Eating Behaviours and Attitudes in Narcolepsy and their Association with Sleepiness and Mood

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Abstract

A number of studies have found maladaptive eating behaviours and attitudes in individuals with narcolepsy, however, few studies have comprehensively investigated possible reasons (other than hypocretin deficiency) for these findings. The aim was to investigate the eating behaviours and attitudes of individuals with narcolepsy. The sample consisted of 73 individuals with narcolepsy and 74 controls. Measures used were the Bulimia Test, Depression Anxiety Stress Scale and Epworth Sleepiness Scale, a one day Food and Drink Intake Diary, a newly developed Meal and Snack Timing Questionnaire and an adapted Meal Choice Questionnaire and Snack and Drink Choice Questionnaire. Results showed that individuals with narcolepsy consumed significantly more snacks and drinks than controls. Controls were more concerned about the impact of snack and drink choice on their weight. Individuals with narcolepsy were more likely to binge eat than controls. Individuals with narcolepsy rated the importance of timing food intake with convenience of sleepiness more highly than controls. Individuals with narcolepsy had significantly higher levels of depression, anxiety and stress than controls. Individuals with narcolepsy with moderate to severe levels of anxiety and stress scored significantly higher on the binge factor than those with normal to mild levels of anxiety and stress. A hypothesis is developed suggesting that two opposing frameworks may be operating in narcolepsy, one of a purposeful eating behaviour and another of uncontrolled eating behaviour. The pattern of timing meals and snacks according to the convenience of sleepiness implies that individuals with narcolepsy are engaging in a purposeful behaviour to control their symptom of daytime sleepiness. Binge eating may be an uncontrolled compensatory factor as a consequence of this purposeful timing of food and sleepiness. Further, this study suggests that this extent to which binge eating is engaged by individuals with narcolepsy may be driven by anxiety and stress.

Doctor of Psychology Declaration

"I, Danielle Gatti, declare that the Doctor of Psychology (Clinical Psychology) thesis entitled Eating Behaviours and Attitudes in Narcolepsy and their Association with Sleepiness and Mood is no more than 40,000 words in length including quotes and exclusive of tables, figures, appendices, bibliography, references and footnotes. This thesis contains no material that has been submitted previously, in whole or in part, for the award of any other academic degree or diploma. Except where otherwise indicated, this thesis is my own work".

Signature: Date:

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Publications

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Chapter 1: Literature Review

1.1. Introduction

"Food affects me terribly. During the day at work I can't eat at all. Not even a handful of peanuts" (Bruck & Broughton, 2001, p. 21). This quote was taken from an individual with narcolepsy. Narcolepsy is a sleep disorder prevalent in 0.02-0.16% of the adult population, presenting equally in both males and females (American Psychiatric Association, 2000). The disorder is characterised by excessive daytime sleepiness and irresistible attacks of sleep (Stores, 1999). Hypocretin, a neuropeptide produced in the lateral hypothalamus, has been hypothesised to be involved in the regulation of sleep/wakefulness and appetite (Scammell, 2001). The recent discovery of a hypocretin deficiency as a possible causal factor of human narcolepsy (Nishino, Ripley, Overeem, Lammers, & Mignot, 2000) has sparked an increased interest in the area of eating behaviours and attitudes of individuals with narcolepsy. However, to date there have been few published studies offering comprehensive insight into this area of narcolepsy. A number of studies have found maladaptive eating behaviours and attitudes in individuals with narcolepsy (Bruck, Armstrong, & Coleman, 1989; Chabas, et al., 2007; Fortuyn, et al., 2008) but these findings are largely inconsistent (not in the predicted direction) with a hypocretin deficiency explanation. Furthermore, there have been no published studies which have empirically measured how additional factors to hypocretin such as reported sleepiness, depression, anxiety and stress may affect the eating behaviours and attitudes of individuals with narcolepsy. The current study will not draw conclusions on the role of hypocretin or the independence from hypocretin for factors explored.

The following literature review will examine research relevant to the issue of whether sleepiness, depression, anxiety and/or stress may affect the eating behaviours and attitudes of individuals with narcolepsy. This literature review will review the symptoms of narcolepsy, the genetic factors in the development of narcolepsy, management of the symptoms of narcolepsy and the treatment of narcolepsy. It will discuss hypocretin functioning in the general population and in individuals with narcolepsy. Additionally this literature review will explore comorbid conditions in individuals with narcolepsy, specifically obesity, diabetes mellitus and mood disorders. It will look into the physiological, behavioural and psychological aspects of food intake including the initiation and selection of food intake, emotionally induced eating, disordered eating and sleep/wake behaviour after food intake. Lastly it will provide an introduction to the current study outlining rationale, aims and hypotheses. Please note, when the current thesis refers to "narcolepsy" it is referring to narcolepsy in broad terms and this may encompass narcolepsy that is both with and without cataplexy, unless otherwise specified.

1.2. Narcolepsy

1.2.1 Diagnosis and Symptomatology

The following section will review diagnostic and associated symptoms of narcolepsy. The symptoms associated with narcolepsy typically (but not necessarily) begin during adolescence to early adulthood (15-25 years of age; Mignot, 2004). On average there tends to be a substantial delay of ten and a half years between the onset of symptoms and the accurate diagnosis of narcolepsy (Morrish, King, Smith, & Shneerson, 2004), although this is likely to vary from region to region. This delay is due, in large part, to

an inadequate understanding of the disorder of narcolepsy (Black, Nishino, & Brooks, 2005).

The International Classification of Sleep Disorders (ICSD-2; American Sleep Disorders Association, 2005) distinguishes between narcolepsy with cataplexy and narcolepsy without cataplexy. Specifically, criteria for narcolepsy with cataplexy is set out as the following; almost daily complaint of excessive daytime sleepiness for a minimum period of three months, unambiguous cataplexy (defined as episodes of loss of muscle that are transient, sudden and emotionally triggered) and diagnosis should be confirmed by nocturnal polysommography and multiple sleep latency test (MSLT). A nocturnal polysomnography must precede a MSLT. The mean sleep latency should be measured at less than or equal to eight minutes and with two or more sleep onset rapid eye movement periods (SOREMPs) on the MSLT to confirm diagnosis of narcolepsy with cataplexy. Alternatively, hypocretin levels in the cerebral spinal fluid may be measured. One hundred and ten pg/mL or one third of control mean values of hypocretin are considered a reliable indicator of narcolepsy with cataplexy based on reference samples from Stanford University. The final criteria to be met for a diagnosis of narcolepsy with cataplexy is that hypersomnia could not otherwise be better explained by another sleep disorder, medical condition, neurological disorder, mental health disorder, medication use and or substance use. Criteria for narcolepsy without cataplexy are identical to that above with the exception of the symptom of cataplexy. Unambiguous cataplexy is not present although atypical cataplexy episodes (episodes that do not meet full definition of cataplexy) may occur.

Individuals with narcolepsy lack the ability to assert control over maintaining alertness (Bruck & Broughton, 2001). They experience excessive daytime sleepiness that is typically heightened during periods of low stimulation or monotonous situations

(Zeman, et al., 2004). The characteristic feature of narcolepsy is irresistible attacks of sleep. Sleep attacks are often unintentional and occur in inappropriate situations (e.g., while driving a car or during a conversation). These sleep attacks usually last 10-20 minutes (American Psychiatric Association, 2000). Usually the symptom of excessive daytime sleepiness develops first with cataplexy and recurrent intrusions of REM developing up to several years later (Zeman, et al., 2004). It is important to note that although often used interchangeably, individuals with narcolepsy experience feeling *sleepy*, not simply *fatigued*. The distinction is important, individuals with narcolepsy have a tendency to fall asleep at inappropriate times. Individuals who are fatigued lack energy and feel restless, however can resist the tendency to fall asleep at unwanted times (Peacock & Benca, 2010). On a MSLT individuals who experience fatigue most likely do not display abnormal results (Wise, Arand, Auger, Brooks, & Watson, 2007).

Intrusions of elements of REM sleep can occur repeatedly during the transitions between sleep and waking. These intrusions can manifest as either hallucinations or sleep paralysis (American Psychiatric Association, 2000). An estimated 50-60% of individuals with narcolepsy experience hallucinations (Stores, 1999). These hallucinations can take one of two forms; hypnagogic or hypnopomic. The Diagnostic and Statistical Manual of Mental Health Disorders (DSM-IV-TR American Psychiatric Association, 2000) provides the following definitions/criteria for hallucinations in individuals with narcolepsy. Hypnagogic hallucinations are dreamlike imagery that occurs right before falling asleep. Hypnopomic hallucinations are dreamlike imagery that occurs right after awakening from asleep. The hallucinations (either of hynagogic or hypnopomic form) can be visual, auditory or kinetic and last from a few seconds to a few minutes, terminating spontaneously.). The other manifestation of REM sleep intrusion, sleep paralysis, presents in approximately 40% of individuals with narcolepsy

(Stores, 1999). During sleep paralysis individuals feel unable to speak or move and may also feel unable to breathe, despite no physical explanation. Sleep paralysis may last from a few seconds to a few minutes, terminating spontaneously (American Psychiatric Association, 2000).

Individuals with narcolepsy commonly experience disruptions in nocturnal sleep. In comparison to the general population they have significantly shorter nocturnal REM sleep latency and higher occurrences of periodic limb movement during nocturnal sleep (Hong, Hayduk, Lim, & Mignot, 2000). Furthermore, 71 percent of individuals with narcolepsy are unable to have nocturnal sleep without awakening, 83 percent awake early and 50 percent feel unrefreshed after awakening in the morning (Sturzenegger & Bassetti, 2004).

Cataplexy is the sudden loss/weakness of muscle tone (e.g., falling to the ground or sagging jaw) while conscious (American Psychiatric Association, 2000). All muscles may be affected or it can be limited only to certain muscle groups (Dauvilliers, Billiard, & Montplaisir, 2003). Episodes of cataplexy typically occur as a result of heightened emotion (e.g., laughter). They can last from a few seconds to thirty minutes and are followed by a full recovery of muscle tone (American Psychiatric Association, 2000). It is difficult to provide an estimate of prevalence rates of the symptom of cataplexy within the narcolepsy. Reported cataplexy estimates within the narcolepsy population vary considerably, ranging between 60 to 90 percent (Bassetti & Aldrich, 1996; Chaudhary & Husain, 1993; Stores, 1999). Percentages vary across ethnic samples and these prevalence estimates are over a decade old. Few studies have investigated the epidemiology of narcolepsy with cataplexy since the criteria distinction of narcolepsy with/out cataplexy in 2005 (American Sleep Disorders Association). A recent study

with a sample of Norway participants found a prevalence rate of 0.022 percent of the general population to have narcolepsy with cataplexy (Heier, et al., 2009).

In addition to the diagnostic criteria set out by the ISCD-2, the disorder of narcolepsy is often accompanied by a much broader range of symptoms or difficulties that impact on everyday functioning. These may include, but are not limited to, cognitive difficulties, automatic behaviour and problems in psychosocial functioning.

Cognitive difficulties are more common in the narcolepsy population in comparison to the general population. Common difficulties include; holding attention and concentration, memory and orientation for people (Ohayon, Ferini-Strambi, Plazzi, Smirne, & Castronovo, 2005). Individuals with narcolepsy often experience states of automatic behaviour which usually occurs after successfully fighting off a sleep attack (Bruck & Broughton, 2001). Automatic behaviour presents as behaviour or speech that occurs without conscious awareness. The individual with narcolepsy commonly does not recall the automatic behaviour (most likely due to their extreme sleepiness; Black, et al., 2005). Individuals with narcolepsy also experience a wide range of psychosocial difficulties such as poor performance at work, poor driving records (Broughton, Geberman, & Roberts, 1984), marital difficulties, embarrassment, reduced academic performance, lowered feelings of self-worth and avoidance of social situations (Broughton & Broughton, 1994).

As demonstrated, individuals with narcolepsy are confronted with numerous symptoms and resulting difficulties. As will become evident throughout this thesis, issues surrounding eating may also impact greatly on the lives of individuals with narcolepsy although this aspect is not as widely researched as other areas. Furthermore, it is documented that depression, anxiety and stress commonly co-occur with narcolepsy.

Issues surrounding eating behaviours and attitudes, depression, anxiety and stress will be discussed in detail later in this review.

1.2.2. Genetic Factors in the Development of Narcolepsy

HLA gene studies and familial studies of individuals with narcolepsy have shown strong suggestion towards a genetic factor contribution to the development of a narcolepsy condition in some cases. However, the development of the disorder of narcolepsy is most likely multifactorial.

The HLA DQB1*0602 genetic marker has been found to be present in the majority of individuals with narcolepsy. In particular this genetic marker is a good indicator of narcolepsy with cataplexy. The prevalence rate of HLA DQB1*0602 in individuals with narcolepsy including the feature of cataplexy is over 90 percent. However, the presence of this marker alone is not sufficient enough to warrant a diagnosis of narcolepsy. Research has also found that between one in ten and one in three individuals without narcolepsy may also present with the HLA DQB1*0602 marker (Peacock & Benca, 2010).

A summary of the literature would reveal approximately 5-15 percent of first degree biological relatives of an individual with narcolepsy also have the sleep disorder. Additionally, data from a collection of HLA studies have estimated 25 to 50 percent of first degree relatives of individuals with narcolepsy have a diagnostic disorder, other than narcolepsy, that is characterised by excessive sleepiness, for example, primary hypersomnia (American Psychiatric Association, 2000). The importance of environmental factors, in addition to genetic, are highlighted by reports that only 25 to 31 percent of monozygotic twins share a diagnosis of narcolepsy (Mignot, 1998).

1.2.3 Measurement of Narcolepsy Symptomatology

A number of measures can be used to measure the symptoms of narcolepsy, in particular excessive daytime sleepiness.. This section briefly outlines the most widely accepted measures namely, polysomnography, MSLT, maintenance of wakefulness test Stanford Sleepiness Scale and Epworth Sleepiness Scale.

A polysomnography is an objective measure used to record sleep patterns as well as physiological process involved in sleep (Parmeggiani & Velluti, 2005). Greater levels of sleepiness are indicated by more rapid sleep onset (Kushida, 2004). Thus, given their excessive levels of daytime sleepiness, individuals with narcolepsy have significantly more rapid sleep latency in comparison to the general population. The polysomnography can aid in differentiating the cause of an individual's excessive daytime sleepiness. Individuals with narcolepsy typically undertake a nocturnal polysomnograph prior to a MSLT. The individual is monitored overnight for at least six hours and information is gathered related to breathing, oxygen desaturation, heart rate, limb movements, eye movements, muscle tension, time in bed and time asleep. An electroencephalogram (EEG) is also required for the identification of sleep stages.. In addition, sleep talking, snoring and complex sleep behaviours or movements can be usually also be monitored (Peacock & Benca, 2010).

The MSLT is the accepted standard to objectively collect information regarding excessive daytime sleepiness and sleep drive. The MSLT involves an interval nap (in a bed in a darkened room) of twenty minutes every two hours repeated five times throughout the day. The individual being tested is instructed to try to fall asleep during scheduled naps and to stay awake between scheduled naps. Data related to sleep onset and REM sleep is collected (Peacock & Benca, 2010). As mentioned earlier, diagnostic

criteria for narcolepsy both with and without cataplexy outlines that the mean sleep latency should be measured at less than or equal to eight minutes and with two or more sleep onset rapid eye movement periods (SOREMPs) on the MSLT as indicative of the condition of narcolepsy (American Sleep Disorders Association, 2005).

Another standard measure of excessive sleepiness is the maintenance of wakefulness test (MWT). In contrast to the MSLT, which provides a measure of an individual's ability to fall asleep, MWT provides a measure on an individual's ability to stay awake (Wise, 2006). Individuals stay in a quiet and non-stimulating room for a set period of time and are instructed to try to stay awake. Recommended protocol involves four trials of forty minutes with two hour periods between each set trial. Measures of nap sleep latencies, stages of sleep, total sleep time, and mean sleep latency are provided (Slinkard, 2006). The MWT has clinical usefulness in evaluating response to treatment following intervention for conditions associated with excessive sleepiness, and in assessing individuals who must remain awake for safety reasons (Wise, 2006).

Measures of general sleepiness can also be administered through self rated questionnaires. The most widely used and accepted measures are the Stanford Sleepiness Scale and the Epworth Sleepiness Scale. The Stanford Sleepiness Scale is a self subjective rating scale based on feelings of sleepiness at a particular time (Hoddes, Zarcone, Smythe, Phillips, & Dement, 1973). Epworth Sleepiness Scale provides a measure of an individual's general level of daytime sleepiness or sleep propensity based on dozing behaviours in recent times, as opposed to current feelings of sleepiness (Johns, 1991).

1.2.4. Treatment of Narcolepsy

Common pharmacological treatment of the symptom of excessive daytime sleepiness in narcolepsy is the use of central nervous system stimulant medication. Typically used stimulant agents include; modafinil, methylphenidate and amphetamines (i.e., dextroamphetamines and methamphetamines). As well as stimulant medication, anticatapletics (medication for cataplexy) may also be needed. Agents include sodium oxybate and low doses of tricyclics and typical (selective serotonin reuptake inhibitors) antidepressants (Black, et al., 2005). Please note sodium oxybate is not available in Australia.

Stimulant medication increases wakefulness, decreases feelings of fatigue, elevates mood and produces behavioural activation and increased performance (Mitler, Aldrich, Koob, & Zarcone, 1994). Although stimulant medication has been shown to significantly improve levels of sleepiness within the narcolepsy population their levels of sleepiness when medicated have not been found to reduce to levels that are comparable to the general population (Bruck, Kennedy, Cooper, & Apel, 2005).

Therefore, in addition to the use of stimulant medication effective control of excessive daytime sleepiness is benefited by structured and regular nocturnal and daytime sleep patterns. Individuals with narcolepsy are encouraged to aim for a period of 8 hours or more for nocturnal sleep, a consistent and scheduled time for falling and awakening from nocturnal sleep and two or more short and regularly timed naps during the day are typically beneficial to enhancing function and controlling daytime sleepiness (Black, et al., 2005).

Side effects are commonly produced by stimulant medication when used as a part of the treatment for narcolepsy. Individuals with narcolepsy are not more prone to the side

effects associated with stimulant medication use than other groups of individuals (Mitler, et al., 1994). Side effects may include; headache, nausea, anxiety, irritability, increased blood pressure, tremors and insomnia (Black, et al., 2005). Stimulant medication has also been found to affect eating behaviours. Modafinil, amphetamines and methylphenidate agents have all been found to have the effect of reducing appetite (Makrisa, 2004; Prescrire International," 2001). A review of studies investigating side effects of stimulant medication in narcolepsy and other hypersomnias by Wise, et al. (2007) found hypertension, anxiety and depression to be common side effects. They also found that information about the side effects of antidepressant medication for the control of cataplexy in individuals with narcolepsy to be sparse. Billiard, et al.'s (2006) review discussed hypertension and gastrointestinal upset as common side effects of anti-depressant medication for treatment of cataplexy for individuals with narcolepsy. Hypertension may lead to the assumption of experience of anxiety and gastrointestinal upset may affect eating patterns.

It is important to note that, as a medication regime is a common part of the daily lives of most diagnosed individuals with narcolepsy, these side effects and residual daytime sleepiness also form a part of their everyday lives. Thus, such issues necessitate addressing from clinicians and researchers.

1.3. Hypocretin

1.3.1 Hypocretin Function in the General Population

Hypocretin (also commonly referred to as orexin in the literature) is a neuropeptide produced in the lateral hypothalamic area of the central nervous system (Sakurai, 2006). Hypocretin is involved in maintaining energy homeostasis and as such serves a number of functions. Hypocretin is believed to be involved in the regulation of

sleep/wakefulness (Scammell, 2001), stress reactions (Berridge, España, & Vittoz, 2010) and appetite (Willie, Chemelli, Sinton, & Yanagisawa, 2001).

Simply summarised, hypocretin containing neurons stimulate brain activity. They increase the firing rates of neurons in the aminergic regions of the brain which promote wakefulness and suppress REM sleep (Scammell, 2001). Thus, as a result hypocretin plays a central role in arousal keeping an individual awake throughout the day. During high levels of arousal, such as stress, hypocretin neurons in areas of the brain (the medial septal area, the general region of the medial preoptic area, substantia innominata and locus coeruleus) associated with responses to stress are activated. As a result hypocretin plays a central role in how individuals respond to stress, both physiologically and behaviourally (Berridge, et al., 2010). Neurons that produce hypocretin project to areas of the brain, such as the diencephalon, which plays a central role in appetite regulation (Samson, Taylor, & Ferguson, 2005). In this respect, hypocretin functions to activate and coordinate food seeking and feeding behaviour (Sakurai, 2006). Hypocretin also plays a similar role in stimulating fluid intake (drinking behaviours; Willie, et al., 2001).

1.3.2. Hypocretin in Individuals with Narcolepsy

Hypocretin in the cerebrospinal fluid has been found to be undetectable in the vast majority of individuals with narcolepsy (Nishino, et al., 2000). These low undetectable levels are due to a hypocretin-ligand deficiency (Fujiki & Nishino, 2007). The majority of research into hypocretin, such as that of Heier, et al. (2007), suggests that a strong association between hypocretin deficiency and narcolepsy with cataplexy. Narcolepsy with cataplexy has been characterized by an approximately 90 percent of hypocretin cell loss (Thannickal, Nienhuis, & Siegel, 2009). The same association has not been

replicated within the narcolepsy without cataplexy population. Therefore, the majority of recent research investigating hypocretin in narcolepsy has focused on narcolepsy with cataplexy only. However, a few recent studies have been conducted on narcolepsy without cataplexy and the role of hypocretin. These have yielded less consistent results in respect to the presence of a hypocretin deficiency than narcolepsy with cataplexy research. For example, Oka, et al. (2006) found that lowered levels of hypocretin in the cerebral spinal fluid of some individuals with narcolepsy without cataplexy could be detected. Thannickal, et al. (2009) found 33 percent of hypocretin cell loss, in comparison to the general population, in the brain of an individual with narcolepsy without cataplexy. These studies concluded the possibility of a causal factor between narcolepsy without cataplexy and a partial loss of hypocretin cells.

The exact cause of a hypocretin deficiency finding still remains uncertain however recent research is indicating autoimmune-mediated postnatal cell death as a likely factor (Cao, 2010). Despite the cause, research indicates that the loss of hypocretin (and thus impaired hypocretin signalling) may be representative of narcolepsy in humans and animals (Scammell, 2001).

A hypothesis within the research is that defects affecting normal hypocretin functioning may be a causal factor in the onset of narcolepsy. Although responsible for the mediation of several functions, the most predominate symtomatology in animals and humans with hypocretin deficits are those associated with the diagnosis of narcolepsy, such as daytime sleepiness, sleep attacks and cataplexy (Kroeger & De Lecea, 2009; Scammell, 2001; Tonokura, Fujita, & Nishino, 2007). If a hypocretin deficiency is the main causal factor of narcolepsy then it would be expected that along with difficulties in sustaining periods of wakefulness and suppressing REM sleep they would experience

impaired (less adaptive) physiological and behavioural responses to stress and a smaller appetite than the general population.

When hypocretin signalling is impaired the fibres that they contain may not produce sufficient activation and firing rates to produce sufficient activation in the aminergic regions of the brain and, therefore, inadequate sustaining of wakefulness and dysregulation of REM sleep may result (Scammell, 2001). Poorly sustained levels of wakefulness and disrupted REM sleep are characteristic features of narcolepsy (American Sleep Disorders Association, 2005).

The role of hypocretin in reactions to stress suggests a hypocretin deficiency may be involved in stress related psychopathology (Berridge, et al., 2010). Individuals with narcolepsy express higher rates of depression (Lindsley & Crawford, 1996), anxiety (Fortuyn, et al., 2010) and stress (Broughton & Broughton, 1994) in comparison to the general population. Depression, anxiety and stress will be explored in detail in section 1.4.3 of this thesis.

The discovery of a hypocretin deficiency in individuals with narcolepsy and its role in coordinating food seeking and feeding behaviours has promoted interest and recent research into the eating behaviours and attitudes of individuals with narcolepsy. Hypocretin functions to activate food seeking and feeding behaviour and thus increase appetite (Sakurai, 2006) and stimulates fluid intake (drinking behaviours; Willie, et al., 2001). Therefore it would be expected, given their hypocretin deficiency, that individuals with narcolepsy would have a smaller appetites and consume less food and drink in comparison to the general population. However, the research to date investigating the eating patterns and behaviours of individuals with narcolepsy are largely inconsistent (not in the predicted direction) with a hypocretin deficiency

explanation. One possible hypothesis in the literature (see below), which remains to be investigated, is that hypocretin deficiency decreases energy expenditure to a greater degree than it decreases appetite, leading to, a positive weight gain.

1.4. Comorbid conditions in Narcolepsy

1.4.1. Obesity

Siegel (1999) hypothesised that given the role of hypocretin in appetite regulation, and the hypocretin deficiency in the narcolepsy, individuals with narcolepsy would have a gross reduction in food intake. A number of studies have since shown contradictory evidence to this hypothesis, suggesting the role of other factors to hypocretin deficiency impacting on the eating behaviours of individuals with narcolepsy. This section will explore one of those lines of contradictory evidence, increased body mass index and obesity in the narcolepsy population. Firstly, it will highlight the research pertaining to the general population and increased body mass index (BMI) and obesity.

In the general Australian population twenty-five percent of adults are considered obese and an additional thirty-seven percent are considered overweight ("National Health Survey: Summary of Results, Australia," 2007-2008). Obesity has been found to be a significant risk factor in the development of a number of chronic physical health conditions including, but not limited to; type two diabetes (Harris, et al., 1998), cardiovascular diseases (Hubert, Feinleib, McNamara, & Castelli, 1983), cancer (Hjartaker, Langseth, & Weiderpass, 2008) and obstructive sleep apnoea (Al Lawati, Patel, & Ayas, 2009). The precise contributors to the cause of being overweight or obese are unclear. However, the most widely researched and generally excepted explanations for weight gain and obesity are reduced physical activity and specific food intake which is considered connected with food manufacturing and marketing practices,

for example, vending machines, increased portion size, fast food availability and use of high fructose corn syrup (Keith, et al., 2006).

Progress has also been made in investigating the relationship between sleep and obesity within the general population. A number of cross cultural studies has shown a negative correlation between sleep and BMI, that is, as sleep decreases BMI increases (Patel, et al., 2008; Theorell-Haglow, Berne, Janson, Sahlin, & Lindberg, 2010). Other research however has shown that a higher BMI is associated with both shorten and prolonged sleep durations (Cappuccio, et al., 2008; Spiegel, 2008). Neurological/biological research suggests that sleep deprivation, in individuals who experience sleep restriction at a chronic level, may contribute to obesity by decreasing levels of leptin. This reduction in leptin in turn decreases levels of hormones involved in satiety and hunger promotion, namely, ghrelin (Spiegel, Tasali, Leproult, & Van Cauter, 2009; Van Cauter, et al., 2007) . It has also been found that insufficient sleep changes impact on insulin resistance (and related hormone levels) leading to an increase in appetite and thus also increasing the risk of obesity (Pack & Pien, 2011).

Given the above, it would be assumed that due to excessive sleepiness and sleep deprivation (not feeling rested) individuals with narcolepsy would be more likely to have higher body mass indexes than the general population. In fact, it has been consistently shown in the research that individuals with narcolepsy have significantly higher body mass indexes and are more likely to be overweight/obese in comparison to the general population and psychiatric controls. (Dahmen, Bierbrauer, & Kasten, 2001; Kotagal, Krahn, & Slocumb, 2004; Schuld, Hebebrand, Geller, & Pollmacher, 2000). Kok, et al. (2003) found forty-three percent of the narcolepsy population to be overweight and thirty-three percent to be obese. Studies investigating obesity in narcolepsy have shown no significant difference between individuals on pharmaceutical

treatment and those not on pharmaceutical treatments on measures of body mass index. This suggests that medication is not a factor contributing to weight gain in individuals with narcolepsy and additionally, no differences based on symptom severity have been found (Dahmen, et al., 2001). As well as adults, studies have found higher body mass indexes than average in children with narcolepsy (Kotagal, Hartse, & Walsh, 1990; Kotagal, et al., 2004). Daniels (1934) was among the first to investigate body mass index in individuals with narcolepsy. He reported that fifty percent of his participants diagnosed with narcolepsy had gained a significant amount of weight, ranging from 5 kilograms to 45 kilograms at onset of the disorder.

A higher body mass index in comparison to the general population has been a consistent finding across cultures. African American, Caucasian, Asian, Latino (Okun, Lin, Pelin, Hong, & Mignot, 2002) and European (Schuld, Hebebrand, et al., 2000) individuals with narcolepsy have all been found to have higher body mass indexes than their respective general population. However, bigger difference (of higher body mass index in comparison to the general population) has been found in African American and Asian populations (Okun, et al., 2002). It has been found that first degree relatives have significantly lower body mass indexes in comparison to their relative with narcolepsy, however, significantly higher body mass indexes in comparison to the general population (Dahmen, et al., 2001). A higher body mass index has been found to be significant irrespective of medication status, gender (Schuld, Hebebrand, et al., 2000), daytime sleepiness (Kok, et al., 2003), symptom severity, presence of auxiliary symptoms (Dahmen, et al., 2001) and HLADQB1*0602 marker (Okun, et al., 2002). A recent investigation however has found a negative correlation between BMI and sleep latency as measured by the MSLT, suggesting the role of sleepiness severity in weight gain and/or vice versa (Sonka, et al., 2010).

