You Decide to Participate?

If you decide to be in the study, you will be asked to complete a short questionnaire, and to wear a special monitor for one night while you sleep at home. You will receive a phone call from the research team in the next few days, asking you if you would like to be in the study. Please feel free to ask any questions you may have. If you decide that you would like to be in the study, we will select an evening that is convenient for you, and two members of the research team will come to your home. They will give you a

questionnaire to fill out, measure your blood pressure, weigh you and measure your height, and set up the sleep monitor. This involves:

Three removable connections taped to your chest, to measure your heart rate;
- a tiny microphone taped just above your collarbone, to measure the sounds you make while you are asleep;
- a little monitor taped to your breastbone that detects whether you are lying on your back, side, or front; and
- a little plastic sleeve that fits over your finger tip and measures the amount of oxygen in your blood

These are attached by fine wires to a small recording box that you can carry around in a shoulder bag until you are ready to go to bed, and then put down beside the bed when you are ready to go to sleep. In the morning, the researchers will come back at a time that suits you to collect all the equipment. Your questionnaire and your sleep recording will be given a matching code number. All the information you give will be strictly confidential and your name will not be attached to it or used in any reports on this study.

Risks and Benefits

There are no personal risks involved in wearing the sleep recording equipment, and similar studies using this equipment have been done in other countries. If your sleep recording suggests that you may have a problem with OSA, we will tell you, and your family doctor if you wish, and suggest where you can go for further evaluation and treatment.

Participation

Being in the study is entirely your own choice. If you do decide to be in the study, you can still choose to pull out at any time without having to give a reason. Whatever you choose, it will in no way affect your future health care.

Contact Phone Numbers

Kara Mihaere	Junior Research Fellow	(04)
Philippa Gander	Associate Professor	(04)

APPENDIX 7
COMMUNITY CONSENT FORM

CONSENT FORM

1. I understand the letter dated _____ for volunteers taking part in the study designed to find out how many people in New Zealand suffer from obstructive sleep apnoea, and whether this has other effects on their health. I have had the opportunity to discuss this study. I am satisfied with the answers I have been given.

2. I understand that taking part in this study is voluntary (my choice), that I may withdraw from the study at any time, and that this will in no way affect my future health care.

3. I understand that my participation in this study is confidential and that no information which could identify me will be used in any reports on the study.

4. I have had enough time to consider whether to take part.

5. I know who to contact if I have any concerns or questions about the study.

6. If the results of my study indicate that I may need further evaluation, I consent to my GP being informed of my results **YES / NO.** If yes, please write your GPs name and center below.

   GP Name _____ Centre _____

7. I _____ hereby consent to take part in this study.

8. If you have any concerns about the study, you may contact: The Wellington Ethics Committee, Wellington Hospital, Phone 385 5999 ext 5185.

Signature _____ Witness Signature _____
Date _____

Contact Numbers

Kara Mihaere
Philippa Gander
Wai Wai

APPENDIX 8
COMMUNITY SLEEPLOG

Te Whare Wānanga o Otāgo

14th May 2001

Hi <<Name>>

Thank you for agreeing to participate in our Sleep study.

Before we come to your home, we would like you to have:

1. Changed into sleepwear (preferably 2 piece)
2. Eaten your dinner, washed dishes etc....
3. Signed consent form (we will have a spare copy)

We would also like you to try and follow your normal daily routine as close as possible.

We have arranged to visit you on: <<Date>><<Time>>

If this time becomes inconvenient please let me know on (Wk) / and another time can be arranged.

Kind regards

Kara Mihaere
Junior Research Fellow

APPENDIX 9
COMMUNITY RESULTS LETTER

WellSleep

Wellington School of Medicine Sleep Investigation Centre

Bowen Hospital
Churchill Drive
Crofton Downs
Wellington
Ph 04
Fax 04

<<Date>>

Dear Dr _____

Re: <<Participants name>>

<<Participants name>> agreed to participate in a research study carried out by the Wellington School of Medicine to measure the prevalence of obstructive sleep apnoea syndrome in the community. She requested the results of her study be forwarded to you.

