The Emotional and Cognitive processes in Obesity

By Iraida Neira [Iraida Neira Miller]

A thesis submitted to

The University of Birmingham

For the degree of

DOCTOR OF PHILOSOPHY

The University of Birmingham

School of Psychology

September 2014

ABSTRACT

This thesis examined the relationship between emotional and cognitive function in obesity. The results from Chapter 2 demonstrated that anxiety and depression exacerbate negative perceptions of quality of life in the severely obese. In Chapter 3 it is reported that Alexithymia, a form of emotional dysfunction, may contribute towards poor lifestyle choices such as unhealthy snacking, less exercise, and lower fruit and vegetable consumption in adolescents. These findings are of particular interest because they suggest one way in which the obesity cycle may begin in youth. Chapter 4 presents the results study which found that the relationship between BMI and cognitive tasks is not as straightforward as suggested in previous literature. When controlling for factors such as impulsivity and self-esteem, many BMI-cognition relationships disappeared. However, a relationship between memory function and BMI was observed that was moderated by age. The studies reported in Chapter 5, investigated individual difference in food memory. Positive food preoccupation was found to influence both food memory and later snack intake. Overall, this thesis reports novel findings that add to the literature documenting emotional and cognitive problems related to obesity.

DEDICATION

I would like to dedicate this thesis to my father Joseph C. Neira. You are in my heart and in my thoughts every day. Your love and belief in me has made all my dreams possible. I will always remember you, and dedicate every achievement to you.

ACKNOWLEDGMENTS

My supervisor Dr. Suzanne Higgs has provided me guidance, advice, and inspiration throughout my PhD. She has encouraged my growth as a researcher and motivated me to overcome obstacles both academic and personal. The completion of my thesis has been immensely aided by her on-going support and ingenuity. My second supervisor Dr. Shahrad Taheri enabled me to work in a clinical setting, which has been a rewarding experience. The time he took to introduce me into the research hospital environment was much appreciated. I would also like to acknowledge the support from my friends and family. Dr. Anna Fogel, who has helped me grow as a teacher which in turn made me better at my work. Dr. Sarah Santos-Murphy who has given me advice during the write-up process and has been so helpful- academically and otherwise! To my family, even though you are far away you always let me know how clever you think I am. Thanks! I would also like to acknowledge Hugh Miller, who has gone above and beyond to make writing this thesis as painless as possible. From early morning cups of tea to (like my family) telling me how clever he thinks I am. I'm noticing a flattering trend… I love you, Hugh! But whose idea was it to submit my thesis 5 days before our wedding?!

DISSEMINATION

The contents of this thesis have been presented at conferences for The British Feeding and Drinking Group (2010-2013), The Society for the Study of Ingestive Behaviour (2012), and The Association for the Study of Obesity (2012).

CONTENTS

TABLES AND FIGURES

CHAPTER 2

CHAPTER 3

CHAPTER 4

CHAPTER 5

CHAPTER 1: INTRODUCTION

1.1 Obesity and its causes

The Health and Social Care Information (HSCI) centre recently published national statistics reporting that 24% of men, 25% of women, and about 14% of children were obese in the UK in 2012 (2014). Obesity is described as an excess of body weight and fat (HSCI 2014). It is usually measured via Body Mass Index (BMI), which is the division of an individual's weight by their height squared. A score of 25 or above is considered overweight and 30 or above is obese (HSCI 2014). The HSCI also reported obesity to be associated with health risks and financial implications for health care systems such as increased hospital visits, lower physical activity rates, higher blood pressure, and limiting long-standing diseases (HSCI 2014). But when did obesity become an issue? A National Health and Nutrition Survey conducted in the United States between 1960 and 1994 identified a significant rise in obesity over this time (Flegal, Caroll, Johnson 1994). Therefore, there is evidence that obesity rates have been rising over the past few decades and that obesity is associated with costs for both the individual and society.

There is evidence that variation in BMI within a population is genetically determined. A classic study on monozygotic and dizygotic male twins found that weight and BMI were highly correlated between identical twins, which supports the idea of a genetic influence on obesity (Stunkard, Foch, and Hrubec 1986. Furthermore, although associations have been found between adopted children's weights and their adoptive parent's weights, stronger correlations were found between biological children and their biological parents (Stunkard, Foch, Hrubec 1986). The specific genes involved in common obesity are unclear, but there is

some evidence that the fat mass and obesity associated (FTO) gene is related to a higher risk of human obesity (Fredriksson et al. 2008, Zabena et al. 2009). A study by Zabena and colleagues (2009), investigated the relationship between obesity and the rs9939609 FTO gene variant. They found that in a sample of 180 participants (75 obese), rs9939609 was associated with obesity, particularly when observing total body fat (in contrast to fat distribution). Scuteri and others (2007) conducted a study on over 4,000 Sardinians and also reported rs9939609 to be associated with higher BMI and body weight, as well as increased hip circumference. Another study on the FTO gene found that in children it predicted BMI, obesity, and adult obesity (Frayling et al, 2007). Furthermore, Wardle and colleagues (2008) reported an association between rs9939609 higher fat mass and decreased satiety in children. Studies such as these indicate that the FTO gene is involved in the development of obesity, although other specific genes remain to be identified.

Some researchers believe the current obesogenic environment may be facilitating weight gain. The obesogenic environment refers to both the food environment and the built environment. For example, it has been argued that the availability of affordable high fat foods, increased portion sizes and food marketing to children contribute to overeating and weight gain (Swinburn 1999, Swinburn et al. 2011, Swinburn 2009, Young and Nestle 2007). An interesting study dissecting what makes an obesogenic environment in rural areas found that obesity was related to distance from recreational areas, unpleasant community for physical activity, fear of crime or traffic, and less non-residential areas. Additionally, expected associations such as with consumption of high fat foods and sedentary lifestyle were seen (Boehmer, Lovegreen, Haire-Joshu, Brownson 2006). Interestingly, one study by Westerterp and Speakman (2008) reported that activity levels have not declined, while another paper by

Church and colleagues (2011) indicated that modern people are employed in more sedentary careers. Another study by Mobley and colleagues (2006) reported lower risk for CVD and higher BMI in women who lived in a maximum mixed land-use environment, had access to additional fitness facilities per 1000 residents, and were living in neighbourhoods with less crime. A review by Chaput, Klingenberg, Astrup, and Sjödin (2011) suggested that sedentariness was promoting overconsumption. Specifically, increased intake occurs during distracting behaviours such as watching television, playing video games, cognitive tasks, listening to music. These data suggest that the obesogenic environment may contribute to weight gain through increasing food intake and reducing physical activity.

The family environment might also play a role in the development of obesity. A study by Wardle and colleagues (2001) found that when comparing children from obese and normal weight families, children with obese parents had a preference for fatty foods, a sedentary lifestyle, lower vegetable liking, and were more likely to have an 'overeating' type of eating style. Another study on mothers of same-sex twins (Wardle et al. 2002), found that obese mothers were more likely to offer food as a reward to children when dealing with distressing situations. Obese mothers were also less likely to monitor children's food intake than lean mothers. However, in these studies it is difficult to disentangle genetic and environmental influences.

An important issue is that not all individuals who experience the obesogenic environment become obese, which suggests that an interaction between genetics and the environment is important in the development of obesity. Indeed, it has been suggested that obesity may be due to a genetic predisposition expressed in a particular environment (Loos and Bouchard

2003; Blakemore and Buxton 2014). A twin study on over 5,000 children in the UK was conducted with the aim of observing how genetic and environmental factors influence BMI and central obesity (Wardle, Carnell, Haworth, and Plomin 2008). Use of quantitative genetic modelling supported that both BMI and waist circumference were hereditary, while environmental effects were much smaller. A study by Ravussin and Borgadus (2007) studied genetic and environmental influences on obesity within a Pima Indian population. They reported that 40% of the variability of BMI was due to genetic factors involved in food intake and physical activity leaving the remainder as non-genetic factors. A longitudinal study on over 7,000 twins indicated that as children age, the genetic influence on BMI increases (Haworth et al. 2008). They further suggested that as children age they may gravitate towards environments which nurture these genetic preferences. A systematic review of twin and adoption studies investigating obesity up to age 18 was conducted by Silventoinen, Rokholm, Kaprio, and Sørensen (2009). They reported that genetics had a significant effect on BMI across ages. Environmental factors were more influential in childhood, and less so as children aged. Therefore, although it is clear that gene-environment interactions are important in the development of obesity, they are complex and not yet fully understood.

1.2 Consequences of obesity

A literature search on ‘obesity’ prior to 1995 demonstrates a focus on the physical consequences of being overweight/obese. This led to studies and findings that emphasized concerns about mortality due to diseases such as heart disease and cancer. In one study, Peters and colleagues (1995) tested thousands of middle aged men across 7 European countries. They reported that that a gain of 7 kg of body weight was associated with an increased risk of death by heart disease. Central obesity in particular has been found to be a risk marker of

diseases such as CVD, diabetes, and dementia (Mcternan, Mcternan, Hart, Levick, Barnett, Kumar 2002; Whitmer, Gustafson,Barrett-Connor, Haan, Gunderson, Yaffe 2008; Kanaya et al. 2006). Young and Gelskey (1995) reported that individuals with non-central/overall obesity (in contrast to centrally obese) were also at risk for poor lipid profiles and glucose status, as well as high blood pressure. Weight-loss has been suggested as the preferred treatment to improve coronary disease in overweight middle-aged to older men (Katzel et al. 1995). However, weight cycling is associated with increased mortality (Peters et al. 1995) and weight variability (intentional or otherwise) has also been reported as a risk factor for disease (French, Jeffrey, Folsom, Williamson, Byers 1995). In addition, Flegal and colleagues (2013), have highlighted that overweight may not be associated with increased mortality. In fact, a systematic review indicated that overweight was associated with lower mortality risk (Flegal et al. (2013). Nevertheless, the evidence suggests that severe obesity is likely to be linked to higher mortality risk.

In has been suggested more recently that obesity is also linked to impairments in cognitive functioning (Volkow et al. 2008, Taki et al. 2008). Other evidence suggests that simply having some excess weight may also result in decreased cognitive abilities (Volkow et al. 2008). Cognitive deficits in obesity have been linked to poorer cardiovascular fitness (Fedor and Gunstad 2013), diabetes (Lasselin et al. 2012; Kaufmann, Pixner, Starke, Zotter, Köhle, Meraner, Kremser, Egger 2012), and depression (Bourke, Porter, Carter, Mcintosh, Jordan, Bell, Carter, Colhoun, Joyce 2012) which may lead to major impacts on quality of life (Cutajar, Ferriani, Scandellari, Sabattini, Trocino, Marchello and Stecchi 2000). Benito-Leon and colleagues (2013) conducted a study on a large sample (1,949) of elderly participants. When adjusting for age, gender, medication, diabetes (among other diseases), waist

circumference and education, overweight and obese participants performed the poorest cognitively, including in immediate logical memory. In one study, (Gunstad, Paul, Cohen, Tate, Spitznagel, Gordon 2007) found that along with obese individuals, overweight individuals also exhibited lower cognitive ability than individuals of normal weight. There are multiple potential causes of cognitive dysfunction, ranging from lifestyle choices, physical trauma, or even predisposition but these factors are poorly understood (Graveline 2004). Given the rising levels of overweight and obesity worldwide (McLellan 2002), it is important that that the relationship between accumulation of body fat and cognitive function is more clearly understood. Investigating the potential factors contributing to the effects of overweight and obesity on cognition are necessary for effective treatment and prevention of cognitive deficits in obesity. This will facilitate understanding of the implications for both individuals and a society with increasing waistlines. Greater reflection on this relationship may also have implications for understanding other neurological disorders such as dementia. This is significant given that over a third of women and just under a quarter of men are estimated to eventually develop dementia (Yaffe 2008). In this review, the relationship between obesity and cognition, along with some of the possible mechanisms, will be discussed.

1.2.2 Body Mass Index (BMI) and Cognition across a lifetime

One common perception of cognitive decline is that it is a natural part of the life cycle and an inevitable consequence of ageing. However, research suggests that a person's weight may play a bigger role than expected in cognitive functioning as people age. Although cognitive dysfunction generally increases with age, significant contributing factors to cognitive decline may be conditions, such as obesity. An interesting study (Galioto, Alosco, Spitznagel, Stanek, and Gunstad 2013) investigated how cognitive reserve, or in this case pre-morbid intelligence, may affect post-morbid cognition. They found that cognitive reserve was in fact related to

how cognitive impairment progresses after obesity. This means that if obesity is causing cognitive impairments, an individual's cognitive ability prior to obesity may significantly influence how obesity affects them. Lasikiewicz, Hendrickx, Talbot, and Dye (2013) investigated cognitive impairment (using the CANTAB test battery) during post-stress situations in centrally obese individuals. They found that centrally obese participants showed cognitive decline, specifically poorer declarative memory performance, after feeling stressed. However, findings in this area have varied; A systematic review by Fitzpatrick, Gilbert, and Serpell (2013) on overweight, obesity, and impairment on behavioural tasks of executive functioning, reported that the obese had more difficulties in decision-making, planning, and problem-solving than normal weight individuals; with fewer difficulties on other tasks such as memory.

Cross sectional studies

Cross-sectional studies have proven useful in obesity research, as they allow comparisons to be made across different groups such as genders, ages, ethnicities, BMI categories. Although they facilitate access to a diverse sample, it should be kept in mind that conclusions are being made based on between-subject data. In a study on an elderly population, obese individuals with metabolic syndrome were found to have higher levels of cognitive decline (Yaffe 2007). In an older out-patient group diagnosed with type II diabetes, central obesity was found to be related to lower cognitive function (Kim et al. 2008). In another cross-sectional study, cognition was evaluated in the elderly, comparing overweight, obese, and lean participants (Benito-Leon, Mitchell, Hernandez-Gallego, and Bermejo-Pareja 2013). They found that overweight and obese participants performed more poorly on neuropsychological tests than their lean counterparts. In a study investigating obesity and Alzheimer's disease (AD), 50 participants who likely had AD were observed. The results supported the claim that AD may

be associated with obesity (Razay, Vreugdenhil, and Wilcock 2006).

Other evidence suggests that there is a link between cognitive function and body weight not only as people age but also during earlier adulthood. In a large study examining healthy adults aged 20 to 82 years, individuals with higher BMIs were found to perform more poorly on cognitive tests than individuals with lower BMIs regardless of age group (Gunstad et al. 2007). A cross-sectional study on 5 and 6 year old German children revealed an association between sleep and BMI (Von Kries, Toschke, Wurmser, Sauerwald, Koletzko 2002) and reported the prevalence of obesity decreased as sleep duration (which may be linked to cognition) increased. Jensen and Friedmann (2002) found that although men with a BMI over 35 displayed a higher risk of functional decline than women, women with a BMI over 40 were at higher risk than men. However, a study on the effects of BMI on cognition by Yesavage and colleagues (2014), did not find this link. Using a sample of 369 individuals aged 50 or older, they assessed cognitive areas such as verbal memory and executive function. They found cognitive deficits to be linked to cardiovascular disease (CVD), but not obesity. This highlights the importance of considering other factors and comorbidities during obesity-cognition research.

Longitudinal studies

Longitudinal studies provide the benefit of following the same subjects for more accurate and long term observation. However, the commitment required by a participant for this long period may lead to high dropout rates. Because participants who keep in contact until the end of a longitudinal study may be more eager or motivated than a typical participant, bias might also be introduced into the study sample. A longitudinal study by The Lothian Birth Cohort of 1936 included IQ data from when participants were 11 years of age, which was compared to

the same IQ measures at ~70 years of age (Corley, Gow, Starr, Deary 2010). It was reported that BMI-cognition relationships in later life could be largely explained by previous cognitive ability and socioeconomic status. This supports previous research on how factors such as pre-morbid cognitive ability may affect cognition after obesity. Additionally it indicates that other factors such as education and income may be related to obesity and cognition. In a study of nearly 11,000 people across a period of 27 years, obese participants were found to have a 74% increased risk of dementia, while overweight participants had a 35% increased risk, when compared with individuals of normal weight (Whitmer et al. 2005). Additionally, these authors suggested that regardless of comorbid conditions, obesity in middle age places an individual at higher risk for dementia.

The Framingham Heart study was run across a period of 18 years, and measures of obesity, hypertension, and cognitive function in healthy men and women were taken. Cognition was measured by assessing functions like memory, learning, executive function, and reasoning. Obesity and hypertension were shown to have negative effects on cognitive function, but only in men (Elias et al. 2003). In the Whitehall II Cohort Study, another long term observation of a population was conducted (Sabia et al. 2008). Cognitive function was assessed by tests of global cognition, short term verbal memory, and executive function. The individuals were assessed during early adulthood, early midlife, and late midlife. It was reported that sustained obesity is related with lower cognitive function in late midlife. Interestingly, being obese at 2-3 different periods of your life was also linked to lower cognitive function. Additionally, a significant increase in BMI during the time period observed resulted in lower executive function (Sabia et al. 2008). This research indicates that a person may potentially be able to influence their own level of mental functioning by maintaining a healthy weight throughout

their life.

Laboratory animal studies

Animal research has become an important part in developing our understanding of obesity in humans. Winocur and colleagues (2005) published a study comparing obese and lean Zucker rats, in which rats undertook variable-interval delayed alternation tests of learning and memory. They found that obese rats were impaired at hippocampal dependent (lengthier) intervals. Winocur and Greenwood (2005) also reported on how rats fed a high fat diet for 3 months were impaired when it came to learning and memory. Rats fed a high calorie diet had decreased ability in discrimination reversal learning and reduced levels of and brain derived neurotrophic factor (BDNF), which is important for neuron growth in the prefrontal cortex and hippocampus. Rats that consumed a high fat diet were also more responsive to reward signals at the start of the discrimination reversal task (Kanoski, Meisel, Mullins, and Davidson 2007). Another study using high fat diet (HFD) obesity induced mice, investigated how HFD affected stress and metabolic hormones, learning, and the hippocampus (Hwang et al. 2009). Obese male rats were seen to gain more weight, and developed more health complications such as hyperglycaemia. They also showed poorer learning performance, and hippocampal synaptic plasticity than normal weight mice. Overall, the study suggested obese male rats were more vulnerable to HFD consequences than obese female and normal weight counterparts. Maternal obesity in rats has also been seen to damage hippocampal BDNF in offspring (Tozuka et al. 2010). In fact, many of the studies on human obesity may have been supported due to evidence found in animal studies. The same links between obesity and diabetes (Xu et al. 2003), memory (Takeda et al. 2010), and depression (Collins, Daniels, Rohlfs 1999) have been found in animals although whether these effects are specific to the

obese state is unclear, because in some cases the effects of consuming a high fat diet on consumption are independent of their effects on body weight gain and obesity (Kanoski 2012).

Cognitive impairment as a consequence or cause of obesity?

A significant point of interest is which condition might be preceding the other; obesity or cognitive impairment? There are studies which contend high fat diet is related to cognitive impairment (Francis and Stevenson 2011); hippocampal deficits have been correlated with high fat diets (Lindqvist et al 2006), which implies that deficits in memory related to diet may precede obesity. Hsu and Kanoski presented a review of dementia and obesity research in which they argued that these increasingly common diseases may be induced by the western diet (2014). They contend that the Western diet, which is high in saturated fat, impairs the hippocampus which increases the chances of both obesity and AD. Kanazawa (2014) suggest that although previous cross-sectional studies have implied that obesity precedes cognitive decline, longitudinal studies are more suited to the question, and indicate that poor cognition comes first. If this is the case, practically, it would mean that lower cognitive function may lead to poorer control of food intake, which in turn results in excess body weight. Belsky and colleagues (2013) conducted a longitudinal study on over 1,000 participants from childhood to adulthood. They reported that although obesity was associated with cognitive decline, there was no indication it was the initial cause of lower intelligence (measured by the Wechsler Intelligence Scale for Children-Revised [Castro, Delfries, Fulker 1995] and Wechsler Adult Intelligence Scale-IV [Hartman 2009]). More plainly, obese individuals appeared to have a lower IQ from childhood (prior to becoming obese). Although the initial cause is unclear, there does appear to be a relationship between cognition and obesity. This links back to the

idea Davidson and colleagues (2005) suggested that diets high in saturated fat impair hippocampal function, degrade the ability to monitor (remember) food intake accurately, and result in higher body mass.

1.3.1 BMI and specific cognitive functions

While various studies have highlighted a relationship between obesity, generally cognitive abilities and dementia there is also now a growing literature documenting relationships between obesity and specific cognitive function that are underpinned by activity in specific neural networks. This research is uncovering specific patters of cognitive impairments associated with overweight and obesity.

1.3.2 Frontal Lobe (Executive functions)

The frontal lobe is one of the four main regions of the cerebral cortex and is located in the anterior of the brain. It is made up of three parts; one of which is the prefrontal cortex (PFC). Frontal lobe function is thought to be significant in spatial planning and working memory tasks, along with decision-making and attention (Smith and Jonides 1999, Chayer and Freedman 2002). Damage to the frontal lobe has been seen to result in impaired spatial working memory capacity (Owen et al. 1990). Kurth and colleagues (2013) investigated whether BMI and waist circumference were correlated with brain volume. They found that in a group of 115 healthy adults, BMI and waist circumference were significantly negatively correlated with gray matter in certain areas of the brain, including the prefrontal cortex. The effects were much more pronounced for waist circumference, especially in females, highlighting that BMI may not always be the most useful measure of obesity. Through magnetic resonance imaging (MRI) Shimoji and colleagues (2013) conducted a whole-brain

analysis of 14 middle aged men, half of which had metabolic syndrome. They found that subjects with metabolic syndrome had significantly lower fractional anisotrophy (FA) values in several brain areas, including the deep white matter in the frontal lobe. Volkow et al. (2008) tested a group of healthy subjects and conducted neuropsychological evaluations and brain scans. A negative correlation was found between BMI and metabolic activity in the prefrontal cortex and cingulate gyrus (Volkow et al. 2008) and it was argued that this may explain the poorer executive function reported in healthy obese populations. A larger study involved data collection from 292 obese and normal weight individuals twice over the course of 5 years (Brooks et al. 2013). Obese individuals had significantly reduced total gray matter in bilateral dorsolateral prefrontal cortex (DLPFC), which is believed to be essential in decision making (Bechara, Tranel, Damasio 2000) and social conduct and emotional processing (Tranel, Bechara, Denburg 2002). Obese participants also had significantly slower reactions times than normal weight participants in a trail-making task related to DLPFC function (Brooks et al. 2013).