Leptin, in part, regulates hypocretin (Sakurai, 2006). Leptin has been found to be reduced by approximately fifty percent in individuals with narcolepsy (Schuld, Blum, et al., 2000). A deficiency in leptin has been found to lead to obesity in both animals and humans (Kok, et al., 2002). A recent study found individuals with narcolepsy and no symptom of cataplexy did not have higher BMI's or higher incidences of obesity compared to those without narcolepsy, unlike their narcolepsy with cataplexy counterparts. Additionally they found significantly higher BMI's in participants with narcolepsy and cataplexy compared to those with narcolepsy and no cataplexy (Sonka, et al., 2010). Based on findings presented above it has been hypothesised that being overweight and/or obesity in individuals with narcolepsy is not secondary to behavioural consequences, however, a high BMI is related to the pathophysiology of the disorder.

However, despite the above suggestion of the role of pathophysiology, research investigating obesity in mice with narcolepsy has shown that environmental factors are also important for the development of obesity in narcolepsy (Sakurai, 2006).There has been some suggestion in the research that narcolepsy disorder related behaviours may be responsible for increased body mass index, such as, reduction in locomotor activity and increased sleepiness (Schuld, Hebebrand, et al., 2000). However, no conclusive evidence can be found. Additionally, no research to date has directly explored the link between disordered eating or maladaptive eating patterns in narcolepsy and the connection with obesity. Section 1.5 of this literature review will explore disordered/maladaptive eating patterns in individuals with narcolepsy in some detail.

1.4.2. Diabetes Mellitus

Diabetes mellitus is a group of metabolic disorders where the primary defect is a deficiency of insulin production. This is characterised by high blood glucose. Prevalence of diagnosed incidences of diabetes mellitus in Australia is 3.6 percent (Australian Institute of Health and Welfare, 2009). There are two main categories/types of diabetes mellitus. Diabetes mellitus type one is a disorder characterised by the pancreas being unable to produce insulin. Without insulin the cells within the body are unable to convert glucose, which is sugar, into energy. Diabetes mellitus type two is defined when the pancreas is able to produce some insulin, however, not to the amount that the body requires. Symptoms of diabetes mellitus include, amongst others; excessive thirst, fatigue, constant feeling of hunger and weight gain (Diabetes Australia, 2011).

Studies have reported prevalence rates as high as 26 percent of comorbid diabetes mellitus type two and binge eating disorder. Those with this comorbidity tend to have higher BMI's (Crow, Kendall, Praus, & Thuras, 2001) and more depressive symtomatology (Wing, Marcus, Epstein, Blair, & Burton, 1989).

Significantly higher frequency rates of diabetes mellitus have been found in the narcolepsy population, in comparison to, the general population. Frequency rated within the population of individuals with narcolepsy has been found at 12.5 percent. This higher incidence has not been attributed to obesity. That is, the significant difference between frequencies of diabetes mellitus was found when there was no difference between obesity indices and frequency of obesity between groups (Honda, Doi, & Ninomiya, 1986).

Interestingly the HLA DQB1*0602 genetic marker which indicates strong susceptibility to narcolepsy (with cataplexy) also operates as a protection against the development of diabetes mellitus type one (Siebold, et al., 2004). This suggests that individuals with narcolepsy would experience lowered rates of diabetes mellitus type one. Unfortunately to date studies separating the prevalence of type one versus type two diabetes mellitus in narcolepsy has not been published. From the evidence available it can be hypothesised that individuals with narcolepsy would have lower rates of diabetes mellitus type one and higher rates of diabetes mellitus type two, in comparison to, the general population. Higher rates of diabetes mellitus type two are associated with an increased body mass index (being overweight or obese). A greater understanding of behavioural eating patterns and attitudes for individuals with narcolepsy would potentially add valuable knowledge to adding in managing and preventing symptoms of diabetes mellitus in this population.

1.4. 3. Mood Disorders

This section of the thesis will focus on three aspects of mood experienced by individuals with narcolepsy (namely, depression, anxiety and stress) at higher rates than the general population.

Symptoms of depression include depressed mood (i.e., feelings of sadness, emptiness and tearfulness), reduction in interest or pleasure, significant weight gain/loss or gain/loss of appetite, insomnia or hypersomnia, psychomotor agitation or retardation, fatigue or loss of energy, feelings of worthlessness or excessive/inappropriate guilt, reduction in ability to think or concentrate, indecisiveness and periodic thoughts of death. Individuals with a major depressive disorder are also likely to have a co-morbid

mental health disorder, commonly anxiety disorders and eating disorders (American Psychiatric Association, 2000).

Community samples from the general population have revealed a major depressive disorder (clinical criteria for diagnosis met) prevalence rate of five to nine percent for females and two to three percent for males. Lifetime risk of developing a major depressive disorder based on community samples ranges from 10 to 25 percent for females and five to 12 percent for males (American Psychiatric Association, 2000).

Research has found a number of vulnerabilities/processes involved in the development of depression. This review will provide a very brief overview of common vulnerabilities; gender, cognition, physiology, life events, physical health and sleep. Ingram, Atchley and Segal (2001) summaries the underlining vulnerabilities of depression neatly, and, as follows; A commonly found vulnerability is being female, research had found that women are more likely than their male counterparts to develop depression, suggesting an underlying gender vulnerability. A negative thinking style also acts a cognitive vulnerability to the development of depression. A physiological vulnerability included the activation of the prefrontal cortex, cingulate gyrus and limbic system (particularly amygdale and hippocampus regions). In individuals with depression the prefrontal cortex and cingulate gyrus are under-activated and the limbic system hyper-activated. This regulation of activation may be either at a chronic level or responsive to negative events, such as, criticism from others. Negative life events, in particular, those related to significant loss (such as the death of a spouse), associated with uncontrollability and chronic in nature (such as, finances and physical health) have been correlated with onset and maintenance of depression.

Another vulnerability to the development of depression is physical health. Approximately 20 to 25 percent of individuals with a medical condition, such as diabetes, develop a major depressive disorder. This is important to note because prognosis of depression is adversely impacted (i.e., poorer response to treatment) when a medical condition is comorbid (American Psychiatric Association, 2000). Risk of the development of depression had been found to be comparable for individuals with normal glucose metabolism, impaired glucose metabolism and undiagnosed diabetes mellitus type two. Those with a diagnosis of diabetes mellitus type two have been found to have an increased risk of developing depression in comparison to those with impaired glucose metabolism and undiagnosed diabetes mellitus type two (Nouwen, et al., 2011).

Disrupted sleep is also considered a vulnerability to the development of depression. Approximately 40 to 60 percent of outpatients and 90 percent of inpatients experiencing a major depressive episode have abnormal sleep recording on an EEG (American Psychiatric Association, 2000). Mamelak (2009) summarises the literature on sleep and depression. He describes that the breakdown of sleep as a fundamental feature of depression that often acts as a precursor to depression. Frequent periods of wakefulness (arousal) and short sleep onset REM latency are common features evident on a polysomnographic measure in individuals with depression. A combination of monoaminergic deficiency and cholinergic sensitivity has been associated with the short latency to REM sleep. In individuals with narcolepsy a similar combination of monoaminergic deficiency and cholinergic sensitivity has been found. This imbalance combination is suggested to result from a hypocretin deficiency.

The comorbidity rate of narcolepsy and depression has been estimated between 30-52% This is substantially above estimated rates of depression found in the general population. It has been found that approximately 46 percent of individuals with a

diagnosis of narcolepsy met a clinical diagnosis of depression prior to their diagnosis of narcolepsy. Symptoms of depression have been found to increase rather than decrease across the life span of individuals with narcolepsy (Lindsley & Crawford, 1996). Research has found that individuals with narcolepsy have significantly more symptoms of depression if they are on a low income, have higher self ratings of symptom severity (Merrit, Cohen, & Smith, 1992) and/or are a female (Jara, Popp, Zulley, Hajak, & Geisler, 2011). Symptoms of depression in individuals with narcolepsy have been found to be lower in older individuals, decrease as level of education increases and be lower in individuals that are married (Merrit, et al., 1992). Self rated symptom severity of depression in individuals with narcolepsy has been found to be independent of disruption of school or work life, medication, reported severity of excessive daytime sleepiness (Lindsley & Crawford, 1996) and the presence of cataplexy (Jara, et al., 2011). Recent research has shown that individuals with narcolepsy that have the symptom of cataplexy are more likely to have higher severity of depression than individuals with narcolepsy and no cataplexy symptomatology. Low levels of hypocretin (typical of narcolepsy with cataplexy) have been hypothesised to correlate with severity of depressive symptoms (Dimitrova, et al., 2011). However, other research has shown the presence of depression independently of cataplexy severity (Lindsley & Crawford, 1996). Additionally, it has been found that risk of an anxiety diagnosis in individuals with narcolepsy increased as severity of depressed mood increased. A similar relationship has been found between cognitive disturbance (difficulty focusing and concentrating). That is, cognitive disturbance increased as symptom severity of depression increased in individuals with narcolepsy (Lindsley & Crawford, 1996). A limitation of previous research into depression and narcolepsy is that the majority of such research had included measurements of depression which include items related to

abnormal sleep patterns and/or fatigue for example, Jara, et al.(2011) used the Beck Depression Inventory and Merrit, et al. (1992) used the Centre for Epidemiologic Studies Depression Scale. This may inflate depression scores as abnormal sleep would more likely be a function of narcolepsy diagnosis than poor affect regulation.

The association between anxiety and narcolepsy has been less frequently studied than the relationship between depression and narcolepsy. However, exploration of the relationship between anxiety and narcolepsy is of interest. Symptoms of anxiety include excessive worry that is hard to control, restlessness, feeling "keyed up", fatigued easily, poor concentration, irritability, muscle tension, sleep abnormality, panic and agoraphobia (American Psychiatric Association, 2000).

Community samples from the general population have found prevalence rates of anxiety disorders to be at 18 percent (Kessler, Chiu, Demler, & Walters, 2005). The lifetime risk of developing an anxiety disorder based on community samples range from 15 to 35 percent (Kessler, Berglund, Demler, Jin, & Walters, 2005).

Anxiety is considered to be the response from overstimulation of the circuits that produce fear. In particular this is the amygdala and extended areas of amygdala, such as the bed nucleus of the stria terminalis. The overstimulation of the fear circuits is expressed as hypervigilance and an increased behavioural response to fearful stimuli. A reduction in thresholds for activation and overstimulation in the fear circuits develop through sensitisation processes or kindling processes. These processes involve neuropeptides, hormones, as well as other proteins (Rosen & Schulkin, 1998)

Vulnerabilities to the development of anxiety include genetic factors such as family history of anxiety, psychological conditioning (particularly a general sense of uncontrollability) and stressful life events (Barlow & Durand, 2005). Women are more

vulnerable to the development of anxiety with females being 60 percent more likely than males to develop an anxiety disorder (Kessler, Berglund, et al., 2005). Anxiety disorders have been found to be more prevalent among individuals with a chronic physical medical condition (i.e., diabetes mellitus, asthma, coronary heart disease, stroke, cancer, arthritis) in comparison to those without a chronic physical condition (Teesson, et al., 2011). Specific research on diabetes mellitus and anxiety has not found an increased probability of anxiety in comparison to those without diabetes mellitus (Paddison, et al., 2011; Wu, et al., 2011). Anxiety disorders have also been found to be frequently comorbid with other mood disorders such as depression and substance related disorders (American Psychiatric Association, 2000). Disrupted sleep may also act as a risk factor in the development of anxiety. Research measuring anxiety after exposure to stressor stimuli has shown anxiety to be exacerbated by disrupted sleep (MacLean & Datta, 2007). The literature shows that individuals in the general population with sleep complaints report higher anxiety than those without sleep complaints. However, it is possible that other aspects in addition to mood disturbances might contribute to sleep-related distress, such as beliefs and attitudes about sleep (Edinger, et al., 2000).

As mentioned earlier, research into anxiety within the narcolepsy has not been as well investigated as depression. However, the research that has been conducted largely suggests high rates of anxiety within the narcolepsy population in comparison to the general population**.** Fortuyn, et al.'s (2010) investigation found more than half of their sample of individuals with narcolepsy reported anxiety and panic attacks. They categorised 35 percent of individuals with narcolepsy to have a diagnosable anxiety disorder. These statistics are greater than that seen in the general population. Lindsley and Crawford (1996) found 32 percent of individuals with narcolepsy to carry a

diagnosis of anxiety prior to the identification of a narcolepsy diagnosis. A twofold explanation of anxiety in individuals with narcolepsy has been positioned by Fortuyn, et al. (2010) to explain the high prevalence of anxiety. Anxiety may be secondary to narcolepsy and as a result of chronic exposure to stress related situations and/or experiences associated with narcolepsy. However, it may also be a primary pathophysiology of the narcolepsy disorder. There has been little research that has investigated anxiety in the narcolepsy without cataplexy population, making it difficult to rule in or out the role of hypocretin. Limited research suggests that diagnostic anxiety in narcolepsy may be associated with increased rates of cataplexy (Flosnik, Cortese, & Uhde, 2009). As mentioned in section 1.3.1, hypocretin plays a role in stress regulation.

Stress is a term used to define physical, psychological and social forces and pressures. Stress can be both the cause of pressure and the effect of pressure (Reber & Reber, 2001). The hypothalamic-pituitary-adrenal axis (HPA) and the sympathetic nervous system (SNS) are central psychological processes in the maintenance and activation of stress. Put simply, the HPA produces adrenal glands and cortisol, also known as the stress hormone. The SNS readies the body at times of stress activating necessary organs and glands. It sets our body into a state of emergency reaction. The peripheral nervous system (PNS) takes over the SNS when activated for a period of time. During chronic stress episodes long term activation of the HPA and SNS is present (Barlow & Durand, 2005). As discussed below, stress can increase the risk of diabetes mellitus type two and obesity. A link has been found between stress and disruptive sleep and - high rates of psychosocial stress are found in individuals with narcolepsy.

General emotional stress has been found to increase the risk of development of diabetes mellitus type two. This has been hypothesised to occur through behavioural and or psychological pathways (Pouwer, Kupper, & Adriaanse, 2010). Behavioural factors

such as poor eating habits (considering quality and quantity of food), reduced exercise, cigarette smoking, abuse of alcohol and general unhealthy lifestyle behaviours associated with emotional stress have been linked to diabetes mellitus type two (Bonnet, et al., 2005; Rod, Gronbaek, Schnohr, Prescott, & Kristensen, 2009). As mentioned above chronic stress is associated with long term activation of the HPA and SNS. These systems have been found to be associated with abdominal obesity. Abdominal obesity increases the risk of diabetes mellitus type two and thus chronic stress would increase the chance of diabetes mellitus type two through this pathway (Björntorp, 2001; Vogelzangs, et al., 2008).

Emotional stress has been found to have a twofold association with sleep. Stress can affect the onset, duration and quality of sleep. Sleep disruption, however, may not be a consequence of stress but the source of the stress itself (Pouwer, et al., 2010). Individuals with narcolepsy are more likely than the general population to experience everyday psychosocial difficulties that are potentially stressful such as; marital difficulties, embarrassment and reduced academic performance (Broughton & Broughton, 1994). Additionally, individuals with narcolepsy have been found to have poor psychosocial adjustment and significantly higher rates of psychosocial adjustment difficulties than other medical disorders including cardiac, mixed cancer or diabetes patients. Furthermore, individuals with narcolepsy are likely to report significant psychological distress and this has been found to be strongly correlated with day time activity disruption due to narcolepsy symptomatology (Bruck, 2001). Research has shown that over course of the disorder of narcolepsy some areas of psychosocial stress tend to decrease for individuals with narcolepsy. For example Costa (2001) found a significant decline in stress related to health care, sexual relationships and extended family relationships over a ten year period.

The findings in relation to depression, anxiety and stress highlight the significance of need to understand and address affective disturbances in the symtomatology and treatments of the disorder narcolepsy. It is possible that affective disturbance may not only be associated with narcolepsy but a component of its presentation.

1.5. Physiological, Behavioural and Psychological Aspects of Food Intake

There are three main theories, developmental, cognitive and psycho-physiological, within the literature that pertain to the initiation and selection of food intake. Developmental theories refer to factors including exposure, social learning and associative learning. Cognitive theorists describe factors such as attitudes, social norms, control (perceived) and ambivalence. Psycho-physiological approaches refer to factors such as neurochemicals, mood, arousal levels and stress. The research however suggests that there is a combination of developmental, cognitive and psycho-physiological factors that contribute to the initiation and selection of food intake (Ogden, 2010). This review will focus on behavioural and psycho-physiological approaches to food intake.

1.5.1 Initiation and Selection of Food Intake

Food intake, the behavioural action of eating food, is not considered a regulated variable. Food intake is defined as an effector or response mechanism. In this sense, it is able to be engaged and disengaged (Woods, 2009).

The role of the stomach has had a long conceptualisation as being central to the initiation of food intake. Based on this, hunger would occur when the stomach was empty. Eating occurs as long as food is made available. With the accumulation of food in the stomach distension during a meal occurs and hunger wanes while satiation takes over, causing eating to ultimately stop. However, further developments in research have

complicated understandings of the process of the initiation and regulation of food intake (Woods, 2009). Current research has tended to focus on the hypothalamus, and interrelated neurons, as the centre for controlling eating.

Brain cells rely on glucose molecules derived from the blood to coerce cellular activities. In this sense low blood glucose is considered a trigger to providing the sensation of hunger and in turn elicits eating (Langhans, 1996). What is of importance is not necessarily the level of blood glucose but rather the ability of the brain cells to obtain sufficient energy from the glucose. It has been proposed that hypocretin plays a central role in shortening energy homeostasis regulation through the initiation of a feeding response that is dictated by the falls of glucose levels followed by a termination of this process after ingestion of food (Cai, et al., 1999). Leptin is believed to mediate energy homeostasis and feeding behaviour through activation of varied sites

within the central nervous system. This activation provides a physiological satiety signal, mediates synaptic plasticity and provides the perception of reward through feeding behaviour (Oswal & Yeo, 2010). Given the hypocretin deficiency in individuals with narcolepsy (as noted in section 1.3.2) and the role of leptin in obesity and high incidence of obesity with individuals with narcolepsy (as discussed in section 1.4.1) the regulation of the discussed processes above may be impaired in individuals with narcolepsy contributing to abnormal feeding initiation and selection.

There is considerable research which suggests that increased dietary variety within individual meals and across meals increases the amount of food that one consumes. This is termed the “variety effect” (Meiselman, De Graaf, & Lesher, 2000; Zandstra, 2000). Variety within and across meals has been shown to lead to hyperphagia in animals. Hyperphagia is a term used to describe an increased appetite for food and an increased

consumption of food. Sensory specific satiety has been proposed as the driving mechanism for the variety effect (Raynor & Epstein, 2001). It is theorised that the sensory specific satiety occurs immediately after food consumption and is therefore not related to absorptive mechanisms (Abigail, Remick, & Patricia, 2009). This also suggests that food choice (selection of nutrients, vitamins and minerals) would vary from one point in time to another, rather than being fixed and stable.

Lammers, et al. (1996) found that individuals with narcolepsy, in comparison to controls, consumed fewer kilojoules. However, other research has presented mixed findings such as reports of no differences in kilojoule intake (Pollak & Green, 1990) and reports of higher kilojoule and carbohydrate intake, especially during lunch and dinner (Bruck, et al., 1989). Studies have also reported that individuals with narcolepsy have higher frequencies of snacking and are more likely to eat when not hungry, in comparison to, the general population (Bell, 1976; Bruck, Armstrong, & Coleman, 1994). Furthermore, these findings did not differ as a function of taking stimulant medication. It has been found that individuals with narcolepsy are more likely to snack in the evening (after dinner; Pollak & Green, 1990). Studies have also found that individuals with narcolepsy are more likely to choose the consumption of particular fluids. Individuals with narcolepsy have been found to be more likely to consume high in sugar content drinks such as soft drink and cordial (Bruck, et al., 1989) and to crave milk and sweets (Bell, 1976). There has been inference made within the literature that timing of food intake may be modulated by levels of sleepiness rather than influenced by the body's glucose levels (Pollak & Green, 1990). Please refer to section 1.4.4 for a further detailed explanation of this relationship.

Research into the initiation and selection of food/fluid intake within the narcolepsy population is currently limited and more than 15 years old. Collectively however, the

findings reported above in relation to consumption of food and drink within the narcolepsy population are inconsistent with the theory that individuals with narcolepsy would have a decreased appetite and /fluid intake due to a hypocretin deficiency.

1.5.2 Sleep/Wake Behaviour After Food Intake

Within the general population there is a body of research about the association between food intake and sleep/wake behaviour. As mentioned earlier even with medication, levels of sleepiness in individuals with narcolepsy have not been found to reduce to levels that are comparable to the general population (Bruck, et al., 2005). Presently, there has been limited research that has empirically measured the effects of sleepiness in individuals with narcolepsy in relation to their eating behaviours and attitudes. However, there is some research suggesting a possible link between the levels of sleepiness in narcolepsy and their eating behaviours and attitudes.

A postprandial dip is a normal state of sleepiness experienced after the consumption of a meal. Both dietary and psychobiological factors impact on the postprandial dip. The postprandial dip is affected by numerous factors including hunger, meal volume, meal constituents, time of day and daytime sleepiness (Stahl, Orr, & Bollinger, 1983). Meals high in carbohydrates have been found to increase subjective sleepiness after intake (Lowden, et al., 2004). A temporary decrease in sleep latency after the consumption of a meal occurs (Harnish, Greenleaf, & Orr, 1998) and the duration of a sleep episode increases during the postprandial dip (Zammit, Kolevzon, Fauci, Shindledecker, & Ackerman, 1995).

There have been very few studies that have investigated the effects of sleep on food consumption or food choice. Wells and Cruess (2006) found that sleep deprived (four hours or less of sleep) university students had a change in food choice, based on

responses to the food choice questionnaire (Steptoe & Pollard, 1995). After sleep deprivation they were less likely to choose food based on health, sensory appeal, natural content, price, weight control, familiarity or ethical concern. It was found however that food choice based on mood and convenience was consistent with when no sleep deprivation occurred. Additionally, it has been found that food intake is likely to increase after just one night of reduced sleep (Brondel, Romer, Nougues, Touyarou, & Davenne, 2010).

Although research is limited, a specific connection between timing of sleep and food intake has been found within individuals with narcolepsy. It has been found that individuals with narcolepsy are more likely to nap after meals, rather than before them. Additionally this tendency to nap after meals is more likely than that found in the general population. Specifically, Pollak & Green (1990) found that unmedicated individuals with narcolepsy with cataplexy had a peak in subjective alertness at meal onset (both scheduled and free running meals). Individuals with narcolepsy spent less time awake after each scheduled meal. There was a 150 minute period of increased tendency to nap and decreased subjective alertness, with tendency to sleep being most predominant after lunch. During the free running meal condition sleep tendency only increased significantly after lunch (or second peak eating time). Pollak & Green (1990) concluded no relationship between meal sizes or content of food with subjective alertness or tendency for sleepiness/naps but rather an underlying body rhythm was in play. Their findings suggest a common connection between the timing of food intake and excessive sleepiness (or intrusive naps) in individuals with narcolepsy.

Lammers et al. (1996) investigated the consumption of carbohydrates in unmedicated individuals with narcolepsy (with cataplexy only) and without comorbid depression. These researchers found that individuals with narcolepsy, in comparison to controls,

consumed fewer kilojoules a day because they were consuming fewer carbohydrates a day. It was concluded that their findings were consistent with reports that individuals with narcolepsy are more susceptible to the sleepiness-inducing effects of carbohydrates in their food**.** This conclusion is also consistent with the research of Bruck, et al. (1994) which measured the effects and differences of sleepiness after glucose (a simple carbohydrate) between individuals with narcolepsy (with cataplexy and not taking medication) and individuals without narcolepsy. They found an association between glucose intake and shortened wake duration and sleep onset latency, increased spontaneous and induced sleep stage changes and an increase in intensity of sleepiness.

Furthermore, qualitative investigations such as Bruck & Broughton (2001) have reported statements by participants with narcolepsy linking their association with sleepiness to food. Bell (1976) reported that their participants with narcolepsy commonly stated that in eliminating carbohydrates from their diets, their levels of sleepiness became less severe. Chabas, et al. (2007) reported several statements made by individuals with narcolepsy, who were drug free for two months, in response to the reasons for their abnormal eating behaviours (these behaviours are explained in detail in section 1.4.6). In summary, their reasons referred to ways of modifying food intake by controlling the timing of their sleepiness (e.g., "quickly eating snacks when feeling a sleep attack coming", "avoiding food at lunch to be more alert" and "skipping lunch to nap during lunch time") rather than for controlling body weight. Fortuyn, et al. (2008) reported that some of their participants with narcolepsy (with cataplexy) had reported that resistance to binge eating weakened after an accumulation of sleep pressure throughout the day. The possibility and recognition by individuals with narcolepsy that food makes them sleepy may be an explanation for the observed abnormal eating behaviours within this population. For example, an individual with narcolepsy may

avoid large meals during the day because it is not feasible to nap after a meal, but may 'binge eat' at night when sleeping is more feasible.

Overall, much of the research into the connection between excessive sleepiness and eating behaviours and attitudes in the narcolepsy population has been qualitative. There has been no research that has empirically measured an intentional/purposeful behavioural connection performed by individuals with narcolepsy between eating behaviours and ways of controlling their effects of sleepiness. However, there is some suggestion within the literature that there may be a possible pattern of behaviour related to eating and sleepiness within individuals with narcolepsy.

1.5.3 Emotionally Induced Eating

A connection between emotions, particularly depression, anxiety and stress, and eating patterns has long been established within the general population and in the population of individuals with eating disorders. However, the connection between depression, anxiety and stress levels and eating patterns within individuals with narcolepsy has not been sufficiently investigated.

Depression has been linked to a number of eating behaviours and attitudes. A change in appetite is a commonly occurring symptom of depression. In particular, patients with melancholic features of depression are characterised by a decrease in appetite and those with atypical features of depression are characterized by an increase in appetite (American Psychiatric Association, 2000). Furthermore, depression has found to be highly comorbid with binge eating disorder (Grucza, Przybeck, & Cloninger, 2007; Paxton & Diggens, 1997) and binge eating disorder has found to be associated with lifetime attempts of suicide (Grucza, et al., 2007). Individuals with depression tend to have a preference for sweets (Kazes, et al., 1994).

Anxiety and stress has been linked with abnormal eating behaviours and attitudes. Seventy-one percent of individuals with bulimia nervosa and seventy-one percent of individuals with anorexia nervosa have a lifetime comorbidity of an anxiety disorder (Godart, et al., 2003). Furthermore, in individuals with bulimia nervosa incidences of binge eating has found to occur when levels of self-rated anxiety are high (Elmore & de Castro, 1990) and binge eating disorder has been found to be highly comorbid with generalised anxiety and panic attacks (Grucza, et al., 2007). Stress is associated with changes in normal eating patterns. A positive correlation between the number of daily stressors and snack consumption has been found (Conner, Fitter, & Fletcher, 1999). However, the effect of stress on overall food intake is variable. Stress can affect overall intake of food in one of two ways; over or under eating (Torres & Nowson, 2007). Under stress individuals are more likely to choose snack foods which are highly palatable, energy dense and easy to prepare (e.g., sweets and chocolate) in comparison to meal foods that are less energy dense and not as easy to prepare (for example, vegetables, meat and fish; Oliver & Wardle, 1999). Stress has also been found to be linked with binge and compulsive eating behaviours (Squires & Kagan, 1986; Wolff, Crosby, Roberts, & Wittrock, 2000) with the tendency to binge eat being increased with interpersonal stress (Hilbert, Vögele, Tuschen-Caffier, & Hartmann, 2011).

Despite the above information, limited research has considered the role of mood in the abnormal eating patterns found within individuals with narcolepsy. Two published studies at the time of writing have quantitatively measured the connection between depression and abnormal eating. Fortuyn, et al. (2008) investigated individuals with cataplexy (participants included both on and off medication) and found that those with depressed mood had high incidences of meeting diagnostic criteria for an eating disorder (not otherwise specified) and reports of uncontrolled binge eating with a lack

of control. Chabas, et al. (2007) however, did not find a correlation between scores on a measure of depressive symptomatology and scores on a measure of disordered eating. Their study included participants with unmedicated (for two months) narcolepsy with and without the symptom of cataplexy. At the time of writing, no published research had quantitatively measured the relationship between anxiety and stress with abnormal eating patterns and/or attitudes within individuals with narcolepsy.

Given the high prevalence rates of depression, anxiety and stress within the narcolepsy population and the effects of these affective disturbances within the general population, it may be possible that the abnormal eating patterns documented in narcolepsy (i.e., changes in food intake, increased snacking, craving for sweets and binging) are related to the comorbid symptomology of depression, anxiety and/or stress in individuals with narcolepsy, rather than an association with the disorder itself. Research in the area of narcolepsy and eating behaviours and attitudes has not investigated the role of anxiety and/or stress and limited research has investigated the role of depression. Such research is needed.