The study was undertaken on the <<Date of study>> and demonstrated:

- ☐ no significant sleep disordered breathing
- ☐ mild obstructive sleep apnoea syndrome
- ☐ moderate obstructive sleep apnoea syndrome *****
- ☐ severe sleep apnoea syndrome *****
- ☐ Other

***** If significant obstructive sleep apnoea is demonstrated during this study you may wish to consider referring your patient for further evaluation at the Department of Respiratory Medicine – Sleep Disordered Breathing Clinic, Wellington Hospital.

Please find attached an information sheet regarding the study. If you have any concerns or would like further information about this study please contact either: Dr Angela Campbell, Laboratory Manager, Wellsleep Clinic – (04) or Dr Alister Neill – Clinical Director, Wellsleep Clinic – (04)

Yours sincerely

Dr Alister Neill

APPENDIX 10
CLINIC CONSENT FORM

WellSleep

CONSENT FORM FOR STUDY
ON OBSTRUCTIVE SLEEP APNOEA

1. I understand the information sheet for volunteers taking part in the study designed to find out how many people in New Zealand suffer from obstructive sleep apnoea, and whether this has other effects on their health. I have had the opportunity to discuss this study. I am satisfied with the answers I have been given.

2. I understand that taking part in this study is voluntary (my choice) and that I may withdraw from the study at any time, and that this will in no way affect my future health care.

3. I understand that my participation in this study is confidential and that no information which could identify me will be used in any reports on the study.

4. I have had enough time to consider whether to take part.

5. I know who to contact if I have any concerns or questions about the study.

6. I consent to my GP being informed of the results of my participation in this study **YES / NO**

7. I _____ hereby consent to take part in this study.

8. If you have any concerns about the study, you may contact: The Wellington Ethics Committee, Wellington Hospital, Phone 385 5999 ext 5185.

Signature _____ Date _____

Contacts

Associate Professor Philippa Gander	Phone
Dr Papaarangi Reid	Phone
Dr Alister Neill	Phone

Project explained by _____ Signature _____
Project role _____ Date _____

APPENDIX 11

1996 CENSUS POPULATION NUMBERS FOR MĀORI AND NON-MĀORI AGED 30-60 YEARS IN THE WELLINGTON REGION

	Māori ethnic group	non-Māori	Total
Male 30-39 years	3669	30354	34023
Male 40-49 years	2361	26040	28401
Male 50-59 years	1359	18270	19629
Female 30-39 years	4125	31902	36027
Female 40-49 years	2433	26364	28797
Female 50-59 years	1314	18384	19698
Total	15261	151314	166575

Source: SNZ 1997b, SNZ 1997c

APPENDIX 12
MULTIVARIATE ANALYSES

This appendix presents results that were not presented in Chapter 6 (Developing a Screening Tool). The results are presented as follows and form part of the discussion in Chapter 7:

Table 1. Possible univariate predictors of OSA (RDIc)