The suggestion that overweight may cause damage in the frontal lobe region is worth noting, since similar damage has been found in individuals suffering from addiction (Bechara et al. 2001). Some behaviours such as impulsivity, binging, and emotional eating are common to both obesity and addiction, and this may relate to a common deficit in frontal lobe function. In a review of the literature Blumenthal and Gold (2010) suggested that dependence on food and drugs may work along similar neural networks. Additionally, Volkow, Wang, Tomasi, and Baler (2013) recently made the argument that the dopamine (DA) system links drug addiction and obesity and this may relate to activity in frontal-striatal circuits. Interestingly, a study on individuals with ventromedial prefrontal cortex damage revealed that following this damage,

insensitivity to future consequences was developed *(*Bechara, Damásio, A. R., Damásio, H., Anderson, 1994*).* It is possible that individuals prone to drug addiction and obesity both share a common feature which is that they are more influenced by immediate rather than delayed consequences of their behavior and discount the future health consequences of overeating and drug taking.

1.3.3 Frontal-Striatal areas (Inhibition and reward)

Fronto-striatal circuits comprise the basal ganglia, which are connected to the prefrontal cortex, and loops to the thalamus (Alexander, DeLong, Strick 1986). Impulsivity has been associated with frontal-striatal brain function and obesity (Dalley, Mar, Economidou, Robbins 2008; Nederkoorn et al. 2007). Impulsivity has been linked to obesity in several studies (Sarisoy, Atmaca, Ecemis, Gumus, Pazvantoglu 2013; Thamotharan, Lange, Zale, Huffhines, Fields 2013; Mobb, Crépin, Thiéry, Golay, Linden 2010; Schag, Schönleber, Teufel, Zipfel, Giel 2013). Studies have specifically been able to link overeating and overweight/obesity to impulsivity when eating, potentially due to dulled responses to food (Davis, Strachan, Berksin 2004; Stice, Spoor, Bohon, Small 2008; Burger and Stice 2014). This may parallel the relationship between drug addicts and addictive substances. In a study which observed impulsivity in overweight/obese and non-obese participants, overweight/obese individuals were seen to have higher levels of specific types of impulsivity (Mobb et al. 2010); these were 'urgency, lack of perseverance, and sensitivity to reward'. In a study examining impulsivity towards food, (Schag et al. 2013), they observed that obese individuals with binge eating disorder (BED) in particular, demonstrated heightened food-related impulsivity. Thamotharan, et al. (2013) found that even overweight/obese children tended to be more impulsive than their normal weight counterparts. A study by Guerrieri et al. (2009) primed impulsive

responding and found that food intake increased significantly in the impulsivity group, and even more so for highly restrained participants.

It has been suggested that an exaggerated reward response to food may underlie difficulties in resisting the temptations of palatable foods cues (impulsivity) and that this may lead to over eating and obesity (Davis et al. 2004). In line with idea, many studies have shown that obese participants have greater activity in areas such as the striatum when viewing food cues (for a recent review see Burger and Bener 2014). However, on the other hand, it has also been shown that obese individuals also show reduced activity in the striatum when actually tasting palatable foods (Stice et al. 2008).

In a study investigating the association between brain dopamine and eating behaviour (Volkow et al. 2003), researchers found that eating behaviour (assessed by the Dutch Eating Behaviour Questions - DEBQ) was related to dopamine changes in the dorsal striatal area of the brain. They found higher restraint to be correlated with higher dopamine changes after food stimulation and higher emotional eating to be associated with lower D2 receptors. These data have been suggested to show that weight gain maybe due to low levels of experienced pleasure due to reduced dopamine function triggering overeating to boost hedonic reactivity (Volkow et al. 2003). In support of this idea, Wang and colleagues (2001) found that obese participants had decreased availability of striatal dopamine D2 receptors relative to lean participants in a PET study. However, this result has not been widely replicated in humans, and it may be that obesity causes changes in dopamine receptor density rather than obesity resulting from reduced dopamine receptor density.

Dopamine reward-related responses may also depend upon factors such as binge eating status, even within an obese population. For example, Balodis and colleagues (2013) compared an obese group seeking treating for BED with non-BED obese participants and normal weight participants during a monetary reward/loss task. Compared to the non-obese group, the non-BED obese group had increased ventral striatal and ventromedial prefrontal cortex activity during phases involving anticipation. When comparing them to the non-BED obese group, the BED obese group had decreased bilateral ventral striatal activity during this phase. There were no ventral striatal differences between the BED obese and normal weight group. Therefore, while there is evidence that formal striatal function is altered in obesity the exact mechanisms involved are complex and may relate to both over and under activity of frontal-striatal reward systems. For example, a heighted reward response to food cues may contribute to overeating but then continued consumption of high calorie foods may down regulate reward responses in obesity and contribute to the maintenance of overeating (Burger, and Stice, 2014).

1.3.4 The Medial Temporal Lobe area including Hippocampus (Memory)

The temporal lobe is another one of the four regions of the cerebral cortex. The medial temporal lobe is believed to contribute in human memory and new learning (Preston and Wagner 2013); so much so that even its width has been suggested as a good 'ante-mortem marker' for Alzheimer's disease (O'Brien et al. 2000). One study showed over a 40% decrease in hippocampal volume in Alzheimer's disease patients when compared to non-Alzheimer's individuals (Scheltens et al. 1992). Visser et al. (2002) conducted a study on patients over 50 years of age with minor cognitive impairment. Medial temporal lobe atrophy was seen to be associated with cognitive decline, and hippocampal volume along with overall medial

temporal lobe atrophy was associated with Alzheimer's type dementia at follow up.

Obesity has been linked to changes in medial temporal lobes. A large study on over 1,400 Japanese participants investigated the relationship between BMI and gray matter volume. In men, increased BMI was seen to be related with overall loss of gray matter, including volume of the medial temporal lobes (Taki et al. 2008). One way in which impaired cognition may cause obesity is through hippocampal deficits, and associated poor memory. There is some evidence that obesity is specially associated with poor memory performance (Lasikiewicz, Hendrickx, Talbot, and Dye 2013; Benito-Leon et al. 2013) and hippocampal damage is associated with reduced satiety (Higgs et al. 2008). Hsu and Kanoski and Davidson and colleagues (2005) have proposed that diets high in saturated fat impair memory, inhibit accurate control over food intake, and result in excess adiposity. There may be a specific role for memory of recent food intake, which has been found to contribute towards overeating (Higgs, 2002). The limited research on food memory indicates that better memory for recent eating behaviour reduces subsequent snacking (Higgs and Donohoe 2011), while poorer memory for meals increases later snack intake (Higgs et al. 2008; Higgs and Woodward 2009; Oldham-Cooper, Hardman, Nicoll, Rogers, Brunstrom 2011; Mittal, Stevenson, Oaten, Miller 2011; Oldham-Cooper et al. 2011).

Other data suggest enhanced reactivity of hippocampus in obesity using fMRI. Some studies have found that obese participants show greater activation in hippocampus in response to viewing of food images. In a study on obese and non-obese adolescents, Wallner-Liebmann and colleagues (2010) found a positive correlation between hippocampal activation after viewing high-caloric food images. In a study analyzing fMRI scans and reaction to food

images (Brooks, Cedernaes, Scioth 2013), obese participants were found to have increased activation in the right parahippocampal gyrus (in addition to other areas such as the left dorsomedial prefrontal cortex) in response to food images when compared to normal weight participants. It may be that activation of the hippocampus in these tasks reflect non-memorial functions of the hippocampus but clearly it would be of interest to explore further the relationship between hippocampal function BMI and obesity to resolve the data.

In summary, there are many indications in the literature that a relationship exists between specific cognitive abilities and obesity. Deficits in executive function, including planning and behavioural inhibition, are commonly reported. There are also problems with memory although this is an understudied area. In addition, the extent to which the reported cognitive deficits are accounted for other factors that associate with obesity such as depression and anxiety is unclear because these factors are not always taken into account. Therefore, further consideration of the specific mechanisms that underlie the relationship between obesity and cognitive function are warranted.

1.4.1 Underlying mechanisms

The precise mechanisms by which obesity may affect cognitive performance are not fully known and there are a number of potential candidates. A review on obesity and the central nervous system stated that the literature suggested that the interaction of obesity and aging may impair cognition and exacerbate the effects of age-related cognitive diseases (Bruce-Keller, Keller, Morrison 2009). This may tie in with the previously presented idea that it is not simply adiposity that is linked to cognitive defects, but more complex processes that are

comorbid with severe obesity.

1.4.2 Co-morbidities

Diabetes and insulin resistance

McNeilly et al. (2011) found that in rats undergoing high fat feeding, insulin sensitivity correlated with negative performance on some cognitive tasks. A review of rat models by Biessels and Gispen (2005) point out that results of investigations on type 2 diabetes and cognition have been varied, and conclusions are difficult to determine. When looking at humans, another study used functional magnetic resonance imaging (fMRI) to compare normal, overweight, and obese individuals during working memory tasks (Gonzales et al. 2010). As expected, the obese group exhibited much lower task-related activation in the right parietal cortex. Interestingly, when they examined insulin sensitivity as a mediator between BMI and brain activation, they found that it was greatly associated. These findings suggest that one of the mechanisms behind brain atrophy in obesity can be found in the long established relationship between CNS function, obesity, and insulin. Greenwood and Winocur (2005) showed older adults with insulin resistance exhibited similar impairments and responses, indicating insulin resistance as a mediator for diet related cognitive decline. A systematic review by van den Berg and colleagues (2009), reviewed cross-sectional and longitudinal studies on vascular diseases and cognition. They used non-demented participants, and controlled for age, gender, and education. They reported a positive relationship between diabetes and hypertension with cognitive deficits, but a weaker relationship with obesity itself. This brings into question what is causing these impairments, especially when cognitive decline has still been found in individuals without comorbidities such as diabetes.

Sleep disruption

Studies have reported that sleep deprivation may degrade cognitive ability, such as decision-making, attention, and working memory (Alhola and Polo-Kantola 2007; Killgore 2010). A decrease in sleep duration and quality has also been linked to obesity (Beccuti and Pannain 2011). This is potentially explained by a decrease in leptin and increases of ghrelin and cortisol levels, which results in increased appetite (Beccuti and Pannain 2011) and increased BMI (Taheri, Lin, Austin, Young, Mignot 2004). Wang and colleagues (2013) also found cognitive impairments (executive function and memory) in obese sufferers of sleep apnea (SA), but reported that this relationship was independent of SA (Wang, Chen, Peng, Zhang, Shen, Li, Han, Liu 2013). McDonald and colleagues (2013) reported that when individuals experienced disruptions in the circadian sleep rhythm, they also suffered reductions in context learning and memory processing ability. On the other hand, Lucassen and colleagues (2014) reported sleep extension as a way to improve cognition in sleep deprived obese individuals. A cohort study on 121 short-sleeping (less than 6 and half hours per night) obese participants, found that at baseline participants suffered from impairments in memory, motor skills, and executive function. Upon improving sleep duration (reported average by 11%); further improvements were seen in global cognition, memory, executive function, and attention.

Even in children, short-sleeping is found to increase the chances of future weight gain and obesity (Patel and Hu 2012). It has been suggested that poor sleep may not only encourage obesity, it may contribute towards cognitive decline within obesity (Hannon, Rofey, Ryan, Clapper, Chakravorty, Arslanian 2012). Another study on morbidly obese children with sleep apnea (which reduces sleep) reported that impairments in memory, learning, attention, and

vocabulary were found (Rhodes, Shimoda, Waid, O'neil, Oexmann, Collop, Willi 1995).

It has been further suggested that sleep deprivation may affect cognition through impairment of emotional processing ability (Killgore 2010). A within participants study compared emotional intelligence (EI) task performance pre and post sleep deprivation (Killgore, Kahn-Greene, Lapizzi, Newman, Kamimori, Balkin 2008); they found that after sleep deprivation participants had lower overall EI, lower interpersonal (such as assertiveness/independence) and intrapersonal (such as empathy) scores, poorer stress management ability, and poorer coping ability (such as less positive thinking). This is interesting because it suggests another mechanism by which poor sleep may contribute to both cognitive problems and weight gain via alterations in emotional processing, which underlines the tight links between these processes.

1.4.3 Obesity-related changes in brain structure

Bruce-Keller, Keller, and Morrison (2008) argued that obesity may impair brain structure and hence increase the risk of diseases, such as Alzheimer's disease. As previously reviewed in this thesis, there is evidence of brain structure changes in obesity including decreases in prefrontal cortex and hippocampal grey matter (e.g. Brooks et al. 2012). Taki et al. (2008), examined brain images for over 1,400 people, along with their heights and weights. Interestingly, a significant negative correlation between BMI and the volume of grey matter was found in men. However, the extent to which these effects are specific to increases in fat mass has yet to be thoroughly investigated and one study on 76 individuals with a range of adiposity found relationships between Fat free mass index (FFMI) and temporal and medial orbitofrontal grey matter volume that were independent of adiposity (Weise, Thiyyagura,

Reiman, Chen, Krakoff 2013). When adjusting for fat free mass (FFM) no relationship was observed between adiposity and grey matter volume, suggesting that is it important to take into account body composition.

Mediators of changes in brain structure and function

Decreases in grey matter may suggest that obese individuals, similarly to older individuals, are at higher risk for cognitive decline. As mentioned previously, the central nervous system may be damaged by obesity. Miller et al. (2009), ran a study on participants with Prader-Willi syndrome and early-onset obesity. Their findings led them to conclude that early childhood obesity may hinder cognition and the development of the cerebellum. Patterns set in early-life are known to leave lasting behavioural impression and the results of this study imply there may also be physical ones.

Ahima (2009) reported that experiments on mice indicate that obesity may affect brain structure and function by causing telomere shortening. Higher BMI in humans has also been associated with advanced telomere shortening (Rode, Nordestgaard, Weischer, Bojesen 2014). Telomeres are stretches of DNA at the end of chromosomes that shorten with age (Houben, Moonen, Schooten, Hageman 2008). Accelerated aging is also associated with vascular events such as cardiovascular disease (Mainous and Diaz 2010) and diabetes (Tamura et al. 2014). A systematic review on telomere length and vascular disease (Butt et al. 2010) concluded that there were consistent associations between telomere shortening and heart disease. They specifically listed risk factors were obesity, diabetes, sex, gender, and age. Interestingly, telomere shortening has also been linked to mood disorders; with one study reporting as much as 10 years accelerated aging on those diagnosed with mood problems (Simon, Smoller, McNamara, Maser, McNamara, Pollack, Nierenberg, Fava, and Wong

2006). Another study found that childhood adversity was related to telomere shortening in adults with anxiety disorder as well as non-anxious controls (Tirka, Price, Kao, Porton, Marsella, and Carpenter 2010). Elvsåshagen and colleagues (2011) reported that when comparing telomere length between Bipolar II Disorder (BD-II) individuals to healthy controls, shortened telomere loads was associated with number of depressive episodes in BD-II participants. One study found that women with the highest level of perceived stress had telomere shortening equivalent to that of 10 years compared to their less stressed counterparts (Epel, Blackburn, Lin, Dhabhar, Adler, Marrow, and Cawthon 2004). These data suggest that obesity and related psychological co-morbidities could result in accelerated brain aging and associated cognitive decline.

Oxidative stress and inflammation

Oxidative stress is defined as 'the disturbance in the balance between the production of reactive... free radicals and antioxidant defenses' (Betteridge 2000), and has been associated with cognitive decline (Barnham, Masters, Bush 2004), which may in part relate to deterioration of telomeres. A comprehensive study on obesity, oxidative stress, and accelerated aging (Tzanetakou, Katsilambros, Benetos, Mikhailidis, Perrea 2012) reported that shortened telomeres were associated with higher BMI, and that oxidative stress enhanced telomere deterioration. A study by Vincent, Innes, Vincent (2009) reported that obesity increased oxidative across three groups (young, old, and a clinical population). They also pointed out the oxidative stress could be elevated by advanced aging and is related to chronic disease in obesity. Bondia-Pons, Ryan, and Martinez (2012) investigated how oxidative stress, obesity, and inflammation interacted. They discussed how oxidative stress in obesity may be

associated with ‘low grade pro-inflammatory state’, which may further promote cardiovascular diseases. Exploring these ideas may be of use in understanding the obesity-oxidative stress relationship.

White and colleagues (2009) conducted a study in which rats were observed pre and postnatal after their dams were given a high fat diet (HFD) or control diet (CD). After weaning, the offspring were then separated into HFD and CD as well. At 20 weeks, the offspring were then placed in a Morris Maze to observe signs for oxidative stress and inflammation in the brain. When compared to rats on CD, rats on HFD demonstrated signs of oxidative stress, which was further exacerbated if they were born to dams consuming a HFD. This resulted in poorer retention of knowledge when completing the Morris Maze task. Morrison and colleagues (2010) also reported that HFD increased hippocampal oxidative stress and decreased cognition in older mice. Similar results were found by Pistell and colleagues (2010). Tucsek and colleagues (2014) compared brain function in young (7 months) and aged (24 months) HFD obese mice. Their findings suggested that aging exacerbated oxidative stress and neuroinflammation in the hippocampus that is caused by obesity. Ferrante (2007) wrote that obesity may produce a state of inflammation which can lead to diseases such as diabetes and heart disease. Wu and colleagues (2009) investigated the relationships between oxidative stress, fat mass, and inflammation, and suggested that inflammation caused by oxidative stress may be contributing towards obesity comorbidities. Regardless of the direction of causation, there appears to be evidence that oxidative stress and inflammation may play a role in obesity health complications, although most of this evidence is currently from animal models.

Appetite hormones

Physiological factors that are related to the accumulation of body fat such as the hormone leptin may contribute to brain changes associated with cognitive decline in obesity. Leptin is a hormone believed to assist in energy/body weight regulation (Friedman and Halaas 1998), and more recently, to be associated with obesity, inflammation, and cardiovascular disease (Martin, Qasim, Reilly 2008). A rat study by Scarpace and Zhang (2008), concluded that leptin resistance (more common in older animals) in particular, may promote obesity. A rat study by Lin and colleagues (2000) made an interesting outline of the stages of leptin levels as obesity sets in. They found that at the start of high fat dieting (HFD) mice remained sensitive to leptin, but that once they begin to increase their HFD intake, they developed leptin resistance. A review by Correia and Haynes (2004) confirms that there appears to be a relationship between leptin resistance, vascular inflammation, and oxidative stress. Kanoski and colleagues (2011), conducted an interesting study in which they investigated how increases in leptin to the hippocampus affected food intake and food memory. They found that ventral hippocampal leptin levels specifically could inhibit food memory and suppress food intake. The hormone ghrelin has also been associated with obesity and increasing ghrelin levels has been suggested as a method of reducing appetite (Patterson, Bloom, Gardiner 2011). It has been reported that ghrelin modulates hippocampal neurogenesis, and is even related to increased cognitive function in dwarf rats (Diano et al. 2006;Li, Kim, Kim, Park 2013). Although some cognitive links have been suggested, more research into human populations are required before firm conclusions can be drawn about the role of leptin ghrelin in cognitive function in obesity.

1.4.4 Other mechanisms/confounders

There is strong evidence that the relationship between obesity and cognition is underpinned by changes in brain structure and function that are due to accumulation of fat tissue and/or a high fat diet but it is also possible that some effects may be due to other factors known to affect cognition that associate with obesity. For example, changes in mood, specific attitudes towards food and eating traits such as dietary restraint (which is related to higher food intake; Mitchell and Brunstrom 2005). The next section will consider these factors in turn.

1.4.5 Eating traits

Overweight and obesity are associated with specific food-related attitudes and cognitions that may contribute to alterations in cognitive function (Oda-Montecinos, Saldana, Andres 2013). Questionnaires such as the Dutch Eating Behaviour Questionnaire (DEBQ) attempt to characterise eating patterns by dividing behaviours into 3 dimensions: restrained eating, external eating, and emotional eating (Van Strien et al. 1986). Various studies have documented relationships between restraint, external and emotional eating and obesity in both children and adults.

Obesity in children and adults has been associated variously with increased restraint (e.g. Herman and Polivy 1976; Shunk and Birch 2004), external eating (e.g. Burton, Smit, and Lightowler 2007) and emotional eating and tendency towards disinhibition (e.g. Ouwens, van Strien, and van der Staak 2003; Van Strien and Cleven, and Schippers 2000; Van Strien, Herman, Verheijden 2009) although other studies have reported that there is little evidence for the popular idea that prior restraint causes disinhibited eating and overweight (Johnson, Pratt, Wardle 2012). The relationships between eating style and obesity are complex and not fully

understood but it is clear that obesity is likely to correlate with eating styles and so it is important to consider whether differences in eating style may be related to altered cognitive function.

Dietary restraint has been associated with impairments in cognitive function (Green, Rogers, Elliman, Gatenby 1994). Even in children, restrained eating has been linked to poorer cognitive performance and longer reaction times in cognitive tasks (Brunstrom, Davison, Mitchell 2005). Silva, Pizzagalli, Larson, Jackson, Davidson (2002) conducted a study in which they found that chronic restrained eating was related to relative right frontal asymmetry. This relationship becomes very relevant in obesity-cognition research when considering that right frontal asymmetry has been linked to lack of empathy (Jones, Field, Davalos 2000), emotional disorders (Coan and Allen 2000), and emotional expression (Harmon-Jones 2003). In the study by Green et al. (1994) participants who were currently dieting exhibited poorer performance on a vigilance task, poorer memory, and longer reaction times when compared to non-dieting low to moderately restrained eaters. In a study on over 100 women, Westenhoefer and colleagues (2013) concluded that rigid restraint was related to 'preoccupying cognitions and attentional bias to food'. This indicates that one reason why restrained eaters may have reduced cognitive function is because of food preoccupation; Jones and Rogers (2003) suggest that dieters are being distracted by thoughts about food, and thereby have difficulty focusing on cognitive tasks.

1.4.6 Cognitive biases

Biases and focus of attention have also been associated with food intake (Soetens and Braet 2010; Vreugdenburg, Bryan, Kemps 2003). A study which supports the idea presented by

Jones and Rogers (2003), is by Tiggemann¸ Kemps, and Parnell (2010) which reported that chocolate cravings had a negative effect on visuospatial working memory ability in undergraduate women. Similar working memory deficits have been found in anorexics, who are thought to be preoccupied with dieting thoughts (Kemps, Tiggemann, Wade, Ben-Tovim, Breyer 2006**).** Current dieters have also been found to have poorer recall than non-dieters and scored higher on preoccupying thoughts (Kemps and Tiggemann 2005). Similarly Braet and Crombez (2003) found that when comparing non-obese to obese children, obese children experienced cognitive interference (intruding thoughts) when presented with food words. They suggested it may be due to an elevated sensitivity to food cues, which may underlie dysfunctional eating thoughts and behaviour (Braet and Crombez 2003). A study by Isreal, Stolmaker, and Adrian (2006) found that food related thoughts were more common among heavier individuals supporting the idea that cognitive function in obesity might also be related to the distracting effect of food preoccupations.