1.5.4 Disordered Eating

Eating disorders are defined by severe disturbances in eating behaviour. Specifically, the DSM-IV (American Psychiatric Association, 2000) defines three forms of eating disorders. Anorexia nervosa is an eating disorder characterised by refusal to sustain a minimum normal body weight. Bulimia nervosa is characterised by binge eating episodes that precede compensatory behaviour for example, use of laxatives or excessive exercise. The third defined eating disorder is eating disorder not otherwise specified (EDNOS). This includes eating disorders that do not meet full criteria for anorexia nervosa or bulimia nervosa. Binge eating disorder is included under the

EDNOS classification. Binge eating disorder is defined by behaviour and subjective lack of control over eating binges with significant distress related to episodes of binge eating (excessive eating in a discrete amount of time). Unlike bulimia nervosa, compensatory behaviours are not associated with binge eating episodes. Limited research has investigated eating disorders within individuals with narcolepsy. However, available research indicates significantly higher prevalence rates of eating disorders within individuals with narcolepsy in comparison to the general population.

Lifetime prevalence of anorexia nervosa in the general population is 0.5 percent and for bulimia nervosa one to three percent in females. Prevalence rates for males are one-tenth of that of females for both anorexia nervosa and bulimia nervosa. Research into binge eating as an eating disorder is less established than anorexia nervosa and bulimia nervosa research. A prevalence rate within the general population is estimated at 0.7 to 4 percent (American Psychiatric Association, 2000). There is also evidence to suggest that individuals with eating disorders are likely to have abnormal sleep patterns. Sleep disturbances have been identified in approximately 50 percent of females with anorexia nervosa or bulimia nervosa. Sleep disturbance has been found more likely to occur when binge eating or purging are predominant symptoms. Higher frequencies of binge eating and purging are subsequently found in those with sleep disturbances and comorbid eating disorders. Kim, et al. (2010) have suggested that, based on their findings presented above, an important clinical indicator of eating disorders is sleep disturbance.

Bulimia nervosa like symptoms have been described within individuals with narcolepsy since the 1930's (Daniels, 1934). Early research also described obsessive and compulsive eating experienced by individuals with narcolepsy (Bell, 1976). However, such observations have not been quantitatively confirmed until recently. Kotagal, et al

(2004) had found that children with narcolepsy (on and off medication) engaged in binge eating behaviours. In adults with unmedicated narcolepsy an increased prevalence rate of EDNOS has been found. Specifically, 54 percent of the narcolepsy sample of Chabas, et al. (2007) had abnormal eating patterns in the clinical range. There was no difference of eating disorder scores between participants with narcolepsy with or without cataplexy or as a function of BMI. Fortuyn, et al. (2008) found 23.3 percent of their participants with narcolepsy (with cataplexy) to meet the clinical criteria for an eating disorder. EDNOS (incomplete binge eating disorder) was the most common classification found. A clinical diagnosis of binge eating disorder was generally considered incomplete due to "marked distress regarding binging" not being present (Fortuyn, Mulders, Renier, Buitelaar, & Overeem, 2011). Overwhelming cravings for food and binge eating behaviours were the most common eating disorder symptoms with 25 percent of participants with narcolepsy reporting to engage in binging behaviour twice or more a week. Medication use was not found to impact on these findings (Fortuyn, et al., 2008). Dimitrova, et al.(2011) found individuals with narcolepsy with cataplexy to be more likely to self report moderate and severe binge eating behaviours than individuals with narcolepsy without cataplexy and individuals without narcolepsy. Participants in this study maintained their regular medications, and no effects of medication on relevant questionnaire scores were found. In contradiction to the above findings, Dahmen, et al. (2001) found no significant difference between individuals with narcolepsy and controls on diagnostic criteria for eating disorders or on the symptom of binge eating behaviour. A recent study by Palaia, et al.(2011) found that individuals with narcolepsy with cataplexy were more likely to engage in nocturnal compulsive behaviours, specifically sleep related eating disorder. Sleep related eating

disorder is a non-REM parasomnia that is defined by involuntary eating and drinking during arousals from sleep.

Overall, above findings generally suggest that the eating behaviours and attitudes of individuals with narcolepsy are not consistent with a simple decrease in food intake and appetite, as would be expected if their hypocretin deficiency was the main controlling factor. Furthermore, neither medication nor the symptoms of cataplexy have been found to impact the above results. Therefore there may be another contributor (besides medication and hypocretin explanations) to the observed eating behaviours and attitudes in individuals with narcolepsy. The current study purposes two possible suggestions based on literature presented above;

1. the level of sleepiness experienced by individuals with narcolepsy

2. mood, in particular levels of depression, anxiety and/or stress, of individuals with narcolepsy.

1.6 The Current Study

1.6.1 Rationale

Overall, evidence suggests a pattern of abnormal eating in individuals with narcolepsy that is inconsistent with a simple decrease in appetite and food intake, as would be expected if their medication and hypocretin deficiencies were the main controlling factors. Furthermore, despite some research suggesting a possible link, little is known about how sleepiness, depression, anxiety and stress may affect the eating behaviours and attitudes of individuals with narcolepsy.

Examination of the differences in eating behaviours and attitudes of individuals with narcolepsy in comparison to individuals without narcolepsy will cover a number of

different areas. Firstly, it is important to examine whether the hypothesised differences in food behaviours and attitudes in people with narcolepsy can be documented in comparison with a matched control group of people without narcolepsy. Secondly, it is of interest to know what attributions and/or motivations are offered by the participants with and without narcolepsy about their eating patterns in relation to a variety of factors, including sleepiness. Thirdly, understanding behavioural and attitude differences with regard to eating patterns between individuals with and without narcolepsy will help medical and mental health professionals in guiding management of symptoms, counselling and potentially preventing comorbidity (e.g., obesity, diabetes and eating disorders). Furthermore, it is of significance to examine the eating behaviours and attitudes of individuals with narcolepsy in regards to their normal everyday functioning, specifically with respect to stimulant medication status. This will assist medical and mental health professionals in providing an understanding and development of symptom management which is applicable to the everyday experience of individuals with narcolepsy receiving treatment.

The current study cannot make specific conclusions on the role of hypocretin for a number of reasons. Firstly, the study focuses on everyday eating patterns and attitudes and does not require withdrawal from medication. It is known that the effects of stimulant medication may decrease food intake. In addition, the hypocretin status of individuals with narcolepsy in Australia is rarely documented and this status of participants in the current research is unknown. Some of the findings will however, be considered as a function of whether the individuals experience the symptom of cataplexy or not (and, as discussed above, lack of hypocretin is typically associated with the presence of the cataplexy symptom).

1.6.2 Aims and Hypotheses

The primary aim of the current study is to examine the differences between the eating behaviours and attitudes of individuals who have been diagnosed with narcolepsy and matched individuals (controls) without narcolepsy or daytime sleepiness (matched on age and sex variables). The secondary aim of this study is to investigate whether there are differences in the eating behaviours and attitudes of individuals with narcolepsy as a function of their reported levels of daytime sleepiness, depression, anxiety and/or stress.

There are 10 hypotheses for the current study;

1) There will be a significant difference between individuals with narcolepsy and controls in the reported number of snacks and drinks consumed. It is expected that individuals with narcolepsy will consume more snacks and drinks.

Rationale: Studies have reported that individuals with narcolepsy have higher frequencies of snacking, are more likely to eat when not hungry and crave drinks such as soft drink, cordial and milk, in comparison to, the general population (Bell, 1976; Bruck, et al., 1989; Bruck, et al., 1994).

2) There will be a significant difference between individuals with narcolepsy and controls in their meal sizes for breakfast and lunch. It is expected that individuals with narcolepsy will have smaller breakfast and lunch meals than controls.

Rationale: Previous studies (such as Chabas, et al., 2007) have quoted qualitative reports from participants that suggest a purposefully timing of food intake to avoid daytime sleepiness. Given Pollak & Green's (1990) findings that individuals with narcolepsy had increased tendency to nap and decreased subjective alertness after meals, with tendency to sleep being most predominant after lunch and the assumption

that people generally have more day time commitments than evening it may be more likely that individuals with narcolepsy would indulge more at dinner time when napping afterwards may be more convenient. Testing this hypothesis 2 may provide valuable information to aid in understanding the eating patterns and timing of food intake in individuals with narcolepsy.

3) There will be a significant difference between individuals with narcolepsy and controls in meal size for dinner. It is expected that individuals with narcolepsy will have larger dinner meals than controls.

Rationale: Same as rationale above for hypothesis 2.

4) There will be no difference between individuals with narcolepsy and controls in their total meal sizes across the whole day.

Rationale: Findings on daily calorie intake has been mixed. It is likely that snacking behaviour is not a compensatory behaviour for meal intake but may be related to mechanisms to control sleepiness or related to depression, anxiety or stress.

5) There will be a difference between individuals with narcolepsy and controls on the mood factors on both the Meal Choice Questionnaire and the Snack and Drink Choice Questionnaire (choosing meals, snacks and drink to aid in altering mood). There will be no significant difference on the other factors in either questionnaire (health, natural content, convenience and weight control).

Rationale: The current study has separated the food choice questionnaire into two questionnaires to measure meal choice and snack and drink choice separately. This is consistent with previous research investigating food and drink consumption in

narcolepsy such Bruck, et al. (1989) and Pollak & Green (1990) whose research separated meal, snack and drink consumption in their investigations.

Sensory specific satiety suggests that food choice (selection of nutrients, vitamins and minerals) varies from one point in time to another, rather than being fixed and stable (Abigail, et al., 2009). Individuals with narcolepsy are more likely to be in a depressed (Lindsley & Crawford, 1996), anxious (Fortuyn, et al., 2010) and stressed (Bruck, 2001) mood and these factors have been found to affect abnormal eating behaviours (American Psychiatric Association, 2000; Godart, et al., 2003; Torres & Nowson, 2007). Therefore individuals with narcolepsy may be more likely to make food and drink choices based on present mood.

6) There will be a significant difference between individuals with narcolepsy and controls on the binge factor (measure of binge eating behaviours) taken from the Bulimia Test (BULIT), with individuals with narcolepsy being more likely to binge eat.

Rationale: The majority of previous research has found an increased prevalence of binge eating behaviours in individuals with narcolepsy in comparison to those without narcolepsy (Chabas, et al., 2007; Dimitrova, et al., 2011; Fortuyn, et al., 2008; Kotagal, et al., 2004)

7) There will be a significant difference between individuals with narcolepsy and controls on the Meal and Snack Timing Questionnaire (MSTQ). It is expected that individuals with narcolepsy will be more concerned with the timing of their meals and snacks in relation to sleepiness.

Rationale: There has been no research that has empirically measured a purposeful behavioural connection between eating behaviours and the effects of sleepiness in

individuals with narcolepsy. However, there is some suggestion within the literature that there may be a possible pattern of behaviour related to eating and sleepiness within individuals with narcolepsy. Previous studies (such as Chabas, et al., 2007) have quoted qualitative reports from participants that suggest a purposefully timing of food intake to avoid daytime sleepiness. This rationale extends that of hypothesis 2.

8) There will be a significant difference between individuals with narcolepsy who report severe daytime sleepiness and individuals with narcolepsy reporting less severe daytime sleepiness (both considered as when on medication) on the binge factor (measure of binge eating behaviours derived from the BULIT), breakfast, lunch and dinner meal size and on the mood factor of the MCQ and SDCQ (choosing meals, snacks and drink to aid in altering mood). The direction of the differences will be consistent with the role of increased sleepiness in the above hypotheses testing these variables.

Rationale: This hypothesis extends the rationale of hypothesis 2 and 7. As research indicates that sleepiness may impact on eating behaviours and choices (Bell, 1976; Bruck & Broughton, 2001; Chabas, et al., 2007) it is assumed that severity of sleepiness would also have an impact. It is of interest to investigate whether poor symptom management of excessive daytime sleepiness may be an influential variable on abnormal eating patterns observed in individuals with narcolepsy. No study at the time of writing has quantitatively investigated severity of sleepiness and abnormal eating behaviours and attitudes within the narcolepsy population.

9) There will be a significant difference between individuals with narcolepsy and controls on their levels of depression, anxiety and stress. It is expected that individuals with narcolepsy will have more depression, anxiety and stress.

Rationale: Previous research has found individuals with narcolepsy to have significantly higher levels of depression, anxiety and stress in comparison to individuals without narcolepsy (Bruck, 2001; Fortuyn, et al., 2010; Lindsley & Crawford, 1996).

10) There will be a difference between individuals with narcolepsy who have normal-to-mild versus moderate-to-severe depression, anxiety and/or stress on the binge factor (measure of binge eating behaviours derived from the BULIT), breakfast, lunch and dinner meal size, on the mood factors of the MCQ and SDCQ (choosing meals, snacks and drink to aid in altering mood). It is expected that individuals with narcolepsy who have normal-to-mild versus moderate- to-severe depression, anxiety and stress would score higher on all the listed variables than individuals with normal-to-mild depression, anxiety and stress levels. As the effect of affective factors on eating can be, the direction of these differences will be exploratory.

Rationale: This hypothesis extends on the rationale of hypothesis 5 and 9**.** It is of interest to investigate whether poor symptom management of depression, anxiety and/or stress may be an influential variable on abnormal eating patterns observed in individuals with narcolepsy. Limited research has investigated the relationship between depression and eating behaviours and attitudes within individuals with narcolepsy and no published research at the time of writing has investigated the relationship between anxiety and/or stress and eating behaviours and attitudes within individuals with narcolepsy.

Chapter 2: Methodology

2.1 Participants

The sample of the current study consisted of 73 individuals with unambiguous narcolepsy (*M* = 58.41 yrs, SD = 18.45) and 74 controls (*M* = 57.35 yrs, SD = 15.35). Response rate was 80 percent. Groups were matched on age and gender.

The addresses of 166 individuals with narcolepsy were provided by the Narcolepsy and Overwhelming Daytime Sleepiness Society (NODSS), an Australian national self help group for narcolepsy. Each year, on completing their membership renewal, members of NODSS are asked whether they wish to opt out of being approached for potential participation in research. Only those members who had not chosen to opt out were approached.

The control group was recruited by asking the 166 individuals with narcolepsy who were provided with a survey package to voluntarily invite an adult spouse, partner or friend who were not blood relatives to participate in the current study. Groups were not matched on social class, however, through use of the above method to enlist a control group it is assumed that participants would be derived from a similar social class (e.g., abundance of food and psychosocial stresses). All participants were over the age of 18. Controls were excluded if they reported a known or suspected sleep disorder, another disorder that restricts food intake (in particular their calorie and sugar intake), such as diabetes or anorexia nervosa (disorders such as lactose intolerant and irritable bowel syndrome were allowed within the sample) or excessive levels of day time sleepiness (> 16 as rated by the Epworth Sleepiness Scale. Scores of 16 or more indicate high levels of daytime sleepiness and are not found in those without sleep disorders such as narcolepsy and/or sleep aponea. Research has shown that scores approximately ranging

between 10 and 15 on the ESS overlap between normal and abnormal sleepiness; Johns, 1991, 1992). Potential narcolepsy participants with a co-morbid disorder that restricted food intake (in particular their calorie and sugar intake), such as diabetes or anorexia nervosa (disorders such as lactose intolerant and irritable bowel syndrome were allowed within the sample). Individuals with narcolepsy were only included in the study if their narcolepsy diagnosis was unambiguous, as was determined by using the diagnostic criteria outlined in the ISCD-2 (American Sleep Disorders Association, 2005). Specifically, all participants with narcolepsy were considered on a case by case basis. In accordance with diagnostic criteria outlined in the ISCD-2 (American Sleep Disorders Association, 2005) all individuals included in the narcolepsy group had an onset of narcolepsy symptomatology for at least three months. Minimal criteria for inclusion included either;

1) excessive daytime sleepiness and cataplexy or

2) excessive daytime sleepiness and a polysomographic finding (MSLT) indicative of narcolepsy.

In order for polysomgraphic findings to be found indicative of narcolepsy a nocturnal polysomnography must be followed by a multiple sleep latency test (MSLT). Sleep latency would need to be measured at less than or equal to eight minutes and with two or more sleep onset rapid eye movement periods (SOREMP's) on the MSLT (American Sleep Disorders Association, 2005).

Seventy-seven percent of the participants with narcolepsy reported that they suffered from cataplexy. Eighty five percent of participants with narcolepsy reported that at the time of the survey they were taking medication for their narcolepsy diagnosis (stimulant or anticataplexy medication). Sixty two percent of participants with narcolepsy were

taking stimulant medication only, four percent were taking anticataplexy medication only and nineteen percent were taking both stimulant and anticataplexy medication. Stimulant medication for this sample included dexamphetamine, ritalin/methylphenidate and modafinil. Anticataplexy medication was normally tricyclic antidepressants. Forty-three individuals with narcolepsy and 40 individuals without narcolepsy reported that they took medication for a condition other than narcolepsy.

Please refer to Appendix F for a summary of the clinical and diagnostic features of the sample of participants with narcolepsy.

2.2 Materials

The following section describes each questionnaire and the scoring procedure for each. Please see Appendix C for a copy of all questionnaires distributed.

2.2.1 General Questionnaire

All participants were given a questionnaire of demographic details and participants with narcolepsy were asked to fill in a questionnaire about their diagnosis and symptoms. The additional questions for the narcolepsy population were to aid in confirming an unambiguous diagnosis of narcolepsy.

Items were given a numerical rating (for example, 1 to 5) for purposes of data entry and interpretation. Body mass index (BMI) was measured using the standard classification as recommended by the World Health Organization and a numerical value given to each category of weight (1 = underweight, 2 = healthy weight, 3 = overweight and 4 = obese; *Australia's Health 2010*, 2010).

2.2.2 The Epworth Sleepiness Scale

The Epworth Sleepiness Scale (ESS; Johns, 1991) consists of eight questions that together provide an overall measure of one's level of general daytime sleepiness. The level of general daytime sleepiness is also referred to as the average sleep propensity. The average sleep propensity, at any given time, is considered the function of the ratio of total sleep drive to the competing total wake drive. This provides the probability of falling asleep in a variety of daytime situations (Johns, 1993). The ESS has established reliability and validity. As measured by Cronbach's alpha, the ESS has a high internal consistency level (0.88). The test-retest reliability is also high with a Pearson's Correlation of 0.822 (Johns, 1992). The significant correlations between the ESS and sleep latency, as measured by the multiple sleep latency test, and an overnight polysomnography (-0.379 and -0.514 respectively) shows the convergent construct validity of the ESS (Johns, 1991).

In the participants with narcolepsy the ESS was completed twice. The ESS was completed once as with stimulant medication and completed again as without stimulant medication. The ESS without stimulant medication score was to provide an indication of underlying sleepiness severity and forms part of the clinical picture of their symptoms (see Appendix F). Control participants are also asked to complete the ESS. The ESS for this group was used as a screening tool so that those who scored within the excessive daytime sleepiness range could be excluded.

In order to aid analysis and interpretation each participant was given a total ESS score based on provided responses. Scoring was based on that outlined by Johns (1991). Levels of sleepiness were divided within the narcolepsy group around the median ESS

score (completed as for sleepiness when medicated). ESS scores used in the screening process (see Appendix F) were based on sleep propensity self report when unmedicated.

2.2.3 The Depression Anxiety Stress Scale

The Depression Anxiety Stress Scale (DASS; Lovibond & Lovibond, 1995b) consists of forty-two questions. The DASS provides measures on three factors; depression, anxiety and stress. These factors are measured as of the past seven days. The DASS has established reliability and validity. High internal consistency, as measured by Cronbach's alpha, has been found for the depression (0.96), anxiety (0.89) and stress (0.93) scales (Brown, Chorpita, Korotitscw, & Barlow, 1997). Convergent validity has been shown through significant correlations of 0.81 with the DASS anxiety scale and the Beck Anxiety Inventory and 0.74 with the DASS depression scale and the Beck Depression Inventory (Lovibond & Lovibond, 1995a).

In order to aid analysis and interpretation each individual was provided with a calculated total score for depression, for anxiety and for stress based on responses to items. Scoring was in accordance with Lovibond and Lovibond (1995b) set criteria. These scores were used when analysis refers to total scores for depression, for anxiety and for stress. Based on these scores participant's levels of depression, anxiety and stress were then divided into normal to little depression/anxiety/stress versus moderate to high depression/anxiety/stress. This division was based on cut-off scores provided for interpretation by Lovibond and Lovibond (1995b) that is, scores between 0 and 13 for depression, 0 and 9 for anxiety and 0 to 18 were categorised as none (normal) to little depression and 14 to highest score for depression, 10 to highest score for anxiety and 19 to highest score for stress were categorised as moderate to high (extremely severe). The

DASS does not provide a cut off score for clinically significant depression, anxiety or stress.

2.2.4 The Bulimia Test

The Bulimia Test (BULIT; Smith & Thelen, 1984) consists of 36 items and measures the symptoms of bulimia. The BULIT provides a measure of symptoms of bulimia nervosa. For the current study the BULIT was used to provide a measure for binge eating, based on a derived "binge" factor (see below). The BULIT has established reliability and validity. Internal consistency as measured by Chronbach's alpha is 0.98 (Wertheim, 1989). Convergent validity has been shown with significant correlation at 0.80 between the BULIT and bulimia nervosa patients and controls (Smith & Thelen, 1984).

In order to assist analysis and interpretation all items that contributed to the BUILT were given a numerical rating (for example, 1 to 5), as set out by Smith & Thelen's (1984). For the purposes of this study an explanatory factor analysis (based on all completed questionnaires) was run on all items of the BULIT in order to derive a "binge factor". The "binge factor" was determined by selection of the factor which most resembled items from the BULIT which referred to behaviours and /or attitudes associated with binge eating. Refer to Table 3.9 for a list of the specific variables that made up the selected binge factor. Reliability was shown for the Binge Factor with Cronbach's Alpha at 0.881. See section 3.2.6 for factor analysis details.

2.2.5 Meal Choice and Snack and Drink Choice Questionnaires

The Meal Choice Questionnaire (MCQ) consisted of 27 questions. The MCQ measures the motives associated with meal choice. All except three of these questions were

adapted from the Food Choice Questionnaire (FCQ; Steptoe & Pollard, 1995). The word "food" in the items taken from the FCQ was replaced with "meals" for the MCQ. The three researcher established questions were; "It is important to me that the meals I eat on a typical day ….."

- "contain little to no sugar"
- "contain complex carbohydrates e.g., wholemeal ingredients"
- "contain a lot of sugar"
- "do not contain items I am sensitive or allergic to"

The Snack and Drink Choice Questionnaire (SDCQ) was developed for this study and consisted of the same questions as the MCQ with one additional item. SDCQ measured the motives associated with snack and drink choice. The word "food" in the items taken from the FCQ was replaced with "snacks and drinks" for the SDCQ. The additional researcher established question was "it is important to me that the snacks and drinks I consume on a typical day …. contain caffeine."

Both the MCQ and SDCQ included questions that make up five factors from the FCQ; health and food content, mood, convenience, weight control and natural content. The factor loadings from the original questionnaire (the FCQ) which were adapted for the current study ranged from 0.57 to 0.87. The FCQ has established reliability and validity. Test-retest reliability for all scales of the FCQ are greater than 0.70. Furthermore, as measured by Cronbach's Alpha the internal consistency of the factors are high (health = 0.81, convenience = 0.84, natural content = 0.86 and weight control = 0.85; Steptoe & Pollard, 1995). For the purpose of the current study the FCQ was reconstructed into two questionnaires, one based on meals and the other snack and drinks. It was of interest to

the researchers whether a different pattern of behaviours may be evident in analysis between meals and snacks and drinks.

For analysis and interpretation purposes all responses to items on the MCQ and SDCQ were given a numerical rating (not at all important = 1, a little bit important = 2, moderately important = 3 and very important = 4). A confirmatory factor analysis was then run on the MCQ and SDCQ to determine factors for the current studies population. These derived factors were then calculated and total scores were used in subsequent analysis. Table 3.3 shows both Steptoe and Pollard's (1995) factor names, as well as revised factor names for the current study. Table 3.5 shows comparisons of variable factor loadings between the FCQ, MCQ and SDCQ. Please refer to Table 3.4 for a comparison of reliability coefficients between the FCQ, MCQ and SDCQ. See section 3.2.5 for factor analysis details.

2.2.6 A Meal and Snack Timing Questionnaire

The Meal and Snack Timing Questionnaire (MSTQ) consisted of six questions that provided a measure of the timing of food intake, in relation to sleepiness. This was developed by the researchers and was based upon issues arising from the literature. The questionnaire was designed in a way to facilitate data collection and analysis for hypotheses related to behavioural importance of timing of food intake in relation as a response or consequence of sleepiness.

To assist with analysis and interpretation each item response on the MSTQ was given a numerical rating (not at all important = 1, a little bit important = 2, moderately important = 3 and very important = 4). A total overall score for the MSTQ was then calculated based on the relevance of participant responses to timing. Higher responses were indicative of higher

importance for timing food intake in relation to sleepiness. Cronbach's alpha for the MSTQ for this sample was good (0.87).

2.2.7 One Day Food and Drink Diary

Participants were asked to fill in a one day food diary, developed by the researchers. This was used to measure frequency of snack and drink intake and frequency and quantity (small, medium or large) of meal intake. Participant's self reported as to whether food was a meal or snack and the size of the meal. Any identified food consumed between meals was considered a snack (i.e., labelled "morning/afternoon tea", supper" and "dessert" or food intake not labelled between meals.). Water was excluded as a drink. When participants did not provide information on the size of the meal, this was considered missing data.

For scoring and interpretation meal sizes were given a numerical value (small = 1, medium = 2 and large = 3). Average meal size was calculated by using a total score of meal size across the day. Total numbers for snacks and drinks were used for analysis.

2.2.8 Information to Participants Involved in Research Sheet

The Information to Participants Involved in Research Sheet described the nature and aims of the study. This was used to assist in explaining the research to potential participants and recruit their participation (see Appendix D).

2.3 Procedure

Prior to commencement of recruitment, approval of the research design was granted by NODSS and ethics approval was granted by The Victoria University Human Research Ethics Committee, Victoria University (please see Appendix B for VU ethics approval letter). The NODSS president, vice president and treasurer provided a joint supporting

letter for the researchers ethics application to The Victoria University Human Research Ethics Committee, Victoria University (please see Appendix A for NODSS supporting letter).

One hundred and sixty six individuals with narcolepsy on the NODSS registration for participation in research list were mailed two survey packages, one for themselves and one for an adult spouse, partner or friend without narcolepsy (please see Appendix D for Information for Participants Involved in Research and Appendix C for questionnaires). A follow up letter was sent after fourteen days and a second follow up letter was sent fourteen days after the first follow up letter (see Appendix E). Two reminder letters were considered necessary as one of the effects of narcolepsy is forgetfulness (Ohayon, et al., 2005). Participants were asked to mail back their filled in questionnaires to the Psychology Department at Victoria University, St Albans Campus (Melbourne Australia), in the self addressed and stamped envelopes provided with the survey packages. To aid in maintaining confidentiality each package included two self addressed and stamped envelopes, one for individuals with narcolepsy and one for control participants.

2.4 Data Analysis

To analyse the data obtained, the PASW Statistic 18.0 (SPSS Statistics) and SPSS Statistics 20.0 program for Microsoft Windows was utilised. Microsoft Excel program was used to study scatter plots. Firstly demographic data was explored through frequency statistics. Prior to analysis for each hypothesis appropriate assumption testing for parametric testing was conducted. When the assumption of normality was violated the Mann-Whitney U Test was used, as opposed to the independent group t-test statistic (or analysis of variance). Assumption testing for normality is not considered critical for

non-parametric statistics (Coakes & Steed, 2007). The Z approximation was used as it provides the inclusion of an adjustment for equal ranks. The current data has many equal ranks. When hypotheses referred to factors, confirmatory and/or explanatory factor analysis was undertaken. Interpretation of factors was based on both statistics and information from previous research. Single variable analyses were chosen to allow exploration of the contribution of factors individually. This was the preferred method because several of the variables measured had not been explored before. Given that a series of statistics was undertaken the issue of alpha inflation was considered. All enquires were pre-planned as part of a set of hypotheses or considered a principal point of the current research study. In view of this, and in order to provide sufficient power to the analyses an alpha level was set at 0.05, (unless otherwise stated) and no further adjustments were deemed necessary (Keppel & Wickens, 2004). A maximum acceptable level of missing data was, somewhat arbitrarily, set at fourteen percent prior to data analysis. If missing data for any variable exceeded this set percent relevant collected data was considered to be of questionable reliability and excluded from analysis.

A power analysis was conducted using the analysis program, G*Power 3 (Faul, Erdfelder, Lang, & Buchner, 2007). This found that given our sample sizes in each group and an alpha of .05, analyses on the differences between individuals with narcolepsy with or without narcolepsy had sufficient power to detect a large (0.8) and medium (0.5) effect sizes, power equalled .998 and .85 respectively. To detect a low effect size (0.2) power was low (0.26).

Chapter 3: Results

3.1 Demographics and Descriptive Data

Analysed data consisted of 73 individuals (53 female and 20 male) with unambiguous narcolepsy and 74 controls (45 female and 29 male). The sample of individuals with narcolepsy consisted of 17 participants without the symptom of cataplexy (12 female and 5 male). The mean age of individuals with narcolepsy was 58.41 years with a standard deviation of 18.45 and for controls the mean age was 57.24 years with a standard deviation of 15.35).