Variable	Description	RDIc ≥ 5			RDIc ≥ 10			RDIc ≥ 15		
		OR	95% CI	p-value	OR	95% CI	p-value	OR	95% CI	p-value
Ethnicity	Māori vs. non-Māori	0.58	0.43-0.81	0.0010	0.58	0.42-0.78	0.0005	0.64	0.46-0.88	0.0066
Sex	men vs. women	2.68	1.97-3.66	<0.0001	3.54	2.60-4.82	<0.0001	3.74	2.67-5.26	<0.0001
Age 1	yearly increments	1.03	1.01-1.05	0.0005	1.02	1.01-1.04	0.0092	1.02	1.00-1.04	0.0255
Age 2	10 year increments	1.31	1.10-1.56	0.0026	1.24	1.05-1.47	0.0141	1.21	1.01-1.45	0.0390
CSC Eligibility	yes vs. no	2.71	1.93-3.81	<0.0001	2.07	1.48-2.90	<0.0001	1.97	1.40-2.76	<0.0001
BMI 1	Increasing	1.19	1.16-1.23	<0.0001	1.15	1.12-1.18	<0.0001	1.14	1.11-1.67	<0.0001
BMI 2	overweight vs. ideal/underweigh	2.30	1.42-3.72	0.0008	2.57	1.55-5.30	0.0008	2.94	1.49-5.82	0.0019
	obese vs. ideal/underweight	12.51	7.84-19.96	<0.0001	16.00	8.95-28.70	<0.0001	14.43	7.59-27.45	<0.0001
Neck 1	cm increments	1.36	1.30-1.42	<0.0001	1.35	1.30-1.41	<0.0001	1.30	1.30-1.43	<0.0001
Neck 2	> national av. vs. < national av.	5.20	3.87-6.98	<0.0001	5.57	4.08-7.60	<0.0001	6.06	4.29-8.57	<0.0001
ESS 1	>10 vs. ≤10	4.68	3.43-6.40	<0.0001	3.83	2.88-5.10	<0.00001	3.74	1.79-5.01	<0.0001
ESS 2	11-15 vs. ≤10	3.16	2.24-4.46	<0.0001	3.11	2.16-4.47	<0.0001	2.91	1.97-4.30	<0.0001
	16+ vs. ≤10	6.16	4.20-9.02	<0.0001	6.05	4.13-8.86	<0.0001	6.51	4.39-9.65	<0.0001
Snore 1	always vs. never/rarely/often	6.12	4.25-8.82	<0.0001	4.84	3.55-6.58	<0.0001	4.60	3.30-6.25	<0.0001
Snore 2	often/always vs. never/rare	7.94	5.63-11.19	<0.0001	9.97	6.44-15.43	<0.0001	12.06	6.99-20.82	<0.0001
Snore 3	rarely vs. never	1.08	0.43-2.66	NS	0.76	0.24-2.37	NS	0.92	0.20-4.30	NS
	often vs. never	5.78	2.40-13.95	<0.0001	5.54	1.88-16.29	0.0019	7.87	1.84-33.70	0.0054
	always vs. never	17.76	7.15-44.12	<0.0001	14.06	4.75-4.61	<0.0001	19.50	4.55-83.50	<0.0001
	don't know vs. never	2.80	0.94-8.27	NS	1.62	0.42-6.21	NS	1.31	0.20-8.41	NS
Observed Apnoea	yes vs. no	8.66	6.25-12.00	<0.0001	8.00	5.90-10.86	<0.0001	4.38	6.72-13.05	<0.0001
Wake feeling refreshed 1	never/rarely vs. often/always	2.34	1.73-3.06	<0.0001	2.15	1.61-2.88	<0.0001	2.33	1.70-3.18	NS
Wake feeling refreshed 2	never vs. always	1.23	0.31-5.17	NS	1.50	0.43-5.19	NS	1.41	0.39-5.08	NS
	rarely vs. always	0.70	0.18-2.66	NS	1.15	0.35-3.83	NS	1.20	0.35-4.16	NS
	often vs. always	0.32	0.08-1.26	NS	0.55	0.16-1.85	NS	0.52	0.15-1.82	NS

Table 1. Possible univariate predictors of OSA (RDIc) (cont…)