Restrained eaters have also been reported to be quicker to detect food words than unrestrained eaters, despite neutral word distracters included in the task (Hollitt, Kemps, Tiggemann, Smeets, Mills 2010). Hence, it is possible that preoccupation with food also leads to attentional biases towards food related cues, which may facilitate consumption (Werthmann et al. 2011). However, this effect may be moderated by dieting success. A study by Papies, Stroebe, and Aarts (2007) found that unsuccessful restrained eaters spontaneously activated hedonic food thoughts when presented with behaviour descriptions of tasty food items whereas successful dieters were more likely to activate dieting goals which protected against overconsumption.

Interestingly, food preoccupation may change with weight interventions. Patients who underwent gastric bypass surgery reported frequent food preoccupation when compared to

morbidly obese controls (Rand, Macgregor, Hankins 1987, Ogden, Clementi, Aylwin, Patel 2005). However, Edwards and colleagues (2005) conducted a study on family based behavioural treatment (FBBT) on 33 families with obese children. Twenty-seven percent of families completed the treated and those children lost an average of 8.4% of their BMI. Although there were improvements in depression and self-esteem scores, no change in food preoccupation was observed. Suggesting that food preoccupation may be a trait associated with a tendency to overweight.

The Food Preoccupation Questionnaire (FPQ) was developed by Tapper and Pothos (2010) to measure how frequently an individual experiences food thoughts, in addition to the emotional association those thoughts carry (positive, negative, neutral). This assessment of food preoccupation and emotional valence, may serve as a useful tool in identifying different kinds of 'food thinkers' and how this may relate to cognitive processing in obesity.

1.5 Obesity, Emotions and cognitive function

In a cross-sectional survey, Jorm et al. (2007) found that obesity is associated with both anxiety, depression, and emotional well-being. Much of the literature supports a link between overweight and anxiety and depression. A large telephone study by Strine et al. (2007) found that adults with depression or anxiety were more likely to be obese and physically inactive. Albert et al. (2002) examined depression and anxiety in abdominally obese middle aged men. When compared to non-obese age matched samples, abdominal obesity correlated highly with scales for anxiety and depression, more so than BMI alone. Anderson et al. (2007) also observed that female adolescent obesity predicted a larger risk for Major Depressive Disorder

and anxiety disorder. However, the role of depression in severe obesity is understudied and the direction of the relationship is unclear.

Depression and anxiety may contribute to the development of obesity and the maintenance of overeating. Masheb and Grilo (2005) observed that emotional overeating was related to binge eating and depression. Goossens, Braet, Van Vlierbergh, and Mels (2008) investigated loss of control when overeating in 188 adolescents aged 8 to 18. They found that emotional eating tended to mediate the relationship between anxiety and loss of control. Increased depression was related to emotional eating as well. They suggested that loss of control when eating is due to poor coping with negative emotions, such as anxiety. In a study by Slochower (1980), obese participants were seen to eat most when they felt anxious, in contrast to non-obese counterparts. Interestingly, a study by Ruderman (1983) found that obese individuals ate significantly less when 'highly' anxious than when 'mildly' anxious. This suggests that specific consideration of the type and extent of emotion is important understanding the role of emotions in overeating and obesity.

The relationship between obesity and emotions may be more complex than anticipated. For instance, alexithymia, a difficulty in emotional processing (Haviland, Warren, Riggs 2000), has also been associated with obesity (Clerici et al. 1992; Morosin and Riva 1997; de Zwaan et al. 1995; Zak-Golab et al 2013; Larsen, van Strien, Eisinga, Engels 2006; Fukunishi and Kaji 1997; Surcinelli et al. 2007; Elfhag and Lundh 2007). Alexithymia is also associated with depression and anxiety (Lumley, Ovies, Stettner, Wehmer, Lakey 1996; Bonnet, Brejard, Pasquier, Pedinielli 2012) and poor sleep quality (Gennaro et al. 2004). The association between alexithymia and obesity may be related to how emotions influence lifestyle habits like disordered eating behaviour (such as emotional eating or binge eating) or lack of exercise (Adami et al. 2001; Larsen, van Strien, Eisinga, Engels 2006; Pinaquy et al. 2003; Ouwens,

van Strien, van Leeuwe 2009; van Strien and Ouwens 2007; De Lenclave, Florequin, and Bailly 2001; Wheeler, Greiner, and Boulton 2005; Noli et al. 2010; Deborde et al. 2006; Nowakowski, McFarlane, and Cassin 2013; Sasai, Tanaka, Hishimoto 2010, Pinna et al. 2011; Pinaquy et al. 2003). These negative emotional states associated with alexithymia and obesity may relate to lower self-esteem and life satisfaction (Carano et al. 2006; Cochrane, Brewerton, Wilson, and Hodges 1993), and predict disordered eating (Laquatra and Clopton 1994).

Interestingly, the role of emotions on eating behaviour is not always in the negative valence spectrum. Bongers et al. (2013) conducted a range of studies linking positive emotions to overeating. Using a measure of emotional eating called the Single Target Implicit Association Test (ST-IAT) and the Dutch Eating Behaviour Questionnaire (DEBQ), they reported that participants who scored high on positive and negative emotional eating on the ST-IAT (not the DEBQ) ate more during positive mood induction than negative food induction. In another study by Bongers et al. (2013), they found that emotional eaters with induced positive mood consumed more food than non-emotional eaters, with no effects in the negative condition.

Cserjesi, Luminet, Poncelet, and Lenard (2009) were interested in the interaction between obesity, depression, and cognition. They found decreased mental flexibility and sustained attention ability in obese individuals with depression. There is also a large literature linking depression and anxiety to poor cognitive performance (van Tol et al. 2010). This literature highlights that the effects of obesity may not be limited to physiological or intellectual consequences and that severe obesity in particular is associated with alterations in emotional

state. A contributing factor may be the high levels of stigma experienced by obese individuals (Lewis and Van Puymbroeck 2008).

1.6 Stigma

With obesity comes stigmatization and often negative attitudes towards the obese state (Brownell, Puhl, Schwartz, Rudd 2001). This is may be due in part to the (likely inaccurate) belief that stigmatization and blame of overweight persons may serve as motivation for lifestyle change (Puhl and Heuer 2010). Frequent stigmatization has been linked to more psychological distress and even more weight gain in obese individuals (Myers and Rosen 1999). One study found that discrimination against obese people may be more accepted socially than discrimination against other groups (Lather, O'Brien, Durso, Brinkman, McDonald 2008).

Research indicates that stigma may come not only from peers, but from the overweight individual themselves (Durso and Latner 2008). Latner, Stunkard, and Wilson (2005) conducted a study on university students, in which they asked them to rate their peers according to appearance. The results indicated that not only was obesity highly stigmatized, overweight and obese people were just as stigmatizing towards obesity as their non-overweight peers. Durso and Latner developed the Weight Bias Internalization Scale (WBIS) which showed high internal consistency. They reported WBIS was correlated with self-esteem, mood measures, and eating disturbance. Multiple regressions showed that WBIS significantly predicted measures of self-esteem, mood, and binge eating. The inevitability of encountering stigma may stem from the finding that even health professionals express anti-fat attitudes (Schwartz, Chambliss, Brownell, Blair, Billington 2003). Schwartz and colleagues

reported that health professionals favoured pro-thin and anti-fat beliefs, while accepting stereotypes such as overweight individuals are lazier and less intelligent.

This stigma may, at least in part, lead to the negative mood experienced in obesity. Anderson and colleagues reported that in adolescent females obesity predicted higher risk for Major Depressive Disorder (MDD) and Anxiety Disorder (AD) (Anderson, Cohen, Naumova, Jacques, Must 2007). In this thesis stigma is not directly investigated. However, self-esteem and depression (which may be related to feeling stigmatized) are controlled for.

1.7 Implications for weight-loss interventions

Obesity is related to anxiety and depression (Strine et al. 2008; Luppino et al. 2010; Anderson, Cohen, Naumova, Must 2006), cognitive deficits (Steffens et al. 2006), sleep problems (Beebe et al. 2005), poorer quality of life (Kolotkin, Meter, Williams 2008; Kolotkin, Crosby, Williams 2002), and physical function (Lang, Llewellyn, Alexander, Melzer 2008; Rolland et al. 2009, Bouchard and Janssen 2010) which is related to decreased use of weight-loss (Inelman et al. 2005). This not only implies that obesity may cause individuals to suffer; it also makes the chances of successful weight-loss less likely via its comorbities. However, it should be noted that improvements in quality of life and anxiety/depression were correlated with weight-loss (Karlsson and colleagues 2007; Averbukh et al. 2003; Faulconbridge et al. 2012).

Current weight loss interventions for obesity comprise lifestyle modification programmes e.g. dietary advice, drug therapies and bariatric surgery. Current drug treatments are limited to only one available treatment in Europe called Orlistat. A review of lifestyle medication

programmes reported that a reduction in 10% body weight in 4-6.5 months was possible when participating in lifestyle interventions (Wadden, Butryn, & Byrne, 2004). In a one year trial by Wadden and colleagues (2005) 224 obese participants were placed on one of two treatment options, either on the anti-obesity drug sibutramine alongside lifestyle modification or lifestyle modification alone. Participants who were given the combination of treatments were seen to have lost the most weight. Despite this, it appears weight loss is hard to maintain. A review of lifestyle modification in obesity demonstrated that in order to maintain weight-loss, contact with the health practitioner, physical activity, and long term use of anti-obesity drugs and lifestyle modification techniques were important (Wadden, Butryn, & Wilson, 2007).

A randomized controlled trial compared Weight Watchers (WW) to a self-help condition (Johnston, Rost, Miller-Kovach, Moreno, & Foreyt, 2013). WW was found to enable significantly greater weight-loss than self-help. However, there is still major dispute as the most beneficial program for weight-loss. Tsai and colleagues (2012) compared WW participants to groups attending weight loss clinics (medically managed) and found that the clinic based intervention was more successful with weight-loss. Hutchesson and colleagues (2014) reported on a more novel approach, in the form of a 12 week commercial web-based weight-loss program. It was conducted on a large group of men and women with a mean BMI of 32.1. They found successful dieters experienced improvement in the quality of dietary intake, but that overall the reported dietary intake was not distinguishable from participants in control groups. While weight loss can be achieved it is hard to maintain and many dieters regain the weight they lost within a few years (Wadden, et al. 2007).

Gastric surgery seems to be effective for weight loss but is associated with risks. A systematic

review and meta-analysis by Buchwald and colleagues (2004), reported that effective weight-loss was achieved for morbidly obese patients in addition to significant improvements/resolutions to comorbidities (diabetes, hypertension, hyperlipidaemia, obstructive sleep apnea). Metabolic Syndrome (MS) is a relevant area of obesity literature as it refers to obese individuals who have diabetes and high blood pressure (Grundy et al. 2004). Gastric bypass has been seen to improve MS (Batsis, Romero-Corral, Collazo-Clavell, Sarr, Somers, Lopez-Jimenez 2008). A study which followed long limb gastric bypass patients found that at follow up there was an MS remission rate of 32.7%, and 59.2% at 36 months, with a significantly decreased number of patients still using anti-diabetic and cardiovascular disease medications (CVD) (Nora et al. 2014). However, not all patients are eligible for gastric surgery and complications such as anastomotic leaks (Mitchell, Pizzitola, Knuttinen, Robinson, Gasparaitis 2005), gastric perforation, deep abscess (Msika 2003), hernias, and small bowel obstruction (Griffith, Birch, Sharma, Karmali 2012). Therefore, there is a need to consider new approaches to weight loss interventions.

Given the relationship between obesity and cognitive function; one possibility would be to develop interventions aims more at improving cognition and supporting psychological outcomes in weight loss programmes. A systematic review of the psychological benefits of weight-loss programs (resulting with and without weight-loss) found that significant improvement in psychological outcomes took place (Lasikeiwicz, Myrissa, Hoyland, Lawton 2014). This included changes in body image and health related quality of life (QOL). A randomized Controlled Trial investigating QOL after weight-loss, observed participants in a 6 month clinical weight-loss program, and then randomized into one of two 6 month extended care groups (Blissmer 2006). The mean age was 50.2, mean was 32.5, and 78% were female.

Although there was some weight regain, significant improvements were noted and maintained for up to 24 months. This included physical function, scores in the mental composite scale, and score in the mental health subscales. Siervo and colleagues (2011) conducted a systematic review on weight-loss and cognition in overweight/obese. Weight-loss was related to minor improvements in executive function, attention, and memory in only the obese participants. Siervo and colleagues (2012) found that middle aged obese individuals (aged 30-59) experienced positive improvements on cognition and physical function post weight-loss. It appears losing weight may be beneficial when attempting to improve cognition and psychological well-being.

One possible approach to improving weight loss programmes might be to include adjuncts that facilitate changes in cognition and emotion because these would be predicted to improve outcomes. One such addition might be exercise. A study on high fat diet induced rats investigated how diet and exercise was related to cognition (Woo, Shin, Park, Jang, Kang 2013). High fat diet induced obesity was associated with decreased cognition function, yet improved with diet change and exercise. Exercise was specifically seen to improve memory and learning (in contrast to diet). Cotman and Engessar-Cesar (2002) reported that animals that exercise express changes in the brain which lead to better learning and decreased cognitive deficits. Additionally, Aguiar and colleagues (2011) observed short bursts of moderate exercise increased spatial learning and memory in older rats. Therefore, exercise might be a helpful adjunct to weight loss diets via its effects on cognitive function (Gomez-Pinilla, Vaynman, Ying 2008). Indeed exercise has been suggested to help with weight loss maintenance. In addition, it has been suggested that strategies focusing on improving food memories such as “attentive eating” might be useful in improving appetite control (Robinson,

Aveyard, Daley, Jolly, Lewis, Lycett, Higgs 2013). While others advocate techniques related to mindfulness meditation. Epel and colleagues (2009) specifically suggested meditation may assist with decreasing stress and telomere shortening associated with overeating and obesity. These data point to possibilities that could stem from further investigating of the relationships between cognition, emotion and obesity.

1.8 Summary

The present literature review demonstrates that there are strong associations between cognition and obesity. How this relationship originates, and which direction it goes, is being debated. In addition, the role played by emotional disturbances, such as depression and anxiety are unclear. A review of the literature suggests that incorporating relevant potential confounds, such as these, may help separate out the underlying mechanisms. More sophisticated investigations including associated factors such as preoccupations, and eating attitudes would be useful in understanding the cognition and eating behaviour cycle and to give a broader overview of the complex relationships between obesity, emotion and cognition.

1.9 Aims of thesis

The literature demonstrates a connection between obesity and both cognitive and emotional dysfunction. This thesis aims to investigate emotional and cognitive function in obesity. Specifically, emotional function in younger and older participants with a large range of BMIs will be investigated to further explore the role of associated factors in obesity. Another primary aim is to investigate the relationship between BMI and specific cognitive performance while taking into account these differences in emotional function and other important confounders. It is predicted that higher BMI will be related to poorer memory,

particularly when a participant is older. Detailed assessments of cognitive function will be conducted alongside measurement of psychological and behavioural measures, such as measures for depression, anxiety, food preoccupation and eating behaviour attitudes. In addition, memory will be investigated both generally and more specifically in relation to food memory. It is predicted that anxiety and depression will be associated with obesity and response to obesity treatment, that emotional dysfunction will be related to poorer lifestyle, that increased BMI will be negatively related to cognitive ability, and that memory for food will be related to food preoccupation.

CHAPTER 2: Study 1 - Depression and anxiety are associated with reduced obesity-related quality of life measures in extremely obese patients attending a specialist weight management service.

2.1 Introduction

Internationally rising rates of obesity have contributed to concerns over its links to health complications such as cancer, diabetes, and heart disease (Pergola and Silvestris 2013, Jemal et al. 2001, Astrup and Finer 2000, Rabkin 2006). With these complications come costs that are leading to considerable spending on medical treatment (Pelone et al. 2012) and discussions on more cost effective strategies to addressing the disease (Lehnert, Sonntag, Konnopka, Riedel-Heller, König 2012).

Obesity is also associated with emotional problems such as anxiety and depression. It has been reported that adults with anxiety are more likely to be obese (Strine et al. 2008), while it also increased the risk of depression (Luppino et al. 2010). In a study that followed 827 individuals (ages ranging: 9 to 40) for twenty years, it was found that in females anxiety and depression were positively correlated with higher BMIs (Anderson, Cohen, Naumova, Must 2007). Similarly, with data from a telephone survey on over 200,000 Americans, Strine et al. (2008) found that adults with depression or anxiety were more likely to be obese, binge on food and drink, and be physically inactive. However, systemic reviews of the relationship between obesity and depression and anxiety have reported that obesity may only increase the risk of depression in subgroups of individuals such as those prone to binge eating (e.g. Atlantis and Baker 2008) and there have been other reports of non-significant associations between obesity and depression in community samples and even posit that obesity is associated with improved emotional functioning (Crisp and McGuinness, 1979; Friedman and

Brownell, 1995). Furthermore, there have been few investigations of the association of obesity and mental health among severely obese populations. This is important because both depression/anxiety and obesity affect quality of life (Katschnig et al. 1997) and it is possible that mental health issues interact with obesity to worsen quality of life, especially for people with extreme obesity.

Many studies have reported an association between obesity and quality of life measures relating to both physical and mental functioning. A study of over 3,000 obese individuals using the IWQOL-Lite, which assesses the impact of obesity on physical function, self-esteem, sexual life, public distress and work, found that impaired quality of life increased as BMI increased and that overall impairment is greater in women (Kolotkin, Crosby, Williams 2002). Higher BMI has also been correlated with impaired physical function (Lang et al. 2008, Rolland et al. 2009), specifically upper body and to a lesser extent lower body function (Apovian et al. 2002). In a 10 year study of over 600 surgically treated cases and their matched controlled counterparts, it was found that sustained weight loss was positively correlated with quality of life (Karlsson et al. 2007). This research suggests that very high adiposity may be causing increased impairment on physical functioning and quality of life in the obese, although this may be reversible with weight loss. However, the extent to which quality of life is further affected by depression and anxiety in a severely obese population is not known.

Mental health issues may also have an adverse effect on an obese person via effects on management of weight loss. There is evidence to suggest that anxiety and depression may lead to difficulties in daily life, and may even cause cognitive deficits, which affect adherence

to weight loss regimens (Steffens et al. 2006). It has been reported the depression and anxiety can worsen outcomes for participants in a weight loss programme (Pagoto et al. 2007). However, study by Averbukh et al. (2003) of 47 patients undergoing a gastric bypass were followed for 1 year post surgery found that patient's pre-surgery Beck Depression Inventory (BDI) score predicted post-surgery weight loss after 1 year. This means that more depressed individuals, lost more weight than less depressed counterparts. It is possible that these results are unique to the morbidly obese population, the only group of dieters with access to weight loss surgery. Therefore, it is of interest to examine the relationship of depression and anxiety to weight management.

We hypothesised that obesity would be associated with depression and anxiety and that quality of life would be impaired in obesity especially for participants with high levels of depression, and anxiety. We further hypothesised that depression and anxiety might be related to poorer outcomes in terms of weight loss on the programme.

2.2 Method

Participants and Procedure

This study was a retrospective analysis of data collected as part of the evaluation of a regional specialist weight management service in the South Birmingham Primary Care Trust (PCT). The service evaluation examined anonymized data; therefore no formal ethical approval was needed. All data collection took place at Heartlands Hospital. Researchers were given access to previously collected questionnaire measures, attendance records, and medical records of participants. All data was then scored and inputted into a secure NHS and anonymized

database, which was used in the analyses. No new measures were added by researchers as all data had been collected beforehand.

Data from 63 men and 181 women between 17 to 80 years of age (mean age = 43y and mean BMI = 46Kg/m^2) was analysed. Participants had been referred to their local weight management service by their GP from January 2009 to October 2011 in Stechford, England. The criteria for referral were as follows: BMI ≥ 35 kg/m2 with a comorbidity (e.g., diabetes mellitus) or BMI ≥ 40 kg/m2 without a comorbidity; to have previously attended other weight management services (commercial, community, or primary care) without success. Participants were seen by a GP and dietician and received advice to promote healthy lifestyle change. They may have also been referred to monthly a weight-loss management group called Slim Group at their local hospital (Heartlands Hospital). The community outreach programme provided a range of services including physical assessment, a tailored diet programme, counselling services, and assistance with bariatric surgery referral.

The objective of the intervention was to lose 10% of body weight, which in some cases could assist in obtaining weight loss surgery funding. The intervention included personalized lifestyle change advice on diet and exercise by a physician or dietician. During an initial introduction to the programme (first advice session), patient demographics, height and weight, and medical information were noted. Any weight related diagnosis (such as diabetes or CVD) was put on file. Patients were also provided with specific treatment of further testing if necessary. For example, some were offered weight loss medication (such as orlistat), or counselling opportunities for underlying psychological issues. Also, if suspected of having sleep apnea they were scheduled to visit the sleep lab for an evaluation. At their first weight management appointment they were asked to fill out all self-report measures. Throughout the

next 12 months patients were scheduled into meetings/check-ups with a GP or dietician for advice and progress monitoring. After 12 months they were discharged from the programme.

Measures

Weight related comorbidities

At baseline, formal diagnoses of the following diseases were recorded by GP for: Diabetes type II, hypertension, depression, blood pressure, obstructive sleep apnea, cardiovascular disease (CVD), polycystic ovary syndrome (PCOS) and arthritis.

Self-report measures

Demographics: Information on the patient's gender, age, marital status, smoking status, alcohol consumption, ethnicity, and occupation were recorded by staff through previous records or one on one discussion.

EuroQol (EQ-5D) Questionnaire (Hurst, Kind, Ruta, Hunter, Stubbings 1997)*:* This is a 6 item questionnaire designed to measure perception of health status by the patient. It consists of 5 subscales: a measure for general health, problems with mobility, problems with self-care, problems with usual activities, and problems with pain and discomfort (EuroQol group, 1990). Each item is on a scale from 1 to 100, with higher scores indicating difficulty in each area. However, higher scores on the health item indicate a better self-rating of overall health. The health scores were of particular interest as quality of life measures such as these have been correlated with obesity and are suspected to influence weight management success (Sach et al, 2007). It has been found to have good internal validity and reliability (Hurst et al.1997).

Impact of Weight on Quality of Life-Lite Questionnaire (IWQOL-Lite) (Kolotkin et al. 2001)*:* This is a 31 item questionnaire used to determine impact of weight on 5 subscales; physical function, self-esteem, public distress, sexual life, and work. This questionnaire was used because obesity has been associated with reduced quality of life (Algul et al. 2009). Each subscale consists of a likert scale ranging from 1 to 5 (from "never true" to "always true"). Higher scores indicate a higher perceived impact of weight on quality of life. It has been reported as having good internal consistency ((Kolotkin et al. 2001) and test re-test reliability (Kolotkin and Crosby 2002). (See appendix 4a)

Hospital Anxiety and Depression Scale (HAD) (Zigmond & Snaith, 1983): This is a 14 item questionnaire used to rate anxiety and depression levels. It consists of two subscales; anxiety (HADS-A) and depression (HADS-D) each including seven items. Each item is measured on a four-point Likert scale, each ranging from 0 to 3, resulting in a maximum score of 21. A score above seven on each subscale implies the presence of potential distress. There has been a suggested relationship between obesity, anxiety, and depression (Jorm et al., 2003). A less documented relationship is the one between these factors and in a severely overweight sample attempting weight management. (See appendix 3a)

Pittsburgh Sleep Quality Index (PSQI) (Buysse et al., 1989)*:* The PSQI is a validated measure for sleep quality. It assesses 7 subscales; subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleep medication, and daytime dysfunction. A global score ranges from 0 to 21, with lower scores indicating poorer sleep quality. A global score of 5 or less signifies severe sleeping problems. (See appendix 1a)

Outcome measures

Height and weight: To assess weight change across the programme, weight was measured by a GP, dietician, or nurse at baseline, during routine care, and at the end of the programme. This was also used to compute Body Mass Index (BMI) by researchers.