An intention of the current study was to control for age and gender between groups. Before the undertaking the analyses of hypotheses the data was analysed to confirm no difference between groups (with narcolepsy and without) on age and gender. Prior to analysis the distribution for age in the narcolepsy group was found to be negatively skewed and flat. The distribution for controls was found to be normal. The results from a Chi-square test for relatedness or independence revealed that there was not a significant difference between individuals with narcolepsy and without narcolepsy on age X^2 (1, $\underline{N}$ = 147) = 2.30, p = .129 or gender X^2 (1, $\underline{N}$ = 147) = 2.25, p = .162.

The mean Epworth Sleepiness Scale (ESS) score for controls was 5.03 with a standard deviation of 3.07 and a range of 15 (0 to 15). This contrasted with a mean ESS (unmedicated) for those with narcolepsy of 19.19 with a standard deviation of 3.55 and a range of 13 (11 to 24).

The body mass index (BMI) of participants was also investigated. Prior to analysis the distribution for BMI in the narcolepsy and control groups was found to be negatively skewed and flat. The results from a Mann-Whitney U Test revealed that there was not a

significant difference between individuals with narcolepsy and without narcolepsy on BMI (U = 2178, Z = -1.064, p = .287). However, a graph of the frequencies shows a trend towards those with narcolepsy moving towards obesity. Please refer to figure 3.1.

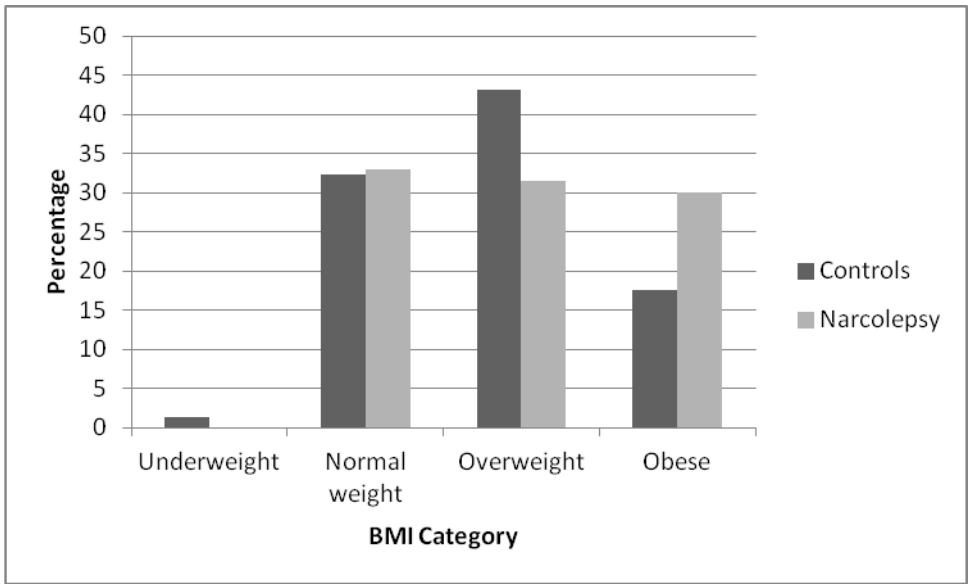

Figure 3.1 Percentage of Narcolepsy and Control Participants in Different Body Mass Index Categories

3.2 Hypothesis Testing

3.2.1 Hypothesis 1: There will be a significant difference between individuals with narcolepsy and controls in the number of snacks and drinks consumed. It is expected that individuals with narcolepsy will consume more snacks and drinks than controls.

Prior to analysis the distribution for snacks and drinks in both the narcolepsy and control groups were found to be positively skewed and peaked. The results from a Mann-Whitney U Test showed that individuals with narcolepsy consumed significantly more snacks and more drinks per day than individuals without narcolepsy (see Tables 3.1 and 3.2).

Table 3.1

Descriptive and Mann-Whitney U Results for Number of Snacks per day for Individuals with Narcolepsy and Controls

Group	Median	Range	Mean (Standard Deviation)	*U*	Z	*p*
Control	2	7	1.76 (1.40)			
Narcolepsy	2	10	2.87 (1.93)			
				1976	-2.02	.043

Table 3.2

Descriptive and Mann-Whitney U Results for Number of Drinks per day for Individuals with Narcolepsy and Controls

Group	Median	Range	Mean (Standard Deviation)	*U*	*Z*	*p*
Control	3	8	3.44 (1.68)			
Narcolepsy	4	12	4.67 (2.62)			
				1690	-3.321	.001

3.2.2. Hypothesis 2: There will be a significant difference between individuals with narcolepsy and controls in their meal sizes for breakfast and lunch. It is expected that individuals with narcolepsy will have smaller breakfast and lunch meal sizes than controls.

Hypothesis 2 was not analysed due to a high proportion of missing data. Specifically, the variable "meal size for breakfast" had 19 percent of data missing for the narcolepsy group and 23 percent of data was missing for the control group. The variable "meal size for lunch" had 16 percent of data missing for the narcolepsy group and 30 percent of data missing for the control group.

3.2.3. Hypothesis 3: There will be a significant difference between individuals with narcolepsy and controls in meal size for dinner. It is expected that individuals with narcolepsy will have larger dinner meal sizes than controls.

Hypothesis 3 was not analysed due to a high proportion of missing data. Specifically, the variable "size for dinner" had 15 percent of data missing for the narcolepsy group and 24 percent of data was missing for the control group.

3.2.4. Hypothesis 4: There will be no difference between individuals with narcolepsy and controls on their average meal size score across the whole day.

Hypothesis 4 was not analysed due to a high proportion of missing data. Specifically, the variable "average meal size across the whole day" had 27 percent of data missing for the narcolepsy group and 39 percent of data was missing for the control group.

3.2.5. *Hypothesis 5*

Prior to the analyses for Hypothesis 5 it was necessary to conduct confirmatory factor analyses on the two questionnaires that were developed by the researchers. This was framed as Hypothesis 5a.

Hypothesis 5a: Factors of the Meal Choice Questionnaire (MCQ) and Snack and Drink Choice Questionnaire (SDCQ) in the sample of this study will be the same as the factors defined by Steptoe & Pollard, 1995 in relation to the Food Choice Questionnaire (FCQ).

A confirmatory factor analysis was run on the MCQ. Multiple variables were positively or negatively skewed, however factor analysis is robust to the assumption of normality (Tabachnick & Fidell, 2007). Although differences in skewness among variables may suggest curvilinearity spot check examinations of randomly selected scatter plots

showed no evidence of true curvilinearity. Furthermore, transformations are not considered favorable due to the goals of analysis (i.e., confirming pre-defined factors; Tabachnick & Fidell, 2007). All outliers were deleted prior to analysis. Outliers were defined as scores appearing significantly inconsistent with the remainder of the data set. This was determined through SPSS box plots (on default setting). Mean substitution was used for missing values.

The principal axis factoring with the eigenvalue higher than one criterion for the choice of a factor number and the varimax normalised rotation was adapted. This was consistent with other confirmatory studies of the FCQ such as Eertmans et al. (2006). Four revised factors were extracted. With extraction of these factors 57.9 percent of the variance could be explained. Criterion for significant correlation was set as .3 prior to analysis. This is a moderate loading and pure variables are considered to have loadings of .3 or greater on one factor (Coakes & Steed, 2007). Analysis revealed eight variables with two or more moderate loadings. A higher criterion for significant correlation was therefore set as 0.45 (20% variance overlap between variable and factor). With use of this criterion only three variables were found to be complex. When complex variables were found the variable was adapted to the factor of highest loading, unless otherwise specified. On examination of factor loadings the below was decided for further analysis.

Revised MCQ factor one consisted of all six variables that were included in the “health” factor of the FCQ and all three variables included in the “natural content” factor of the FCQ. In addition, two researcher’s questions loaded onto factor one; “It is important to me that the meals I eat on a typical day”… “contain complex carbohydrates e.g. wholemeal ingredients” and “contain little/no sugar”. All eleven variables were given the collective revised factor title of “revised health and natural content”.

Revised factor two consisted of all six variables that were included in the "mood" factor of the FCQ and thus was titled under the "mood" factor for the current study.

Revised factor three consisted of all five variables that loaded on the FCQ "convenience" factor and consistently was titled "convenience" factor for the current study.

Revised factor four consistent of all three items that loaded onto the FCQ "weight control" factor. Consistent with the FCQ, this factor was titled "weight control".

The researcher's devised question "It is important to me that the meals I eat on a typical day"… "do not contain items I am sensitive or allergic to" did not load on any factor.

As hypothesised the mood, convenience and weight control factors of the MCQ in the sample of this study was found to be the same as the factors defined by Steptoe & Pollard, 1995 in relation to the FCQ. However, the health and natural content factors of the FCQ acted as a single combined factor in the sample of the current study.

A confirmatory factor analysis was also run on the SDCQ. Multiple variables were positively or negatively skewed, however, factor analysis is robust to the assumption of normality (Tabachnick & Fidell, 2007). Although differences in skewness among variables may suggest curvilinearity spot check examinations of randomly selected scatter plots showed no evidence of true curvilinearity. Furthermore, transformations are not considered favourable due to the goals of analysis (i.e., confirming pre-defined factors; Tabachnick & Fidell, 2007). Examination of box plots, on SPSS default settings, showed four outliers. All outliers were deleted prior to analysis. Mean substitution was used for missing values.

The principal axis factoring with the eigenvalue higher than one criterion for the choice of a factor number and the varimax normalised rotation was adapted. This was consistent with other confirmatory studies of the FCQ such as Eertmans et al. (2006). Four revised factors were extracted. With extraction of these factors 63.2 percent of the variance could be explained. Criterion for significant correlation was set as .3 prior to analysis. This is a moderate loading and pure variables are considered to have loadings of .3 or greater on one factor (Coakes & Steed, 2007) Analysis revealed six variables with two or more moderate loadings. A higher criterion for significant correlation was therefore set as 0.45 (20% variance overlap between variable and factor). With use of this criterion only two variables were found to be complex. When complex variables were found the variable was adapted to the factor of highest loading, unless otherwise specified. On examination of factor loadings the below was decided for further analysis.

Revised SCDQ factor one consisted of all six variables that were included in the "health" factor of the FCQ and all three variables included in the "natural content" factor of the FCQ. In addition, three researcher's questions loaded onto factor one; "It is important to me that the snacks and drinks I consume on a typical day"… "contain complex carbohydrates e.g. wholemeal ingredients", "contain little/no sugar" and "do not contain items I am sensitive/allergic to". Interestingly, all the same variables that loaded on to the MCQ "revised health and natural content" factor loaded onto the SDCQ factor one. This is with the exception of the variable "do not contain items that I am sensitive/allergic to" question was not included in any factors of the SDCQ. Thus the twelve variables were given the collective revised factor title of "revised health and natural content", consistent with the MCQ.

Revised factor two consisted of all six variables that were included in the "mood" factor of the FCQ and thus was titled under the "mood" factor for the current study. This was consistent with the MCQ.

Revised factor three consisted of all five variables that loaded on the FCQ "convenience" factor and was titled "convenience" factor for the current study. This was consistent with the MCQ.

Revised factor four consistent of all three items that loaded onto the FCQ "weight control" factor. Although the factor "It is important to me that the snacks and drinks I consume on a typical day"… "are low in calories" loaded slightly higher on revised health and natural content, it was chosen to be part of factor four as it made more intuitive sense. The loading difference was minimal (.585 versus .581). Consistent with the FCQ, this factor was titled "weight control". This factor was also consistent with the MCQ.

The researcher's devised questions "It is important to me that the snacks I eat on a typical day"… "contain a lot of sugar" and "contain caffeine" loaded separately on a fifth factor for the SDCQ. As only two variables loaded together, this was not considered a separate fifth factor. When used in further analyses these two variables were used with a combined total score and titled "alerting" variable.

As hypothesised the mood, convenience and weight control factors of the MCQ in the sample of this study was found to be the same as the factors defined in Steptoe and Pollard (1995) in relation to the FCQ. Contrary to the hypothesis the health and natural content factors of the FCQ acted as a single combined factor in the sample of the current study. Please refer to Table 3.3 for the Variable Loadings on Factors of the FCQ, MCQ and SDCQ and Table 3.4 for reliability coefficients of the factors.

Table 3.3

Variables Loadings on Factors of the Food Choice Questionnaire, Meal Choice Questionnaire and Snack and Drink Questionnaire

The letters in brackets symbols refer to the factors associated with the variable loadings; (H) Health, (RHN) Revised health and natural content, (N) Natural content, (M) Mood, (C) Convenience, (W) Weight control, (A) Alerting variable

Variable	Loadings FCQ	Loadings MCQ	Loadings SDCQ
It is important to me that…	(Steptoe & Pollard, 1995)		
the food I eat on a typical day… (FCQ)			
the meals I eat on a typical day… (MCQ)			
the snacks and drinks I consume on a typical day…(SDCQ)			
Contains a lot of vitamins and minerals	.77 H	.65 RHN	.62 RHN
Keeps me healthy	.75 H	.77 RHN	.71 RHN
Is/are nutritious	.75 H	.71 RHN	.74 RHN
Is/are high in protein	.72 H	.63 RHN	.73 RHN
Is/are good for my skin/teeth/hair/nails etc	.68 H	.57 RHN	.66 RHN
Is high in fiber and roughage	.66 H	.63 RHN	.70 RHN

Variable	Loadings FCQ	Loadings MCQ	Loadings SDCQ
It is important to me that…	(Steptoe & Pollard, 1995)		
the food I eat on a typical day… (FCQ)			
the meals I eat on a typical day… (MCQ)			
the snacks and drinks I consume on a typical day…(SDCQ)			
Contain/s no additives	.81 N	.74 RHN	.76 RHN
Contain/s natural ingredients	.72 N	.79 RHN	.85 RHN
Contain/s no artificial ingredients	.71 N	.75 RHN	.81 RHN
Contain complex carbohydrates e.g., wholemeal ingredients	-	.60 RHN	.72 RHN
Contain little/no sugar	-	.55 RHN	.61 RHN
Do not contain items that I am sensitive/allergic to	-	-	.55 RHN
Help/s me cope with my stress	.79 M	.70 M	.75 M
Help/s me cope with life	.79 M	.70 M	.75 M
Help/s me relax	.78 M	.75 M	.72 M

Variable	Loadings FCQ	Loadings MCQ	Loadings SDCQ
It is important to me that…	(Steptoe & Pollard, 1995)		
the food I eat on a typical day… (FCQ)			
the meals I eat on a typical day… (MCQ)			
the snacks and drinks I consume on a typical day…(SDCQ)			
Keep/s me awake/alert	.60 M	.55 M	.46 M
Cheer/s me up	.60 M	.71 M	.72 M
Make/s me feel good	.57 M	.68 M	.69 M
Is/are easy to prepare	.82 C	.84 C	.73 C
Can be cooked very simply	.81 C	.79 C	.77 C
Take/s no time to prepare	.76 C	.74 C	.74 C
Can be bought in shops close to where I live or work	.65 C	.47 C	.80 C
Is/are easily available in shops and supermarkets	.59 C	.49 C	.76 C
Is/are low in calories	.87 W	.75 W	.58 W

Variable	Loadings FCQ	Loadings MCQ	Loadings SDCQ
It is important to me that…	(Steptoe & Pollard, 1995)		
the food I eat on a typical day… (FCQ)			
the meals I eat on a typical day… (MCQ)			
the snacks and drinks I consume on a typical day…(SDCQ)			
Help/s me control my weight	.79 W	.75 W	.66 W
Is/are low in fat	.74 W	.70 W	.72 W
Contain a lot of sugar	-	-	.61 A
Contain coffee	-	-	.58 A

Table 3.4

Reliability of the Factors from the Food Choice Questionnaire (Cronbach's alpha) for Steptoe & Pollard's (1995) Sample and from the Meal Choice Questionnaire and the Snack and Drink Questionnaire for the Current Sample.

Factor	Food Choice Questionnaire (Steptoe & Pollard, 1995)	Meal Choice Questionnaire (Current Sample)	Snack and Drink Choice Questionnaire (Current Sample)
Mood	.83	.89	.91
Health	.87	-	-
Natural Content	.84	-	-
Revised Health and Natural Content	-	.92	.94
Convenience	.81	.84	.91
Weight Control	.79	.84	.91

Hypothesis 5 b: There will be a difference between individuals with narcolepsy and controls on the mood factors on both the MCQ and the SDCQ. It is expected that individuals with narcolepsy will score higher on the mood factors of the MCQ and SDCQ than controls. There will be no significant difference on the other four factors in either questionnaire.

Prior to analysis a normal distribution was found for the narcolepsy group on all MCQ factors. However, the distribution for the control group was only normal for the convenience factor. The distribution for the revised health and natural content factor and the weight control factor was found to be positively skewed and peaked for the control group. The distribution for the mood factor of the control group was found to be

positively skewed and flat. Results from a Mann-Whitney U test showed no significant difference between individuals with narcolepsy and controls on the MCQ mood factor, revised health and natural content, convenience factor or weight control factor. Please refer to Table 3.5 for descriptive results of the MCQ and Table 3.6 for significant testing between groups on the MCQ.

Table 3.5

Descriptive Results for Meal Choice Questionnaire Factors for Individuals with Narcolepsy and Controls

Factor	Descriptive	Narcolepsy	Control
Mood	Median	31.50	33
	Range	38	31
	Mean (Standard Deviation)	30.31 (7.32)	31.58 (8.39)
Revised Health and Natural Content	Median	13.50	11.50
	Range	18	18
	Mean (Standard Deviation)	15.11 (5.05)	12.62 (5.64)
Convenience	Median	13	13
	Range	15	15
	Mean (Standard Deviation)	13.27 (4.21)	12.94 (3.97)
Weight Control	Median	8	9
	Range	9	9
	Mean (Standard Deviation)	8.82 (2.49)	8.67 (2.52)

Table 3.6

Significant Testing (Man Whitney U Test) between the Narcolepsy and Control Groups on Factors of the Meal Choice Questionnaire

Factor	*U*	*Z*	*p*
Mood	1866	-1.415	.157
Revised Health and Natural Content	1823.5	-1.062	.288
Convenience	2195.5	-.498	.625
Weight Control	2127	-1.211	.226

Prior to analysis of the SDCQ the distribution of scores for both groups was found to be negatively skewed and flat on the convenience and weight factor. Distribution for the mood factor and alerting grouped variable was positively skewed and flat for the control group. The revised health and natural content factor was flat and negatively skewed for narcolepsy and negatively skewed for controls. Interestingly, a normal distribution was found for both groups on the revised health and natural content factor and for narcolepsy only on the mood factor. Results from a Mann-Whitney U test showed a significant difference between individuals with narcolepsy and controls on the SDCQ weight factor. Controls were more likely to rate the weight factor higher than individuals with narcolepsy. This indicates that controls were more concerned about the body weight implications of the snacks and drinks they were consuming than people with narcolepsy. Other results revealed no significant differences on the SDCQ revised health and natural content, convenience factor and mood factor between groups. Additionally, no significant difference between individuals with narcolepsy and controls was found on the alerting (combined) variable. Although there was not a significant difference between those with and without narcolepsy on the mood factor of the SDCQ

screening of the descriptive statistics shows a trend towards those with narcolepsy scoring higher on the mood factor. Please refer to Table 3.7 for descriptive results of the SDCQ and Table 3.8 for significant testing between groups on the SDCQ.

Table 3.7

Significant Testing (Man Whitney U Test) between the Narcolepsy and Control Groups on Factors of the Meal Choice Questionnaire

Factor	Descriptive	Narcolepsy	Control
Weight Control	Median	8	9
	Range	9	9
	Mean (Standard Deviation)	7.97 (2.69)	8.81 (3.05)
Mood	Median	15	12
	Range	18	18
	Mean (Standard Deviation)	14.77 (5.18)	13.05 (5.63)
Revised Health and Natural Content	Median	29.50	33
	Range	36	36
	Mean (Standard Deviation)	29.15 (9.93)	31.82 (10.66)
Convenience	Median	15	14
	Range	15	15
	Mean (Standard Deviation)	14.31 (4.79)	13.08 (4.57)
Alerting (combined variable)	Median	4	3
	Range	6	5
	Mean (Standard Deviation)	3.70 (1.78)	3.23 (1.38)

Table 3.8

Significant Testing (Mann Whitney U Test) between Individuals with Narcolepsy and Controls on Factors of the Snack and Drink Choice Questionnaire

Factor	*U*	*Z*	*p*
Weight Control	1801.5	-1.981	.048
Mood	1698.5	-1.932	.053
Revised Health and Natural Content	1788	-1.493	.136
Convenience	1859	-1.528	.126
Alerting (combined variables)	1812.5	-1.729	.084

3.2.6. Hypothesis 6: There will be a significant difference between individuals with narcolepsy and controls on the binge factor (measure of binge eating behaviours) taken from the Bulimia Test (BULIT). It is expected that individuals with narcolepsy will score higher on the BULIT.

An exploratory factor analysis was run on the BUILT. Multiple variables were positively or negatively skewed, however, factor analysis is robust to the assumption of normality (Tabachnick & Fidell, 2007). Although, differences in skewness among variables may suggest curvilinearity, spot check examinations of randomly selected scatter plots showed no evidence of true curvilinearity. Furthermore, transformations were not considered favourable due to the goals of analysis (i.e., confirming pre-defined factors; Tabachnick & Fidell, 2007). All outliers were deleted prior to analysis. Outliers were defined as scores appearing significantly inconsistent with the remainder of the data set. This was determined through SPSS box plots (on default setting). Mean substitution was used for missing values.

The principal axis factoring with the eigenvalue higher than one criterion for the choice of a factor number and the varimax normalised rotation was adopted. Criterion for significant correlation was set as .3 prior to analysis. This is a moderate loading and pure variables are considered to have loadings of .3 or greater on one factor (Coakes & Steed, 2007). Analysis revealed eight variables with two or more moderate loadings. A higher criterion for significant correlation was therefore set as 0.45 (20% variance overlap between variable and factor). With use of this criterion only one factor was found to be complex. When complex factors were found the variable was adapted to the factor of highest loading, unless otherwise specified. Refer to Table 3.9 for a list of the specific variables that made up the selected binge factor.

Prior to analysis the distribution of scores on the Binge Factor was found to be positively skewed and flat for the narcolepsy group and positively skewed and peaked for the control group. Results from Mann-Whitney U tests revealed that individuals with narcolepsy scored significantly higher on the total binge items than individuals without narcolepsy. To aid in interpretation further analyses were conducted on the individual variables that made up the Binge factor. Distribution among these variables were found to be positively or negatively skewed. All, with the exception of the variable, "have you ever kept eating until you thought you'd explode?", were found to be scored significantly higher in the narcolepsy group than the control group. Please refer to Table 3.9 for the descriptive results of the binge eating variables and Table 3.10 for significant testing between groups on binge eating.

Table 3.9

Descriptive Results for Binge Eating Variables for Individuals with Narcolepsy and Controls

Variable	Descriptive	Narcolepsy	Control
Binge Factor	Median	10	9
	Range	17	21
	Mean (Standard Deviation)	12.70 (4.78)	10.28 (2.92)
Do you ever eat uncontrollably to the point of stuffing yourself?	Median	1	1
	Range	4	4
	Mean (Standard Deviation)	1.46 (.89)	1.12 (.56)
Have you ever kept eating until you thought you'd explode?	Median	1	1
	Range	3	2
	Mean (Standard Deviation)	1.34 (.62)	1.14 (.39)
Would you presently call yourself a “binge eater”?	Median	1	1
	Range	4	3
	Mean (Standard Deviation)	1.39 (.89)	1.16 (.63)
Do you ever eat to the point of feeling sick?	Median	1	1
	Range	2	1
	Mean (Standard Deviation)	1.22 (.46)	1.07 (.26)
I am afraid to eat anything for fear that I won't be able to stop	Median	1	1
	Range	2	2
	Mean (Standard Deviation)	1.22 (.46)	1.04 (.27)
I would presently label myself a “compulsive eater	Median	1	1
	Range	3	2
	Mean (Standard Deviation)	1.28 (.67)	1.06 (.29)
Which of the following describes your feelings after binge eating……?	Median	1	1
	Range	4	3
	Mean (Standard Deviation)	1.63 (.98)	1.16 (.56)
Do you feel you have control over the amount of food you consume?	Median	1	1
	Range	3	2
	Mean (Standard Deviation)	1.51 (.79)	1.22 (.45)
I eat a lot of food when I'm not even hungry.	Median	1	1
	Range	3	2
	Mean (Standard Deviation)	1.64 (.83)	1.30 (.55)

Table 3.10

Significant Testing (Mann Whitney U Test) between the Narcolepsy and Control Groups on Variables Related to Binge Eating

Variable	*U*	Z	*p*
Binge Factor	1624.5	-3.177	.001
Do you ever eat uncontrollably to the point of stuffing yourself?	2116	-3.17	.002
Have you ever kept eating until you thought you'd explode?	2315	-1.876	.061
Would you presently call yourself a "binge eater"?	2261.5	-2.404	.016
Do you ever eat to the point of feeling sick?	2289	-2.282	.022
I am afraid to eat anything for fear that I won't be able to stop	2223	-3.029	.002
I would presently label myself a "compulsive eater	2162	-2.746	.006
Which of the following describes your feelings after binge eating……?	1971	-.3628	.000
Do you feel you have control over the amount of food you consume?	2038	-2.515	.012
I eat a lot of food when I'm not even hungry	1944	-2.488	.013

3.2.7. Hypothesis 7: There will be a significant difference between individuals with narcolepsy and controls on the meal and snack timing questionnaire (MSTQ). It is expected that individuals with narcolepsy will score higher on the MSTQ.

The MSTQ was a researcher's developed questionnaire, Cronbach's alpha for this sample was good (0.87). Prior to analysis the distribution of scores on the MSTQ were found to be negatively skewed and flat for the narcolepsy group and positively skewed and peaked for the control group. The results from a Mann-Whitney U test revealed that individuals with narcolepsy scored significantly higher on their overall rating on the MSTQ than individuals without narcolepsy. To aid in interpretation further analyses were run on the individual variables that made up the MSTQ. Distribution among these variables were found to be positively or negatively skewed. The results from a Mann-Whitney U test revealed that individuals with narcolepsy scored significantly higher on all individual items that constituted the MSTQ than individuals without narcolepsy. Please refer to Table 3.11 for descriptive of the MSTQ and Table 3.12 for significant testing between groups on the MSTQ.

Table 3.11

Descriptive Results for Meal and Snack Timing Variables of Individuals with Narcolepsy and Controls

Variable	Descriptive	Narcolepsy	Control
Total snack and meal timing score	Median	16	8
	Range	18	18
	Mean (Standard Deviation)	15.51 (4.95)	8.60 (3.41)
It is important to me that the timing of my meals/snacks on a typical day ….			
does not increase my sleepiness when this is inconvenient	Median	3	1
	Range	3	3
	Mean (Standard Deviation)	2.90 (1.22)	1.47 (.85)
increases my sleepiness when this is convenient	Median	2	1
	Range	3	3
	Mean (Standard Deviation)	1.91 (1.08)	1.21 (.56)
are at regular and predictable times	Median	3	2
	Range	3	3
	Mean (Standard Deviation)	2.72 (1.10)	2.11 (1.15)
does not interfere with a time to nap	Median	2	1
	Range	3	3
	Mean (Standard Deviation)	2.21 (1.76)	1.27 (.72)
helps avoids sleepiness in public places	Median	3	1
	Range	3	3
	Mean (Standard Deviation)	2.94 (1.28)	1.24 (.73)
helps prevent the sudden onset of sleep	Median	3	1
	Range	3	3
	Mean (Standard Deviation)	2.84 (1.20)	1.29 (.76)

Table 3.12

Significant Testing (Man Whitney U Test) between the Narcolepsy and Control Groups on Variables related to Snack and Meal Timing

Variable	*U*	*Z*	*p*
Total snack and meal timing score	653	-7.342	.000
It is important to me that the timing of my meals/snacks on a typical day ….			
does not increase my sleepiness when this is inconvenient	1005.5	-5.26	.000
increases my sleepiness when this is convenient	1545.5	-4.557	.000
are at regular and predictable times	1816	-2.977	.003
does not interfere with a time to nap	616.5	-8.316	.000
helps avoids sleepiness in public places	653	-7.342	.000
helps prevent the sudden onset of sleep	827.5	-7.420	.000

3.2.8. Hypothesis 8: There will be a significant difference between individuals with narcolepsy who report severe daytime sleepiness and individuals with narcolepsy reporting less severe daytime sleepiness (both considered as when on medication) on the binge factor (measure of binge eating behaviours derived from the BULIT), breakfast, lunch and dinner meal size and on the mood factor of the MCQ and SDCQ (choosing meals, snacks and drink to aid in altering mood). The direction of the differences will be consistent with the role of increased sleepiness in the above hypotheses testing these variables.

The section of this hypothesis eight referring to the independent variables of breakfast, lunch and dinner meal size was not analysed due to a high proportion of missing data.