Variable	Description	RDIa≥ 5			RDIa≥ 10			RDIa≥ 15		
		OR	95% CI	p-value	OR	95% CI	p-value	OR	95% CI	p-value
Getting enough sleep 1	never/rarely vs. often/always	0.49	0.37-0.64	<0.0001	0.55	0.42-0.72	<0.0001	0.53	0.40-0.71	<0.0001
Getting enough sleep 2	never vs. always	1.06	0.38-2.93	<0.0001	0.77	0.72-1.54	NS	0.73	0.31-1.75	NS
	rarely vs. always	0.57	0.24-1.36	NS	0.37	0.16-0.82	0.0144	0.40	0.19-0.86	0.0185
	often vs. always	0.31	0.13-0.74	0.00821	0.21	0.10-0.53	0.0004	0.25	0.11-0.53	<0.0001
Asthma	yes vs. no/don't know	1.31	0.90-1.92	NS	0.85	0.61-1.27	NS	0.63	0.42-1.00	NS
Hypertension	yes vs. no/don't know	3.12	2.07-4.66	<0.0001	2.45	1.73-3.46	<0.0001	2.56	1.81-3.61	<0.0001
Heart Trouble	yes vs. no/don't know	3.04	1.73-5.34	0.0001	2.49	1.56-3.98	0.0001	2.29	1.45-3.62	0.0004
Diabetes	yes vs. no/don't know	6.43	2.53-16.38	<0.0001	3.21	1.73-5.98	0.0002	3.00	1.67-5.37	0.0002
Stroke	yes vs. no/don't know	2.68	0.57-12.68	NS	1.95	0.55-6.96	NS	2.83	0.80-10.11	NS
Thyroid problem	yes vs. no/don't know	0.66	0.27-1.60	NS	1.30	0.54-3.15	NS	0.80	0.30-2.10	NS
Psychological problem	yes vs. no/don't know	1.80	0.99-3.26	NS	1.91	1.11-3.28	0.011	2.12	1.23-3.60	0.0063
Sleep problem	yes vs. no/don't know	1.45	0.74-2.84	NS	1.24	0.66-2.33	NS	0.97	0.50-1.88	NS
Smoking 1	regular/occasional vs. other	1.57	0.71-3.47	NS	1.30	0.63-2.70	NS	1.25	0.60-2.64	NS
Smoking 2	regular vs. other	1.08	0.75-1.54	NS	1.04	0.73-1.47	NS	1.08	0.75-1.56	NS
Smoking 3	regular vs. non-smoker	1.41	0.98-2.06	NS	1.31	0.90-1.91	NS	1.34	0.95-1.88	NS
	occasional vs. non-smoker	2.06	0.92-4.60	NS	1.64	0.78-3.44	NS	1.56	0.73-3.33	NS
	ex-smoker vs. non-smoker	2.02	1.46-2.80	<0.0001	1.77	1.30-2.41	0.0004	1.66	0.20-2.30	0.0020
Alcohol 1	exceed rec. limits vs. non-drinkers	1.17	0.78-1.74	NS	1.07	0.72-1.57	NS	1.40	0.94-2.07	NS
Alcohol 2	daily vs. non-drinkers	0.80	0.49-1.33	NS	0.57	0.33-0.96	0.0363	0.67	0.38-1.16	NS
Alcohol 3	moderate vs. non-drinkers	0.70	0.53-0.94	0.0154	0.73	0.56-0.97	0.0271	0.73	0.56-0.76	0.0049

Table 2. RDIc≥ 5: Summary of fitted models

Model	Model predictors*	Model Statistics§†									
		-2 log L	Likelihood	Wald	DF	H&L	Pearson	Deviance	Con	Dis	AUC
1	sex, age1, CSC, BMI1, ESS1, snore1, apnoea, refreshed1, enough1, hypertension, heart, diabetes stroke, psych.	722.76	388.26	209.21	14	6.30	841.90	722.76	87.10	12.80	0.87
1a	sex, age1, BMI1, ESS1, snore1, apnoea	726.37	385.65	210.55	6	5.83	859.39	859.39	87.00	12.90	0.89
1b	sex, age1, BMI2, ESS1, snore1, apnoea	753.52	364.37	213.27	6	6.72	887.72	753.52	86.10	13.80	0.86
1c	sex, age1, BMI1, ESS1, snore1, apnoea, hypertension	725.96	386.06	210.66	7	4.15	855.06	725.96	87.00	12.90	0.87
1d	sex, age1, BMI1, BMI1xSex, ESS1, snore1, apnoea	724.77	387.25	210.24	7	4.73	853.39	724.77	87.10	12.80	0.87
2	sex, age1, CSC, Neck1, ESS1, snore1, apnoea, refreshed1, enough1, hypertension, heart, diabetes stroke, psych.	712.34	372.76	207.41	14	10.23	798.56	712.34	86.80	13.00	0.87
2a	age, neck1, ESS1, snore1, breath	716.43	369.66	207.75	5	6.09	814.74	716.43	86.70	13.10	0.87
2b	age, neck2, ESS1, snore1, breath	721.81	326.27	204.64	5	4.69	793.65	719.04	85.20	14.60	0.85
2c	sex, age1, neck1, ESS1, snore1, apnoea, hypertension	715.78	370.32	207.81	6	4.25	810.78	715.78	86.80	13.10	0.87