Programme attendance: Programme participation was initially recorded by hospital staff, dietician, or GP. The measure was then recorded by researchers through observing programme start and end date, and total number of appointments throughout the course of several months. This information was stored via an online database and spread sheets which indicated total months on programme, every date each participant attended, cancelled, or did not attend the clinic. Attendance was defined as actively participating in scheduled programme appointments. Non-attendance was defined as missing an appointment without rebooking and attending another one in its place. A participant was considered a 'drop-out' if they missed 3 or more consecutive appointments without rebooking.

2.3 Statistical Analysis

To determine depression and anxiety levels participants were grouped according to high (11-21), and low to normal levels (0-10) using the *Hospital Anxiety and Depression Scale (HAD)* questionnaire categories. This approach was chosen due the guidelines outlined on the questionnaire diagnostic scoring section, which is standard practice at UK hospitals. Weight change was calculated as percentage body weight change, increasing when a participant gained weight, and going below zero (no change) when the participant lost weight. T-tests were used to compare differences on measures for high/low depressed and anxious groups. Predictors of quality of life measures and weight change were assessed using regression. All

statistical analyses were conducted using SPSS (version 17.0).

2.4 Results

Participant Characteristics

Of the original 270 participants, 26 participants were removed due to high amounts of missing information. This left 244 participants, 63 males and 188 females. The mean age of the sample was 43.4 years (SD = 12.7), mean BMI = 47 (SD = 7.8). The mean anxiety score was = 10.5/21 (SD = 4.5), and the mean depression score was = 9.11/21 (SD = 4.0) (See Table 1 below). 63% of patients had above normal anxiety scores, while 53% of patients had above normal depression scores. There were gender differences for age (males = 48.5 (± 13.4), females =41.6 (±11.9), $p<.001$) , anxiety (males = 9.15 (±5.0), females =10.9 (±4.3), $p<.05$), sleep (males = 6.8 (± 4.6), females= 8.7 (±5.4), $p<.05$) , self-esteem (males =24.8 (±9.3), females =28.9 (±6.8), $p<.001$), and sexual life (males = 11.7 (±5.7), females =13.6 (±5.8), $p<.05$).

When comparing Quality of life scores between anxious and non-anxious participants, scores were significantly higher/more impacted for the anxious participants (Table 2 below). When comparing Quality of life scores between depressed and non-depressed participants, scores were significantly higher/more impacted for the depressed participants (Table 3 below). Participants did not vary significantly in age, BMI, weight change %, or total number of comorbidities.

Table 1. Participant Characteristics in a severely obese population

Variables	Overall
Age (years)	43.4 (± 12.7)
BMI	47.0 (± 7.8)
Weight Change %	-3.0(±7.0)
Hypertension %	60.7%
Diabetes %	50.4%
OSA %	50.8%
Smokers %	40.7%
HAD Anx (0-21)	10.5 (±4.5)
HAD Dep (0-21)	9.11 (±4.0)
PSQI Sleep (0-21)	8.2 (± 5.2)
EQ-5D:	
Health (1-100)	44.3 (±20.1)
IWQOL 5 subscales:	
PF (1-55)	34.3 (±13.1)
Self-Esteem (1-35)	36.1 (±11.4)
Sexual Life (1-25)	13.1 (±5.8)
PD (1-25)	17.0 (±5.8)
Work Life (1-25)	10.9 (±4.6)

OSA: Obstructive Sleep Apnea
HAD: Hospital Anxiety and Depression Scale
PSQI : Pittsburgh Sleep Quality Index
EQ-5D: EuroQol 5D Questionnaire
IWQOL-Lite: Impact of Weight on Quality of Life-Lite Questionnaire
Dep: Depressed Anx: Anxious
PF: Physical Function PD: Public Distress

Table 2. Participant Characteristics and Quality of Life measures for anxious and non-anxious in a severely obese population

Variables	**Non-Anx**	**Anxious**	**t**	**P-values**
Age (years)	44.4(±13.4)	40.1(±11.7)	2.083	.04
BMI	47.1 (±6.9)	46.9 (±8.4)	.221	.83
Weight Change %	-2.8(±2.2)	-2.7(±6.8)	-.135	.89
HAD Anx (0-21)	**6.7 (±2.5)**	**14.4 (±2.4)**	**-24.396**	**<.001**
HAD Dep (0-21)	**6.8 (±3.4)**	**11.4 (±3.1)**	**-10.635**	**<.001**
PSQI Sleep (0-21)	**7.1 (± 5.5)**	**9.7 (±5.0)**	**-3.844**	**<.001**
EQ-5D:				
Health (1-100)	**50.7(±19.2)**	**35.9(±18.1)**	**5.633**	**<.001**
IWQOL 5 subscales:				
PF (1-55)	**33.4(±10.8)**	**39.3(±11.1)**	**-3.977**	**<.001**
Self-Esteem (1-35)	**24.5(±7.9)**	**31.6 (±5.2)**	**-7.996**	**<.001**
Sexual Life (1-25)	**11.3(±5.6)**	**14.7 (±5.6)**	**-4.334**	**<.001**
PD (1-25)	**15.4 (±5.4)**	**18.7 (±5.6)**	**-4.362**	**<.001**
Work Life (1-25)	**9.25 (±4.0)**	**12.6 (±4.4)**	**-5.303**	**<.001**

HAD: Hospital Anxiety and Depression Scale, PSQI : Pittsburgh Sleep Quality Index
EQ-5D: EuroQol 5D Questionnaire, Dep: Depressed, Anx: Anxious
IWQOL-Lite: Impact of Weight on Quality of Life-Lite Questionnaire

Table 3. Participant Characteristics and Quality of Life measures for depressed and non-depressed in a severely obese population

Variables	**Non-Dep**	**Depressed**	**t**	**P-values**
Age (years)	43.8 (± 12.6)	41.1 (± 12.4)	1.576	.12
BMI	46.9 (±7.9)	47.5 (±7.3)	-.634	.53
Weight Change	2.8 (±7.6)	2.8 (±2.8)	-.032	.97
HAD Anx (0-21)	**8.8 (±4.2)**	**13.1 (±3.6)**	**-7.999**	**<.001**
HAD Dep (0-21)	**6.5 (±2.6)**	**12.9 (±1.9)**	**-20.316**	**<.001**
PSQI Sleep (0-21)	**7.7 (±5.5)**	**9.4 (±5.2)**	**-2.329**	**.02**
EQ-5D:				
Health (1-100)	**49.3(±19.6)**	**34.4 (±17.4)**	**5.461**	**<.001**
IWQOL 5 subscales:				
PF (1-55)	**33.8(±10.9)**	**40.6 (±10.3)**	**-4.491**	**<.001**
Self-Esteem (1-35)	**25.0 (±7.7)**	**32.0 (±3.7)**	**-8.646**	**<.001**
Sexual Life (1-25)	**11.0 (±5.6)**	**15.9 (±4.6)**	**-6.572**	**<.001**
PD (1-25)	**15.4 (±5.6)**	**19.8 (±4.7)**	**-5.904**	**<.001**
Work Life (1-25)	**9.5 (±3.9)**	**13.2 (±4.5)**	**-5.735**	**<.001**

HAD: Hospital Anxiety and Depression Scale, PSQI : Pittsburgh Sleep Quality Index
EQ-5D: EuroQol 5D Questionnaire, Dep: Depressed
IWQOL-Lite: Impact of Weight on Quality of Life-Lite Questionnaire
Anx: Anxious, PF: Physical Function, PD: Public Distress

Correlations for BMI, Depression, and Anxiety

There were no significant relationships between BMI and depression and anxiety (See Table 4). However, there was a significant positive relationship between BMI, total number of comorbidities, physical function, and public distress, and Total IWQOL. BMI was negatively correlated with weight change percentage. It was not correlated with age in this sample. Weight change percentage was negatively correlated with months on programme, BMI, and age. It was not correlated with anxiety or depression. Anxiety and Depression were correlated with almost all quality of life measures (See Table 4).

Table 4. Pearsons correlations between Weight Change %, BMI, and comorbidities

	MOP	BMI	Co	Anx	Dep	PS	PF	HS	SE	SL	PD	WL	Age	IW
WC	**-.41****	**-.17****	-.12	.05	-.00	.013	-.10	.03	.06	.06	.01	.05	**-.13***	.06
MOP		.30	.80	**-.16***	-.03	.02	.04	-.04	-.09	.04	-.01	-.04	**.14***	-.02
BMI			**.22****	-.22	.18†	.04	**.28****	-.07	.08	.11	**.38****	.10	.02	**.22****
Co				-.04	-.01	.09	**.33****	**-.15***	**-.13***	**.14***	.07	-.06	**.45****	.09
Anx					**.65****	**.30****	**.33****	**-.39****	**.56****	**.37****	**.37****	**.46****	**.18****	**.47****
Dep						.**25****	**.39****	**-.48****	**.60***	**.54****	**.44****	**.55****	**-.12**	**.60****
PS							**.23****	**-.14***	**.20****	**.24****	**.20**	**.20****	-.07	**.31****
PF								**-.33****	**.34****	**.51****	**.47****	**.46****	**.25****	**.80****
Health									**-.35****	**-.30****	**-.29****	**-.36***	.02	**-.42****
Self-Esteem (SE)										**.57****	**.57****	**.62****	**-.29****	**.77****
Sexual Life (SL)											.**50****	**.50****	.04	**.76****
Public Distress (PD)												**.55****	**-.13***	**.73****
Work life (WL)													**-.12***	**.75****
IWQOL (IW)														1

*p <.05, ** p <.01, † trending
WC: Weight Change percentage, MOP: Months attending programme

BMI: Body Mass Index, Co: Total number comorbidities
PS: Pittsburgh Sleep Quality Index, PF: Physical Function Score on the IWQOL
Anx: HAD Anxiety Score, Dep: HAD Dep Score
IWQOL (IW): Impact or Weight on Quality of Life-Lite Questionnaire

Regressions Analysis for predictors of QOL

To test the hypothesis that comorbidities such as depression and anxiety predict quality of life and weight change in the severely obese, a multiple linear regression analysis approach was used. This was done due to the ability to control effects of covariates while testing the effects of specific predictors while excluding the influence of others (Lindenberger and Potter 1998). Backward elimination was then used, which involved deleting the variable that least fit the data until no further improvement could be made. This was chosen because the sample was representative of the population and the risk of over fitting the model would be minimal. This also follows the principle of Occam's Razor, by identifying the best (or simplest) model while removing 'noise' caused by unnecessary predictors, and avoiding collinearity (Tattar 2013). Based on the correlations observed, the following predictor variables were chosen; gender, age, BMI, depression, anxiety, months on program, and ethnicity. The predictors selected were correlated with the outcome variables (quality of life and weight change percentage). In a regression observing potential predictors for quality of life (via the IWQOL-lite) (See Table 5), depression was the strongest predictor, followed BMI, and then anxiety. However no interactions were found between age and BMI, age and depression, or depression and BMI when predicting quality of life. A second regression on predictors for weight change % was also carried out (Table 6), which controlled for gender, age, and ethnicity. Months on programme was the most significant predictor of weight change, followed by age.

Table 5. Summary of multiple linear regression analysis for variables predicting quality of life within a severely obese sample			
	B[1](±SD)	ß[2]	P-value
Depression	**3.07(±.79)**	**.48**	**< .001**
Anxiety	**1.5 (±.81)**	**.24**	**.07**
Gender	-5.91(±5.3)	-.11	.28
Age	.38(±.24)	- .15	.13
Ethnicity	-4.7(±3.1)	-.01	.94
BMI	**.73(±.33)**	**.21**	**.03**
[1]Unstandardized regression coefficient [2]Standardized regression coefficient R^2=.67			

Table 6. Summary of multiple linear regression analysis for variables predicting Weight Change % within a severely obese sample			
	B[1](±SD)	ß[2]	P-value
MOP	**-.24(±.07)**	**-.32**	**<.001**
Gender	-.63(±1.3)	-.04	.63
Age	**-.09(±.05)**	**- .18**	**.04**
BMI	-.09(±.07)	-.11	.21
[1]Unstandardized regression coefficient [2]Standardized regression coefficient R^2=.41 MOP: Months on programme			

2.5 Discussion

In this sample of severely obese participants attending a weight loss management clinic we observed high levels of depression and anxiety, which supports previous literature on the association between obesity and anxiety/depression (Jorm et al. 2007; Strine et al. 2007). However, BMI was not correlated with depression and anxiety, which was not predicted. This lack of association may be explained by the overall high BMI of the sample and lack of range in BMI scores. Overall, the results are consistent with the suggestion that severe obesity is associated with mental health issues, which should be taken into account in any assessment of the consequences of obesity.

We also observed a relationship between BMI and quality of life as measured by the IWQOL-Lite. BMI was particularly associated with impaired physical function and public distress, as well as sleep problems which may in turn exacerbate further physical and mental issue. These findings coincide generally with the literature on obesity and quality of life (Algul et al. 2009), along with previous work on obesity and sleep (Patel and Hu 2008). That physical function is strongly associated with increasing BMI in this group may relate to the severe nature of the obesity possibly affecting mobility and perhaps being related to the increased number of comorbidities with increasing BMI.

An interesting and important finding was that for measures of quality of life, participants with high levels of depression and anxiety reported much more of a negative impact of their weight on quality of life than participants with who scored low on depression and anxiety. Due to there being no significant differences in BMI, this implies that their perception of day to day life may be different and that mental health problems exacerbate existing issues with quality

of life in obesity. These findings add to the idea that the obese experience is variable and complicated by its comorbidities. Depression was also found to be the best predictor of quality of life in this sample. These findings suggest that BMI is important when considering quality of life, but it may not be as important as depression. Overall, very obese individuals tend to suffer from anxiety/depression, which affects their self-reported quality of life. Individuals of the same weight with similar co-morbidities, but excluding anxiety/depression, do not seem to have such negatively impacted perspectives on quality of life.

Additionally, number of comorbidities (such as diabetes and hypertension) were correlated with several QOL measures (Physical Function, Health Score, Self-esteem, Sexual Life). They were not, however, correlated with anxiety and depression in this population. This raises an interesting point on how physical health outcomes may alter an individual's view on their quality life. As the population is more anxious, depressed, and ill than the non-severely obese population, this data may not be suited to detect a physical health-QOL relationship. Asking how participants feel about their ailments upon first diagnosis may be a more accurate way of assessing the impact their physical health has had on their QOL outlook.

On average, participants in the present weight loss programme did lose weight. Weight change was associated with some of the expected measures such as months on programme and BMI, but not found to be related to other measures such as depression and anxiety. With both these factors being associated with diet failure (Gluck, Geliebter, Satov 2001; Swinburn and Egger 2004), not observing an impact on weight change was surprising. More anxious individuals would have been expected to lose less weight than less anxious participants (Anderson, Cohen, Naumova, Must 2007). A possible explanation may be that

due to the population's highly depressed/anxious nature, these effects simply cannot be seen. The impact of anxiety on weight-loss within a severely obese sample may also differ from the impacts in a less overweight population. In addition, quality of life measures were not associated with weight change in the present study, suggesting that although quality of life is impaired in sever obesity it does not affect weight loss outcomes.

The first limitation of this study was the inability for researchers to choose the measures. As it was a retrospective study, all data had already been collected. Nevertheless, the main areas of interest to this thesis were included in the previously selected measures (obesity, BMI, anxiety, and depression). If the study were to be repeated, measures for eating behaviour would be added (DEBQ) along with cognitive tasks. However, this would have taken away from the simplicity of the original study, and may have resulted in far less participants. Additionally, the sample may not be entirely representative of the very obese population. This is because we were only able to include information on participants who stayed in the programme. Many very obese individuals do not attend weight management and others may attend and then drop out. It is important to consider that this data focus' on individuals who are willing to stay on a weight management programme. This may be due to higher motivation to improve their health, which could affect scores on QOL, anxiety, and depression. Another limitation is that outside of weight change information, mainly baseline data was analysed. Access to questionnaire responses at the end of weight management would have provided a more complete view of their role in weight loss. Additionally, the limitations of using stepwise regression with backward elimination must be considered as generalizability of data may be an issue. However, the sample was representative of the population and so generalizability issues would not be substantial. This method also allows

for identifying the most important predictors within this sample. Another weight management study following diabetics with successful weight-loss experienced significant improvements in quality of life (Williamson et al. 2009). Similar results for improved quality of life were found in a study observing women on a 12 week weight-loss program (Lean, Han, Seidell 1998). Another limitation is that mainly several self-report measures were used, but all of the questionnaire have been previously validated and widely used. Strengths of the study include the variety of measures recorded (ranging from sleep to sex life) and the assessment of a large population of severely overweight participants. Further research into how these factors may be implicated in weight gain and lifestyle management is needed to develop successful long term intervention strategies.

Chapter 2 Summary

This study aimed to evaluate how anxiety and depression affect quality of life and weight change within a severely obese population. The study found that super-morbidly obese patients who participated in a 12 month weight management programme lost weight. Body Mass Index (BMI) was positively correlated with weight-loss, and total number of comorbidities. Weight change, however, was not correlated with depression and anxiety. Clinically obese individuals were found to be at higher risk for anxiety and depression. Anxious and depressed patients reported significantly poorer quality of life (such as physical function and health score) and sleep efficiency scores. The findings indicate psychological perspective is associated with poorer views on quality of life. In addition, quality of life was found to be the best predictor of depression in this sample. These findings add to the idea that the obese experience is variable and complicated by comorbidities.

CHAPTER 3: Study 2 - Relationships between Alexithymia and unhealthy eating habits in adolescents

3.1 Introduction

The results from Chapter 2 suggest that depression is common in a severely obese adult population and that the presence of depression and anxiety affects quality of life measures. Just as there has been little investigation of emotional problems in a severely overweight adult population, there is also a scarcity of research on emotional problems and overweight in young adults. Alexithymia is a deficit in the ability to recognize and express emotion (Haviland, Warren, Riggs 2000) is quite common among young people (Karukivi et al. 2010; Säkkinen et al. 2007) and has been suggested to be a factor that predisposes towards depression and anxiety (Lumley, Ovies, Stettner, Wehmer, Lakey 1996; Bonnet, Brejard, Pasquier, Pedinielli 2012). Higher scores on a measure of alexithymia have been associated with depression and poor sleep quality (Gennaro et al. 2004), along with severe disruptive disorder in adolescents (Mannimen et al. 2001). A link between alexithymia and eating disorders, including obesity has also been proposed (Zak-Golab et al 2013; Larsen, van Strien, Eisinga, Engels 2006), but there have been few investigations of the links between alexithymia and specific factors linked to obesity such as eating and exercise patterns.

The relationship between obesity and alexithymia has been studied in the past with mixed results. For instance, Morosin and Riva (1997) and de Zwaan et al. (1995) did not find a direct link with obesity, but found higher rates of alexithymia in obese subgroups with particular psychological traits such as interpersonal distrust and ineffectiveness. On the other hand, when using a Rorschach test with alexithymia variables Clerici and colleagues (1992) found

severely obese patients to be more alexithymic than lean counterparts. However, the Alexithymia Rorschach test may not reliably measure the exact constructs as questionnaire measures such as the Toronto Alexithymia Scale 20 (TAS-20) for children (Rieffe, Oosterveld, and Terwogt 2006).The TAS is made up of 20 items and three scales: Difficulty Identifying Feelings (DIF), Difficulty Describing Feelings (DDF), and Externally Oriented Thinking (EOT). Fukunishi and Kaji (1997) have reported higher alexithymia scores (using the TAS-20) in an obese versus non-obese sample. Surcinelli and colleagues (2007) compared obese to non-obese participants also confirmed a higher prevalence of alexithymia in the obese sample. Adami and colleagues (2001) suggested that obesity itself does not cause alexithymia, but that when an obese individual attempts lifestyle change, the presence of alexithymia may make their attempts more difficult. Elfhag and Lundh (2007) conducted a study on 259 obese participants and found a clear relationship between obesity and alexithymia. They also pointed out that obese males had higher scores on Externally Oriented Thinking (EOT) than females. Therefore, there is evidence that alexithymia is associated with obesity.

The relationship between alexithymia and obesity may be explained by its association with emotional eating. Larsen, van Strien, Eisinga, Engels (2006) investigated gender differences in alexithymia and emotional eating (EE) in 343 obese females and 70 obese males with a mean age of 43 years. They observed more difficulty in identifying (DIF) and describing feelings (DDF) to be associated with higher EE in men. Although the overall sample was large, a larger sample of male participants would have made a better proportioned comparison. Some studies have examined whether alexithymia mediates relationships between depression and emotional eating (Pinaquy et al. 2003). Depression was found to be

positively correlated with emotional eating (EE) and indirectly related to EE through alexithymia and impulsivity (Ouwens, van Strien, van Leeuwe 2009). In support of this finding, when females were given a distress manipulation (impending public speaking task) before an ad lib taste task, higher alexithymia scores moderated the association between food consumption and stress. This meant that as stress levels went up, higher alexithymia scores were related to higher food consumption. In addition, the alexithymia females in the distress condition ate the same or more than their non-alexithymia counterparts (van Strien and Ouwens 2007). De Lenclave, Florequin, and Bailly (2001) conducted a study investigating alexithymia within obesity and found that although depression was associated with alexithymia and binge-eating disorder, there was no direct relationship between binge eating and alexithymia diagnosis. However, Wheeler, Greiner, and Boulton (2005) found a strong relationship between binge eating and alexithymia. Noli et al. (2010) suggested that although alexithymia did not appear to specifically influence eating behaviour in the obese, it was generally associated with disordered eating behaviours. Deborde et al. (2006) observed eating disordered and non-eating disordered females and also reported higher rates of alexithymia in disordered eaters. The literature indicates that alexithymia may sometimes interact with eating behaviour.