Daytime sleepiness was measured using scores on the ESS, completed when on medication. Groups were split into high and low using the median ESS score of 12. Prior to analysis the distribution of the binge factor was found to be positively skewed and flat for the ESS low group and positively skewed and peaked for the ESS high group. Results from a Mann-Whitney U test revealed no significant difference between individuals with narcolepsy with ESS high and low scores on the binge factor (U = 438.5, Z = -1.255, p =.209). An independent groups t-test revealed no significant difference between individuals with narcolepsy with ESS high and low scores on the mood factor of the MCQ or on the mood factor of the SDCQ (t(62) = .229, p =.819; t (62) = .345, p =.731). Please refer to Table 3.13 for descriptive results.

Table 3.13

Descriptive Results for the Binge Factor and Mood Factor of the Meal Choice Questionnaire (MCQ) and Snack and Drink Choice Questionnaire (SDCQ) for Individuals with Narcolepsy who have High and Low Levels of Sleepiness as measured by the Epworth Sleepiness Scale.*

Factor	Descriptive	Low Sleepiness	High Sleepiness
Binge	Median	10	11.5
	Range	13	17
	Mean (Standard Deviation)	12.12 (4.06)	13.61 (5.64)
Mood (MCQ)	Median	14	16
	Range	18	18
	Mean (Standard Deviation)	14.62 (5.24)	15.08 (5.26)
Mood (SDCQ)	Median	14	16
	Range	18	18
	Mean (Standard Deviation)	14.62 (5.25)	15.08 (5.26)

* Johns, M. W. (1991). A new method for measuring daytime sleepiness: The Epworth Sleepiness Scale. *Sleep, 14*(6), 540 - 545.

3.2.9. Hypothesis 9: There will be a significant difference between individuals with narcolepsy and controls on their depression, anxiety and stress scores. It is expected that individuals with narcolepsy will score higher depression, anxiety and stress than controls.

Depression, anxiety and stress scores were measured by the DASS. Prior to analysis the distribution of depression and anxiety scores were found to be positively skewed and peaked for both the narcolepsy and control group. The distribution of stress scores was found to be positively skewed and flat for the narcolepsy group and positively skewed and flat for the control group. The results from a Mann-Whitney U test revealed that individuals with narcolepsy scored significantly higher on levels of depression, anxiety and stress then individuals without narcolepsy. Please refer to Table 3.14 for descriptive results and Table 3.15 for significance testing between groups on levels of depression, anxiety and stress. Although a significant difference between groups was shown, levels of depression, anxiety and stress based on means were within the "normal" range for both individuals with narcolepsy and individuals without narcolepsy (Lovibond & Lovibond, 1995b). Range of scores for both groups include "normal" to "severe" scores.

Table 3.14

Descriptive Results for Levels of Depression, Anxiety and Stress in Individuals With and Without Narcolepsy as Measured by the Depression Anxiety Stress Scale

Variable	Descriptive	Narcolepsy	Control
Depression	Median	5	1
	Range	40	27
	Mean (Standard Deviation)	8.55 (9.19)	3.15 (5.48)
Anxiety	Median	5	2
	Range	22	20
	Mean (Standard Deviation)	6.62 (5.45)	2.88 (4.01)
Stress	Median	7	4
	Range	35	37
	Mean (Standard Deviation)	10.19 (5.45)	6.54 (7.31)

Table 3.15

Significant Testing (Man Whitney U Test) between the Narcolepsy and Control Groups on Levels of Depression, Anxiety and Stress as measured by the Depression Anxiety and Stress Scale

Variable	*U*	*Z*	*p*
Depression	1381.5	-5.159	.000
Anxiety	1395.5	-5.093	.000
Stress	2083.5	-2.401	.016

3.2.10. Hypothesis 10: There will be a difference between individuals with narcolepsy who have normal-to-mild versus moderate-to-severe depression, anxiety and/or stress on the binge factor, breakfast, lunch and dinner meal size, on the mood factor of the MCQ, and on the mood factor of the SDCQ. As the effect of affective factors on eating can be variable across different individuals, the direction of these differences will be exploratory. It is expected that individuals with narcolepsy who have moderate–to–severe depression, anxiety and stress would score higher on all the listed variables than individuals with normal- to–mild depression, anxiety and stress levels.

The section of this hypothesis eight referring to the independent variables of breakfast, lunch and dinner meal size was not analysed due to a high proportion of missing data.

Levels of depression, anxiety and stress were measured using the DASS manual guidelines (Lovibond & Lovibond, 1995b). Normal to mild levels for depression included scores between zero and 13, for anxiety zero to nine and for stress zero to 18. Moderate to severe levels of depression included scores of 14 plus, for anxiety ten plus and for stress 19 plus.

Prior to analysis the distribution of binge factor scores of individuals with narcolepsy who scored normal to mild levels of depression was found to be positively skewed and peaked. The distribution of binge factor scores for individuals with narcolepsy who scored moderate to severe levels of depression was found to be positively skewed and flat. Results from a Mann-Whitney U test indicated no significant difference between individuals with narcolepsy and who scored normal to mild levels of depression versus moderate to severe levels of depression on the binge factor. Please refer to table 3.16 for descriptive and 3.17 for significant testing.

Prior to analysis the distribution of binge factor scores of individuals with narcolepsy who scored normal-to-mild levels of anxiety was found to be positively skewed and peaked. A normal distribution was found for individuals with narcolepsy who scored moderate to high levels of anxiety. Results from a Mann-Whitney U test indicated a significant difference between individuals with narcolepsy and who scored normal to mild levels of anxiety and moderate to severe levels of anxiety on the binge factor. Individuals with narcolepsy with moderate to severe levels of anxiety scored higher on the binge factor than individuals with narcolepsy with normal to mild levels of anxiety. Please refer to table 3.16 for descriptive and 3.17 for significant testing.

Prior to analysis the distribution binge factor scores of individuals with narcolepsy who scored normal to mild levels of stress was found to be positively skewed and peaked. A normal distribution was found for individuals with narcolepsy who scored moderate to high levels of stress. Results from a Mann-Whitney U test indicated a significant difference between individuals with narcolepsy and who scored normal to little levels of stress and moderate to severe levels of stress on the binge factor. Individuals with narcolepsy with moderate to severe levels of stress scored higher on the binge factor than individuals with narcolepsy with normal to mild levels of stress. Please refer to Table 3.16 for descriptive and 3.17 for significant testing.

Table 3.16

Descriptive Results of Binge Factor Scores in the Narcolepsy Sample Based on Levels of Depression, Anxiety and Stress in Individuals as Measured by the Depression Anxiety Stress Scale

	Median	Range	Mean (Standard Deviation)
normal to mild depression	10	17	12.29 (4.53)
moderate to severe depression	14	16	14.13 (5.37)
normal to mild anxiety	10	13	11.44 (3.48)
moderate to severe anxiety	17	17	16.41 (6.03)
normal to mild stress	10	17	11.44 (3.71)
moderate to severe stress	18	17	18.5 (4.8)

Table 3.17

Significance Testing (Man Whitney U Test) of Binge Scores between the Individuals with Narcolepsy Based on Levels (Normal to Mild versus Moderate to Severe) of Depression, Anxiety and/or Stress, as Measured by the Depression Anxiety Stress Scale and Binge Factor Scores

	U	*Z*	*p*
Depression	323.5	-1.03	.302
Anxiety	202.5	-3.31	.001
Stress	90	-4.052	.000

Distribution was found to be normal on normal to mild levels of depression/anxiety/stress and moderate to severe levels of depression/anxiety/stress on the mood factor. Results from a series of independent groups t-test revealed no significant difference between individuals with narcolepsy and who scored normal to mild levels of depression versus those scoring moderate to severe levels of depression on the mood factor of the MCQ or the mood factor of the SDCQ (t (62) = .908, *p* =.368; t (63) = .592, *p* = .556). There was no significant difference between individuals with narcolepsy and who scored normal to mild levels of anxiety versus moderate to severe levels of anxiety on the mood factor of the MCQ or the mood factor of the SDCQ (t (62) = 1.013, *p* =.315; t (63) = .993, *p* = .325). There was also no significant difference between individuals with narcolepsy and who scored normal to mild levels of stress versus moderate to severe levels of stress on the mood factor of the MCQ or the mood factor of the SDCQ (t (62) = .0832, *p* =.409; t (63) = .357, *p* = .722).

A post-hoc analysis was performed to investigate whether there was a correlation between the mood factors of the MCQ and SDCQ and depression, anxiety and stress scores (DASS raw scores) for individuals with and without narcolepsy.

Prior to analyses the mood factor of the MCQ and the SDCQ were found to have normal distribution for both groups. The distribution of depression scores and anxiety scores were found to be positively skewed and peaked for both groups. The distribution of stress scores were positively skewed and flat for individuals with narcolepsy and positively skewed and peaked for controls.

Results from Spearman's rank order correlations showed that for individuals with narcolepsy there were no significant correlations between the mood factor of the MCQ and anxiety or depression (*r* = .200, p =.113; *r* = .220, p =.081). A positive and weak

correlation was found between the mood factor of the MCQ and stress for individuals with narcolepsy (r = .275, p =.029).

Results from Spearman's rank order correlations showed that for individuals with narcolepsy there were no significant correlations between the mood factor of the SDCQ and anxiety, stress or depression for individuals with narcolepsy (r = .141, p =.263; r = .114, p =.366; r = .078, p =.538).

Results from Spearman's rank order correlations showed that for controls there were no significant correlations between the mood factor of the MCQ and anxiety (r = .188, p =.124). Positive and weak correlations were found between the mood factor of the MCQ and stress and depression for controls (r = .301, p =.013; r = .366, p =.002).

Results from Spearman's rank order correlations showed that for controls there was not a significant correlation between the mood factor of the SDCQ and anxiety (r = .205, p =.105). Positive and weak correlations were found between the mood factor of the SDCQ and stress and depression for controls (r = .296, p =.017; r = .391, p =.001).

A post-hoc analysis was performed to assess whether there was a significant difference between individuals with narcolepsy and controls that scored within the normal to mild range on depression, anxiety and stress (as measured by the DASS) on the binge factor.

Prior to analyses the distribution of the binge factor scores was found to be positively skewed and peaked for both group participants scoring in the normal to mild range on depression. Results from a Mann-Whitney U test revealed a significant difference between individuals with narcolepsy and controls scoring in the normal to mild range of depression on the binge factor (U = 1214, Z = -2.67, p = .008), with individuals with narcolepsy scoring higher on the binge factor.

Prior to analyses the distribution of the binge factor scores was found to be positively skewed and peaked for both group participants scoring in the normal to mild range on anxiety. Results from a Mann-Whitney U test revealed no significant differences were found between individuals with narcolepsy and controls scoring in the normal to mild range of anxiety on the binge factor (U = 1281, Z = -1.706, p = .088).

Prior to analyses the distribution of the binge factor scores was found to be positively skewed and peaked for both group participants scoring in the normal to mild range on stress. Results from a Mann-Whitney U test revealed no significant differences were found between individuals with narcolepsy and controls scoring in the normal to mild range of stress on the binge factor (U = 1449, Z = -1.928, p = .054).

3.3. Post-hoc Analyses Regarding Cataplexy Status

As discussed in the literature review (section 1.6) the current study did not set out to examine eating behaviours and attitudes in narcolepsy in the light of their presumed hypocretin status. However, given the strong association between cataplexy and a hypocretin deficiency in narcolepsy with cataplexy that has not been replicated in narcolepsy without cataplexy (as discussed in section 1.4) it was considered of interest, as post-hoc analyses, to investigate whether possible differences existed between those individuals with narcolepsy who did and did not have the symptom of cataplexy (n= 56 and 17 respectively) on a selected group of variables.

Only selected variables were analysed as a function of cataplexy status: BMI, depression, anxiety, stress, frequency of snacks and drinks and binge eating. These were chosen on the basis that the known action of hypocretin may be expected to influence these variables or previous research has shown a stronger presence of these variables in

individuals with cataplexy as opposed to individuals with narcolepsy without the symptom of cataplexy (see sections 1.3, 1.4 and 1.5 of chapter one).

A post-hoc power analysis was conducted using the analysis program, G*Power 3 (Faul, et al., 2007). This found that given our sample sizes in each group and an alpha of .05, analyses on the differences between individuals with narcolepsy with or without cataplexy only has sufficient power (0.8) to detect a large effect size (0.8). The analyses below do not have sufficient power to detect low (0.2) or medium (0.5) effect sizes, with power equalling 0.1 and 0.4 respectively.

3.3.1 Hypothesis 11: There will be a significant difference between individuals with narcolepsy with cataplexy and without cataplexy on body mass index (BMI) category scores. It is expected that individuals with narcolepsy with cataplexy will have a higher category than individuals with narcolepsy without cataplexy.

Prior to analysis the distribution was found to be flat and positively skewed for those with narcolepsy without cataplexy on BMI and positively skewed and peaked for those with narcolepsy with cataplexy. A Mann-Whitney U test revealed no significant difference between those with narcolepsy who suffer from cataplexy and those who do not suffer from cataplexy on BMI category (U = 362, Z = -1.182, p = .237). Please see Table 3.18 for descriptive results.

Table 3.18

Descriptive Results of Body Mass Index Category Scores in the Narcolepsy Sample both With and Without Cataplexy

	Median	Range	Mean (Standard Deviation)
With Cataplexy	3	2	3.04 (.84)
Without Cataplexy	3	2	2.76 (.75)

3.3.2 Hypothesis 12: There will be a significant difference between individuals with narcolepsy with cataplexy and without cataplexy on the numbers of snacks and drinks consumed. It is expected that individuals with narcolepsy without cataplexy will have a higher frequency of consumed snacks and drinks than individuals with narcolepsy with cataplexy.

Prior to analysis the distribution was found to be normal for those with narcolepsy without cataplexy for both number of snacks and number of drinks consumed. For those with narcolepsy with cataplexy distribution was found to be positively skewed and peaked for both number of snacks and number of drinks consumed. A Mann- Whitney U test revealed no significant difference was found between those with narcolepsy who suffer from cataplexy and those who do not suffer from cataplexy on number of snacks (U = 325.5, Z = -1.181, p = .237) and drinks (U = 412.5, Z = -.276, p = .783) consumed. Please see table 3.19 for descriptive results.

Table 3.19

Descriptive Results of Number of Snacks and Drinks Consumed in the Narcolepsy Sample both With and Without Cataplexy

		Snacks	Drinks
With Cataplexy	Median	2.37	4
	Range	10	12
	Mean (Standard Deviation)	2.37 (2.12)	4.78 (2.66)
Without Cataplexy	Median	3	5
	Range	3	9
	Mean (Standard Deviation)	2.53 (1.06)	4.31 (2.52)

3.3.3 Hypothesis 13: There will be a significant difference between individuals with narcolepsy with cataplexy and without cataplexy on their binge factor score. It is expected that individuals with narcolepsy with cataplexy will have a higher binge factor score than individuals with narcolepsy without cataplexy.

Prior to analysis the distribution was found to be positively skewed and flat for those without cataplexy and positively skewed and peaked for those with cataplexy on the binge factor. A Mann-Whitney U test revealed no significant difference between those with narcolepsy who suffer from cataplexy and those who do not suffer from cataplexy on their binge factor score (U = 339.5, Z = -784, p = .433). Please see table 3.20 for descriptive results.

Table 3.20

Descriptive Results of Binge Factor Scores in the Narcolepsy Sample both With and Without Cataplexy

	Median	Range	Mean (Standard Deviation)
With Cataplexy	10	17	12.40 (4.49)
Without Cataplexy	11	17	13.73 (5.60)

3.3.4 Hypothesis 14: There will be a significant difference between individuals with narcolepsy with cataplexy and without cataplexy on their depression, anxiety and stress scores. It is expected that individuals with narcolepsy with cataplexy will have a higher depression, anxiety and stress scores than individuals with narcolepsy without cataplexy.

Prior to analysis distribution was found to be normal for those with narcolepsy without cataplexy on scores of depression, anxiety and stress. Distribution was found to be

positively skewed and peaked for individuals with narcolepsy with cataplexy on scores of depression, anxiety and stress. A Mann- Whitney U test revealed no significant difference between those with narcolepsy who suffer from cataplexy and those who do not suffer from cataplexy on scores of depression (U = 340.5, Z = -1.775, p = .076), anxiety (U = 467.5, Z = -.111, p = .911) and stress (U = 385.5, Z = -1.185, p = .236). Please see Table 3.21 for descriptive Results.

Table 3.21

Descriptive Results of Depression, Anxiety and Stress Scores in the Narcolepsy Sample both With and Without Cataplexy

	Median	Range	Mean (Standard Deviation)
With Cataplexy			
Depression	4	40	7.77 (9.26)
Anxiety	5	22	8.05 (5.46)
Stress	6.5	35	9.45 (9.15)
Without Cataplexy			
Depression	9	31	11.12 (8.75)
Anxiety	6	18	6.71 (5.55)
Stress	14	32	12.65 (9.64)

3.4. Summary of Significant Findings

A summary of the significant findings is as follows;

- Individuals with narcolepsy consumed significantly more snacks and drinks per day than individuals without narcolepsy as reported in the One Day Food and Drink Diary.
- There was a significant difference between individuals with narcolepsy and controls on the weight control factor within the snack and drink choice

questionnaire. Controls were more likely to rate the weight factor higher than individuals with narcolepsy, meaning that they were more likely to rate the importance of choosing snack and drinks based on weight control properties.

- Individuals with narcolepsy scored significantly higher on the total binge items than individuals without narcolepsy on the binge factor extracted from the Bulimia Test questionnaire, meaning that they were more likely to report binge eating behaviours compared to controls.
- Individuals with narcolepsy scored significantly higher on their overall rating on the Meal and Snack Timing Questionnaire than controls. This means that individuals with narcolepsy were more likely to rate the importance of timing their meal and snack intake in accordance with convenience of sleepiness.
- Individuals with narcolepsy scored significantly higher on levels of depression, anxiety and stress on the Depression Anxiety and Stress Scale than individuals without narcolepsy, indicating higher levels of depression, anxiety and stress in individuals with narcolepsy.
- Individuals with narcolepsy that scored moderate to severe anxiety on the Depression Anxiety Stress Scale scored significantly higher on the binge factor extracted from the Bulimia Test, compared to individuals with narcolepsy that scored normal to low anxiety on the Depression Anxiety and Stress Scale. This means that individuals with narcolepsy that have moderate to severe anxiety are more likely to engage on binge eating behaviours compared to individuals with narcolepsy who have normal to mild anxiety.
- Individuals with narcolepsy that scored moderate to severe stress on the Depression Anxiety Stress Scale scored significantly higher on the binge factor extracted from the Bulimia Test, compared to, individuals with narcolepsy that

scored normal to low stress on the Depression Anxiety and Stress Scale. This means that individuals with narcolepsy that have moderate to severe stress are more likely to engage on binge eating behaviours compared to individuals with narcolepsy who have normal to mild stress.

- A positive and weak significant correlation was found for individuals with narcolepsy between the mood factor of the MCQ and stress. This means that as scores for stress increase, scores for the mood factor of the MCQ also increase for individuals with narcolepsy. A positive and weak significant correlation for controls was found between the mood factor of the MCQ and stress, between the mood factor of the MCQ and depression, between the mood factor of the SDCQ and stress and between the mood factor of the SDCQ and depression. This means that as scores for depression and or stress increase scores for the mood factor of the MCQ and SDCQ tend to also increase for controls.
- Results from a Mann-Whitney U test revealed a significant difference between individuals with narcolepsy and controls scoring in the normal to mild range of depression on the binge factor ($U = 1214$, $Z = -2.67$, $p = .008$) . This means that individuals with narcolepsy and normal to mild levels of depression are more likely to binge eat than controls with normal to mild levels of depression.

Chapter 4: Discussion

The discussion of hypotheses and findings of the current study are organised into five key sections within section 4.1 for clarity, that is; 1. body mass index, 2. consumption and timing of meals, snacks and drinks, 3. binge eating, 4. mood and 5. sleepiness. Limitations and directions for future research and conclusions are discussed in parts 4.2 and 4.3 respectively.

4.1 Discussion of Hypotheses and Findings

4.1.1 Body Mass Index (BMI)

Analyses revealed no significant difference between the BMIs of those with and without narcolepsy. At first glance this seems contradictory to previous research, such as that of Kotagal et al. (2004), which found individuals with narcolepsy to have significantly higher body mass indexes and to be more likely to be overweight/obese in comparison to the general population (without narcolepsy). However, further thought/investigation shows this finding to be more complicated.

Kok, et al. (2003) found 33 percent of the population with narcolepsy to be obese. This statistic is remarkably similar to the current sample of individuals with narcolepsy where 30 percent were found to be obese. Kok, et al. (2003) found 12.5 percent of their control sample to be obese compared to the current sample which found 17.6 percent of the control group to be obese. It is possible that the control population of the current study may have higher BMIs than controls in previous research (as in Kok, et al., 2003). This would influence the finding of a non significant relationship between the individuals with narcolepsy and the control sample. A possible explanation for the current study having a control group with higher BMI's is that they were drawn from an

Australian population. Australia has been found to be ranked the fifth highest country, under the Organisation for Economic Co-operation and Development, for obesity ("Overweight and Obesity in Adults in Australia: A Snapshot," 2011). There is a current trend towards a growing culture of increased BMI's in the general Australian population. For example, the prevalence of obesity in Australia has more than doubled in the twenty years preceding the year 2000 (Cameron, et al., 2003). Additionally, the control group of the current study was recruited by the individuals with narcolepsy through their asking of family or friends to participate. This may have influenced the results through limiting variability of biological and social aspects of body mass index between individuals with and without narcolepsy.

Another possible explanation for the current study not having finding a difference between individuals with narcolepsy and without narcolepsy on BMI category may be due the set exclusion criteria for individuals with diabetes mellitus (type one or two). Higher rates of diabetes mellitus have been found within individuals with narcolepsy in comparison to individuals without narcolepsy (Honda, et al., 1986). Weight gain is considered both a symptom of diabetes mellitus and a risk factor for diabetes mellitus (Diabetes Australia, 2011). Therefore, it may be that by excluding individuals with narcolepsy the current study also excluded a comorbid subgroup of individuals with narcolepsy, diabetes mellitus and overweight/obesity.

The current study found no significant difference between individuals with narcolepsy with and without cataplexy on BMI category scores (Hypothesis 11). This is in contrary to the hypothesis and previous research, such as that of Sonka et al. (2010), which found higher BMIs in individuals with narcolepsy with cataplexy compared to those with narcolepsy and no cataplexy. Findings of the current study also seem counter intuitive given the connecting roles of hypocretin and leptin in controlling body weight (Sakurai,

2006) and their found deficiencies in narcolepsy, particularly in narcolepsy with cataplexy (Kok, et al., 2002; Schuld, Hebebrand, et al., 2000). It may be possible that a low or medium effect size difference was present in the current study but unable to be detected. If this was the case, it is possible that the inclusion of participants with narcolepsy and no cataplexy may have reduced the difference between groups (individuals with narcolepsy and individuals without narcolepsy) examined in the current study in relation to BMI differences. The finding of no BMI difference between individuals with and without narcolepsy in the current study should be taken into consideration when reflecting on other eating behaviours and attitudes explored in the current study.

4.1.2 Consumption and Timing of Meals, Snacks and Drinks

Three main hypotheses were tested in relation to the consumption and timing of meals, snacks and drinks. It was found that individuals with narcolepsy consumed significantly more snacks and drinks per day in comparison to those without narcolepsy (Hypothesis 1). There was no significant difference between individuals with narcolepsy and without narcolepsy on factors measuring mood, health and natural content, convenience or weight control on measurement of meal choice (Hypothesis 5). Additionally it was found that no significant relationship existed between individuals with narcolepsy and without narcolepsy on factors measuring mood, health and natural content or convenience on measurement of snack and drink choice (Hypothesis 5). However, a significant difference was found between individuals with narcolepsy and without narcolepsy on the weight control factor on measurement of snack and drink choice (Hypothesis 5). Individuals without narcolepsy were more likely to rate the weight control factors higher than individuals with narcolepsy on measurement of snack and drink choice. Three further hypotheses in relation to the consumption and timing of

meals were not analysed due to a high proportion of missing data (Hypotheses 2, 3 and 4), see section 3.2 for details and please refer to section 4.2 for an explanation of the limitations pertaining to these hypotheses. This section of the discussion will further explore the above listed findings in relation to consumption and timing of meals, snacks and drinks.

As predicted, the current study found individuals with narcolepsy consumed significantly more snacks and drinks per day than individuals without narcolepsy. This is consistent with limited early research into this area (Bell, 1976; Bruck, et al., 1989) that found individuals with narcolepsy consumed a greater number of snack and drinks than the general population. It is also consistent with research such as that of Pollak and Green (1990) suggesting that individuals with narcolepsy eat more frequently than those without narcolepsy. Interestingly however, increased snacking is the opposite as to what we would expect given the medication status of our sample. Eighty one percent of individuals with narcolepsy were on stimulant medication. As discussed in chapter one stimulant medication can reduce hunger (Makrisa, 2004; Prescrire International," 2001). The finding that individuals with narcolepsy consumed significantly more snacks and drinks per day than individuals without narcolepsy also contrasts with the predicted direction of possible hypocretin deficiency induced behaviour of reducing food/drink seeking and feeding/drinking behaviour (Sakurai, 2006; Willie, et al., 2001). Interestingly, the current study found no significant difference between individuals with narcolepsy with and without cataplexy on number of snacks or drinks per day (Hypothesis 12). Given the strong association of narcolepsy with cataplexy and a hypocretin deficiency which is not found in narcolepsy without cataplexy (Thannickal, et al., 2009) these findings are also in contrast to predications based on a hypocretin deficiency. This alludes to the possibility of other controlling factors being in play.

However, it is possible that a low to medium effect size difference was present but unable to be detected by the current study.

Specifics in relation to the types of snacks and drinks chosen were not investigated in the current study (with the exception of water being excluded). Other research has found that individuals tend to crave sweets, soft drink and cordial (Bruck, et al., 1989). Thus they may consume snacks and drinks of high sugar content. Symptoms of diabetes include an increase in thirst and feelings of hunger (Diabetes Australia, 2011). Effort was made to exclude individuals with diagnosed diabetes mellitus from the current study. However, given the demonstrated high diabetes rate within the narcolepsy population (Honda, et al., 1986) it may be possible that the current study includes a significant proportion of individuals with narcolepsy who have undiagnosed diabetes mellitus or have only an early stage of the disorder. Additionally insufficient sleep has been associated with increased rates of insulin resistance. Insufficient sleep impacts on insulin resistance (and related hormone levels) leading to an increase in appetite (Pack & Pien, 2011). Given the sleepiness status of individuals with narcolepsy, even when medicated (Bruck, et al., 2005) it may be possible that this phenomena was present in the current sample of individuals with narcolepsy. The intertwining of these various factors may have contributed to the results of increased snacking and drinking found within the individuals with narcolepsy sample of the current study.

Two points of interest into the findings of individuals with narcolepsy consuming significantly more snacks and drinks than individuals without narcolepsy are that; 1) such patterns may be related to choosing snacks or drinks to help with mood (i.e., decrease depression, anxiety and/or stress) and/or 2) patterns may be related to timing of snacks and drinks with convenience or inconvenience of sleepiness.

The current study measured the importance of the role of meals and snacks and drinks that are consumed by those with narcolepsy in comparison to those without narcolepsy.

As hypothesised the factors within the meal choice questionnaire and the snack and drink choice questionnaire (adapted from the food choice questionnaire of (Steptoe & Pollard, 1995) were broadly consistent with the original questionnaire.

The current study found no significant difference between individuals with narcolepsy and without narcolepsy on the mood, health and natural content and convenience factors relating to meals or snacks and drinks. This suggests that the importance of consuming meals and/or snacks and drinks that alleviate negative mood, maintain health and natural content and are convenient to obtain/cook were not different between individuals with narcolepsy and individuals without narcolepsy. Further, no significant difference between individuals with narcolepsy and without narcolepsy on the alerting variable relating to snacks and drinks was found. This suggests that the importance of consuming meals and/or snacks and drinks that increase alertness was not different between groups.

Of particular interest are the mixed findings of relationships when it came to the importance of mood, depression, anxiety and stress when choosing meals, snacks and/or drinks. Positive weak relationships were found for stress and mood based meal choice for individuals with narcolepsy. Additionally, positive weak relationships were found for individuals without narcolepsy between stress and depression and mood based meal and snack and drink choice (Hypothesis 10, post hoc analysis). Such relationships are broadly consistent with previous research (see below). However, inconsistent with suggestions in previous research are the various comparisons of these variables for both groups where no relationships were found and furthermore, that no significant

difference between individuals with and without narcolepsy between mood and choice of food and/or drink consumption was found.