*Predictors in grey indicate non-significance (p>0.05)

§ Model statistics in grey indicate the tests did not achieve goodness of fit significance

† -Model statistics names in full: -2 Log L model fit statistics, Likelihood ratio test, Wald test, Hosmer-Lemeshow test, Pearson Chi-Square and Deviance Goodness of fit statistics, % Concordant, % Discordant, Area Under the Curve (AUC)

Table 3. RDIc≥ 10: Summary of fitted models

Model	Model predictors*	Model Statistics§†									
		-2 log L	Likelihood	Wald	DF	H&L	Pearson	Deviance	Con	Dis	AUC
1	sex, age1, CSC, BMI1, ESS1, snore1, apnoea, refreshed1, enough1, hypertension, heart, diabetes stroke, psych.	758.12	379.49	208.05	14	11.62	826.43	758.12	86.20	13.70	0.86
1a	sex, age1, BMI1, ESS1, snore1, apnoea	767.04	372.19	208.00	6	8.98	809.60	767.04	85.80	14.10	0.86
1b	sex, age1, BMI2, ESS1, snore1, apnoea	795.83	350.61	213.22	6	13.93	822.10	793.06	84.90	14.90	0.85
1c	sex, age1, BMI1, ESS1, snore1, apnoea, hypertension	766.87	372.36	208.35	7	10.49	807.69	766.87	85.80	14.10	0.86
1d	sex, age1, BMI1, BMI1xSex, ESS1, snore1, apnoea	761.81	377.42	211.40	7	9.95	781.77	761.81	86.00	13.90	0.86
2	sex, age1, CSC, Neck1, ESS1, snore1, apnoea, refreshed1, enough1, hypertension, heart, diabetes stroke, psych.	735.02	377.11	208.35	14	3.64	810.93	735.02	86.50	13.40	0.87
2a	age, neck1, ESS1, snore1, breath	743.83	369.92	209.03	5	10.52	789.03	743.83	86.10	13.80	0.86
2b	age, neck2, ESS1, snore1, breath	788.74	299.09	192.20	5	3.63	761.11	783.19	83.20	16.70	0.83
2c	sex, age1, neck1, ESS1, snore1, apnoea, hypertension	743.68	370.07	208.55	6	9.25	792.21	743.68	86.10	13.80	0.86

*Predictors in grey indicate non-significance (p>0.05)

§ Model statistics in grey indicate the tests did not achieve goodness of fit significance

† -Model statistics names in full: -2 Log L model fit statistics, Likelihood ratio test, Wald test, Hosmer-Lemeshow test, Pearson Chi-Square and Deviance Goodness of fit statistics, % Concordant, % Discordant, Area Under the Curve (AUC)