Alexithymia has also been associated with eating disorders. Nowakowski, McFarlane, and Cassin (2013) conducted an extensive literature search and found evidence that individuals with eating disorders are more likely to be alexithymic than controls (For example Sasai, Tanaka, Hishimoto 2010), and specifically more likely to score higher on DIF and DDF. Indeed, Alexithymia has been associated with both Anorexia (Bourke, Taylor, Parker, Bagby

1992; Schmidt, Jiwany, Treasure 1993; Corcos et al. 2000) and Binge Eating Disorder (BED). Pinna et al. (2011) compared two large groups (293 obese patients to 293 non-obese subjects) and found a 12.9% alexithymia prevalence in the first group, versus nearly half that at 6.9% in controls, along with a high BED-alexithymia relationship. Pinaquy and colleagues (2003) tested 169 obese women using self-report measures including the Beck Depression Index (BDI), Dutch Eating Behaviour Questionnaire (DEBQ), TAS, and Binge Eating Disorder (BED) via interview. Approximately one-third of the sample was found to have BED, and this subgroup had the highest TAS, BDI, and emotional and external eating scores. Within this subgroup, alexithymia was the main predictor for emotional eating. The use of interview to assess BED may be worth noting, as interviewer bias and a subject's social behaviour may influence results. However, Carano and colleagues (2006) measured 101 BED patients using the TAS and found a 39.6% prevalence of alexithymia. Individuals with alexithymia were also found to have higher rates of body dissatisfaction, low self-esteem, and depressive symptoms. DIF and DDF were found to be predictors for BED severity. Hence, it may be that that alexithymia is a predictor or mediator of disordered eating generally regardless of the type (i.e. binging and restricting) (Laquatra and Clopton 1994). However, this relationship may not be direct. Cochrane, Brewerton, Wilson, and Hodges (1993) reported a correlation between alexithymia and self-ratings of affective symptoms, and not with binge-purge frequencies. In addition, alexithymia and EDI (Eating Disorder Inventory) scores have been found to be related to psychological traits of ED patients, not their actual attitudes or behaviours towards food and food intake (Taylor, Parker, Bagby, Bourke 1996). This suggests that any relationship between alexithymia and unhealthy or disordered eating might be indirect. When controlling for psychological distress (such as depression), some studies have found the relationships between alexithymia and disordered eating disappear while others observed no

change (Nowakowski, McFarlane, and Cassin 2013).

Fewer studies have examined relationships between alexithymia and eating problems in a young people. In a sample of 729 non-clinical late adolescents TAS and eating disorder symptoms results suggested that ED symptoms, as in adults, are also more common in alexithymics (Karukivi et al. 2010). A smaller study on 30 ED adolescent girls and 31 healthy adolescent girls also found higher TAS scores within ED, along with worse performances on emotion recognition tests (Zonnevijlle-Bendek et al. 2002). These studies appear to mirror results found in adult alexithymia research.

The literature indicates that alexithymia is common in young people (Karukivi et al. 2010), obese populations (Pinna et al. 2011), and may predispose young people to depression and anxiety (Lumley et al. 1996). Alexithymia has also been linked to disordered eating, such as obesity (Zak-Golab et al. 2013; Larsen, Van Strien, Eisinga, Engels 2006). It is predicted that that alexithymia and unhealthy lifestyle may also be correlated. This has been demonstrated through studies in the areas of obesity, eating disorders, emotional dysfunction, and lifestyle. However, evidence is lacking on the nature of the specific relationships between alexithymia, obesity and eating, especially for adolescent populations. This study aims to investigate whether a specific kind of emotional dysfunction, Alexithymia, may be related to unhealthy eating behaviours and lifestyle. We predicted that higher alexithymia scores would be positively correlated with unhealthy lifestyle and BMI, and that this relationship might be related to a measure of emotional problems (psychological distress).

3.2 Method

Participants

50 adolescent males and 60 adolescent females (mean age = 12.24 SD+/- .6, mean BMI = 19.24Kg/m^2 SD +/- 3.1) were selected from a co-educational secondary school. This was a subset of pool of 600 students for a larger project. However, for this study only the 110 students from one secondary school were included. Ethical approval was obtained from the Coventry and Warwick Research Ethics Committee (REC), as a sub-study under an obesity and adolescence investigation, which took place across 9 schools in the West Midlands, UK and investigated the relationship between sleep and obesity in adolescents. This included the collection of self-report data on eating habits. Only the subset of 110 adolescents was measured for Alexithymia (TAS-20) data.

Procedure

Three researchers visited a co-educational secondary school, with an age range of 11 to 16 years old. Only year 7's (approximately 12 years of age) were included in the study. At the school, all researchers collected questionnaire and physiological data on the approved adolescent sample. This experiment was a separate Alexithymia study (conducted by one researcher), in which 110 of the student participants were administered an additional electronic TAS-20 (Alexithymia) questionnaire during their computer task. The Alexithymia measure was the only measure selected by the researcher for this study. This was done by meeting students during an afternoon computer lab class. First, they were greeted and seated at an available desktop. Each student was allowed to log on and directed to the electronic questionnaires on survey monkey. Survey Monkey is a commercial website that allows researchers to upload questionnaires electronically, enabling them to administer them to

participants through a web link. Data was then collected online through the website. As part of the wider study, researchers also collected demographic, lifestyle, and all other self-report measures via Survey Monkey. Researchers also individually fitted participants with wristband sleep monitors. Researchers observed participants as they completed the computer task and assisted when necessary. Each student completed the task at their own pace. Lastly, participants were also weighed on a scale and measured for height to later calculate Body Mass Index (BMI).

Self-report measures

Demographics: A section with questions on demographic information was included. This collected data on the participant's gender, age, ethnicity, educational performance, family history on diabetes and cardiovascular disease, parent employment status and parent educational background.

Alexithymia: The Alexithymia Questionnaire for children is a validated alexithymia measure (Rieffe, Oosterveld, and Terwogt 2006) based on the Toronto Alexithymia Scale 20 (TAS-20) (Bagby, Parker, Taylor 1994) and was used to assess the students for alexithymia. This was chosen because it was an available adolescent-friendly alexithymia measure. It is made up of 20 items with a minimum score of 20 and maximum score of 60. There are three scales: Difficulty Identifying Feelings (DIF) with 7 items, Difficulty Describing Feelings (DDF) with 5 items and Externally Oriented Thinking (EOT) with 8 items. Children were instructed to score each item on a 3-point response scale (0 = not true; 1 = a bit true; 2 = true). This validated children's version of the scale has been used due to its use of simpler language compared to the TAS-20, which has been reported to have inconsistencies in younger populations. In 2007 Moriguchi and colleagues conducted a cross-validation study of the

TAS-20 on a large Japanese sample ranging from 14-84 years old and confirmed it to be valid for Japanese samples. However, the study did not test participants under 14 years old. In 2002, the reliability and validity of the TAS-20 was investigated (Parker, Taylor, Bagby 2002). They found the TAS-20 demonstrated good internal validity in a large co-ed adult Canadian sample, but did not test this in children. Rieffe, Oosterveld, and Terwogt (2006) reported that the Alexithymia Questionnaire for Children showed good predictive validity in children for at least the first two factors, with (DIF) and (DDF), showing good psychometric properties. However, (EOT) was not found to be as reliable. (See Appendix 1b)

Healthy lifestyle habits: Fruit and vegetable daily intake was scored on a 4 point likert scale; with one equalling 0, two equalling 1-2, three equalling 3-4, and four equalling 5 or more portions. Exercise frequency outside school was scored on a 5 point likert scale; with one equalling very often, two equalling often, three equalling sometimes, four equalling rarely, and five equalling never. The results for this measure were reported reverse scored, meaning a high score of 5 was scored as very often, and the lower score of 1 was interpreted as never. Unhealthy snacking (e.g. crisps, chocolate, cake) was scored on a 4 point likert scale; with one equalling daily, two equalling almost daily, three equalling sometimes, and four equalling rarely; this was reported reverse scored, meaning a high score of 4 was scored as a daily, and a low score of 1 was scored as rarely. (See Appendix 2b)

Psychological distress: Psychological distress was measured with four items: Do you feel hopeless about the future (in last two weeks); do you feel unhappy; sad, or depressed (in last two weeks); do you worry too much about things (in last two weeks); do you feel nervous or tense (in last two weeks). Each item was on a 3 point Likert scale ranging from one equalling

'a lot', two equalling 'somewhat', three equalling 'not at all'. All 4 items were previously selected in the wider study. They were selected due to their focus on emotional states (depression, hopelessness, and anxiety) and were the most closely related to the feeling of depression and anxiety, which are factors that may be related to eating behaviour. Due to all the items being closely related in nature and highly correlated with one another, it was deemed appropriate to combine them into one and to reverse scored so that a high score meant a lot of distress. (See Appendix 3b). The psychological distress tool was not based on a previous measure, it was developed by combining individual question items that had been included in the online questionnaire portion of the study. These items were not from a previous questionnaire, they were created by researchers when designing the original study.

Other measures

Body Mass Index/Z-score: Height and weight were measured by a scale and tape measure by researchers to compute Body Mass Index (BMI). BMI in addition to gender and age were used to compute a z-score for each participant.

3.3 Statistical Analysis

Scores for each alexithymia subscale were calculated for each participant, giving each subject their own value for three Alexithymia (Rieffe, Oosterveld, and Terwogt 2006) categories; DIF, DDF, EOT. A score was also calculated for every student's BMI and z-score, vegetable intake, exercise outside of school, and daily unhealthy snacking. Body Mass Index (BMI) z-scores were calculated using height and weight (for BMI) and Excel Macro used to determine a z-score. The z-score is a comparison of a child’s BMI to other children within their sex/age group. Each participant z-score was calculated and used for analysis. All statistical analyses

were conducted using SPSS (version 17.0).

3.4 Results

Participant Characteristics

All participants were randomly selected from the 9 schools that were visited during the larger study. Sixty-seven percent of participants were of white British/white non-British descent, seventeen percent were Indian or Pakistani descent, while the remainder with a mixture of mixed race, Chinese, and other ethnicities. There were no gender differences in age, BMI z-score, or alexithymia score, which was out of a minimum score or 20 and maximum score of 60 (See Table 1). Out of a scale of 1 through 4, with one being 0 and four being 5 or more portions per day , the mean fruit and vegetable intake on the Likert scale was 3.1 SD+/-.718 which corresponds with response number 3 (three to four per day). Out of a scale of 1-4, the mean daily unhealthy snack intake was 2.7 (±.9), which corresponds to between ‘almost daily’ and ‘sometimes’. Out of a scale of 1-5, the mean exercise frequency was 3.9 (±.9), which corresponds to just under ‘rarely’.

Table 1. Participant Characteristics, Alexithymia scores, and lifestyle measures in Adolescents

Means	**Male**	**Female**	**Total**	**t**	**P-values**
Participants Age (years)	60 12.1 (± .6)	50 12.4 (±.6)	110 12.2 (± .6)	-2.098	.40
Body Mass Index (BMI)	18.8 (±2.9)	19.8 (±3.2)	19.3 (±3.1)	-1.734	.09
BMI Z-score	.33 (±1.2)	.34 (±1.2)	.3 (±1.2)	-.067	.94
Alexithymia (Total) (20-60)	33.6 (±4.9)	34.0 (±5.0)	33.8 (±4.9)	-.475	.63
Subscales: Difficulty Identifying Feelings (DIF) (7-	10.7 (±2.9)	11.4 (±2.7)	11.0 (±2.8)	-1.166	.24

21)					
Difficulty Describing Feelings (DDF) (5-15)	8.9 (± 2.6)	9.2 (±2.6)	9.0 (±2.6)	-.552	.58
Externally Oriented Thinking (EOT) (8-24)	15.8 (± 2.0)	15.4 (±2.5)	15.6 (±2.2)	.888	.38
Unhealthy Snack intake (1-4)	2.7 (±.9)	2.7 (±.9)	2.7 (±.9)	.038	.97
Fruit & Vegetable intake (1-4)	3.1 (±.7)	3.2 (±.7)	3.1 (±.7)	-.701	.48
Exercise frequency (1-5) Psychological Distress (1-12)	4.0 (±.8) 6.2 (±2.3)	3.7 (±1.0) 7.0 (±2.4)	3.9 (±.9) 6.6 (±2.4)	2.012 -1.770	.62 .08

Correlations for BMI z-score, Alexithymia, and lifestyle habits

There were no significant relationships between BMI z-score and most measures. However, self-reported unhealthy snacking was significantly negatively correlated with BMI z-score (see Table 2). Alexithymia total score was significantly negatively correlated with self-reported fruit and vegetable intake. Furthermore, Difficulty Identifying Feelings (DIF) was significantly negatively correlated with self-reported fruit and vegetable intake and self-reported exercise frequency outside school. Difficulty Describing Feelings (DDF) was similarly negatively correlated with self-reported fruit and vegetable intake and self-reported exercise frequency outside school, while unhealthy snacking appears to be trending towards a positive correlation. EOT was not correlated with any of the outcome measures. Psychological distress (PD) did not correlate with alexithymia total scale, alexithymia DIF, alexithymia EOT, or with self-reported unhealthy eating habits or fruit and vegetable intake. PD did however correlate negatively with exercise and Alexithymia DDF, and positively correlated with BMI z-score (Table 2).

Table 2. Pearson's correlations between Alexithymia, Psychological distress, Lifestyle habits, and Z-scores

	Alex	DIF	DDF	EOT	Unhealthy Ss	F&V	Exercise	BMI Z-score	PD
Alex	1	**.81****	**.82****	**.39****	.11	**-.019***	-.16	-.11	-.09
DIF		1	**.70****	-.06	.16	**-.19***	**-.19***	-.05	-.10
DDF			1	-.04	**.17†**	**-.24****	**-.28****	**-.17†**	**-.19***
EOT				1	-.09	.06	.14	-.05	.11
Unhealthy Snacks					1	.12	-.05	**-.21***	.10
F&V						1	**.20***	-.08	.02
Exercise frequency							1	-.04	**-.23***
BMI Z-score								1	**.20***

*p <.05, ** p <.01, † trend
DIF: Difficulty Identifying Feelings, DDF: Difficulty Describing Feelings
EOT:Externally Oriented Thinking, F&V: Fruit & Vegetable daily
Ss: Snacks, BMI: Body Mass Index, Alex: Alexithymia Total Score

Regressions predicting Alexithymia and lifestyle habits

To test the hypothesis that alexithymia DDF predicts fruit and vegetable intake, exercise frequency, and BMI z-score, a multiple linear regression analysis approach was used. Based on the correlations observed, the following predictor variables were chosen; gender, age, BMI z-score (except for the BMI z-score regression model), and psychological distress (PD) and ethnicity. The predictors selected were correlated with the outcome variables (fruit and vegetable intake, exercise frequency, and BMI z-score). When predicting fruit and vegetable consumption, alexithymia DDF was most significant factor with a p-value of .06, while alexithymia DIF was not a significant predictor (See Table 3 below). When predicting exercise frequency, alexithymia DDF was once again the most significant predictor with a p-value < .01 at first, while alexithymia DIF and fruit and vegetable intake were not significant predictors. (See Table 4 below). When predicting BMI z-score, neither alexithymia DDF, psychological distress (PD), nor unhealthy snacking was a significant factor (See Table 5 below). When predicting unhealthy snacking (See Table 6 below), neither BMI z-score nor alexithymia DDF were significant factors. Checks demonstrated no multicollinearity issues with all variable VIF's = < 2.

Table 3. Summary of multiple linear regression analysis for Alexithymia variables predicting Fruit and Vegetable Intake

	B[1](±SD)	ß[2]	P-value

DIF	-.01(±.03)	-.03	.78
DDF	**-.07(±.03)**	**-.25**	**.06**

[1]Unstandardized regression coefficient
[2]Standardized regression coefficient
R^2=.10
DIF: Difficulty Identifying Feelings
DDF: Difficulty Describing Feelings

Table 4. Summary of multiple linear regression analysis for Alexithymia variables predicting Exercise frequency

	B[1](±SD)	ß[2]	P-value
Model 1a			
DIF	.01(±.04)	.04	.77
DDF	**-.12(±.05)**	**-.34**	**.01**
F&V	.16(±.12)	.13	.15
Model 2b			
DDF	**-.11(±.03)**	**-.32**	**<.001**
F&V	.17(±.12)	.13	.16

[1]Unstandardized regression coefficient

[2]Standardized regression coefficient

1a R^2=.24

b R^2=.23

DIF: Difficulty Identifying Feelings

DDF: Difficulty Describing Feelings

F&V: Fruit & Vegetable daily

Table 5. Summary of multiple linear regression analysis for Alexithymia and PD variables predicting Body Mass Index (BMI) Z-score

	B[1](±SD)	ß[2]	P-value
PD	.17(±.13)	.13	.19
DDF	-.14(±.12)	-.12	.24

Unhealthy Snacks	-.55(±.33)	-.16	.10

[1]Unstandardized regression coefficient

[2]Standardized regression coefficient

1a R^2=36

DDF: Difficulty Describing Feelings

Table 6. Summary of multiple linear regression analysis for Alexithymia and BMI predicting Unhealthy Snacking

	B[1](±SD)	ß[2]	P-value
Model 1a			
BMI z-score	-.12(±.08)	-.15	.14
DDF	-.05(±.03)	.15	.13

[1]Unstandardized regression coefficient

[2]Standardized regression coefficient

1a R^2=.31

DDF: Difficulty Describing Feelings

F&V: Fruit & Vegetable daily

3.5 Discussion

The primary aim of the current study was to ascertain whether alexithymia was associated with BMI and having an unhealthy lifestyle and whether this relationship was mediated by psychological distress. Unhealthy lifestyle was measuring eating habits (fruit and vegetable intake and unhealthy snacking), and exercise frequency. Distress was measured by a

psychological distress (PD) scale. It was hypothesized that participants with higher alexithymia scores would have higher BMI z-scores, poorer eating habits (less fruit and vegetable intake, more unhealthy snacking), lower exercise frequency, and have higher psychological distress.

When compared to other adolescent studies on alexithymia, mean alexithymia scores for this sample were lower (Karukivi et al. 2010; Putte et al 2007; Tozzi et al. 2013; Joukamaa, Taanila, Miettunen, Karvonen, Koskinen, Veijola 2007). However, these studies are based around research investigating issues such as chronic fatigue and anxiety, which may be related to higher alexithymia scores. Additionally, most adolescent studies on alexithymia observe an older age group of adolescents (some as old as 18 years old) which may affect alexithymia levels. Scores for fruit and vegetable intake (about 3-4 servings daily), unhealthy snacking (almost daily), and exercise (sometimes to rarely) were similar to other reports in previous research (Larson, Neumark-Sztainer , Hannan, Story 2007; Boynton-Jarrett, Thomas, Peterson, Wiecha, Sobol, Gortmaker 2003;)

In accordance with the hypothesis, Alexithymia total score was negatively correlated with fruit and vegetable intake. It was not correlated with BMI z-score, unhealthy snacking, exercise frequency, or PD. However, when the scale is broken down into its three subscales (DIF, DDF, EOT), some of the predicted relationships are visible. This may be due to the Externally Oriented Thinking (EOT) subscale, which has been previously mentioned as less effective by Rieffe and colleagues (2006), potentially diluting the alexithymia measure. Interestingly, alexithymia EOT was not found to be correlated with the other subscales (DIF and DDF), which supports the findings of Rieffe and colleagues (2006).

Alexithymia DIF was found to be negatively correlated with both fruit and vegetable intake and exercise frequency. It was not correlated with BMI z-score, unhealthy snacking, or PD. In accordance with the hypotheses, alexithymia DDF was negatively correlated with fruit and vegetable intake, and exercise frequency. This indicates than both fruit and vegetable consumption and exercise outside of school are associated with DIF and DDF alexithymia scores. When identifying the strongest predictors for lifestyle measures, the alexithymia DDF scale was the most significant factor for both fruit and vegetable intake and exercise frequency. In a study by Helmers and Mente (1999) on males aged 18-45, subjects completed the TAS-26 along with questionnaires measuring lifestyle habits; alcohol and drug intake, sedentary lifestyle, nutrition, and risky sexual behaviour. They reported that alexithymia and its sub-scale (DIF) were associated with poor nutrition, and the DIF was associated with greater alcohol and drug intake. Difficulty Communicating Feelings (DCF) (which corresponds with the children's Difficulty Describing Feelings DDF) was associated with sedentary lifestyle. Therefore, the present results add to evidence that alexithymia effect poor lifestyle choices, such as sedentary habits.

Previously mentioned literature (Mazzeo and Espelage (2002), Hund and Espelage (2005) indicated that PD and measures such as depression may have a relationship with alexithymia and eating behaviours. It may also be the case that the PD measure used here did not accurately measure PD; however PD is positively correlated with BMI z-score which is consistent with the previous findings (Sjoberg, Nilsson, Leppert 2005). An alternative explanation is that the relationship is associated with other factors that relate to alexithymia such as disruptive behaviour (Mannimen et al. 2001), impulsivity (Ouwens, van Strien, van

Leeuwe 2009) which may lead to reduced motivation to engage in social behaviours such as group exercise (Helmers and Mente 1999). Another possibility is that difficulty in identifying feelings, which has been related to general difficulties in appreciating bodily sensations (interoception), is a factor influencing food choices, although this remains to be specifically investigated.

An important result to address is the lack of relationship between alexithymia and BMI z-score. As mentioned earlier, the alexithymia scale was broken down into its three subscales for analytical purposes. This revealed a subscale (EOT) which did not correlate with the other items, and possibly decreased the significance of the alexithymia total score correlations. This led to identifying alexithymia DDF as the strongest factor when predicting lifestyle outcomes, excluding BMI z-score. However, alexithymia DDF was trending toward a positive relationship with BMI z-score. An explanation as to why a clearer relationship was not found may be due to a relatively lean sample (mean BMI z-score .3). Use of a population with a wider BMI z-score range may have provided a more pronounced effect during analysis of this measure. As discussed above with the Helmers and Mente (1999) findings, alexithymia appears to be related to an unhealthy lifestyle generally although in the present study it was not associated with self-reported intake of unhealthy snacks. It may be that in this population alexithymia is more associated with under nutrition rather than overeating. It is also possible that a more significant relationship between BMI and alexithymia would emerge later during adolescence as a result of continuing unhealthy behaviours. No gender differences were observed for alexithymia scores which does coincide with previous research for adolescents (Joukamaa et al. 2007; Karukivi et al. 2010; Tozzi et al. 2010).