Previous research has found a positive and moderate correlation between the number of daily stressors and snack consumption in healthy individuals within the general population (Conner, et al., 1999). Additionally under stress individuals are more likely to choose snack foods which are highly palatable, energy dense and easy to prepare (e.g., sweets and chocolate, Oliver & Wardle, 1999). It is possible that the current mixed findings are a result of one, or both, of the following issues. Firstly, the current study found that individuals with narcolepsy had higher levels of depression, anxiety and stress than those without narcolepsy, as discussed further in section 4.1.4 below. However, as the mean of scores for both individuals with and without narcolepsy remained in the normal range a floor effect may be operating. This may have led to the non-significant findings in relation to mood and snack and/or drink consumption and with regard to depression, anxiety and stress and mood based food and drink consumption. The second possibility is that consumption is not based on a conscious choice of importance (as measured in the current study through self-report) but may be less purposeful in nature, such as being emotionally driven or impulsive. Indeed an interplay of purposeful choice and less purposeful mood reaction may be an explanation for the mixed relationship findings for those with and without narcolepsy on depression, anxiety and stress and mood based meal, snack and drink consumption. Section 4.1.4 will discuss mood in relation to food and drink in detail.

The current study did not show a significant difference between the self-report of individuals with and without narcolepsy on the weight control factor in relation to consumption of meals. Interestingly, however, a significant difference between individuals with narcolepsy and without narcolepsy on the weight factor in relation to

the consumption of snacks and drinks (not meals) was found. Individuals without narcolepsy rated the importance of choosing snacks and drinks that aided in weight control higher than individuals with narcolepsy. Thus the former group were more concerned about the body weight implications of the snacks and drinks they were consuming than people with narcolepsy. Bruck et al., (1989) found that individuals with narcolepsy were more likely to crave sweets and sugar filled drinks such as cordial and soft drink. The finding of weight control being rated lower in importance for those individuals with narcolepsy implies that such individuals may be more likely to give in to such cravings and choose sweet snacks and drinks than those without narcolepsy. A possible explanation for this finding is the high levels of psychosocial stressors faced by those with narcolepsy (Bruck, 2001). It may be possible that given their ongoing stressors and struggle with sleepiness, weight control simply does not present itself as a priority.

Wells and Cruess (2006) found that sleep deprived (four hours or less of sleep) university students had a change in food choice. After sleep deprivation they were less likely to choose food based on weight control properties, compared to when no sleep deprivation occurred. Given that the controls in the current sample were screened for the absence of excessive sleepiness levels, it can be assumed, that they were not sleep deprived. In comparison, however, excessive daytime sleepiness is still prevalent in a medicated group of individuals with narcolepsy (Bruck, et al., 2005) and thus assumed within the current study sample of individuals with narcolepsy. The symptom of excessive daytime sleepiness found within narcolepsy may act in a similar manner to the sleep deprivation in the Well and Cruess (2006) study. This would provide the explanation that individuals with narcolepsy would be less concerned with choosing food (meals, snacks and possibly drinks) based on weight control properties due to high

levels of daytime sleepiness, a phenomenon consistent with individuals without narcolepsy.

In accordance with hypothesis seven, a significant difference was found between individuals with narcolepsy and individuals without narcolepsy on the meal and snack timing questionnaire (MSTQ). Individuals with narcolepsy rated the importance of timing of meals and/or snacks on a typical day in relation to sleepiness higher than those without narcolepsy. Specifically, individuals with narcolepsy rated the importance of timing meals and snacks on a typical day so that they do not increase sleepiness when inconvenient, increase sleepiness when convenient, do not interfere with an nap, are at regular and predictable times, help to avoid sleepiness in public places and prevent sudden onset of sleep. This is consistent with the qualitative reports such as that found by Chabas, et al. (2007) of individuals with narcolepsy using food as a method to manage daytime sleepiness.

A possible limitation to the findings of a significant difference between those with and without narcolepsy on the MSTQ is that participants without narcolepsy were excluded if they had excessive levels of sleepiness. It could be assumed that lower scores on the MSTQ for individuals without narcolepsy may be an outcome of having low or no sleepiness, and therefore no need to control sleepiness through behaviours such as timing meals and snacks. This may have allowed a floor effect to be present in the current results. However, a significant finding still suggests that individuals with narcolepsy engage in the particular behaviour of timing meals, snacks and sleepiness suggesting an important connection between sleepiness and food intake for individuals with narcolepsy. Future studies may benefit from investigating/comparing narcolepsy on the MSTQ with other sleep disorders or disorders that present with high levels of sleepiness, for example untreated sleep apnoea or chronic fatigue syndrome.

The above findings of the current study show that individuals with narcolepsy place importance on engaging in a purposeful pattern of feeding behaviour with regard to timing. As highlighted in chapter one, qualitative research such as that of Bruck and Broughton (2001) have long made reference to the connection between symptoms of daytime sleepiness and food in narcolepsy. The current study shows that individuals with narcolepsy are purposefully using the timing of their meals and snacks as a mechanism of controlling the levels of sleepiness that they experience. Individuals with narcolepsy may be delaying or overindulging in eating at specific times of the day or week as convenience or inconvenience with being sleepy allows. In this way they use the timing of their food intake to decrease or increase (presumably to power nap) their sleepiness. For example, as the opening quote to this thesis shows, individuals with narcolepsy may limit eating during the working day (due to inconvenience of sleepiness) and delay food gratification until when home in the evening when sleepiness is considered convenient. Findings of the current study reinforce that for individuals with narcolepsy there is an important association between food intake and sleep. Such behaviour is consistent with research which suggests a link between the impact of food on sleepiness. In particular it supports Pollak and Green's (1990) suggestion that meals and sleeping patterns are timed by a common physiological mechanism (i.e., second circadian oscillator or homeostatic processes), as opposed to meal content producing sleepiness. Consistent with this Pollak and Green (1990) found meals are likely to be followed by a nap despite size or nutrient content within individuals with narcolepsy. A common physiological mechanism would explain not only the strong tendency to nap after a meal, as in the Pollak and Green (1990) study, but the purposeful use of meals and snacks in accordance with convenience of periods of sleepiness and wakefulness. Wakefulness would be maintained with the predisposition to eat, and after the onset of

eating both the predisposition of wakefulness and to eat would decrease, offering an increase in sleepiness and need for a nap, at least for individuals with narcolepsy. Additionally, the current study's findings that alertness variables did not impact on choice of snack/drink adds further weight to these connections. It may be that individuals with narcolepsy have noted a connection between timing of meals/snacks and sleepiness, and thus weigh the importance of the same and use this connection to their advantage. The choice of meals/snacks types (i.e., content) may not have been as relevant as the timing of the meal and snack.

In summary, individuals with narcolepsy were more likely to consume greater amounts of snacks and drinks compared to individuals without narcolepsy. Individuals with narcolepsy were less concerned about the choice of snacks and drinks that aided weight control. Interestingly, choice of meals or snacks and drinks that aided in coping with negative mood was not found and relationships between mood and depression, anxiety and stress were inconsistent. Findings suggest that individuals with narcolepsy use the timing of their food intake to decrease or increase (presumably to power nap) their sleepiness as convenient. A connection between food and drink intake, timing of food intake and sleepiness was shown within the individuals with narcolepsy sample. This suggests that the management of a sleep-wake schedule may influence the eating behaviours in individuals with narcolepsy and vice versa.

4.1.3 Binge Eating

The current study found a significant difference between individuals with narcolepsy and without narcolepsy on the binge factor (Hypothesis 6). This indicated that individuals with narcolepsy were more likely to self report engaging in binge eating behaviours. Binge eating in the current study was defined by questions referring to

uncontrolled eating, compulsive eating, over eating and eating when not hungry. The finding of a tendency for individuals with narcolepsy to engage in binge eating behaviours is consistent with the previous research of Kotagal, et al. (2004) and Fortuyn, et al. (2008).

As discussed in chapter one, a high comorbidity rate of binge eating has been found in obese individuals with diabetes mellitus type two (Crow, et al., 2001). However, the current study excluded participants with diabetes mellitus and found no significant difference between those with and without narcolepsy on BMI in the sample used. It is possible there is a more complicated, specific and narcolepsy-related function that is operating in the production of binge eating behaviour in this group. The current study therefore supports the suggestion made by Fortuyn (2008) that disordered eating in individuals with narcolepsy is a key aspect of the narcolepsy phenotype and not a mere consequence of obesity. Additionally, the current study suggests that disordered eating is not merely a consequence of diabetes mellitus disorders either. The current study also found no significant difference between individuals with narcolepsy with cataplexy and without cataplexy on binge eating scores (Hypothesis 13) suggesting factors outside of a hypocretin deficiency or cataplexy symtomatology being involved in the phenomenon of binge eating behaviour within individuals with narcolepsy. However, it is possible that a low or medium effect size difference was present but unable to be detected by the current study. Given the basic necessity of eating for survival, it is not surprising that a number of different physiological and behavioural mechanisms would be involved, of which hypocretin is just one. However, the question remains as to why the eating behaviour in individuals with narcolepsy would be experienced as uncontrolled, compulsive and overindulgent, consistent with binge eating behaviour.

Taken together, the findings of (i) increased binge eating, snack consumption and drink consumption and (ii) timing meals and snacks according to sleep related convenience has shown two defined eating behaviours that are categorised in an opposite framework. One is a purposeful eating behaviour (related to timing) and the other can be considered an uncontrolled eating behaviour (binge eating). The hour time of binge eating episodes was, unfortunately, not measured and therefore the current study cannot rule out the presence of a nocturnal eating disorder. Nocturnal eating disorders have been found prevalent in individuals with narcolepsy with cataplexy (Palaia, et al., 2011). However, the pattern of timing meals and snacks according to the convenience of sleepiness implies that individuals with narcolepsy were engaging in a purposeful behaviour (separate to uncontrolled binges) to control their symptom of daytime sleepiness. Binge eating (whether nocturnal or not) may be an uncontrolled compensatory factor as a consequence of purposeful timing of food and sleepiness. This is consistent with the qualitative finding by Fortuyn, et al. (2008) that when there was a build up of pressure from sleepiness during the day, the ability to resist binge eating would weaken. There may also be a bilateral or separate factor to the timing of food and sleepiness that is complicated by emotionally driven attributes (i.e., depression, anxiety, stress) and/or daytime sleepiness experienced by the individuals with narcolepsy. The role of mood and daytime sleepiness in eating behaviours and attitudes in individuals with narcolepsy is further discussed in detail in sections 4.1.4 and 4.1.5 respectively.

4.1.4 Mood

The finding of higher rates of depression symtomatology/diagnostic depression in the individuals with narcolepsy sample compared to individuals without narcolepsy sample in the current study (Hypothesis 9) replicates past research, such as, that of Lindsley and Crawford (1996). In the current study individuals with narcolepsy scores on depression

ranged from normal to severe, however, investigation showed the individuals with narcolepsy group had a mean score within the normal (non-clinical) range. Results from this study may possibly present an underestimation of depressive symtomatology within the narcolepsy population, as individuals with high ratings on depression would be less motivated to complete the somewhat lengthy questionnaire provided to them. Additionally, the current study included individuals with narcolepsy that did not have the symptom of cataplexy (23 percent). Recent research has shown that individuals with narcolepsy that have the symptom of cataplexy are more likely to have higher severity of depression than individuals with narcolepsy and no cataplexy symtomatology. Low levels of hypocretin (typical of narcolepsy with cataplexy) have been hypothesised to be associated with severity of depressive symptoms (Dimitrova, et al., 2011). However, other research has shown the presence of depression independent of cataplexy severity (Lindsley & Crawford, 1996). Interestingly, the current study however found no significant difference between individuals with narcolepsy with and without cataplexy on depression (Hypothesis 14). However, as already mentioned, the current study was unable to detect medium or low effect size differences between individuals with narcolepsy with cataplexy and without cataplexy. It may be possible that a low or medium effect size difference was present but unable to be detected. Despite this, the current study suggests that individuals with narcolepsy (with or without cataplexy) experience higher levels of depression in comparison to individuals without narcolepsy.

No significant difference between those individuals with narcolepsy who scored normal to mild levels of depression versus moderate to severe levels of depression on the binge eating factor was found (Hypothesis 8). This suggests that the presence and/or severity level of depression in individuals with narcolepsy does not impact on the eating behaviour of binging observed. Thus, depression is not a risk factor for binge eating in

individuals with narcolepsy. This finding is in contrast to the original hypothesis of the current study and suggestions by other researchers including Fortuyn, et al. (2008). However, the finding in the current study of binge eating being independent of severity of depression in individuals with narcolepsy is consistent with findings of Chabas, et al. (2007) which found features of bulimia nervosa (of which binge eating is one) to be independent of depressed mood. At the time of writing no published studies had quantitatively investigated the relationship between depression and binge eating (independently, rather than as a feature of bulimia nervosa) in individuals with narcolepsy. Chabas, et al. (2007) suggested that disordered eating in individuals with narcolepsy may be strategy for managing daytime sleepiness. However given the "uncontrolled" aspects in the definition of binge eating the relationship between sleepiness and binge eating may be better explained as a consequence of delaying or minimising food intake at times that are inconvenient. Due to this delay or minimisation of food intake a compensatory mechanism is activated at times when convenient (and the individual with narcolepsy engages in eating). Stress and anxiety, rather than depression, may place one at greater risk of engaging in this compensatory behaviour.

A significant difference in binge eating behaviours was found between individuals with and without narcolepsy who scored in the normal to mild range of depression (Hypothesis 10, post-hoc analysis). This suggests that individuals with narcolepsy may engage in binge eating behaviours even when depression is normal or mild, highlighting that factors other than depression may be impacting on binge eating. Alternatively, it is possible that the presence of even mild depression may impact on binge eating behaviours in narcolepsy and this presence, rather than severity, is of importance. However, the design of the current study cannot make any sound conclusions on this. It would be of interest for further research to investigate such issues. It would also be

beneficial for future research to isolate depression, anxiety and stress to rule out the impact of possible co-morbidities on findings. No significant differences in binge eating behaviours was found between individuals with and without narcolepsy who scored in the normal to mild range of anxiety and stress (Hypothesis 10, post-hoc analysis) this was consistent with other findings of the current study, as discussed below, in emphasising the role of severity of anxiety and stress symptoms in individuals with narcolepsy engaging in binge eating behaviours.

The finding of higher rates of anxiety symtomatology/diagnostic anxiety in the narcolepsy sample compared to the control sample (Hypothesis 9) in the current study replicates past research such as that by Fortuyn, et al. (2010). In the current study individuals with narcolepsy scores on anxiety ranged from normal to severe, however, investigation showed individuals with narcolepsy to have a mean score with the normal (non-clinical) range. The majority of previous research investigating the connection between anxiety and narcolepsy has not included participants with narcolepsy without the symptom of cataplexy (Fortuyn, et al., 2010; Lindsley & Crawford, 1996). The current study found no significant difference between individuals with narcolepsy with and without cataplexy on anxiety (Hypothesis 14). It may be possible that a low or medium effect size difference was present but unable to be detected. Regardless, the current study shows an increase in anxiety symtomatology in the individuals with narcolepsy (with or without the symptom of cataplexy).

Consistent with hypothesis ten, the current study found a significant difference between individuals with narcolepsy who reported normal to mild levels of anxiety versus moderate to severe levels of anxiety on the binge factor. Individuals with narcolepsy with moderate to severe levels of anxiety scored higher on the binge factor than individuals with narcolepsy with normal to mild levels of anxiety. Thus for individuals

with narcolepsy as anxiety increases so too does binge eating behaviour and vice versa. At the time of writing no published research, known to the author, had investigated quantitatively the association between anxiety and binge eating in individuals with narcolepsy. The current study shows that anxiety may be a risk factor for binge eating behaviours in the narcolepsy population.

The finding of higher rates of stress related symtomatology in the individuals with narcolepsy sample compared to individuals without narcolepsy sample in the current study (Hypothesis 9) replicates past research such as that by Broughton and Broughton (1994) showing significantly higher psychosocial stressors experienced by individuals with narcolepsy. In the current study individuals with narcolepsy scores on stress ranged from normal to severe, however, investigation showed individuals with narcolepsy to have a mean score within the normal (non-clinical) range. Individuals with narcolepsy and clinically high stress levels may be less motivated to complete the somewhat lengthy questionnaire provided to them and as a consequence the current study may have underestimated the severity of stress experienced by individuals with narcolepsy. Additionally the current study has an older population sample of individuals with narcolepsy with a mean age of 58 years. Research has shown that over time some areas of psychosocial stress tend to decrease for individuals with narcolepsy. For example Costa (2001) found a significant decline in stress related to health care, sexual relationships and extended family relationships over a ten year period. The older participant sample in the current study may reflect the higher stress levels in a younger potential narcolepsy sample.

The current study found no significant difference between individuals with narcolepsy with and without cataplexy on stress scores (Hypothesis 14). This is in contrast to what would be expected given the predominant role of hypocretin in the regulation of

response to stress (Berridge, et al., 2010) and the presumed high rates of a hypocretin deficiency in the current study. This suggests other mediating roles of induced stress may be in action. Additionally, a low to medium effect size difference may be present but unable to be detected by the current study.

Consistent with hypothesis ten the current study found a significant difference between individuals with narcolepsy who scored normal to mild levels of stress versus moderate to severe levels of stress on the binge factor. Individuals with narcolepsy with moderate to severe levels of stress scored higher on the binge factor than individuals with narcolepsy with normal to mild levels of stress. These results suggest that for individuals with narcolepsy as stress increases so too does binge eating behaviour and/or vice versa. At the time of writing no published research, known to the author, had investigated quantitatively the association between stress and binge eating in individuals with narcolepsy. The current study shows that stress may be a risk factor to binge eating behaviours in individuals with narcolepsy.

In a circular notion, vulnerability to stress and anxiety may be enhanced by the lack of control that an individual with narcolepsy has over their symtomatology such as sleep-wake cycles, cataplexy and binge eating (Fortuyn, et al., 2010; Fortuyn, et al., 2011). These uncontrolled behaviours could increase their levels of stress, anxiety and vice versa. Interestingly, a cognitive demand for control is often represented as a cognitive distortion in a clinical anxiety population (O'Kelly, 2010). It may be that individuals with narcolepsy use the timing of their meal and snack intake as a method to gain control. This behaviour may be more likely to occur in individuals with narcolepsy with heightened anxiety about their narcolepsy symtomatology.

Unexpectedly no significant differences were found between individuals with narcolepsy who had normal-to-mild versus moderate-to-severe depression, anxiety and/or stress on the mood factor of the meal choice questionnaire (MCQ) or the mood factor of the snack and drink choice questionnaire (SDCQ) (Hypothesis 10). This adds to the findings of the current study of no significant difference between individuals with and without narcolepsy on the importance of choosing meals based on mood control properties discussed in section 4.1.2. These results suggests that individuals with narcolepsy are not more likely than individuals without narcolepsy to make choices regarding consumption of meal, snacks and drinks based on mood, particularly the experience of depression, anxiety and/or stress. However, interestingly given the relationship between binge eating and anxiety and stress in individuals with narcolepsy, the uncontrolled behaviour of binge eating may be emotionally driven.

In summary the current study found that individuals with narcolepsy are more likely to have more symptoms of depression, anxiety and stress in comparison to individuals without narcolepsy. Anxiety and stress may act as a risk factor to binge eating within individuals with narcolepsy and the greater the anxiety and/stress severity levels the greater the risk of engaging in binge eating behaviour. Interestingly, depression is no more likely a risk factor for binge eating in individuals with narcolepsy than in individuals without narcolepsy. Meal, snack and drink choice is no more likely based on mood in individuals with narcolepsy than in individuals without narcolepsy.

4.1.5. Sleepiness

The current study found no significant difference between individuals with narcolepsy with high versus low Epworth Sleepiness Scale scores (on medication) on the binge factor (Hypothesis 8). As discussed in chapter one, individuals with narcolepsy will

experience daytime sleepiness even when on medication (Mitler, et al., 1994). These results suggest that binge eating behaviour does not vary as a function of the severity of daytime sleepiness in narcolepsy. That is, individuals with narcolepsy and high daytime sleepiness are not more likely to engage in binge eating behaviours than individuals with narcolepsy and a lower level of daytime sleepiness. This is somewhat inconsistent with suggestions made in previous research, such as that of Chabas, et al. (2007) that general eating patterns of individuals with narcolepsy may be a strategy to control daytime sleepiness. Logically, if this was the case the less daytime sleepiness that was experienced by an individual with narcolepsy the less need there would be for the engagement in behaviour to control the same. This control may be through either purposefully controlled behaviour or uncontrolled behaviour. The finding of no relationship between severity level of sleepiness and binge eating is also interesting because excessive sleepiness is believed to be associated with other forms of uncontrolled behaviour in narcolepsy such as automatic behaviour. What is more interesting here is that automatic behaviour in narcolepsy is often not remembered by the individual with narcolepsy (due to sleepiness; Black, et al. 2005). However, as binge eating was self reported in the current study it is presumed that individuals with narcolepsy (or at least most) can remember engaging in binge eating behaviour. So although "uncontrolled", binge eating is a conscious behaviour in individuals with narcolepsy separating it from automatic behavioural symptoms in narcolepsy. At the time of writing no published research known to the author had quantitatively measured the relationship between severity of sleepiness and binge eating behaviour. The current study cannot rule out the role of daytime sleepiness itself (regardless of severity) on binge eating behaviours as it compared two levels of severity rather than a correlation between sleepiness and binge eating scores.

The current study found no significant difference between individuals with narcolepsy on high versus low Epworth Sleepiness Scale scores (on medication) on the mood factor of the MCQ or on the mood factor of the SDCQ. That is, individuals with narcolepsy and high daytime sleepiness were not more likely to make mood-based meal, snack and/or drink choices than individuals with narcolepsy and a low sleepiness score. These findings are not consistent with the hypotheses of the current study. However, they are consistent with the previously reported findings of the current study that individuals with narcolepsy were not more likely to make meal, snack and drink mood-based choices than those without narcolepsy. At the time of writing no published research known to the author had quantitatively measured severity of sleepiness and mood based choices for meals, snacks and drinks for individuals with narcolepsy.

In summary the current study found that severity of daytime sleepiness does not impact on the likelihood to engage in binge eating behaviour in individuals with narcolepsy. It was also found that severity of daytime sleepiness did not impact on mood based choices for meals, snacks and drinks in individuals with narcolepsy.

4.2 Limitations and Directions for Future Research

It may be argued that the sample size of the current study is small, however, power was sufficient enough to detect medium and large effect sizes. Power was low to detect small effect sizes and future research may benefit from increasing the sample size in order to investigate whether additional differences with low effect sizes may be occurring in relation to eating behaviours in individuals with narcolepsy.

A limitation of the current study is that, with the exception of the Epworth Sleepiness Scale (Johns, 1991), none of the questionnaires/measures had been validated for specific use in the narcolepsy population. However, as set out in the results section

confirmatory factor analyses and internal reliability checks were performed where possible. Care was taken in the current study in choosing measures that limited symptom overlap. For example within the Depression Anxiety Stress Scale (DASS; Lovibond & Lovibond, 1995b) the depression scale does not include items referring to symptoms of fatigue or sleep duration. However, it should be noted that the DASS does contain items that may be influenced by the symptom of cataplexy and sleepiness and by the use of stimulant medication (for example, "feeling of shakiness e.g. legs going to give way", "dryness of my mouth", "I found myself getting agitated"). Such items may have inflated depression, anxiety and/or stress scores in individuals with narcolepsy. Future research would benefit from the controlling of such items or the development of a specific tool to be used within the population of individuals with narcolepsy.

A possible limitation of the current sample was that the mean age of individuals with narcolepsy was 58 years old. The current findings may be more applicable to older individuals with narcolepsy than younger individuals with narcolepsy. Interestingly though, as age was a matched variable across groups in the current study, if patterns are indicative of aging in narcolepsy the same pattern was not found in the individuals without narcolepsy sample.

Presumably, the sample of participants used for the current sample was adequately self motivated to complete the somewhat lengthy questionnaire and return it to the sender. The individuals with narcolepsy sample of the current study may represent those that had more time available, were more organised, less forgetful and/or sufficiently functional to complete the questionnaires. Additionally, the mean depression scores for the individuals with narcolepsy was within the average range, and this is at variance with reports in the literature of clinical ranged depression being more likely to occur within individuals with narcolepsy than in individuals without narcolepsy (for example,

Lindsley & Crawford, 1996). It is possible that those individuals with high/severe levels of depression were less likely to complete the questionnaire package and return it to the sender. The same argument may stand for anxiety and stress. Another limitation of using questionnaires was that they relied on self reported diagnosis of narcolepsy and co-morbidities. Although questions were tailored to meet diagnostic criteria for narcolepsy, and this was examined closely by researchers, it is always possible that inaccurate information may be collected through self report. Questionnaires were designed to be brief to minimise difficulties arising from possible attention, concentration and memory deficits known to occur in narcolepsy (as discussed in section 1.2.1.). However, future research may benefit from more detailed questions relating to the symptoms of narcolepsy (specifically cataplexy, sleep paralysis and hallucinations) and/or cross reference of diagnosis and co-morbidities through one on one interview or confirmation by the participant's treating practitioner. Possible misclassification of narcolepsy diagnosis may have influenced the results.

Another limitation of the current study was that hypotheses two to four (relating to meal sizes) were not analysed. These hypotheses were unable to be analysed due to a high proportion (15 percent or more) of missing data on information pertaining to the sizes of breakfast, lunch and dinner meals. This was despite the inclusion of a detailed example within the questionnaire package, please refer to appendix C. It may be argued that memory deficits known to commonly occur in narcolepsy (Ohayon, et al. 2005) may have affected particpants with narcolepsy to accuately report information on food and drink intake resulting in high missing values. However, missing data propotions for breakfast, lunch and dinner sizes were still high for indidviuals without narcolepsy. In order to maintain confidentiality participants were not asked to provide personal details on their returned questionnaires and therefore no follow up on missing data was

possible. Future research may benefit from considering other methods of collecting data pertaining to the sizes of breakfast, lunch and dinner meals such as telephone, face to face interviews and observation through clinical trials or a voluntary request to provide contact details for further follow up if required. It is of interest for future research to look into these hypotheses as it may provide valuable information to aid in further understanding the eating patterns and timing of food intake in individuals with narcolepsy. For example, are individuals with narcolepsy reducing daytime gratification of meals resulting in overindulgence or binging behaviour in the evening?

Post-hoc analyses were conducted to investigate whether there was any significant differences between those with narcolepsy who suffer from cataplexy and those who do not suffer from cataplexy. Variables of measure were chosen based on the premise of the known action of hypocretin which may be expected to influence these variables or previous research has shown a stronger presence of these variables in individuals with narcolepsy with cataplexy in comparison to individuals with narcolepsy with no cataplexy: BMI (Sakurai, 2006), frequency of snacks and drinks (Sakurai, 2006; Willie, et al., 2001), depression (Dimitrova, et al., 2011), anxiety (Flosnik, et al., 2009), stress (Berridge, et al., 2010) and binge eating (Dimitrova, et al., 2011). Given that current results were contrary to the literature, it may be likely that low or medium effect size differences are present but unable to be detected by the current study. Further research would be of interest particularly given the stronger link between hypocretin deficiency in individuals with narcolepsy with cataplexy compared to individuals with narcolepsy without cataplexy (Thannickal, et al., 2009). Distinguishing eating patterns and behaviours in those with and without cataplexy would provide valuable input into the possible role of hypocretin in eating patterns and behaviours. However, the high proportion of participants with narcolepsy and cataplexy (77 percent) in the current

study provides the expectation that the majority of the narcolepsy participants would have low or no hypocretin levels in their cerebral spinal fluid (Thannickal, et al., 2009). Some interesting questions for further research are raised by this; Is there a compensatory appetite regulation system as a response to the hypocretin deficiency? If so, does this predispose binge eating and or increased snacking and drinking in those with narcolepsy? What is the role, if any, of increased anxiety and stress in modulating this system? Does the purposeful timing of meals and snacks in relation to sleepiness impact on this system?

It should also be noted that the current study cannot make specific conclusions on the role or effects of medication. Narcolepsy medication may also have a relationship with food intake, in particular stimulant medications have been found to reduce appetite (Makrisa, 2004; Prescrire International," 2001). Interestingly however, the current study found an increased frequency in snack and drink consumption (assuming an increased appetite) in narcolepsy in comparison to individuals without narcolepsy. Furthermore, a relatively large proportion of individuals with narcolepsy (43 percent) and without narcolepsy (40 percent) where taking medication for a condition other than narcolepsy. The current study did not assess whether these medications were comparable across groups or whether these medications themselves may have a known influence on eating behaviour and therefore it is unknown whether this may have influenced results. Future research may benefit from controlling for such medication.

An aim of the current study was to examine the eating behaviours and attitudes of individuals with narcolepsy in regards to their normal everyday functioning, specifically with respect to stimulant medication status and irrespective of cataplexy status. Such an examination adds to the clinical relevance of the findings. The proportions of the individuals with narcolepsy sample with and without cataplexy in the current study

approximate the proportions reported in the literature across narcolepsy populations. The literature generally reports a range between 60 to 90 percent of individuals with narcolepsy having the symptom of cataplexy (Bassetti & Aldrich, 1996; Chaudhary & Husain, 1993; Stores, 1999). While this more clinically based approach may limit conclusions about putative physiological mechanisms, it enhances the external validity of the results to the clinical population of individuals diagnosed with narcolepsy. It is hoped that the findings of the current study will assist medical and mental health professionals in providing an understanding and development of symptom management, which is applicable to the everyday experience of individuals with narcolepsy receiving treatment.