Table 4. RDIc≥ 15: Summary of fitted models

Model	Model predictors*	Model Statistics§†									
		-2 log L	Likelihood	Wald	DF	H&L	Pearson	Deviance	Con	Dis	AUC
1	sex, age1, CSC, BMI1, ESS1, snore1, apnoea, refreshed, enough, hypertension, heart, diabetes stroke, psych.	711.44	369.37	196.73	14	7.76	768.93	711.44	86.80	13.10	0.87
1a	sex, age1, BMI1, ESS1, snore1, apnoea	723.68	359.18	195.40	6	3.61	774.50	723.68	86.20	13.60	0.86
1b	sex, age1, BMI2, ESS1, snore1, apnoea	741.26	348.37	203.43	6	16.55	802.28	738.48	85.90	13.90	0.86
1c	sex, age1, BMI1, ESS1, snore1, apnoea, hypertension	722.98	359.88	196.31	7	6.35	772.52	722.98	86.30	13.60	0.86
1d	sex, age1, BMI1, BMI1xSex, ESS1, snore1, apnoea	709.63	373.23	202.78	7	3.39	709.63	744.56	86.90	13.00	0.87
2	sex, age1, CSC, Neck1, ESS1, snore1, apnoea, refreshed, enough, hypertension, heart, diabetes stroke, psych.	683.95	371.84	198.47	14	3.39	727.19	683.95	87.10	12.80	0.87
2a	age, neck1, ESS1, snore1, breath	695.48	362.36	198.04	5	7.41	735.98	695.48	86.70	13.20	0.87
2b	age, neck2, ESS1, snore1, breath	766.73	276.41	179.24	5	5.32	737.20	761.18	82.90	16.90	0.83
2c	sex, age1, neck1, ESS1, snore1, apnoea, hypertension	695.40	362.45	197.61	6	6.95	738.30	695.40	86.70	13.20	0.87

*Predictors in grey indicate non-significance (p>0.05)

§ Model statistics in grey indicate the tests did not achieve goodness of fit significance

† -Model statistics names in full: -2 Log L model fit statistics, Likelihood ratio test, Wald test, Hosmer-Lemeshow test, Pearson Chi-Square and Deviance Goodness of fit statistics, % Concordant, % Discordant, Area Under the Curve (AUC)

Table 5. Model 1a (RDIa≥ 5): Model parameters

Explanatory variable	DF	Estimate (β)	Standard error	Chi-square	p-value
Intercept	1	-10.5015	0.92	130.09	<0.0001
Sex	1	1.14	0.24	22.32	<0.0001
Age	1	0.053	0.01	19.44	<0.0001
Body mass index (BMI)	1	0.167	0.02	100.95	<0.0001
Observed apnoea	1	1.37	0.21	44.05	<0.0001
Excessive daytime sleepiness	1	0.56	0.216	7.96	0.0048
Habitual snoring	1	1.01	0.20	24.96	<0.0001

Table 6. Model 1a (RDIa≥ 10): Model parameters

Explanatory variable	DF	Estimate (β)	Standard error	Chi-square	p-value
Intercept	1	-10.47	0.96	227.86	<0.0001
Sex	1	1.44	0.26	29.82	<0.0001
Age	1	0.05	0.01	13.96	<0.0001
Body mass index (BMI)	1	0.15	0.02	87.16	<0.0001
Observed apnoea	1	1.43	0.22	41.51	<0.0001
Excessive daytime sleepiness	1	0.56	0.20	27.42	0.0063
Habitual snoring	1	1.07	0.20	27.42	<0.0001

Table 7. Model 2a (RDIa≥ 5): Model parameters

Explanatory variable	DF	Estimate (β)	Standard error	Chi-square	p-value
Intercept	1	-14.59	1.28	128.94	<0.0001
Age	1	0.05	0.01	17.36	<0.0001
Neck circumference	1	0.26	0.03	96.30	<0.0001
Observed apnoea	1	1.27	0.20	40.08	<0.0001
Excessive daytime sleepiness	1	0.60	0.20	9.57	0.0020
Habitual snoring	1	0.86	0.20	96.30	<0.0001

Table 8. Model 2a (RDIa≥ 10): Model parameters

Explanatory variable	DF	Estimate (β)	Standard error	Chi-square	p-value
Intercept	1	-15.44	1.38	125.62	<0.0001
Age	1	0.04	0.01	10.29	0.0013
Neck circumference	1	0.27	0.03	97.20	<0.0001
Observed apnoea	1	1.38	0.22	38.32	<0.0001
Excessive daytime sleepiness	1	0.54	0.21	97.20	0.008
Habitual snoring	1	0.91	0.21	6.86	<0.0001