There are some limitations to consider when evaluating the study outcome measures. Firstly, all measures (excluding BMI z-score) were self-report. The scores do not indicate over/under reporting, but self-report based studies may sometimes be inaccurate. A study that assessed eating disorders through interview and questionnaire format found similar scores overall, except questionnaires reported higher scores in binge eating and body shape concerns (Fairburn, Beglin, Phil 2006). In future studies, the use of different measures may prove beneficial. For instance, food laboratory based studies would enable researchers to monitor exact quantities and nutritional content of the food consumed. In slightly older populations, food diaries may also be of use. Another option would be to enlist the help of parents in monitoring food intake in adolescents, or to simply compare their reports to their children's. However, self-report measures are still seen as useful tools as long as response accuracy is maximized (Boca and Noll 2002). To address this in the current study, researchers were on hand throughout to provide supervision and assistance during questionnaire administration. The Alexithymia Questionnaire for children (Rieffe, Oosterveld, and Terwogt 2006) is based on the Toronoto Alexithymia Scale 20 (TAS-20) (Bagby, Parker, Taylor 1994) and is therefore relatively new. However, it is very similar to its predecessor but with simpler language to make it more accessible to adolescents and has been validated (Rieffe, Oosterveld, and Terwogt 2006). The limitations of using stepwise regression with backward elimination must be considered as generalizability of data may be an issue. However, the sample was representative of the population and so generalizability issues would not be substantial. This method also allows for identifying the most important predictors within this sample. Another clear limitation was the inability to choose more of the questionnaires and being limited to adding the Alexithymia questionnaire. For instance, the lifestyle measures used for fruit and vegetable intake, unhealthy snacking, exercise frequency, and psychological distress items

were designed for the larger study to be concise and easy to complete among a battery of other measures. In the case of fruit, vegetable, and unhealthy snack intake, it may have been more useful for the parents to report the intake on behalf of the adolescents. At this young age parents may provide a more accurate report as they could be more aware of how food is categorized into 'healthy' and unhealthy' categories. That said, parental reporting may also be inaccurate due to parents over reporting healthy food and underreporting unhealthy food, due to embarrassment. Another preferable measure would have been to request specific recall of all food items eaten the previous day. That may have been a less biased way of assessing typical daily intake. Psychological distress (PD) was a measure created by combining four previously included items. A more appropriate measure may have been a questionnaire such as the Children's Depression Inventory (CDI) (Kovacs 2005). However, the items used were straightforward self-report. For example, 'Do you feel unhappy, sad, or depressed?' The items used were also all highly correlated and the relationships expected due to previous literature were seen; PD such as depression has been found to be related to higher BMI/Obesity (Sjoberg, Nilsson, Leppert 2005) and less exercise (Field, Diego, Sanders 2001) in children, and the same was found in this study. Some researchers recommend the use of waist circumference as a better predictor of physical health within children than BMI. For instance, Savva and colleagues (2000) reported that waist circumference in children was a better predictor than BMI for cardiovascular disease. However, a study by Reilly and colleagues (2010) found that the use of waist circumference measurement had no advantage over BMI z-score when diagnosing higher fat mass in children; Garnett and colleagues (2007) reported the same results when investigating cardiovascular disease risk in children. Therefore, BMI is probably a reasonable measure to use and is easy to assess.

A clear advantage of the present study is the large sample size. In addition, to our knowledge it is the first of its kind to collect alexithymia and lifestyle and eating behaviour information in adolescents. This novel study provides evidence that alexithymia DDF is related to some lifestyle choices, specifically lower fruit and vegetable intake, unhealthy snacking, and lower exercise frequency in adolescents. The results also provide support for a potential relationship with unhealthy snacking and BMI z-score. Additionally, the results highlight a distinction between alexithymia (a form of emotional dysfunction) and psychological distress. Due to the scarcity of research in this area, the current study may be used as a basis for future investigations on alexithymia, eating behaviour, and BMI within adolescent and adult populations. The present findings support the idea that the observation and treatment of alexithymia, particularly in adolescence, may assist in improving health outcomes. Emotional problems should be taken into account when examining cognitive function and obesity in young people.

Chapter 3 Summary

The study predicted that higher Alexithymia scores would be associated with poorer lifestyle habits, such as lower fruit and vegetable intake, lower exercise frequency, and higher BMI z-score. Results indicated that higher Alexithymia total score was negatively correlated with self-reported fruit and vegetable intake. The Alexithymia DDF measure was the most aligned with study predictions. Alexithymia DDF was negatively correlated with fruit and vegetable intake, and exercise frequency, and trended towards being positively correlated with BMI z-score and unhealthy snacking. Psychological Distress was correlated (negatively) with Alexithymia DDF, but did not mediate the Alexithymia DDF and lifestyle relationship.

Findings suggest that the measure for Alexithymia DDF may be associated with lifestyle habits in adolescent populations.

CHAPTER 4: Study 3 - Using the Cambridge Neuropsychological Test Automated Battery (CANTAB) to assess cognition in lean, overweight, and obese participants.

4.1 Introduction

The results from Chapters 2 and 3 suggest that obesity and behaviours associated with obesity are linked to problems with emotional processing. There is also an expanding literature suggesting that obesity is associated with impairments in a range of cognitive functions such as memory (Farr et. al 2008) and executive function (such as planning and attention; Gunstad, Paul, Cohen, Tate, Spitznagel, Gordon 2007). Furthermore, cognitive dysfunction may even be responsible for the significant incidence of poor weight-loss outcomes post-bariatric surgery; patients who were not achieving or maintaining the desired weight loss, may be finding it difficult to follow the necessary guidelines due to cognitive deficits (Galioto, Gunstad, Heinberg, Spitznagel 2013). If this is the case, continued investigation into the nature of these obesity related comorbidities is necessary. The current study will assess cognition through the use of a comprehensive cognitive test battery, the Cambridge Neuropsychological Test Automated Battery (CANTAB).

Cognitive efficiency is crucial in determining how well an individual performs on a day to day basis, and thereby in their quality of life (Cutajar et al.2000). Cognitive processes such as memory, attention, and decision making are necessary for strategizing, accomplishing tasks, and noting mistakes. Perhaps most importantly, our memory makes up a large part of who we are. As seen through degenerative disorders such as Dementia, cognitive degradation can cause fear, depression and stress for the sufferers loved ones (Kaiser and Panegyres 2007). Findings on the relationship between overweight and impaired cognition have been reported for several years. It has been found in individuals who are by all other accounts healthy

(Gunstad et al. 2007); even in athletes, cognitive decline has been seen to relate to elevated BMI (Fedor and Gunstad 2013).

There are two important issues that have yet to be fully addressed by previous research. The first issue is whether all the cognitive impairments seen in obesity are related specifically to the obese state or whether co-morbidities and/or confounding variables also account for a large portion of the effect. There are some reports that when confounding variables are controlled for then the relationship between cognitive deficits and obesity is modest at best. Stanek and colleagues (2013) recently published a study detailing observations between cognitive behaviour and Body Mass Index (BMI). Measures included language, memory, motor function, and executive function, along with demographic information. Participants were excluded when they presented neurological damage, common obesity medical issues, or previous psychosis. They found obese individuals were affected when it came to motor and attention/processing speed. In addition, an interaction between BMI and age was reported. However, BMI was not seen to otherwise affect cognition. It should also be noted that Gunstad and colleagues (2007) had previously found no interaction between Body Mass Index (BMI) and age. However, the measures taken did not include traits such as eating behaviour, impulsiveness, self-esteem, or current depression state.

There is further evidence that comorbidities associated with obesity may contribute to cognitive breakdown. Saczynski and colleagues (2008) reported that type II diabetics (mean BMI greater than 25) are at higher risk for cognitive deficits. Through the CANTAB Spatial Working Memory Task (SWM) and other batteries they observed speed, memory, and executive function in 1,917 male and female diabetics (without dementia). They controlled for

demographic differences and medical conditions. The results indicated that varying levels of impairment appeared across time in diabetics, undiagnosed diabetics, and medically managed diabetics. Undiagnosed diabetics performed the worst overall. It has also been reported that that patients with type II diabetes had longer reaction times and poorer spatial planning (Lasselin et al. 2012). Another study using the CANTAB (SWM), reported cognitive dysfunction for type 1 Diabetic children when compared to healthy controls (Kaufmann et al. 2012). Interestingly, type II diabetics (mean BMI of 27 or higher) being treated with both rosiglitazone and glyburide had improved working memory, particularly on the CANTAB task Paired Associates Learning (PAL) (Ryan, Freed, Rood, Cobitz, Waterhouse, Strachan 2006). This implies that if there are in fact deficits in cognition, they may be reversible. Nevertheless, the presence of diabetes should be controlled for in any study of cognitive function in obesity.

Anxiety and depression have strong ties to obesity and cognitive performance (Jorm et al. 2007). Van Tol and colleagues (2011) conducted a study analysing functional MRI during performance of a Tower of London Task (Shallice, 1982) in participants with Manic Depressive Disorder (MDD), MDD and Anxiety Disorder (AD), and AD without MDD. They concluded that no differences appeared between controls and participants with AD, though depending on the state of MDD, a slight lag in performance may occur. This lag was accompanied by increased left dorsolateral prefrontal activity. Another study (Bourke et al. 2012) using the CANTAB test battery, found that when compared to healthy controls and participants with Social Anxiety Disorder (SAD), participants with Major Depressive Episode (MDE), were impaired in verbal learning and SWM. The SAD participants had more difficulty accurately classifying neutral words (grouping them as angry or sad) when

compared to MDE and healthy control counterparts. Therefore, any assessment of cognitive function in obesity should also control for depression.

The second issue yet to be fully addressed in research into links between obesity and cognitive function is whether obesity is associated with a specific pattern of cognitive deficits or whether it is associated with a more general decline in cognitive function across several cognitive domains. Here, it is important to consider how cognition is measured. One study (Galioto, Garcia, Spitznagel, Strain, Devlin, Crosby, Mitchell, Gunstad 2013) reported that the Mini-Mental State Exam (MMSE) in particular, was not sensitive to impaired cognition in a sample of obese individuals. This may imply that other cognitive measures, such as the previously documented CANTAB, are better suited to obesity-cognition research.

As discussed earlier, cognitive reserve (or pre-morbid intelligence) may be related to how cognitive deficits are affected by obesity (Galioto et al. 2013). It should also be noted that central obesity has specifically been associated with cognitive impairment (using a CANTAB test battery for declarative memory) when stressed (Lasikiewicz, Hendrickx, Talbot, and Dye 2013). However, findings in this area have varied, as the aforementioned review by Fitzpatrick, Gilbert, and Serpell (2013) reported that the obese had more difficulties in other cognitive tasks than normal weight individuals; with fewer difficulties on memory.

Overall, the evidence suggests that deficits in cognitive processes, such as executive function/frontal lobe and memory are related to obesity (Wolf et al. 2007; Cserjesi, Luminet, Poncelet, Lenard 2009; Sabia, Kivimaki, Shipley, Marmot, Singh-Manoux 2009; Jurdak, Lichenstein, Kanarek 2008). Much of the previous literature is limited to observations of

specified groups (for example: older adults or morbidly obese) and a small number of measures (for example: depression or frontal-striatal function). Previous investigations have also neglected to consider several potential confounds. For instance, some studies control for age and depression, but not for other factors that could affect performance such as impulsivity and self-esteem. Due to this, information is missing on the cognitive performance within one sample that represents diversity in age and weight, while considering relevant confounds (depression, impulsivity, education, self-esteem, gender, age) and eating behaviours. This study is designed to investigate the nature of the obesity-cognition relationship across a diverse sample, while controlling for these confounds. We will also be assessing a range of cognitive functions at once, in contrast to limiting testing to one domain. Based on previous findings, it is predicted that, when controlling for these confounds, BMI will be related to poorer cognitive performance, particularly in memory, impulsivity, and strategizing (executive function), while no impairments will be observed for simple task performance that requires little cognitive processing.

4.2 Method

Participants

One-hundred and fifty-three participants (45 males and 108 females) were recruited for this study. Participants were recruited from both the University of Birmingham and Heartlands hospital. However, 145 of the participants were from the University of Birmingham. This was due to difficulties recruiting larger numbers from Heartlands Hospital. The university participants consisted of both staff and students, while the Heartlands Hospital participants were weight management clinic patients. The university students were recruited through the Research Participation Scheme (RPS), while staff were recruited through university

advertising. Staff members with a BMI higher than 25 were included in the study, and paid £10 for their participation. The 8 unpaid participants from Heartlands Hospital were included due to their high Body Mass Index (BMI), which was of particular interest in this study. Further considerations on participant inclusion can be found in the discussion. Ethical approval was obtained from the NRES Committee West Midlands-South Birmingham and the University of Birmingham. The mean age was 31.3 years and mean BMI was 26.1.University recruitment took place through an online portal and posters. Participants were reimbursed with course credits or £10 cash. Recruitment at Heartlands Hospital took place at weight management clinics. These participants were part of a long term weight loss program provided by the hospital. They were provided with travel expenses. Informed consent was obtained, ethical approval was provided by the South Birmingham Research Ethics Committee (National Research Ethics Service), and the study was conducted in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki. The participant information sheet (PIS) can be seen in appendix 6c.

Self-Report Measures

The Beck Depression Inventory (BDI) (Beck et al. 1961) may be used to measure symptoms of depression. It consists of 21 questions and responses are in a Likert scale format. For example, the first item can be answered with 'I do not feel sad' for a score of 0, or 'I am so sad or unhappy that I can't stand it' for the highest score of 3. The questionnaire should take approximately 5 minutes to complete. This measure was selected due to the documented relationship between BMI and Depression (Dragan and Akhtar-Danesh 2007). Obesity and depression have been found to be associated in a number of studies (Onyike et al. 2003;

Stunkard, Faith, Allison 2003; Taylor et al. 2013). A systematic review on obesity and depression reported that obesity increased the risk of depression and that depression was predictive of obesity (Luppino et al. 2010). An interesting study by Roberts and Duong (2013) examined obese adolescents and depression, and reported that obese adolescents were not more likely to be depressed, but that depressed adolescents were more likely to be obese. (See Appendix 1c)

The Self-Esteem Scale (SE) (Rosenberg 1965) may be used to measure a patient's self-esteem. Self-esteem has been linked to depression (Brown, Harris, Adler, Bridge 1986). The patient answers 10 questions asking how they feel about themselves e.g. 'On the whole, I am satisfied with myself.' Responses are in Likert scale format, ranging from strongly agree to strongly disagree. The questionnaire takes approximately 5 minutes to complete. A correlation between weight and self-esteem has been noted (Israel and Ivanova 2002, Kimm et al. 1997), which may begin in childhood. Strauss (2000) conducted a large study on over 1500 children and the results indicated that obese female Hispanic and White children tended toward lower self-esteem. Obese children with lower self-esteem were also more prone to negative feelings such as loneliness and sadness. Franklin and colleagues (2006) investigated self-esteem in obese Australian children and results showed obese subjects were more likely to have lower global self-worth. However, some research has found differently; Kaplan and Wadden (1986) reported little correlation between BMI and self-esteem in inner-city children; Mendelson and White (1992) found a correlation between body esteem and weight but not self-esteem and weight. Also, Wardle and Cook (2005) pointed out a lack of evidence for a relationship within a non-clinical population. (See Appendix 5c)

The Dutch Eating Behaviour Questionnaire (DEBQ) (Van Strien et al. 1986) is used to assess a patient's eating behaviours. Eating behaviour such as restrained eating has been found to be related to higher BMI (Snoek, Engels, Van Strien, Otten 2013). The DEBQ consists of 33 questions which address a variety of eating attitudes and habits. There are 3 subscales: restrained eating, emotional eating, and external eating. An example of an item from the restrained scale would be 'When you have put on weight, do you eat less than you usually do?' Responses are in a Likert format ranging from 1 for 'never' to 5 for 'very often'. (See Appendix 3c)

The Barratt Impulsiveness Scale-11 (BIS-11) (Patton, Stanford, Barratt 1995) can be used to measure a patient's impulsive personality traits. Impulsivity was seen as a relevant measure due to its association with overeating (Guerrierri, Nederkoorn, Schrooten, Martjin, Jansen 2009). This contains 30 questions investigating the patient's thoughts on and behaviours during daily activities and decisions. An example of an item would be 'I plan tasks carefully.' with responses in a Likert format ranging from 1 for 'rarely/never' to 4 for 'almost always/always'. (See Appendix 2c)

CANTAB test battery

The Cambridge Neuropsychological Test Automated Battery (CANTAB) has been used extensively in cognitive research, ranging from the study of executive function and cognitive aging, to learning and memory, and to impairment via neurodegenerative disease (Robbins et al. 1994; Fray, Robbins, Sahakian 1996; Robbins et al. 1998; Egerhazi, Berecz, Bartok, Degrell 2007).

The Frontal Lobe (including Ventral and Medial Prefrontal Cortex)

The Stockings of Cambridge task (SOC) assesses spatial planning ability, which is associated with frontal lobe function. SOC has been used to measure impairments within a variety of populations, such as individuals with depression (Egerhazi, Balla, Ritzl, Varga, Frecska,Berecz 2013; McIntyre, Cha, Soczynska, Woldeyohannes, Gallaugher, Kudlow, Alsuwaidan, Baskaran 2013) and individuals with dementia (Dubbelink, Hillebrand, Twisk, Deijen, Stoffers, Schmand, Stam, Berendse 2014). Attention Go/No-Go (AGN), can be used to detect positive or negative biases, which are associated with the ventral and medial prefrontal cortex. This is particularly useful in depression research, as individuals with anxiety and depression have been found to have a negative word bias when compared to non-anxious and non-depressed controls (Mogg and Bradley 2005; Rude, Wenzlaff, Gibbs, Vane, Whitney 2002). This task has been used to assess information processing bias within different groups, such as teachers (Flook, Goldberg, Pinger, Bonus K, Davidson 2013), adults with ADHD (Surman, Hammerness, Petty, Spencer, Doyle, Napolean, Chu, Yorks, Biederman 2013), and adolescents with major depression (Kyte, Goodyer, Sahakian 2005)

SOC TASK: The participant is presented with two displays (one on the upper screen, one on the lower screen), each containing three coloured balls (See Figure 1 below). The display is to be seen as stacks of coloured balls held in stockings or socks suspended from a beam. This makes the 3-D concepts easier for participants to understand along with oral instructions. First, the test administrator demonstrates to the participant how they can move the balls in the lower screen to copy the pattern on the upper screen. This first trial only requires one move. The participant must then manipulate the display on the lower half of the screen to match the

display on the top half of the screen for a trial that requires 2 moves, 3 moves, and then 4. After this, it is the participant must work out in their head how many moves are required to match the following displays. There are a total of 20 trials. The first 6 trials are used as practice and not assessed. The amount time taken to solve the trial, number of correctly solved trials, and number of moves required to solve the trial are recorded as measures of the participant's planning ability.

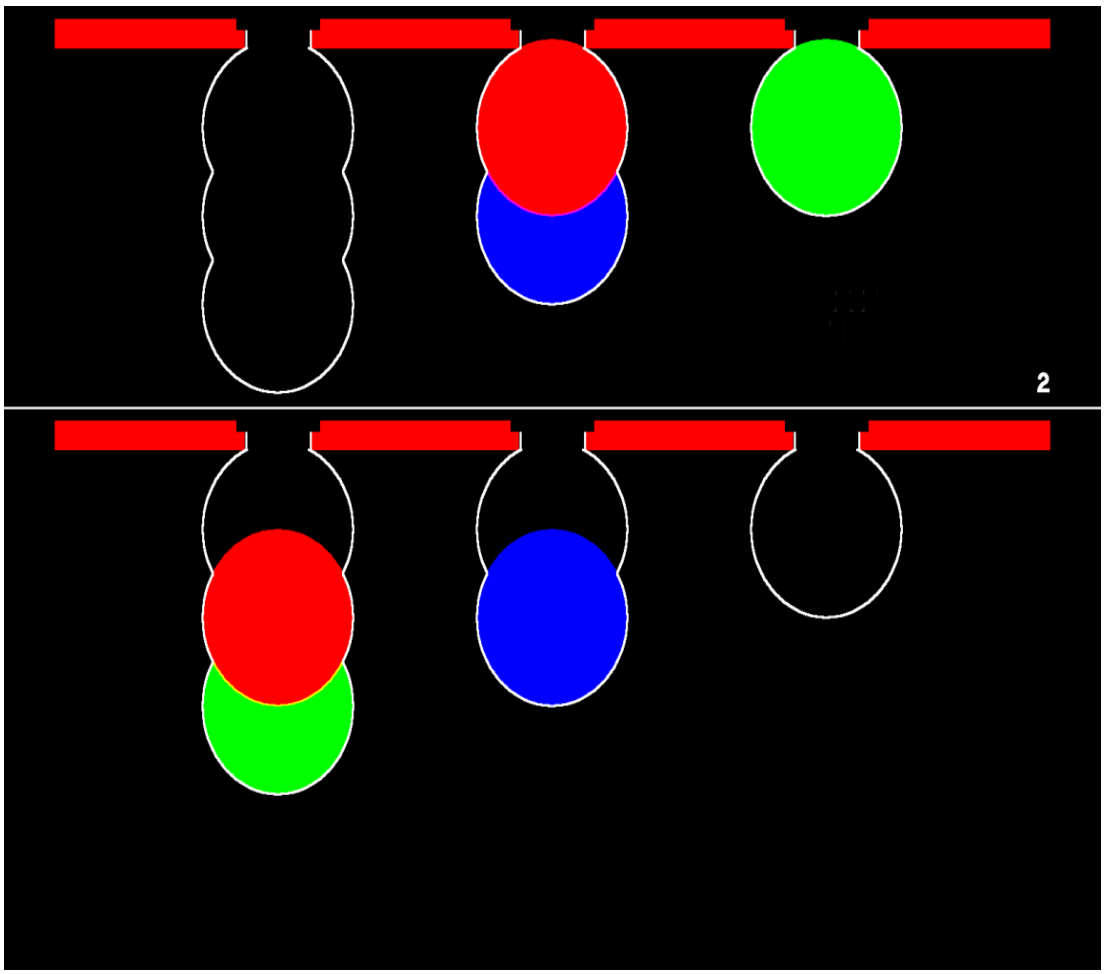


Figure 1. Stockings of Cambridge (SOC) Task: This trial requires 2 moves to solve. First, the red ball must be placed above the blue ball. Second, the green ball must be placed in the empty slot on the far right of the screen.

AGN TASK: The participant is informed that they will be presented with positive (for example: happy, smile, gratitude) or negative (for example: crying, sadness, gloom) words. Each word is presented on the screen for 300 ms followed by a 900 ms break between words (See Figure 2 below). 18 randomly automated blocks are used, each block with a target affective category. At the start of each block, the target affective category is revealed (positive or negative). The participant is directed to press the right button on the press pad when they see a word matching their target category. They are also told to press nothing if the word on the screen does not match their current target category. There are 10 different blocks, consisting of 18 words each, a mixture of positive and negative. The first two trials are for practice, and not scored. In addition number of correct responses (hitting the button when your target category is on the screen), the test monitors false alarms (hitting the button incorrectly when your target category is not on the screen), and misses (not hitting the button when you should). The task takes approximately 10 minutes to complete.