Overall, this study has provided a valid and reliable contribution to the research currently available on eating patterns, behaviours and attitudes in the narcolepsy population. As there is limited research into the area of eating patterns and behaviours in narcolepsy further research and replication of current findings are needed. This would aid in building, and ultimately establishing, an understanding of the mechanisms and/or connections between behavioural, physiological and psychological principles of increased snacking and drinking, timing of meals and snacks in relation to sleepiness and between binge eating and anxiety and stress in those who suffer from narcolepsy. Such an understanding would assist medical and mental health professionals in providing an understanding and development of symptom management, which is applicable to the everyday experience of individuals with narcolepsy receiving treatment.

Further extended areas of research that may be of benefit/interest include the investigation of influences of anxiety and stress on the frequency and severity of binge

eating, the role of increased snaking and drinking fluids and the role of timing food with sleepiness convenience:

- are individuals with narcolepsy who have high BMIs and comorbid anxiety and/or stress more likely to engage in binge eating than individuals with narcolepsy who have normal or low BMIs ?
- are individuals with narcolepsy who have higher BMIs and comorbid anxiety and/or stress more likely to have higher frequencies of snacking and drinking than individuals with narcolepsy who have normal or low BMIs?
- are individuals with narcolepsy that engage in binge eating more likely to engage in starving themselves during the day in comparison to individuals with narcolepsy that do not engage in binge eating?
- are binge eating behaviours in individuals with narcolepsy more likely to occur in the evening?
- are individuals with narcolepsy who engage in binge eating more likely to be concerned about the timing of their food intake in accordance to sleepiness convenience than individuals with narcolepsy that do not binge eat?
- are individuals with narcolepsy who engage in binge eating more likely to be sensitive to the sleep inducing properties of nutrients such as carbohydrates compared to individuals with narcolepsy who do not binge eat?
- and can the use of psychological therapies (i.e., cognitive behavioural therapy) to treat anxiety/stress influence binge eating and vice versa in individuals with narcolepsy?

Further, many questions remain open in regards to the higher rates of obesity and diabetes mellitus, mood dysfunction and maladaptive eating behaviours and attitudes experienced by individuals with narcolepsy.

4.3. Conclusions

This is the first study to show a connection between food and drink intake, timing of food intake and sleepiness within individuals with narcolepsy. This suggests that the management of a sleep-wake schedule may influence the eating behaviours in individuals with narcolepsy and vice versa. The current study also found individuals with narcolepsy were more likely to consume greater amounts of snacks and drinks compared to controls. Individuals with narcolepsy were less concerned about the choice of snacks and drinks that aided weight control than those without narcolepsy. Interestingly, no difference between individuals with and without narcolepsy in choice of meals or snacks and drinks that aided in coping with negative mood was found. Taken together, the findings of increased binge eating and snack/drink intake in the current study indirectly support the suggestion of a qualitative change in eating patterns within individuals with narcolepsy compared to individuals without narcolepsy. Whether or not an overall quantitative difference in eating patterns in narcolepsy also exists is not known as calorie intake was not directly assessed in the current study.

Importantly, this study has revealed two eating behaviours in narcolepsy that are indicative of opposing frameworks. One framework is of a purposeful eating behaviour and the other framework is of an uncontrolled eating behaviour. The pattern of timing meals and snacks according to the convenience or otherwise of sleepiness, implies that individuals with narcolepsy are engaging in a purposeful behaviour to control their symptom of daytime sleepiness. Binge eating may be an uncontrolled compensatory

factor as a consequence of this purposeful timing of food and sleepiness. Further, this study has shown that the extent of binge eating engaged in by individuals with narcolepsy is driven by the emotional attributes of anxiety and stress. The proposed interactions of these variables are shown graphically in Figure 4.1.

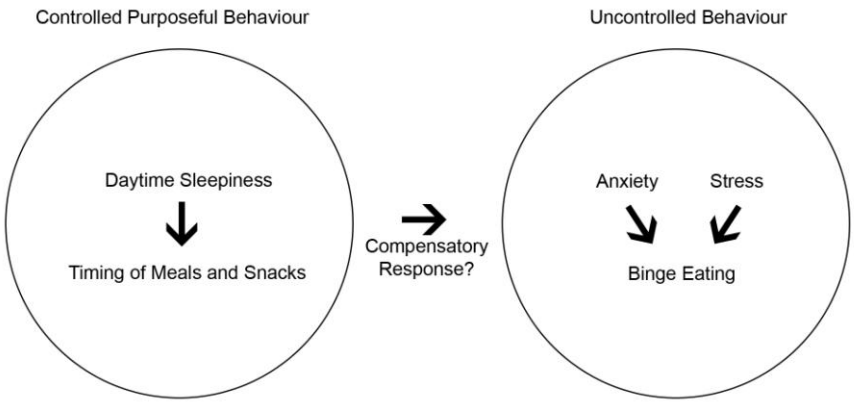

Figure 4.1 Proposed Interactions between Selected Controlled and Uncontrolled Behaviours in Narcolepsy

Individuals with narcolepsy are more likely to have more symptoms of depression, anxiety and stress in comparison to the general population. Anxiety and stress may act as a risk factor to binge eating for individuals with narcolepsy. Additionally, the greater the anxiety and/stress levels the greater the risk of engaging in binge eating behaviour. Interestingly, depression is no more likely a risk factor to binge eating in individuals

with narcolepsy than in individuals without narcolepsy. The uncontrolled nature of binge eating behaviours could increase levels of stress and anxiety and vice versa.

The current study found that severity of daytime sleepiness does not impact on the likelihood to engage in binge eating behaviour. It was also found that severity of daytime sleepiness did not impact on mood based choices for meals, snacks and drinks.

The findings of increased snacking and drinking, importance of timing meal/snack intake with sleepiness convenience, prevalence of binge eating, prevalence of mood dysfunction (depression, anxiety and stress) and the association between binge eating and anxiety and stress are of clinical importance. Professionals working with individuals with narcolepsy should discuss issues around sleep-wake cycles, eating behaviours/attitudes and mood. Referral to a specialist psychiatrist and/or psychologist for cognitive behavioural therapy, education and management of maladaptive psychological issues may be indicated.

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Appendix A: Supporting Letter from the Narcolepsy and Overwhelmining

Daytime Sleepiness Society

NODSS - AUSTRALIA

NODSS - Narcolepsy & Overwhelming Daytime Sleep Society of Australia
ABN 74 398 69? 595

18th June, 2008

The Chairperson
Ethics Committee
Victoria University

Dear Sir/Madam RE: The proposed research on narcolepsy and dietary issues by postgraduate doctoral student: Ms Danielle GATTI

Our society "NODSS-Australia" was founded by psychologist Dorothy Bruck in 1986 to function not only as a self-help support group for persons with narcolepsy and related disorders of somnolence (overwhelming daytime sleepiness), but also to facilitate research into narcolepsy.

As a result, over the last 20 years or so many worthwhile studies on narcolepsy have been conducted under her auspices. Indeed, both Professor Bruck and her former colleague Dr Bernadette Hood (who was herself one of Dorothy's postgraduate doctoral students) have received awards for excellence in their research, from international journals.

Because narcolepsy is considered to be one of the "orphan disorders" even today, the research results produced from Victoria University, School of Psychology, are greatly valued, and have provided us with much needed data/information.

This has been very encouraging. We, the patients with narcolepsy, continue to hope for better understanding from our specialist physicians who (in turn) are vindicated in pursuing treatment regimes to more effectively treat our condition, thus to help us maintain proper alertness.

"NODSS Australia" as an organisation therefore gives full support to Professor Bruck and her postgraduate student Ms Gatti, in undertaking this proposed research study.

Note that NODSS members will be asked for their consent to participate, on an individual basis. Furthermore, participants are informed that they have the right to withdraw from the study, at any time.

We hope you are able to approve and support Professor Bruck and Ms Gatti in this proposed research venture.

Yours sincerely

[redacted]

Judith Tinney *President* PH: 03 9870 1950
Carole Silly *Vice-President*
Elizabeth Hickey *Treasurer/Mail-Secretary*

for "NODSS Australia":
Board Members (2007-2008)

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All donations $2.00 and over tax-deductible

Appendix B: Ethics Approval Letter

VICTORIA UNIVERSITY | A NEW SCHOOL OF THOUGHT

MEMO

TO	Prof. Dorothy Bruck School of Psychology St Albans Campus	DATE	4/09/2008
FROM	Prof. Carolyn Noble Chair Arts, Education & Human Development Human Research Ethics Subcommittee		
SUBJECT	Ethics Application – HRETH 08/156		

Dear Prof. Bruck,

Thank you for submitting this application for ethical approval of the project:

HRETH08/156 Eating behaviours and attitudes in individuals with narcolepsy

The proposed research project has been accepted and deemed to meet the requirements of the National Health and Medical Research Council (NHMRC) 'National Statement on Ethical Conduct in Human Research (2007)', by the Faculty of Arts, Education & Human Development Human Research Ethics Subcommittee. Approval has been granted from 04/09/2008 to 31/01/2010.

Continued approval of this research project by the Victoria University Human Research Ethics Committee (VUHREC) is conditional upon the provision of a report within 12 months of the above approval date (by ***4 September 2009***) or upon the completion of the project (if earlier). A report proforma may be downloaded from the VUHREC web site at: http://research.vu.edu.au/hrec.php

Please note that the Human Research Ethics Committee must be informed of the following: any changes to the approved research protocol, project timelines, any serious events or adverse and/or unforeseen events that may affect continued ethical acceptability of the project. In these unlikely events, researchers must immediately cease all data collection until the Committee has approved the changes. Researchers are also reminded of the need to notify the approving HREC of changes to personnel in research projects via a request for a minor amendment.

If you have any further queries please do not hesitate to contact me on 9919 2917.

Prof. Carolyn Noble
Chair
Faculty of Arts, Education & Human Development Human Research Ethics Subcommittee

Appendix C: Questionnaires

General Information Questionnaire for Control Participants

Questionnaire ID 00XX

The following are some general questions about you. Please tick the appropriate box.

1. Sex:

Female ☐
Male ☐

2. Age: _____year's _____months

3. Highest Educational Qualification:

Year 11 or Below ☐
Year 12 Completed ☐
Apprenticeship/TAFE ☐
University Degree ☐
Higher University Degree ☐

4. Employment Status:

Employed:
Full time ☐
Part time ☐

Not Employed ☐

Not in Labor Force:
Retired ☐
Stay Home Parent ☐
Student ☐

5. Occupation Type:

Senior Management & Professionals ☐

Managers & Associate Professionals ☐

Trades persons, Clerks, Skilled, Office & Sales ☐

Machine Operators, hospitality, assistants, labourers ☐

6. How tall are you with your shoes off?

☐☐☐ cm or ☐ ft ☐ inches

7. How much do you weigh with your clothes off?

☐☐☐ kg or ☐☐ stones or ☐☐☐ pounds

8. Please tick one box in each row.
In the past month how <u>dissatisfied</u> have you felt about your...

	Not at all	Slightly	Moderately	Markedly
Weight				
Body Shape				

9. During the past month, how would you rate your night time sleep quality overall?
Very Good ☐

Fairly Good ☐

Fairly Bad ☐

Very Bad ☐

10. Do you have a sleep disorder (e.g., narcolepsy, sleep apnea or insomnia)? (Note: if you have narcolepsy you should complete a different questionnaire)
Yes ☐ Please Specify

_____
_____
_____

No ☐

11. Do you have any medical/psychological conditions other than a sleep disorder (e.g., diabetes, bulimia nervosa, depression, heart condition)?
Yes ☐ Please Specify

_____
_____
_____

No ☐

12. Are you currently taking any medication?
Yes ☐ Please
Specify_____
_____
_____

No ☐

General Information Questionnaires for Participants with Narcolepsy

Questionnaire ID: 00XX.X

The following is some general questions are about you. Please tick the appropriate box.

1. Sex:

Female ☐
Male ☐

2. Age: _____year's _____months

3. Highest Educational Qualification:

Year 11 or Below ☐
Year 12 Completed ☐
Apprenticeship/TAFE ☐
University Degree ☐
Higher University Degree ☐

4. Employment Status:

Employed:
Full time ☐
Part time ☐

Not Employed ☐
Not in Labor Force:
Retired ☐
Stay Home Parent ☐
Student ☐

5. Occupation Type:

Senior Management & Professionals ☐

Managers & Associate Professionals ☐

Trades persons, Clerks, Skilled, Office & Sales ☐

Machine Operators, hospitality, assistants, labourers ☐

6. How tall are you with your shoes off?

☐☐☐ cm or ☐ ft ☐ inches

7. How much do you weigh with your clothes off?

☐☐☐ kg or ☐☐ stones or ☐☐☐ pounds

8. Please tick one box in each row.
In the past month how <u>dissatisfied</u> have you felt about your...

	Not at all	Slightly	Moderately	Markedly
Weight				
Body Shape				

9. During the past month, how would you rate your night time sleep quality overall?

Very Good ☐

Fairly Good ☐

Fairly Bad ☐

Very Bad ☐

The following questions relate to your diagnosis and symptoms of narcolepsy.

1. At what age were you diagnosed with narcolepsy?

_____

2. Looking back, at what age do think the symptoms of narcolepsy started?

_____

3. What type of medical professional diagnosed you with narcolepsy (e.g., general practitioner, sleep physician, neurologist)?

_____

Comments (if any):

_____

_____

_____

4. Are you currently taking medication for your narcolepsy?

No ☐

Yes ☐ Please specify: Type of medication Dosage

_____ _____

_____ _____

_____ _____

5. Have you previously had a sleep test (e.g., Multiple Sleep Latency Test) in a clinic?

No ☐

Yes ☐ Did the sleep test indicate narcolepsy? Yes ☐

No ☐

Please give the approximate date of the sleep test (month and year if known) Month _____ Year _____

6. Did you have any other tests in the course of being diagnosed with narcolepsy (e.g., blood, cerebral spinal fluid)?

No ☐

Yes ☐ Please specify: What was the test? To your knowledge, was the result consistent with narcolepsy? (Y/N)

_____ _____

_____ _____

_____ _____

7. Do you suffer from cataplexy (a sudden loss of muscle tone, usually triggered by an emotion such as laughter?)

No ☐

Yes ☐ Please specify how frequently _____

8. Upon falling asleep or awakening have you ever felt unable to move (sleep paralysis)?

No ☐

Yes ☐ Please specify how frequently _____

9. Have you ever experienced dream-like imagery (hallucinations) when you know you are awake?

No ☐
Yes ☐ Please specify how frequently _____

10. Please estimate to what extent the symptoms of narcolepsy (include sleepiness, cataplexy and other related symptoms) currently interfere with your day to day activities?

Not at all ☐ Slightly ☐ Moderately ☐ Quite a Bit ☐ Extremely ☐

11. Do you have any medical/psychological conditions other than narcolepsy (e.g., sleep apnea, diabetes, bulimia nervosa, depression, heart condition)?

No ☐
Yes ☐ Please Specify: _____

_____

_____

12. Do you take your stimulant medication for narcolepsy with food? (leave blank if not taking stimulants)

No ☐
Yes ☐ When and which medication?

_____
_____
_____

Why?

_____
_____
_____
_____

13. Are you currently taking any medication other than for narcolepsy?

No ☐
Yes ☐ Please Specify:_____
_____
_____

Epworth Sleepiness Scale for control participants

The following questionnaire will help you measure your general level of daytime sleepiness. You are to rate the chance that you would doze off or fall asleep during different routine daytime situations.

How likely are you to doze off or fall asleep in the following situations, in contrast to just feeling tired? Even if you haven't done some of these activities recently, think about how they would have affected you. Use this scale to choose the most appropriate number for each situation:

0 = would never doze

1= slight chance of dozing

2 = moderate chance of dozing

3 = high chance of dozing

It is important that you circle a number (0 to 3) on each of the questions.

Situation	**Chance of Dozing (0-3)**			
Sitting and reading	0	1	2	3
Watching television	0	1	2	3
Sitting inactive in a public place for example, a theatre or meeting	0	1	2	3
As a passenger in a car for an hour without a break	0	1	2	3
Lying down to rest in the afternoon	0	1	2	3
Sitting and talking to someone	0	1	2	3
Sitting quietly after lunch (when you've had no alcohol)	0	1	2	3
In a car while stopped in traffic	0	1	2	3

Epworth Sleepiness Scales for participants with narcolepsy (two sections)

The following questionnaire will help you measure your general level of daytime sleepiness. You are to rate the chance that you would doze off or fall asleep during different routine daytime situations.

(1) Answer the following questions in relation to when *not* taking stimulant medication

How likely are you to doze off or fall asleep in the following situations, in contrast to just feeling tired? Even if you haven't done some of these activities recently, think about how they would have affected you. Use this scale to choose the most appropriate number for each situation:

0 = would never doze
1= slight chance of dozing
2 = moderate chance of dozing
3 = high chance of dozing

It is important that you circle a number (0 to 3) on each of the questions.

Situation	Chance of Dozing (0-3)			
Sitting and reading	0	1	2	3
Watching television	0	1	2	3
Sitting inactive in a public place-for example, a theatre or meeting	0	1	2	3
As a passenger in a car for an hour without a break	0	1	2	3
Lying down to rest in the afternoon	0	1	2	3
Sitting and talking to someone	0	1	2	3
Sitting quietly after lunch (when you've had no alcohol)	0	1	2	3
In a car while stopped in traffic	0	1	2	3

(2) Answer the following questions in relation to while <u>taking your typical daily dose of stimulant</u> medication (leave blank if you do not take stimulant medication)

How likely are you to doze off or fall asleep in the following situations, in contrast to just feeling tired? Even if you haven't done some of these activities recently, think about how they would have affected you. Use this scale to choose the most appropriate number for each situation:

0 = would never doze
1= slight chance of dozing
2 = moderate chance of dozing
3 = high chance of dozing

It is important that you circle a number (0 to 3) on each of the questions.

Situation	Chance of Dozing (0-3)			
Sitting and reading	0	1	2	3
Watching television	0	1	2	3
Sitting inactive in a public place-for example, a theatre or meeting	0	1	2	3
As a passenger in a car for an hour without a break	0	1	2	3
Lying down to rest in the afternoon	0	1	2	3
Sitting and talking to someone	0	1	2	3
Sitting quietly after lunch (when you've had no alcohol)	0	1	2	3
In a car while stopped in traffic	0	1	2	3

Meal Choice Questionnaire for all participants

The following questions are about the **meal** (e.g., breakfast, lunch and dinner and including light meals) choices you make on a typical day. Please tick the one box in each row that you feel is most appropriate. Your responses should reflect your actual meal choices, not what you think you should be eating.

It is important to me that the meals I eat on a typical day.....	Not At All Important	A Little Bit Important	Moderately Important	Very Important
1. Contain a lot of vitamins				
2. Help me cope with my stress				
3. Are easy to prepare				
4. Contain no additives				
5. Are low in calories				
6. Keep me healthy				
7. Can be cooked very simply				
8. Help me cope with life				
9. Are nutritious				
10. Contain little/no sugar				
11. Are high in protein				
12. Take no time to prepare				
13. Help me control my weight				
14. Are good for my skin/teeth/hair/nails etc.				
15. Contain natural ingredients				
16. Help me relax				
17. Are low in fat				
18. Can be brought in shops close to where I live or work				
19. Keep me awake/alert				
20. Contain no artificial ingredients				
21. Cheers me up				

It is important to me that the meals I eat on a typical day.....				
	Not At All Important	A Little Bit Important	Moderately Important	Very Important
22. Are high in fiber and roughage				
23. Do not contain items I am sensitive/allergic to				
24. Make me feel good				
25. Are easily available in shops and supermarkets				
26. Contain complex carbohydrates e.g., whole meal ingredients				
27. Contain a lot of sugar				

Snack and Drink Choice Questionnaire for all participants

The following questions are about the **snack** and **drink** choices you make on a typical day. A snack is food that you eat between meals (e.g., smaller than a light meal but more than a single lolly). Drinks include any beverage, whether hot or cold, except water. Please tick the box that you feel is most appropriate. Your responses should reflect your actual snack and drink choices, not what you think you should be eating.

It is important to me that the snacks and drinks I consume on a typical day.....				
	Not At All Important	A Little Bit Important	Moderately Important	Very Important
1. Contain a lot of vitamins				
2. Help me cope with my stress				
3. Are easy to prepare				
4. Contain no additives				
5. Are low in calories				
6. Keep me healthy				
7. Can be cooked very simply				
8. Help me cope with life				
9. Are nutritious				
10. Contain little/no sugar				
11. Are high in protein				
12. Take no time to prepare				
13. Help me control my weight				
14. Are good for my skin/teeth/hair/nails etc.				
15. Contain natural ingredients				
16. Help me relax				
17. Are low in fat				
18. Can be brought in shops close to where I live or work				

It is important to me that the snacks and drinks I consume on a typical day.....				
	Not At All Important	A Little Bit Important	Moderately Important	Very Important
19. Keep me awake/alert				
20. Contain no artificial ingredients				
21. Cheer me up				
22. Are high in fiber and roughage				
23. Do not contain items I am sensitive/allergic to				
24. Make me feel good				
25. Are easily available in shops and supermarkets				
26. Contain complex carbohydrates e.g., whole meal ingredients				
27. Contain a lot of sugar				
28. Contain caffeine				

DASS for all participants

Please read each of the following statements and circle a number 0, 1, 2 or 3 which indicates how much the statement applied to you **over the past week.**
The rating scale is as follows:

0 Did not apply to me at all
1 Applied to me to some degree, or some of the time
2 Applied to me a considerable degree, or a good part of the time
3 Applied to me very much, or most of the time

	Did not apply	to some degree	to a considerable degree	applies very much
1. I found myself getting upset by quite trivial things	**0**	**1**	**2**	**3**
2. I was aware of dryness of my mouth	**0**	**1**	**2**	**3**
3. I couldn't seem to experience any positive feeling at all	**0**	**1**	**2**	**3**
4. I experienced breathing difficulty (e.g., excessively rapid breathing, breathlessness in the absence of physical exertion)	**0**	**1**	**2**	**3**
5. I just couldn't seem to get going	**0**	**1**	**2**	**3**
6. I tended to over-react to situations	**0**	**1**	**2**	**3**
7. I had a feeling of shakiness (e.g., legs going to give way)	**0**	**1**	**2**	**3**
8. I found it difficult to relax	**0**	**1**	**2**	**3**
9. I found myself in situations that made me so anxious I was most relieved when they ended	**0**	**1**	**2**	**3**
10. I felt that I had nothing to look forward to	**0**	**1**	**2**	**3**
11. I found myself getting upset rather easily	**0**	**1**	**2**	**3**
12. I felt that I was using a lot of nervous energy	**0**	**1**	**2**	**3**
13. I felt sad and depressed	**0**	**1**	**2**	**3**
14. I found myself getting impatient when I was delayed in any way (e.g., lifts, traffic, lights, being kept waiting)	**0**	**1**	**2**	**3**
15. I had a feeling of faintness	**0**	**1**	**2**	**3**

	Did not apply	to some degree	to a considerable degree	applies very much
16. I felt that I had lost interest in just about everything	**0**	**1**	**2**	**3**
17. I felt I wasn't worth much as a person	**0**	**1**	**2**	**3**
18. I felt that I was rather touchy	**0**	**1**	**2**	**3**
19. I perspired noticeably (e.g., hands sweaty) in the absence of high temperature	**0**	**1**	**2**	**3**
20. I felt scared without good reason	**0**	**1**	**2**	**3**
21. I felt that life wasn't worthwhile	**0**	**1**	**2**	**3**
22. I found it hard to wind down	**0**	**1**	**2**	**3**
23. I had difficulty in swallowing	**0**	**1**	**2**	**3**
24. I couldn't seem to get ant enjoyment out of the small things I did	**0**	**1**	**2**	**3**
25. I was aware of the action of my heart in the absence of physical exertion (e.g., sense of heart rate increase, heart missing a beat)	**0**	**1**	**2**	**3**
26. I felt down-hearted and blue	**0**	**1**	**2**	**3**
27. I found that I was very irritable	**0**	**1**	**2**	**3**
28. I felt I was close to panic	**0**	**1**	**2**	**3**
29. I found it hard to calm down after something upset me	**0**	**1**	**2**	**3**
30. I feared that I would be “thrown” by some trivial but unfamiliar task	**0**	**1**	**2**	**3**
31. I was unable to become enthusiastic about anything	**0**	**1**	**2**	**3**
32. I found it difficult to tolerate interruptions to what I was doing	**0**	**1**	**2**	**3**
33. I was in a state of nervous tension	**0**	**1**	**2**	**3**
34. I felt I was pretty worthless	**0**	**1**	**2**	**3**
35. I was intolerant of anything that kept me from getting on with what I was doing	**0**	**1**	**2**	**3**
36. I felt terrified	**0**	**1**	**2**	**3**
37. I could see nothing in the future to be hopeful about	**0**	**1**	**2**	**3**

	Did not apply	to some degree	to a considerable degree	applies very much
38. I felt that life was meaningless	**0**	**1**	**2**	**3**
39. I found myself getting agitated	**0**	**1**	**2**	**3**
40. I was worried about situations in which I might panic and make a fool of myself	**0**	**1**	**2**	**3**
41. I experienced trembling (e.g., in the hands)	**0**	**1**	**2**	**3**
42. I found it difficult to work up the initiative	**0**	**1**	**2**	**3**

The BT Questionnaire for all participants

Please answer each question by putting a tick in the bracket beside the answer which best applies to you. Please respond to each question as honestly as possible.

1. Do you ever eat uncontrollably to the point of stuffing yourself (i.e., going on eating binges)?
( ) Once a month or less (or never)
( ) 2-3 times a month
( ) Once or twice a week
( ) 3-6 times a week
( ) Once a day or more

2. I am satisfied with my eating patterns today.
( ) Agree
( ) Neutral
( ) Disagree a little
( ) Disagree
( ) Disagree strongly

3. Have you ever kept eating until you thought you'd explode?
( ) Practically every time I eat
( ) Very frequently
( ) Often
( ) Sometimes
( ) Seldom or never

4. Would you presently call yourself a "binge" eater?
( ) Yes, absolutely
( ) Yes
( ) Yes, probably
( ) Yes, possibly
( ) No, probably not

5. I prefer to eat:
( ) At home alone
( ) At home with others
( ) In a public restaurant
( ) At a friends house
( ) Doesn't matter

6. Do you feel you have control over the amount of food you consume?
( ) Most or all of the time
( ) A lot of the time
( ) Occasionally
( ) Rarely
( ) Never

7. I use laxatives or suppositories to help control my weight.
( ) Once a day or more
( ) 3-6 times a week
( ) Once or twice a week
( ) 2-3 times a month
( ) Once a month or less (or never)

8. I eat until I feel too tired to continue.
( ) Once a day or more
( ) 3-6 times a week
( ) Once or twice a week
( ) 2-3 times a month
( ) Once a month or less (or never)

9. How often do you prefer eating ice cream, milk shakes, or puddings during a binge?
( ) Always
( ) Frequently
( ) Sometimes
( ) Seldom or never
( ) I don't binge

10. How much are you concerned about your eating binges?
( ) I don't binge
( ) Bothers me a little
( ) Moderate concern
( ) Major concern
( ) Probably the biggest concern of my life

11. Most people I know would be amazed if they knew how much food I can consume at one sitting.
( ) Without a doubt
( ) Very probably
( ) Probably
( ) Possibly
( ) No

12. Do you ever eat to the point of feeling sick?
( ) Very frequently
( ) Frequently
( ) Fairly often
( ) Occasionally
( ) Rarely or never

13. I am afraid to eat anything for fear that I won't be able to stop.
( ) Always
( ) Almost always
( ) Frequently
( ) Sometimes
( ) Seldom or never

14. I don't like myself after I eat too much.
( ) Always
( ) Frequently
( ) Sometimes
( ) Seldom or never
( ) I don't eat too much

15. How often do you intentionally vomit after eating?
( ) 2 or more times a week
( ) Once a week
( ) 2-3 times a week
( ) Once a month
( ) Less than once a month (or never)

16. Which of the following describes your feelings after binge eating?
( ) I don't binge eat
( ) I feel Okay
( ) I feel mildly upset with myself
( ) I feel quite upset with myself
( ) I hate myself

17. I eat a lot of food when I'm not even hungry.
( ) Very frequently
( ) Frequently
( ) Occasionally
( ) Sometimes
( ) Seldom or Never

18. My eating patterns are different from eating patterns of most people.
( ) Always
( ) Almost always
( ) Frequently
( ) Sometimes
( ) Seldom or never

19. I have tried to lose weight by fasting or going on crash diets.
( ) Not in the past year
( ) Once in the past year
( ) 2-3 times in the past year
( ) 4-5 times in the past year
( ) More than 5 times in the past year

20. I feel sad or blue after eating more than I'd planned to eat.
( ) Always
( ) Almost always
( ) Frequently
( ) Sometimes
( ) Seldom, never, or not applicable

21. When engaged in an eating binge, I tend to eat foods that are high in carbohydrates (sweets and starches).

( ) Always
( ) Almost always
( ) Frequently
( ) Sometimes
( ) Seldom, or I don't binge

22. Compared to most people, my ability to control my eating behaviour seems to be:

( ) Greater than other's ability
( ) About the same
( ) Less
( ) Much less
( ) I have absolutely no control

23. One of your best friends suddenly suggests that you both eat at a new restaurant buffet that night. Although you'd planned on eating something light at home, you go ahead and eat out, eating quite a lot and feeling uncomfortably full. How would you feel about yourself on the ride home?

( ) Fine, glad I tried that new restaurant
( ) A little regretful that I'd eaten too much
( ) Somewhat disappointed in myself
( ) Upset with myself
( ) Totally disgusted with myself

24. I would presently label myself a "compulsive eater" (one who engages in episodes of uncontrolled eating).

( ) Absolutely
( ) Yes
( ) Yes, probably
( ) Yes, possibly
( ) No, probably not

25. What is the most weight you've ever lost in one month?

( ) Over 20lbs or 10kgs
( ) 12-20lbs or 6-10kgs
( ) 8-11lbs or 4-5kgs
( ) 4-7lbs or 2-3kgs
( ) Less than 4lbs or 2kgs

26. If I eat too much at night I feel depressed the next morning.

( ) Always
( ) Frequently
( ) Sometimes
( ) Seldom or never
( ) I don't eat too much at night

27. Do you believe that it is easier for you to vomit than it is for most people?

( ) Yes, it is no problem at all for me
( ) Yes, it's easier
( ) Yes, it's a little easier
( ) About the same
( ) No, it's less easy

28. I feel that food controls my life.

( ) Always
( ) Almost always
( ) Frequently
( ) Sometimes
( ) Seldom or never

29. I feel depressed immediately after eating too much.

( ) Always
( ) Frequently
( ) Sometimes
( ) Seldom or never
( ) I don't eat too much

30. How often do you vomit after eating in order to lose weight?

( ) Less than once a month (or never)
( ) Once a month
( ) 2-3 times a month
( ) Once a week
( ) 2 or more times a week

31. When consuming a large quantity of food, at what rate of speed do you usually eat?

( ) More rapidly than most people have ever eaten in their lives
( ) A lot more rapidly than most people
( ) A little more rapidly than most people
( ) About the same rate as most people
( ) More slowly than most people (or not applicable)

32. What is the most weight you've ever gained in 1 month.

( ) Over 20lbs or 10kgs
( ) 12-20lbs or 6-10kgs
( ) 8-11lbs or 4-5kgs
( ) 4-7lbs or 2-3kgs
( ) Less than 4lbs or 2kgs

33. Females only. My last menstrual period was.

( ) Within the past month
( ) Within the past 2 months
( ) Within the past 4 months
( ) Within the past 6 months
( ) Not within the past 6 months

34. I use diuretics (water pills) to help control my weight.

( ) Once a day or more
( ) 3-6 times a week
( ) Once or twice a week
( ) 2-3 times a month
( ) Once a month or less (or never)

***Please note due to administrative error item 35 and 36 of the original questionnaire were left out of the survey package**

Meal and Snack Timing Questionnaire for all participants

The following questions are about the timing of your meals and/or snacks on a typical day. Please tick the box that you feel is most appropriate.

Note: sleepiness – refers to the tendency to fall asleep, not just a feeling of tiredness
meals – for example, breakfast, lunch and dinner and including light meals
snacks - food that you eat between meals for example, smaller than a light meal but more than a single lolly and excludes drinks

It is important to me that the **timing** of my meals and/or snacks on a typical day.....				
	Not At All Important	A Little Bit Important	Moderately Important	Very Important
1. Does not increase my sleepiness when this is inconvenient				
2. Increases my sleepiness when this is convenient				
3. Does not interfere with a time to nap (e.g., lunch time at work)				
4. Are at regular and predictable times				
5. Helps avoid sleepiness in public places				
6. Helps prevent the sudden onset of sleep				

One Day Food and Drink Intake Diary for all participants

At the end of the day please write down everything you ate or drank (do not include water) and the approximate time. Please include as much detail as possible.

An example:

2 pieces of toast with butter and jam	7:00 a.m	Medium
1 white coffee with 1 sugar	7:30 a.m	
1 can of diet coke	11:00 a.m	
steak, peas, potato and carrots	6:00 p.m	Large
1 mars bar	10:30 p.m	

Food or Drink	Approximate times	If this is a meal (e.g., breakfast, lunch, dinner and including light meals) please specify whether it is small, medium or large.

Food or Drink	Approximate times	If this is a meal (e.g., breakfast, lunch, dinner and including light meals) please specify whether it is small, medium or large.

Appendix D: Information for Participants Involved in Research

For Narcolepsy Participants

VICTORIA UNIVERSITY | A NEW SCHOOL OF THOUGHT

INFORMATION TO PARTICIPANTS INVOLVED IN RESEARCH

You are invited to participate

You are invited to participate in a research project entitled **'Eating Attitudes and Behaviours in Individuals with Narcolepsy'.** You **must** be 18 years of age or over to participate**.**

This project is being conducted by a student researcher Danielle Gatti as part of a Doctorate Degree at Victoria University under the supervision of Professor Dorothy Bruck from the School of Psychology, Victoria University. This research project is supported by the Narcolepsy and Overwhelming Daytime Sleep Society of Australia (NODSS) and your contact details have been provided to us by NODSS. If you no longer wish to receive invitations to participate in research please write to NODSS Australia, PO Box 100, Rosanna, 3084.

Project explanation

The aim of the current study is to examine the differences between the eating behaviours and attitudes of individuals with and without narcolepsy.

Research into the eating behaviours of individuals with narcolepsy has been prompted by the discovery that almost all individuals with narcolepsy have little or no hypocretin neurons in their cerebrospinal fluid. This deficiency in hypocretin leads to disruptions in sleep/wake cycles and may, theoretically, be associated with changes in food intake. However, recent findings into the eating patterns in individuals with narcolepsy suggest that there might be other factors that influence their eating behaviours.

It is of interest to know what attributions and/or motivations are offered by individuals with and without narcolepsy about their eating patterns in relation to a variety of factors. Understanding the eating behaviours and attitudes of individuals with narcolepsy may help medical and allied professionals in guiding management of symptoms and counseling.

What will I be asked to do?

You are invited to participate in answering the set of **blue** questionnaires within this package. The questionnaires will take approximately 20-30 minutes to complete. Firstly, you will be asked to answer some demographic questions and some questions about your diagnosis of and medication for narcolepsy. Next you will be asked a series of questions relating to your eating behaviours and attitudes, sleepiness and your stress and emotions. It should be noted that in addition to the questionnaires we ask you to write out a one day food diary and it is asked that you complete this at the end of the day. On completion of the questionnaires and food diary please mail the questionnaire in the self addressed and stamped envelope provided with your package. We would appreciate if you could complete the package **within 10 days**.

You are also invited to give the second **yellow** questionnaire package to a willing **adult** spouse, partner or friend. It is asked that your spouse, partner or friend be no more than 10 years older or younger than yourself and be 18 years of age or older. It is also asked that this individual does not have a known or suspected sleep disorder or another disorder that may restrict food intake for example, diabetes, bulimia

nervosa or anorexia nervosa. Please do not ask a blood relative (e.g. son, mother, sister, uncle) to complete the yellow questionnaire.

Please note that your participation in this project is optional. Also if you would like to participate but do not wish to pass a questionnaire package onto a spouse, partner or friend you can participate without doing this.

What will I gain from participating?

Your participation in this project will help towards gaining insight into eating behaviours and attitudes and how this impacts on the life style of individuals with narcolepsy. A summary of the findings of this research will be included in a NODSS newsletter in due course.

How will the information I give be used?

The data collected will be used solely for the purposes of research, for the completion of Danielle Gatti's university degree and a publication in a scientific journal.

In no way will your personal information appear on any public document and it is asked that you do not mark any personal information on the questionnaires so that you will not be identifiable to the researchers.

What are the potential risks of participating in this project?

Some people may find the content of some of the questions to be sensitive. While we would like to have as many fully completed questionnaire packages as possible, should you find that you would like to omit answering one or more questions, please do so. If any distress arises from the questionnaires, or you would like to discuss a related issue of concern, please contact Dr Gerard Kennedy, whose details are at the bottom of this letter.

Furthermore, if both questionnaire packages (one for you and the one for a spouse, partner or friend) are posted by the same person in the same envelope it is possible that information on the questionnaires may be viewed by the person posting the questionnaire. If you do not wish for this to occur, it is recommended that the questionnaire surveys are placed in different envelopes. Two self addressed and stamped envelopes are included in the package.

How will this project be conducted?

You are asked to fill in the questionnaires and mail it in the self addressed and stamped envelope within 10 days of receiving the package. Your participation is voluntary. The return of the questionnaires will infer consent for your information on the questionnaires to be used for research purposes, for the completion of Danielle Gatti's University Degree and for publication in a scientific journal. If you wish to withdraw your questionnaire after it has been posted please contact the Principal Researcher, Professor Dorothy Bruck, whose details are at the bottom of this letter and quote your questionnaire ID number, which is on the top left hand side of your questionnaire package.

Who is conducting the study?

This study is being conducted by Danielle Gatti and Professor Dorothy Bruck.

Any questions or enquires about your inclusion in this research-study may be directed to the Principal Researcher Professor Dorothy Bruck. Her contact details are (03) 9919 2336 or dorothy.bruck@.vu.edu.au. Alternatively you can contact the student researcher Danielle Gatti on danielle.gatti@live.vu.edu.au. If you would like to discuss any stress related concerns that may arise as a result of your participation please contact Dr. Gerard Kennedy on (03) 9919 2481 or Gerard.Kennedy@vu.edu.au for independent advice.

Any queries about your participation in this project may be directed to the Principal Researcher listed above.

If you have any queries or complaints about the way you have been treated, you may contact the Secretary, Victoria University Human Research Ethics Committee, Victoria University, PO Box 14428, Melbourne, VIC, 8001 phone (03) 9919 4781.

Information for Participants Involved in Research - For Control Participants

INFORMATION TO PARTICIPANTS INVOLVED IN RESEARCH

You are invited to participate

You are invited to participate in a research project entitled **'Eating attitudes and Behaviours in Individuals with Narcolepsy'.** You **must** be 18 years of age or over to participate and do not have a known or suspected sleep disorder or another disorder that may restrict food intake for example, diabetes, bulimia nervosa or anorexia nervosa. If you have narcolepsy please refer to the blue package.

This project is being conducted by a student researcher Danielle Gatti as part of a Doctorates Degree at Victoria University under the supervision of Professor Dorothy Bruck from the School of Psychology Victoria University.

Project explanation

The aim of the current study is to examine the differences between the eating behaviours and attitudes of individuals with and without narcolepsy (a disorder of excessive daytime sleepiness).

Research into the eating behaviours of individuals with narcolepsy has been prompted by the discovery that almost all individuals with narcolepsy have little or no hypocretin neurons in their cerebrospinal fluid. This deficiency in hypocretin leads to disruptions in sleep/wake cycles and may, theoretically, be associated with changes in food intake. However, recent findings into the eating patterns in individuals with narcolepsy suggest that there might be other factors that influence their eating behaviours.

It is of interest to know what attributions and/or motivations are offered by individuals with and without narcolepsy about their eating patterns in relation to a variety of factors. Understanding the eating behaviours and attitudes of individuals with narcolepsy may help medical and allied professionals in guiding management of symptoms and counseling.

What will I be asked to do?

You are invited to participate in answering the set of **yellow** questionnaires within this package. The questionnaires will take approximately 20-30 minutes to complete. Firstly, you will be asked to answer some demographic questions. Next you will be asked a series of questions relating to your eating behaviours and attitudes. Next you will be asked a series of questions relating to your eating behaviours and attitudes, sleepiness and your stress and emotions. It should be noted that in addition to the questionnaires we ask you to write out a one day food diary and it is asked that you complete this at the end of the day. On completion of the questionnaires and food diary please mail the questionnaire in the self addressed and stamped envelope provided with your package. We would appreciate if you could complete the package within 10 days.

Please note that your participation in this project is optional.

What will I gain from participating?

Your participation in this project will help towards gaining insight into eating behaviours and attitudes and how this impacts on the life style of individuals with narcolepsy.

How will the information I give be used?

The data collected will be used solely for the purposes of research, for the completion of Danielle Gatti's university degree and a publication in a scientific journal.

In no way will your personal information appear on any public document and it is asked that you do not mark any personal information on the questionnaires so that you will not be identifiable to the researchers.

What are the potential risks of participating in this project?

Some people may find the content of some of the questions to be sensitive. While we would like to have as many fully completed questionnaire packages as possible, should you find that you would like to omit answering one or more questions, please do so. If any distress arises from the questionnaires, or you would like to discuss a related issue of concern, please contact Dr Gerard Kennedy, whose details are at the bottom of this letter.

Furthermore, if both questionnaire packages (the one for you and the person who gave you the questionnaires) are posted by the same person in the same envelope it is possible that information on the questionnaires may be viewed by the person posting the questionnaire. If you do not wish for this to occur it is recommended that the questionnaire surveys are placed in different envelopes. Two self addressed and stamped envelopes are included in the package.

How will this project be conducted?

You are asked to fill in the questionnaires and mail it in the self addressed and stamped envelope. Your participation is voluntary. The return of the questionnaires will infer consent for your information on the questionnaires to be used for research purposes, for the completion of Danielle Gatti's University Degree and publication in a scientific journal. If you wish to withdraw your questionnaire after it has been posted please contact the Principal Researcher, Professor Dorothy Bruck, whose details are at the bottom of this letter and quote your questionnaire ID which is on the top left hand side of your questionnaire package.

Who is conducting the study?

This study is being conducted by Danielle Gatti and Professor Dorothy Bruck.

Any questions or enquires about your inclusion in this research-study may be directed to the Principal Researcher Professor Dorothy Bruck. Her contact details are (03) 9919 2336 or dorothy.bruck@.vu.edu.au. Alternatively you can contact the student researcher Danielle Gatti on danielle.gatti@live.vu.edu.au. If you would like to discuss any stress related concerns that may arise as a result of your participation please contact Dr. Gerard Kennedy on (03) 9919 2481 or Gerard.Kennedy@vu.edu.au for independent advice for independent advice.

Any queries about your participation in this project may be directed to the Principal Researcher listed above.
If you have any queries or complaints about the way you have been treated, you may contact the Secretary, Victoria University Human Research Ethics Committee, Victoria University, PO Box 14428, Melbourne, VIC, 8001 phone (03) 9919 4781.

Appendix E: Follow-up Letters

first follow up

VICTORIA UNIVERSITY | A NEW SCHOOL OF THOUGHT

Eating Behaviours and Attitudes in Individuals with Narcolepsy Study

Recently you received a package from us in relation to a study on the eating behaviours and attitudes of individuals with narcolepsy. This letter is a reminder.

We are hoping to achieve a good response rate for our research, so that we can be as confident as possible about the conclusions that we draw. We would greatly appreciate it if you could take some time within the next week to complete the package, and also, if possible, hand out a package to a spouse, partner or friend without narcolepsy. The original package included self addressed and stamped envelopes.

We remind you that your participation is voluntary and entirely confidential.

If you have already completed and sent back your questionnaires, thank you, and please disregard this letter.

With best wishes,

Danielle Gatti and Professor Dorothy Bruck

Any questions or enquires about your inclusion in this research-study may be directed to the Principal Researcher Professor Dorothy Bruck. Her contact details are (03) 9919 2336 or dorothy.bruck@.vu.edu.au. *Alternatively you can contact the student researcher Danielle Gatti on danielle.gatti@live.vu.edu.au.* If you would like to discuss any stress related concerns that may arise as a result of your participation please contact Dr. Gerard Kennedy on (03) 9919 2481 or Gerard.Kennedy@vu.edu.au for independent advice
Any queries about your participation in this project may be directed to the Principal Researcher listed above.
If you have any queries or complaints about the way you have been treated, you may contact the Secretary, Victoria University Human Research Ethics Committee, Victoria University, PO Box 14428, Melbourne, VIC, 8001 phone (03) 9919 4781.

Second Follow-up Letter

Eating Behaviours and Attitudes in Individuals with Narcolepsy Study

About a month ago you received a package from us in relation to a study on the eating behaviours and attitudes of individuals with narcolepsy. This letter is a **final** reminder.

Your participation in this project will help towards gaining insight into eating behaviours and attitudes and how this impacts on the life style of individuals with narcolepsy.

It would be greatly appreciated if you could take some time within the next week to complete the package, and also, if possible, hand out a package to a spouse, partner or friend without narcolepsy. The original package included self addressed and stamped envelopes. If you have misplaced the original package but would like to participate please contact us (see below) and we will send out a replacement.

We remind you that your participation is voluntary and completely confidential.

If you have already completed and sent back your questionnaires, thank you, and please disregard this letter.

With best wishes,

Danielle Gatti and Professor Dorothy Bruck

Any questions or enquires about your inclusion in this research-study may be directed to the Principal Researcher Professor Dorothy Bruck. Her contact details are (03) 9919 2336 or dorothy.bruck@.vu.edu.au. *Alternatively you can contact the student researcher Danielle Gatti on danielle.gatti@live.vu.edu.au.* If you would like to discuss any stress related concerns that may arise as a result of your participation please contact Dr. Gerard Kennedy on (03) 9919 2481 or Gerard.Kennedy@vu.edu.au for independent advice.

Any queries about your participation in this project may be directed to the Principal Researcher listed above.
If you have any queries or complaints about the way you have been treated, you may contact the Secretary, Victoria University Human Research Ethics Committee, Victoria University, PO Box 14428, Melbourne, VIC, 8001 phone (03) 9919 4781.

Appendix F: Narcolepsy Participant Exclusion/Inclusion Criteria

ID	Sex	Age in years	Narcolepsy diagnosed by...	Currently taking medication for narcolepsy/ cataplexy	Multiple Sleep Latency Test that indicated narcolepsy	Other tests that indicated narcolepsy	Cataplexy	Sleep paralysis	Hallucinations	ESS: not taking stimulant medication	Other information or comments	Included in sample
001	M	44	Sleep Physician	Y	Y	N	N	Y	N	14		**Y**
003	F	72	Neurologist	Y	N	Y- not specified	Y	N	Y	20		**Y**
004	M	82	Neurologist	Y	Y	Y –EEG & CSF	Y	Y	Y	16		**Y**
006	M	76	Neurologist	Y	Y	N	Y	Y	Y	19		**Y**
008	F	77	Neurologist	N	NA	Y-EEG	Y	Missing data	Missing data	19	Diagnosed at age 24 prior to MSLT	**Y**
009	F	70	Sleep Physician	Y	Y	N	Y	Y	Y	18		**Y**
0010	M	69	Neurologist	Y	NA	N	Y	Y	Y	15		**Y**
0011	M	34	Sleep Physician	Y	Y	N	N	Y	Y	8	Excluded because low ESS & has a drug & alcohol addiction	**N**
0012	F	57	Neurologist	NA	N	N	Y	Y	N	24		**Y**
0016	F	57	Neurologist	Y	NA	N	Y	Y	Y	2	Excluded because has no MSLT, no sleepiness & no stimulant medication	**N**

ID	Sex	Age in years	Narcolepsy diagnosed by...	Currently taking medication for narcolepsy/ cataplexy	Multiple Sleep Latency Test that indicated narcolepsy	Other tests that indicated narcolepsy	Cataplexy	Sleep paralysis	Hallucinations	ESS: not taking stimulant medication	Other information or comments	Included in sample
0020	F	72	Sleep Physician	Y	NA	Y -Blood	Y	Y	N	11		**Y**
0021	M	76	Neurologist	N	N	Y- EEG	Y	Y	Y	22		**Y**
0022	M	72	Neurologist	Y	Y	N	Y	Y	N	19		**Y**
0024	F	75	Neurologist	Y	NA	N	Y	Y	Y	21		**Y**
0026	M	16	General Practitioner	Y	Y	N	Y	N	N	20	Excluded because age < 18	**N**
0031	F	20	Paediatrician	Y	Y	Missing data	Y	Y	Y	22		**Y**
0032	F	34	Sleep Physician	N	Y	N	N	N	N	13		**Y**
0036	F	79	Sleep Physician	Y	Y	N	Y	N	N	16		**Y**
0037	F	32	Sleep Physician	Y	Y	Y - Blood	N	Y	Y	18		**Y**
0041	F	67	Neurologist	Y	Y	N	Y	Y	Y	23		**Y**
0042	F	55	Not diagnosed with narcolepsy	N	NA	N	N	Y	N	20	Excluded - diagnosed with hypsomlence not narcolepsy	**N**
0043	M	78	Neurologist	Y	Y	Y- not specified	Y	Y	Y	21		**Y**
0045	F	65	Neurologist	N	NA	N	Y	Y	Y	23		**Y**
0049	F	80	General Practitioner	N	NA	N	N	N	Y	19	Excluded because diagnosed by GP & has had no MSLT	**N**
0050	F	57	Missing data	Y	Y	Missing data	Y	Y	N	18		**Y**
0051	F	79	Neurologist	Y	Y	N	Y	Y	Y	21		**Y**
0052	F	72	Neurologist	Y	Y	N	Y	Y	Y	21		**Y**

ID	Sex	Age in years	Narcolepsy diagnosed by...	Currently taking medication for narcolepsy/ cataplexy	Multiple Sleep Latency Test that indicated narcolepsy	Other tests that indicated narcolepsy	Cataplexy	Sleep paralysis	Hallucinations	ESS: not taking stimulant medication	Other information or comments	Included in sample
0053	M	31	Thoracic Physician	N	Y	N	Y	Y	Y	16		**Y**
0055	F	81	Neurologist	N	Missing data	Missing data	N	N	N	13	Excluded has no auxiliary symptoms, no MSLT & no medication	**N**
0056	F	74	Neurologist	Y	N	N	N	Y	Y	17	Excluded - MSLT was negative & no cataplexy	**N**
0057	F	61	General Practitioner	Y	Y	N	N	N	Y	19		**Y**
0058	F	38	Immunologist	Y	Y	Y- not specified	Y	Y	Y	11		**Y**
0060	F	66	General Practitioner	Y	NA	N	Y	Y	Y	18	Exclude because she has diabetes	**N**
0064	F	67	Psychiatrist	Y	Y	N	Y	Y	N	22		**Y**
0066	F	77	Neurologist	N	Y	N	Y	Y	N	16		**Y**
0067	M	32	Sleep Physician	Y	Y	N	N	Y	Y	17	Lactose Intolerant	**Y**
0072	M	68	Neurologist	Y	N	N	Y	Y	N	16		**Y**
0073	F	44	Sleep Physician	Y	Y	N	Y	Y	Y	24		**Y**
0074	F	86	Sleep Physician	Y	Y	N	N	Y	Y	20		**Y**
0077	F	51	Neurologist	Y	N	Y – blood test	Y	Y	Y	17		**Y**
0078	M	71	Neurologist	Y	NA	N	N	Y	Y	18	Included - diagnosed prior to MSLT on medication for auxiliary, high ess score symptoms	**Y**

ID	Sex	Age in years	Narcolepsy diagnosed by...	Currently taking medication for narcolepsy/ cataplexy	Multiple Sleep Latency Test that indicated narcolepsy	Other tests that indicated narcolepsy	Cataplexy	Sleep paralysis	Hallucinations	ESS: not taking stimulant medication	Other information or comments	Included in sample
0079	M	54	Neurologist	Y	NA	N	N	N	N	16	Excluded because he has obsessive compulsive disorder & no auxiliary symptoms	**N**
0080	F	26	Sleep Physician	Y	Y	N	Y	Y	Y	18		**Y**
0083	M	63	Sleep Physician	Y	Y	N	Y	Y	Y	8	Exclude because he has diabetes, low ESS score	**N**
0085	M	78	Neurologist	Y	NA	Y- blood test	Y	N	N	16		**Y**
0086	F	60	Sleep Physician	Y	NA	N	N	N	N	22	Excluded because she has comorbid sleep apnea & depression, no MSLT and no auxiliary symptoms	**N**
0087	F	58	Sleep Physician	Y	Y	N	N	N	Missing data	22		**Y**
0088	F	76	Neurologist	Y	Y	Y – EEG, MRI	Y	Y	Missing data	18		**Y**
0089	F	85	Neurologist	Y	NA	N	Y	Y	Y	11	Diagnosed prior MSLT introduction	**Y**
0090	F	28	Neurologist	Y	Y	Y- blood test	Y	Y	Y	22		**Y**
0094	F	67	Neurologist	Y	Y	N	Y	Y	Y	21	Irritable Bowel Syndrome	**Y**
0095	F	61	Sleep Physician	Y	Y	N	N	Y	Y	24		**Y**

ID	Sex	Age in years	Narcolepsy diagnosed by...	Currently taking medication for narcolepsy/ cataplexy	Multiple Sleep Latency Test that indicated narcolepsy	Other tests that indicated narcolepsy	Cataplexy	Sleep paralysis	Hallucinations	ESS: not taking stimulant medication	Other information or comments	Included in sample
0097	M	55	Sleep Physician	Y	Y	N	N	Y	N	11		**Y**
0098	F	53	Neurologist	Y	Y	Y- not specified	Y	Y	Y	22		**Y**
00106	M	74	General Practitioner	Y	Y	N	Y	N	Y	19	Excluded because he has diabetes	**N**
00107	M	85	Neurologist	Y	NA	N	Y	Y	Y	20		**Y**
00108	F	Missing data	immunologist	Y	Y	N	N	Y	N	14		**Y**
00110	F	54	Sleep Physician	Y	Y	N	Y	Y	Y	24		**Y**
00111	M	79	Neurologist	Y	NA	N	N	N	N	24	Excluded because he has comorbid lopus and depression	**N**
00112	M	30	Neurologist	Y	NA	Y - EGG	Y	Y	Y	23		**Y**
00116	F	62	Sleep Physician	Y	Y	Y – blood test	Y	Y	N	24		**Y**
00118	F	26	Neurologist	Y	Y	Y - MRI	Y	Y	Y	21		**Y**
00120	F	68	Psychiatrist	Y	Y	N	Y	Y	Y	18	Excluded because has diabetes	**N**
00124	F	27	Sleep Physician	N	Y	N	N	Y	Y	16		**Y**
00129	F	35	Neurologist	Y	Y	Y – not specified	Y	Y	Y	24		**Y**
00132	M	60	Neurologist	Y	Y	N	Y	Y	N	24		**Y**
00135	M	56	Psychiatrist	Y	Y	Y – EGG, blood test	Y	Y	N	14		**Y**

ID	Sex	Age in years	Narcolepsy diagnosed by...	Currently taking medication for narcolepsy/ cataplexy	Multiple Sleep Latency Test that indicated narcolepsy	Other tests that indicated narcolepsy	Cataplexy	Sleep paralysis	Hallucinations	ESS: not taking stimulant medication	Other information or comments	Included in sample
00137	F	43	Psychologist	Y	Y	N	Y	N	N	15		**Y**
00139	M	57	Sleep Physician	N	Y	N	N	N	N	23		**Y**
00141	F	Missing data	Sleep Physician	Y	Y	N	N	Y	N	19		**Y**
00142	M	29	Sleep Physician	Y	Y	N	Y	Y	Y	19	Excluded because she has diabetes	**N**
00143	F	40	Neurologist	N	NA	Y- CSF, blood test	Y	Y	Y	18		**Y**
00144	M	43	Neurologist	Y	Y	Y – blood test, MRI	Y	N	N	18		**Y**
00146	F	40	Sleep Physician Neurologist	Y	Y	Y – blood test	N	Y	N	22		**Y**
00150	F	53	Sleep Physician	Y	Y	N	N	N	Y	21		**Y**
00159	F	78	Neurologist	Y	NA	N	Y	Y	Y	24		**Y**
00160	F	60	Neurologist	Y	Y	Y- HLA, CSF	Y	Y	Y	22		**Y**
00161	F	69	Neurologist	Y	NA	N	Y	Y	Y	19		**Y**
00162	F	46	Sleep Physician	N	NA	N	N	N	Y	15	Excluded because MSLT was negative, low sleepiness, no ctaplexy	**N**
00168	F	77	General Practitioner	Y	Y	Y- EGG	Y	Y	Y	22		**Y**
00169	M	62	General Practitioner	Y	Y	N	Y	Y	Y	16		**Y**
00173	F	29	Neurologist	Y	Y	N	Y	N	Y	16		**Y**
00174	F	72	Neurologist	Y	Y	Y- EGG	Y	Y	Y	22		**Y**
00175	F	69	Neurologist	Y	Y	N	Y	Y	Y	21		**Y**

ID	Sex	Age in years	Narcolepsy diagnosed by...	Currently taking medication for narcolepsy/ cataplexy	Multiple Sleep Latency Test that indicated narcolepsy	Other tests that indicated narcolepsy	Cataplexy	Sleep paralysis	Hallucinations	ESS: not taking stimulant medication	Other information or comments	Included in sample
00176	F	Missing data	Neurologist	N	Y	N	N	N	N	21		**Y**
00177	F	67	Neurologist	Y	Y	N	Y	N	N	13	Excluded because has diabetes	**N**
00178	F	40	Sleep Physician Neurologist	Y	Y	N	Y	Y	Y	22		**Y**
00179	F	47	Sleep Physician	Y	Y	N	Y	Y	N	16		**Y**
00302	F	73	Neurologist	Y	Y	N	Y	Y	Y	19		**Y**
00308	F	68	Neurologist	Y	Y	Y	Y	Y	N	22		**Y**
00309	F	62	Sleep Physician	Y	Y	N	N	Y	N	7	Excluded because ESS score is low and has a diagnosis of sleep apnea	**N**