Figure 2. Attention Go/No-Go (AGN)

Frontal-Striatal areas

The Intra-Extra Dimensional Set Shift (IED) assesses set shifting and mental flexibility through tasks on acquisition and reversal. These functions are associated with activity in frontal-striatal areas of the brain. This task was chosen because it assessed visual discrimination ability, perceptual awareness, and flexibility of attention. It has previously been used in studies to identify signs of dementia (Dubbelink, Hillebrand, Twisk, Deijen, Stoffers, Schmand, Stam, Berendse 2014) and poorer mental flexibility in bipolar disorder (Linke, King, Poupon, Hennerici, Gass, Wessa 2013). Big/Little Circle (BLC) task is a practice task for IED and serves as a simplified introduction into the exercise.

For the IED Task (See Figure 3 below), the participant is first trained on the CANTAB Big/Little Circle (BLC) task (See Figure 4 below). This is a simplified visual discrimination task used to familiarize the participant with identifying a rule to follow, and recognizing when the rule has been reversed. This is done by presenting them with a big circle and a little circle, a number of times. The participant must then select the circle they believe is smaller for 20 trials. After the first 20 trials, they are told they must now select the circle they think is larger. This task is also being used as a control measure to examine differences between overweight and lean groups on simple cognitive function. The IED test has a similar premise, but is more complex. It includes visual discrimination, attentional set formation, and shifting and flexibility of attention. The participant is presented with two simple colour-filled shapes, and must guess which one is correct by touching it. The test notifies the participant when they have selected the correct stimulus. At the start, the participant is guessing the rule. Through feedback, they should become aware of the current rule. After six correct selections, the stimuli and/or rules are altered. The shifts are initially intra-dimensional (colour filled shapes are the relevant dimension), then later extra-dimensional (white lines are the relevant dimension). There are 63 trials within 9 blocks, with the last few trials having the extra-dimensional shapes as the correct selection. Participants move forward by providing 6 consecutively correct responses at each stage. The test terminates if, after 6 trials, the participant is unable to meet this goal for a stage.

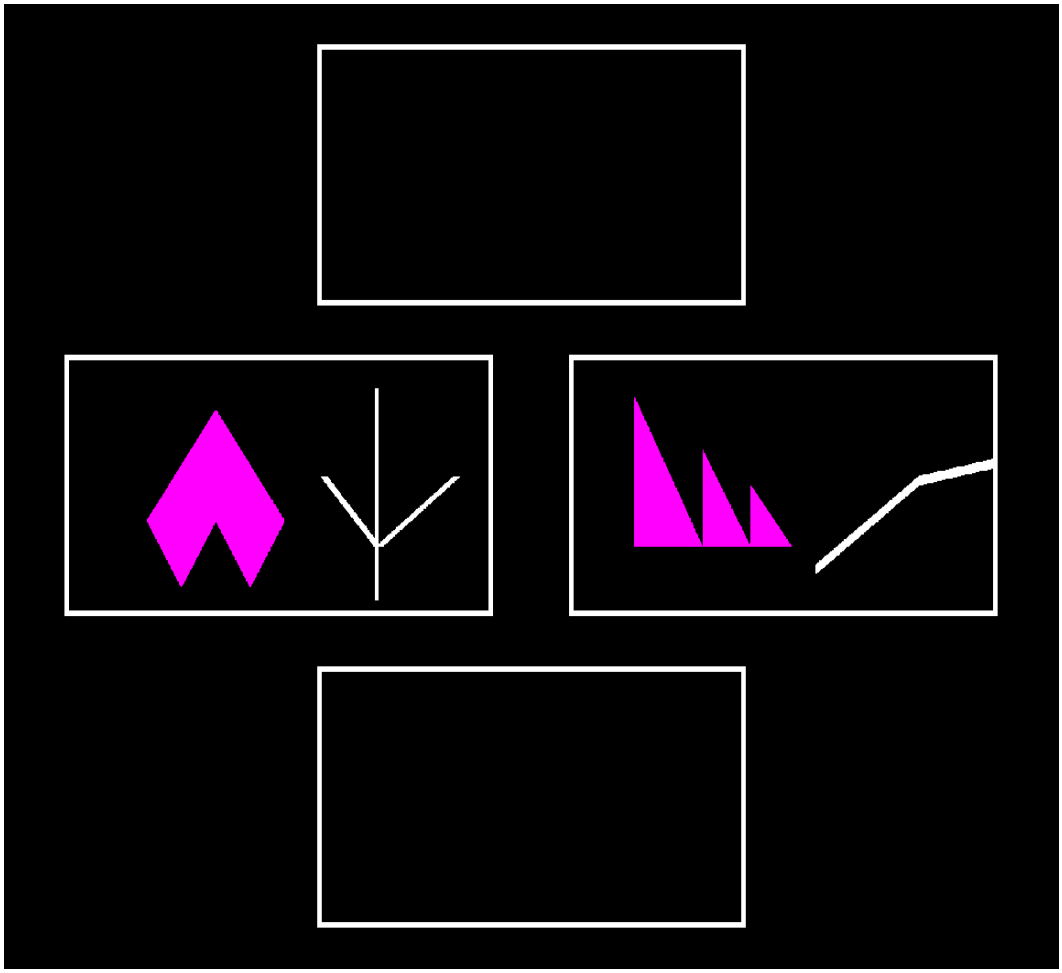


Figure 3. Intra-Extra Dimensional Set Shift (IED) Task: As the task advances a second dimension (white lines) are added to the shapes. These become important when identifying the rule which dictates the correct response.

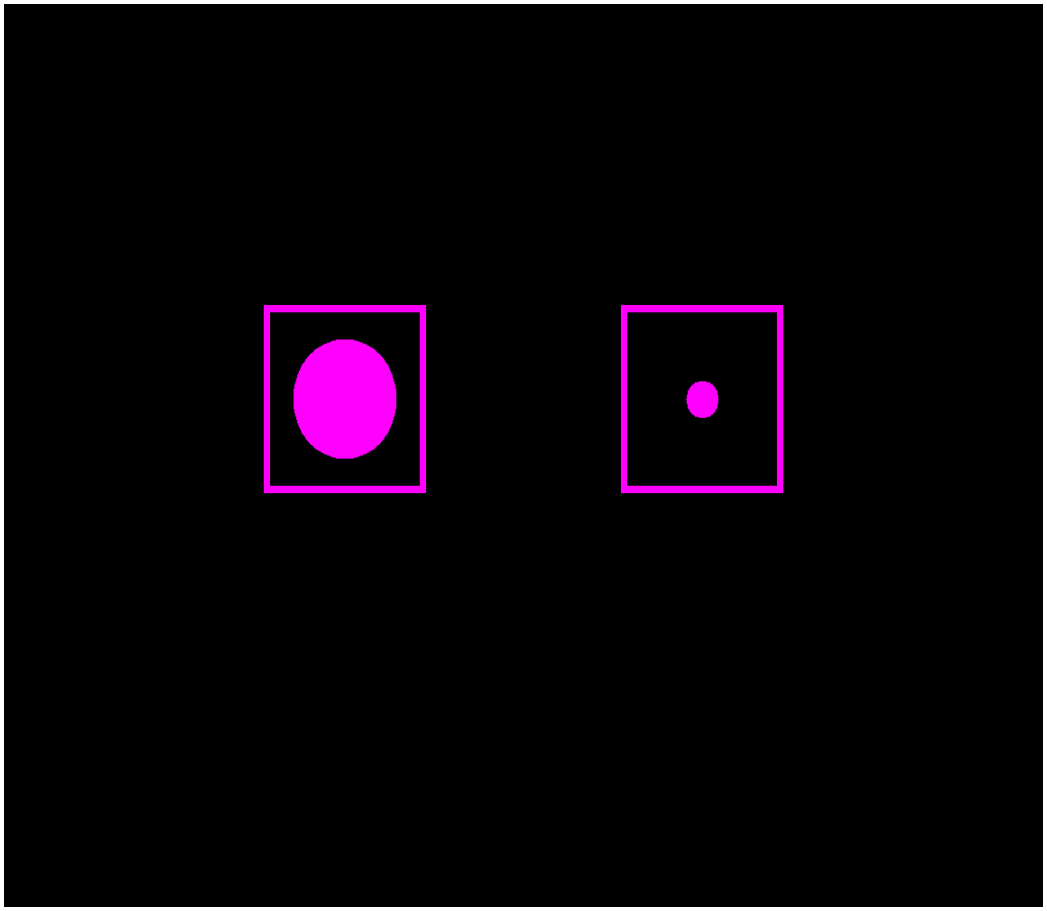


Figure 4. Big/Little Circle (BLC) Task: Practice task for Intra-Extra Dimensional Set Shift (IED) Task. Also used as a control between overweight and lean groups.

The Medial Temporal Lobe area (including Hippocampus)

The Delayed Matching to Sample (DMS) is sensitive to medial temporal lobe damage, and assesses choice recognition memory and short term visual memory. Paired Associates Learning (PAL) detects medial lobe function and hippocampal function, and assesses visual memory and new learning. PAL may also be used to detect diseases that involve impaired memory (Dementia, Alzheimer's, etc.). In previous studies these tasks have been used to detect memory performance in gamblers (Grant, Odlaug, Chamberlain, Schreiber 2012),

individuals with antisocial personality disorder (Dolan 2012), and young binge drinkers (Scaife and Duka 2009).

DMS Task: The participant is shown one sample visual pattern. They are instructed to remember the exact appearance of this pattern as best they can. For the practice trial, there is then a short delay followed by four similar patterns appearing beneath the original sample. The participant must touch the pattern which matches the original sample. This first trial is simple because the participant can still see the original pattern they are asked to remember (See Figure 5 below). The test is made up of 3 stages, and as it progresses, it becomes more complex. For instance, after the practice trial the task goes as follows: The participant is shown one sample visual pattern. They are instructed to remember the exact appearance of this pattern as best they can, because it will now disappear after the delay. After the delay, four similar patterns appearing beneath where the original sample once was. The participant must touch the pattern which matches the original sample. There are a total of 43 trial items. The delay between being shown the original pattern and the four similar answer choices lengthen from 0 at the simultaneous stage, 4000 ms at stage 2, and 12000 ms in stage 3. This task takes approximately 10 minutes to complete.

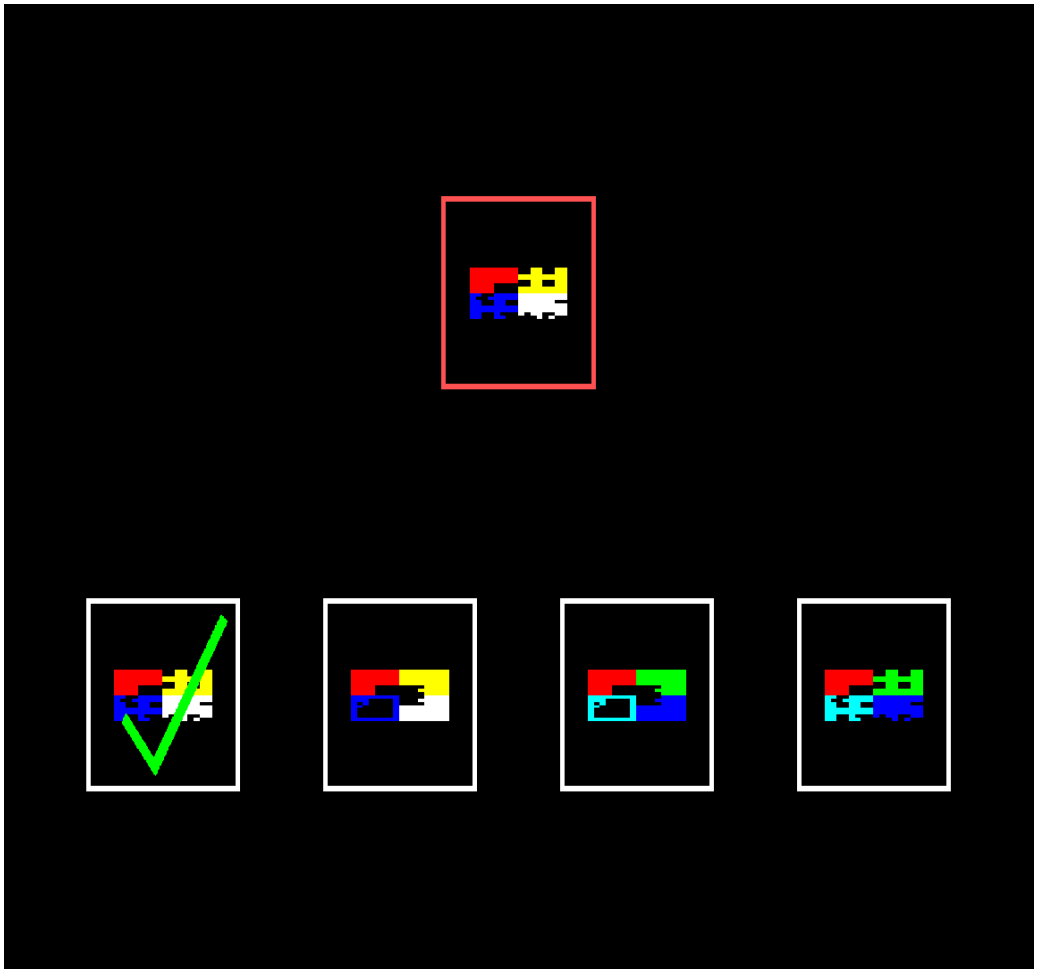


Figure 5. The Delayed Matching to Sample (DMS) Task

PAL Task: The participant is presented with boxes on the screen (See Figure 6 below). They open at random to reveal their contents. The first trial reveals that only 1 box contains a pattern. The participant is instructed to remember the location of this, and any other pattern that may appear within a box. At the end of the trial, the previously revealed patterns appear in the middle of the screen. The participant must then select the box in which the pattern currently displayed was originally located. If the participant makes a mistake, the exact same trial and patterns repeats itself for the participant to try again. The test is made up of 5 stages, with the first serving as a practice trial with the test administrator. As the test progresses, difficulty increases. The first stage requires the participant to remember 1 pattern hidden

among 6 boxes. The first stage requires the participant to remember 1 pattern hidden among 6 boxes. The second stage requires the participant to remember 2 patterns hidden among 6 boxes. The third stage requires the participant to remember 3 patterns hidden among 6 boxes. The fourth stage requires the participant to remember 6 patterns hidden among 6 boxes. The final stage requires the participant to remember 8 patterns hidden among 8 boxes. There are a total of 8 trials, each with a maximum of 10 attempts allowed before failing. Amount of time taken to complete each trial, memory load (the number of items which can be remembered), amount of attempts taken to complete each trial, and whether each trial was completed successfully is recorded.

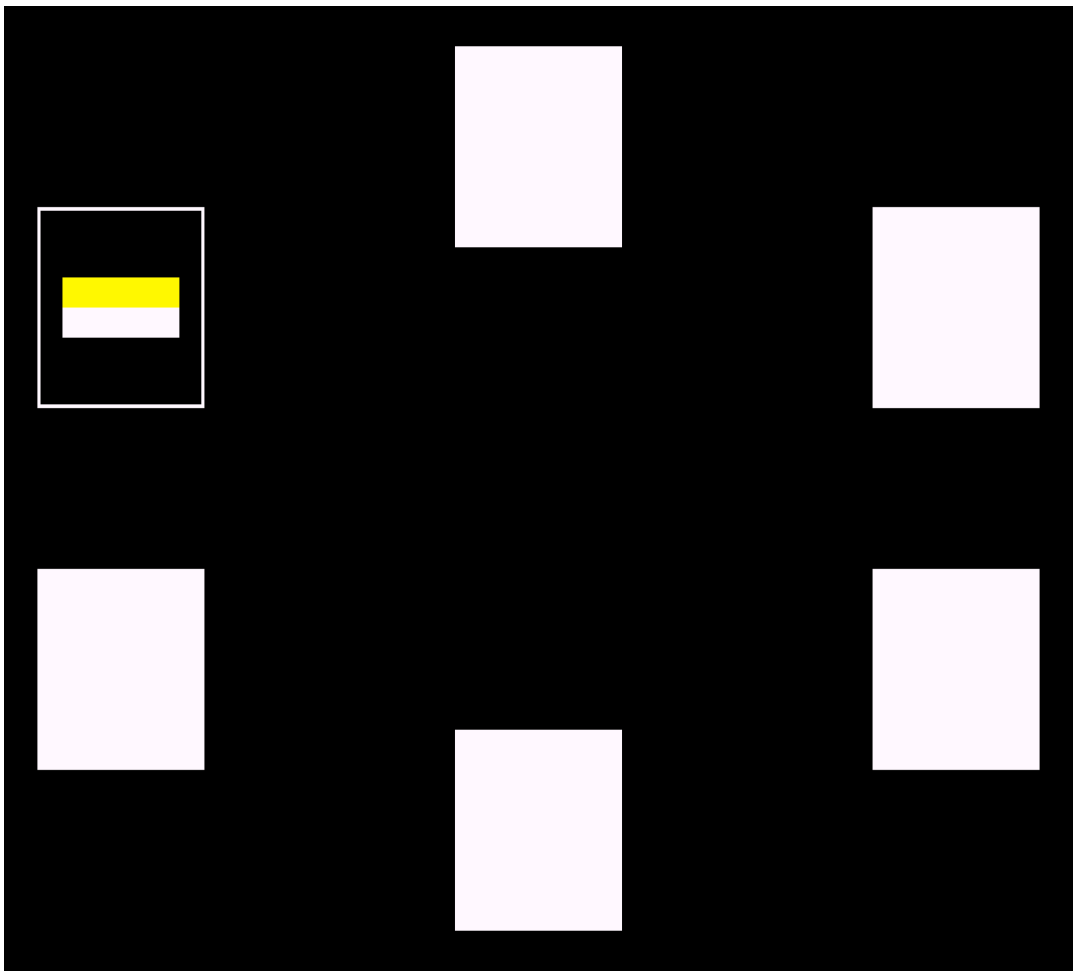


Figure 6. Paired Associates Learning (PAL)

4.3 Statistical Analysis

The aim of this study was to compare individuals across age and Body Mass Index (BMI) categories. It was predicted that participants with higher BMI would vary in performance (when compared to participants with lower BMI) on cognitive tasks (CANTAB). Participants with a BMI over 24.5 were classed as overweight, above 29.5 were classed as obese and 24.4 and under were classed as lean. Deficits in memory, impulsivity, and strategizing (executive function) were expected. These subjects were also predicted to have higher scores in the DEBQ, impulsivity as measured by the BIS, lower self-esteem as measured by SE, and higher scores on depression as measured by BDI. Questionnaire responses were used to calculate individual scores for each of these self-report measures. The same was done for each CANTAB test (5 in total). This was used to estimate several mean scores for each subject. T-tests were run to identify any significant variance between the group's results (Michael and Proschan 2010). The relationships were also observed through correlation and regression analyses. Covariates were used to control for effects of gender, education, self-esteem, impulsivity, age, depression, and ethnicity. Missing data was dealt with by excluding the participant from analysis when their data was not available, and including them in analysis for which their data was available. When considering the participants there is the question of whether the hospital subjects should be included in the analysis because they are from a distinct population. However, when comparing the hospital participants to the university participants (both staff and students), there are no differences between them outside of BMI. When observing the results without the hospital participants, they remain the same but less pronounced. Therefore, it was decided to conduct the analysis on the whole group. SPSS Exploratory analysis was run to check for outliers, which were recognized by being 3

standard deviations above or below the typical mean of the group. These were then removed. All statistical analyses were conducted using SPSS (version 17.0).

4.4 Results

Participant Characteristics

A diverse participant sample was obtained with sixty-two percent of participants of white British/white non-British descent; eighteen percent were Indian or Pakistani descent, while the remainder were a mixture of mixed race, Chinese, and other ethnicities. Few gender differences appeared when observing the self-report measures. Males were slightly older and had higher BMI (not significant), while females scored significantly higher on emotional eating (See Table 1). It is important to note that only 29.4% of the sample was male. 5.9% of the sample reported they were smokers, 75.2% of the sample reported that they drank alcohol. The sample was relatively well educated with 5.2% at the GCSE level, 49% at A-level, and 45.7% at degree level or above. An ANOVA indicated that there was an interaction between BMI and gender when observing education $F(1,149)= .135$, $p= .02$. Further inspection demonstrated that lean females had the lowest education score (2.3 (±.16); equivalent to A-levels), followed by overweight females (2.7 (±.18); equivalent to just under a university degree) and males have very similar education scores (2.8 (±.18); equivalent to just under a university degree), with lean males being the most educated (3.0 (±.13); equivalent to a university degree).

Table 1. Participant characteristics, BMI, and behavioral measures for CANTAB study

Means	Male	Female	Male & Female	**t**	P-values
Participants Age (years)	45 34.3 (± 13.8)	108 30.0 (±13.7)	153 31.3 (±13.8)	1.768	.08
BMI	27.7 (±6.4)	25.5 (±6.9)	26.1 (±6.8)	1.879	.06
DEBQ (1-5): Res	2.4 (±.8)	2.6 (±.9)	2.6 (±.87)	-1.872	.06
Ext	3.0 (±.5)	3.1 (±.6)	3.0 (±.6)	-1.048	.30
Emo	**2.2 (±.8)**	**2.7 (±1.1)**	**2.6 (±1.1)**	**-3.026**	**<.01**
BIS (1-4)	2.0 (± .32)	2.1 (±.37)	2.1 (±.35)	-.685	.49
BDI (0-63)	7.4 (± 5.5)	8.4 (±8.9)	8.0 (±8.0)	-.680	.49
SE (0-30)	20.5 (±5.1)	20.3 (±6.0)	20.4 (±5.7)	.122	.90

DEBQ: Dutch Eating Behaviour Questionnaire
Res, Ext, Emo: Restraint scale, External scale, Emotional scale
BIS: Barratt Impulsiveness Scale
BDI: Beck Depression Inventory
SE: Rosenberg Self-esteem Scale

When separated into weight categories (Table 2) overweight and obese individuals had significantly higher BMI, age, dietary restraint scores, and depression scores than the lean participants.

Table 2. Participant Characteristics and behavioral measures, lean versus overweight participants

Means	Lean	O&O	**t**	P-values
Participants	75	78		
Age (years)	**24.9 (± 9.3)**	**37.4 (±14.8)**	**-6.272**	<.**001**
Body Mass Index (BMI)	**21.5 (±1.7)**	**30.6 (±6.9)**	-11.034	**<.001**
DEBQ (1-5):				
DEBQ Res	**2.4 (±.9)**	**2.7 (±.8)**	-2.111	**<.05**
DEBQ Ext	3.0 (±.58)	3.0 (±.6)	-.114	.90
DEBQ Emo	2.5 (±1.0)	2.6 (±1.1)	-6.17	.53
BIS (1-4)	2.1 (± .33)	2.0 (±.37)	.965	.34
BDI (0-63)	**6.6 (± 6.7)**	**9.5 (±8.9)**	-2.337	**<.05**
SE (0-30)	21.1 (±5.7)	19.6 (±5.7)	1.637	.10

DEBQ: Dutch Eating Behaviour Questionnaire
Res, Ext, Emo: Restraint scale, External scale, Emotional scale
BIS: Barratt Impulsiveness Scale
BDI: Beck Depression Inventory
SE: Rosenberg Self-esteem Scale
O&O: Overweight & Obese

Cognitive outcomes by weight category

Performance on the CANTAB according to weight category was analysed (lean and overweight BMI categories). Overall, overweight and obese participants appeared to lag in performance latency times, significantly so on DMS overall latency, PAL overall latency, PAL number of trials to locate pattern, SOC thinking time, AGN misses on sad words, and IED latency for the later stages (See Table 3).

Table 3. CANTAB cognitive task performance means for lean versus overweight participants

Means	**Lean**	**O&O**	**Overall**	**t**	**P-values**
DMS: Overall Latency	**3075.3**	**3383.5**	**3232.4**	**-2.254**	**<.05**
Memory load	10.4	8.2	9.3	-1.487	.14
Total Errors	3.6	3.1	3.4	1.197	.23
PAL: Overall Latency	**1140.1**	**1246.8**	**1195.2**	**-2.670**	**<.05**
Total Errors	6.2	7.6	6.9	-1.156	.06
# of trials to locate pattern	**6.3**	**7.1**	**6.7**	**-2.038**	**<.001**
SOC: Problems solved in Minimum moves	8.9	8.4	8.6	1.877	.06
Thinking time total	**2663.7**	**3032.7**	**2853.3**	**-3.321**	**<.001**
AGN: False Alarms	7.5	8.1	7.8	-.569	.57

Total Misses (sad)	**1.4**	**4.1**	**2.7**	**-3.268**	**<.001**
Total Misses (happy)	3.1	4.7	3.8	-1.297	.20
Latency (sad)	959.5	998.7	977.4	-1.871	.06
Latency (happy)	957.0	988.2	971.0	-1.590	.11
BC/LC errors:	1.6	1.7	1.6	-.549	.58
IED: Latency (stages 8-9)	**2035.9**	**2265.4**	**2157.6**	**-2.743**	**<.001**
IED Total Errors	13.9	14.2	14.1	-.220	.83

DMS: Delayed Match to sample task
PAL: Paired Associates Learning task
SOC: Stockings of Cambridge task
AGN: Attention Go/No Go task
BC/LC: Big Circle/Little Circle control task
IED: Intra-Extra Dimensional Set Shift

Correlations between BMI, age, and personality variables

BMI was positively correlated with age, DEBQ emotional eating, impulsivity (BIS), and depression (BDI). BMI was negatively correlated with self-esteem (SE) and education level. It was not correlated with DEBQ restrained and external eating (See Table 3).

Table 4. Pearson's correlations between BMI, Age, and behavioural measures

	BMI	Age	Res	Ext	Emo	BIS	BDI	SE	Edu
BMI	1	**.50****	.11	-.04	**.17***	.02	**.31****	**-.17***	**-.284****
Age		1	.08	-.12	.02	**-.21****	.14	-.01	.08
Res			1	-.01	**-.19***	-.12	**.23****	**-.26****	-.04
Ext				1	**.41****	**.21****	**.17****	-.00	-.15†
Emo					1	**.25****	**.37****	**-.31****	**-.33****
BIS						1	**.25****	**-.23****	**-.34***
BDI							1	**-.67****	-.15†
SE								1	**.19***

*p <.05, ** p <.01, † trending
BMI: Body Mass Index
DEBQ (Dutch Eating Behaviour Questionnaire): Res, Ext, Emo: Restraint scale , External scale , Emotional scale
BIS: Barratt Impulsiveness Scale
BDI: Beck Depression Inventory
SE: Rosenberg Self-esteem Scale Ss: Snacks

BMI was correlated with some measures of performance on the CANTAB, particularly latency. For the Delayed Match to Sample (DMS) measure, BMI was significantly correlated with overall latency (r= .360; p<.001). For the Paired Associates Learning (PAL) measure, BMI was significantly correlated with overall latency at (r= .207, p<.05), total errors (r=.218, p<.01), and number of trials to complete all stages (r=.209, p<.01). For the Attention Go/No-Go (AGN), BMI was correlated with total misses for sad words (r=.197, p<.05) and total latency for sad words (r=.171, p<.05), but not with any other measures. For the Intra-Extra Dimensional Set Shift (IED) measure, BMI was correlated with mean latency at stages 8-9 (r=.267; p<.01), but not with total errors (r=.119). For the Stockings of Cambridge (SOC) measure, BMI was correlated with total mean thinking time (r= .252, p<.01), but not with

problems solved in minimum moves (r=-.073).

Regressions predicting cognitive performance

To test the hypothesis that BMI predicts cognitive performance, a multiple linear regression analysis approach was used. Based on the correlations observed, the following predictor variables were chosen; age, gender, BDI, BIS, self-esteem (SE), education level (Edu), and ethnicity. The predictors selected were correlated with the outcome variables for cognitive performance, which were DMS latency, AGN total misses for sad words, and IED latency. For DMS latency, age was the most significant predictor followed by BMI and gender (see Table 5). However, DMS latency scores did not vary significantly between males and females. For AGN, gender was the best predictor for total misses for sad words (see Table 6). Scores did vary significantly between males and females, with females averaging fewer misses in the sad words than males. For IED latency in stages 8-9, age was the most significant predictor followed by gender (see Table 7). Females had significantly faster latency times than males in these stages. Age was the main predictor for latency, errors, and number of trials on PAL. Age was also the main predictor on SOC thinking time. Multiple regressions were carried out on each of the measures to check for BMI-Gender interactions, of which none were found. Some interactions between age and BMI were found for the following: IED latency in stage 6 (R^2=.19, ß = .218, p= .050), DMS Total Errors (R^2=.08, ß = .319 p= .007), and DMS Memory Load (R^2=.09, ß = -.342, p= .003). These were then plotted and can be seen in Figures 7-9. Age groups were divided via a median split which resulted in age 27 and above falling into the older group, and 26 and below falling into the younger category. Participants with a BMI of 25 or higher were classed into the overweight category while participants with a BMI lower than 25 were in the lean category. These variables were then observed via univariate

GLM analysis on SPSS. Means and standard errors were used to plot the data. Figure 7 indicates that the overweight age group has longer IED latency times in stages 6 than the younger age group in both lean and overweight categories. Post hoc tests were conducted by splitting the file according to age group, and then observing mean differences between lean and overweight performance on tasks. For IED latency stage 6, higher BMI significantly increased latency times for younger/overweight participants (young/overweight mean latency =1145.4ms (±345.7), p=.024), (young/lean mean latency = 968.01ms (±234.2). BMI did not significantly affect other age/BMI groups for IED. Figure 8 indicates that the younger age group has more DMS Errors in the lean category. Post hoc tests for Figure 8 indicated that higher BMI did not trend toward an increase in errors for older/overweight participants (younger/lean mean errors = 3.88 (±2.75), older/overweight mean errors =3.22 (±2.4), p=.096). Figure 9 indicates that the younger age group had better DMS Memory Load in mainly the lean category. Post hoc tests for Figure 9 indicated that age significantly increased Memory Load for younger/lean participants (young/lean mean Memory Load = -11.05 (±1.33), older/overweight mean latency =-8.68 (±1.24), p=.092).

Table 5. Linear regression analysis for variables predicting DMS Stages Overall latency (Memory) with BMI, demographics, and behavioural measures

	B[1](±SD)	**ß[2]**	**P-value**
Ethnicity	37.8(±93.3)	.03	.70
Self-Esteem	6.0(±15.0)	.04	.70
BDI	6.0(±11.1)	.06	.60

Education	-108.2(±79.2)	-.11	.17
BIS	379.9 (±193.2)	.16	.05
Gender	**-300.03(±142.9)**	**-.16**	**.04**
Age	**15.9 (±5.6)**	**.26**	**.01**
BMI	**26.5(±11.4)**	**.23**	**.01**

[1]Unstandardized regression coefficient

[2]Standardized regression coefficient

R^2=.23

Table 6. Linear regression analysis for variables predicting AGN (attention, impulsivity, executive function) with BMI, demographics, and behavioural measures

	B[1](±SD)	ß[2]	P-value
AGN-TMS			
Ethnicity	-.69(±.61)	-.1	.27
SE	.07(±.1)	.08	.47
BDI	.08(±.07)	.13	.26

Edu	-.22(±.6)	-.04	.69
BIS	.14(±1.3)	.01	.91
Gender	**2.8(±.97)**	**.26**	**.01**
Age	-.01(±.04)	-.02	.82
BMI	.11(±.08)	.14	.16
AGN-TLS			
Ethnicity	-15.5(±15.4)	-.09	.32
SE	2.3(±2.5)	.11	.36
Edu	7.6(±14.0)	.05	.59
BIS	-8.3(±32.4)	-.03	.80
Gender	14.3(±24.6)	.05	.56
BDI	.25(±1.9)	.02	.90
BMI	2.9 (±2.0)	.14	.14
Age	.25(±.97)	.03	.80

[1]Unstandardized regression coefficient
[2]Standardized regression coefficient
1a R^2=.11
2b R^2=.07
AGN-TMS: Total Misses for sad words, TLS: Latency for sad words

Table 7. Linear regression analysis for variables predicting IED 8-9 (mental flexibility) mean latency with BMI, demographics, and behavioural measures

	B[1](±SD)	ß[2]	P-value
Ethnicity	64.5(±64.9)	.09	.32
SE	-7.7(±9.6)	-.09	.42
BDI	-3.01(±7.8)	-.04	.70

Edu	-69.6(±51.6)	-.123	.18
Gender	**178.6(±90.9)**	**.17**	**.05**
BIS	17.8(±120.9)	-.01	.88
BMI	6.7(±7.4)	.09	.36
Age	**9.3(±3.6)**	**.26**	**.01**

[1]Unstandardized regression coefficient

[2]Standardized regression coefficient

R^2=.16

Figure 7. Interaction between age and BMI for performance (latency) in IED stage 6 (mental flexibility)

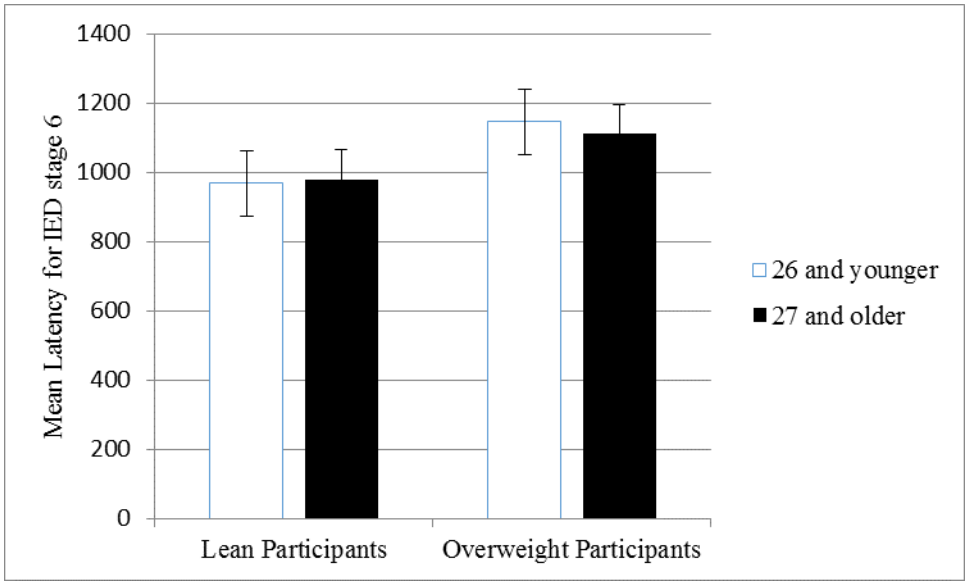


Figure 8. Interaction between age and BMI for DMS Total Errors (Memory) on task

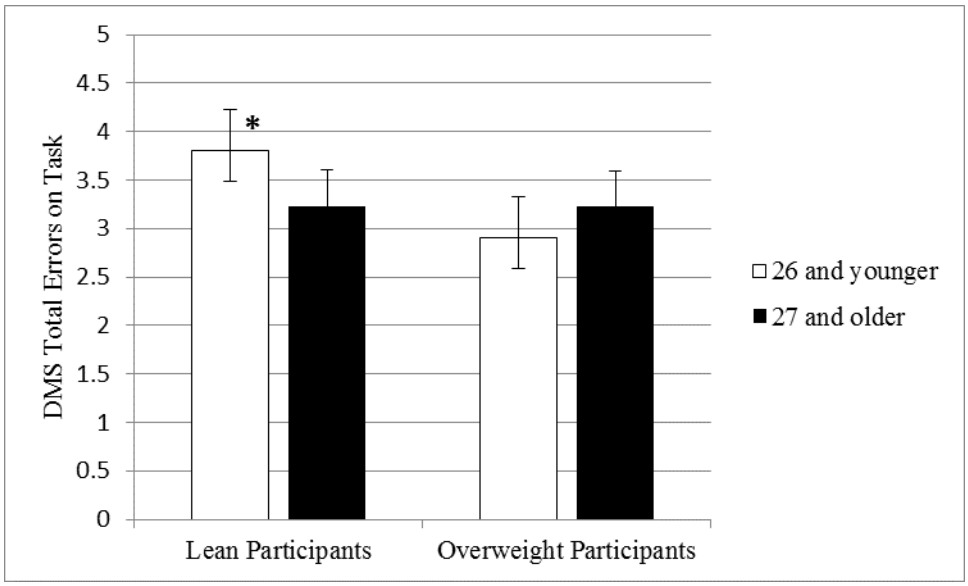
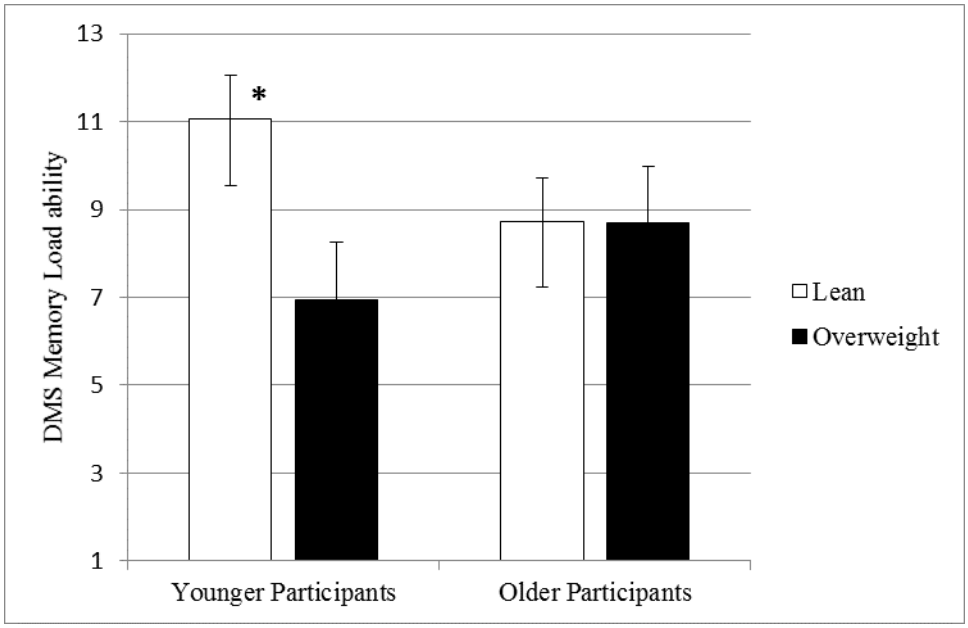


Figure 9. Interaction between age and BMI for DMS Memory Load (Memory) Task



4.5 Discussion

The primary aim of the current study was to assess whether BMI is related to cognitive performance when controlling for the predicted confounds of age, gender, depression, education, impulsivity, and self-esteem. Higher BMI was positively associated with emotional eating, depression, and age, but not correlated with impulsivity. It was negatively associated with self-esteem and education. Overall, participants with higher BMI did not perform more poorly on cognitive tasks, which contradicts much of the literature (Farr et. al 2008; Gunstad, Paul, Cohen, Tate, Spitznagel, Gordon 2007; Fedor and Gunstad 2013; Gunstad et al. 2007; Stanek et al 2013).

BMI was correlated with depression, age, education, impulsivity, and self-esteem. Age was close to, but not significantly correlated to depression. Restrained eating was correlated with emotional eating, depression, and self-esteem. Emotional eating was correlated with external eating, depression, impulsivity, self-esteem, and education. Impulsivity was correlated with

age, emotional eating, external eating, depression, self-esteem, and education. These correlations suggest the study is detecting trends found in prior literature. For instance, BMI is correlated with the expected measures of depression (Farbricatore, Wadden, Sarwer, Faith 2005), age (Rothman 2008), education (Arendt 2005), impulsivity (Nederkoom, Braet, Van Eijs, Tanghe, Jansen 2006), and self-esteem (Strauss 2000), and emotional eating (Van Strien, Herman, Verheijden 2009). This confirms that when addressing previously posed research questions, findings are supported. Importantly, these findings provide further evidence that when testing in a more diverse sample (educated, varied BMI, age, and gender), findings on emotions and behaviour remain the same. They also imply that when using the same sample to answer the same questions while controlling for confounds, findings will be reliable.

The correlations for CANTAB cognitive measures were mixed. The expected relationships between BMI and task accuracy were not found across the data. In terms of task accuracy BMI was correlated with: one measure for visual memory (PAL Total Errors, PAL number of trials to complete all stages) and a measure for impulsivity, attention, and executive function (AGN total misses for sad words, or the inhibition of pre potent responses). Otherwise, it was correlated with more measures for latency: memory (DMS overall latency, PAL overall latency), impulsivity, attention, and executive function (AGN latency for sad words), mental flexibility (IED latency stages 8-9), and spatial planning (SOC mean thinking time). One reason why the BMI-cognition relationship was not found across tasks may be due to previous findings being limited to use of general cognitive measures and specific sample populations. This would indicate that when more ability-specific measures are used, the difference between cognitive function and latency is highlighted. Furthermore, when controlling for confounds such as ethnicity, education, BIS (impulsivity), BDI (depression), SE (self-esteem), age, and

gender, BMI was not the best predictor for cognitive performance. One exception was a memory measure (DMS latency), for which BMI was still a significant predictor. As no studies have previously used these control measures, the findings are novel. This data indicates that accounting for comorbid factors may alter BMI-cognition relationships previously suggested in the literature. Age and gender, well documented correlates of BMI and cognition, were the top predictors for performance overall. Despite this, no interactions were found between BMI and gender. However, BMI and age did appear to interact when predicting latencies in mental flexibility (IED), and memory (DMS). This leads back to previous discussions on accelerated aging due to higher BMI. For the mental flexibility measure, IED, the younger age overweight group had the longer latency times than all groups, significantly so when compared both older and younger lean groups. For memory, DMS, the younger and lean group had the best memory capability in one task, while having the most errors in it as well. This points out that any potential effects age and overweight have on memory, may not apply across all cognitive tasks. Additionally, it may be that older participants prefer to take their time with these tasks, and are not slower due to poorer cognition. Generally, age does seem to affect memory. It is not clear whether this is exacerbated when an individual is overweight, although the older/overweight subjects did have the longest latencies overall in these tasks. However, post-hoc tests of these interactions indicated some potential effects. Overall, BMI may have an impact on memory, depending on the task and age of the individual. Data suggests that the BMI-cognition relationship is not a direct one, nor is it simple. Confounding variables, some of which may cause preoccupations, determine cognitive efficiency. Higher BMI itself does not appear to be related to lower cognitive ability.

Previously Taki et al. (2008) reported that BMI was correlated with overall loss of medial temporal lobe volume in men. They collected a wealth of clinical data such as alcohol intake, blood pressure, diabetes, and heart disease. However, although the study excluded participants with a history of psychiatric disease, they did not control for current mental health or psychological traits, which could explain their results. Similarly, Shimoji and colleagues (2013) reported changes in microstructural white matter in individuals with metabolic syndrome but although these authors collected collect data on many biological measures (blood pressure, cholesterol, insulin levels) they did not account for behaviourial/psychological measures. Brooks et al. (2013) reported that obese individuals have reduced total brain volumes (specifically in the prefrontal cortex) and although they were able to control for age, education, and gender, no controls for confounds such as depression or impulsivity were included. When Wallner-Liebmann and colleagues (2010) found a positive correlation between hippocampal activation and waist circumference (but not BMI), they had more extensive psychological measures. They collected data for age, gender, ethnicity, education, brief symptom inventory, and behavioral inhibition/activation scale. Interestingly, they did not suggest hippocampal deficits, but activation. Although these studies did consider potential confounds, they excluded factors that are known to have significant impacts cognitively such as depression (Lichtenburg, Ross,Mills, Manning 1994) and self-esteem (Campbell, Chew, Scratchley 2006). Without these potential confounds, it is difficult to conclude if BMI, and not psychological characteristics, is the cause of the observed relationships.

The present results suggest that when confounding variables are taken into account, there are few associations between BMI and cognitive function. However, it should be noted that

limitations with the sample may have contributed to the findings. An important distinction between the current sample and the general population is the level of education, with 45.7% of participants being at degree level or above. To adjust for this education level was controlled for when running regressions. There were also significantly more females (108 compared to 45 males), which may have affected correlations. However, gender was controlled for in all other instances. The limitations of using stepwise regression with backward elimination must also be considered as generalizability of data may be an issue. However, the sample was an attempt at a diverse population and should representative a group with variability. This method also allows for identifying the most important predictors within a diverse sample such as this. Previous literature has observed a link between high fat diets and poorer cognition (Francis and Stevenson 2011). The current study did not include consumption of high fat foods, but it is of interest and recommended in future research. Another limitation may have been not including an anxiety measure. Anxiety may also influence cognitive function although it is usually correlated with measures of depression that were included. In addition it would also have been informative to include other measures of adiposity such as waist circumference. A common noted limitation in obesity research, and even in obesity-cognition research, is that waist circumference is a more sensitive measure than BMI (Kurth et al. 2010). Kurth and colleagues (2013) had previously found a significant negative relationship between BMI and gray matter in some areas of the brain. The effects were much more pronounced when using waist circumference, especially in females. This implies that waist circumference may be a more sensitive measure than BMI. However, claiming waist circumference is more sensitive, should not be confused with a claim that BMI is does not detect weight related comorbidities. Abdominal obesity, which is certainly related to BMI, may simply be a more specific (and risky) form of obesity. Additionally, the measure Big

Circle/Little Circle was used to detect any potential differences in attention or interest in the CANTAB tasks. As no differences between groups were found, this indicates that participants experienced a similar level of motivation to complete the tasks.

In summary, as has been reported in the past, age and gender (significant correlates of depression) are associated with poorer scores in cognition. When controlling for confounding variables, there are few relationships between BMI and cognitive function, although there is some evidence of subtle impairments in memory that are greater in older participants. It is therefore unlikely that increased weight alone is responsible for cognitive decline associated with obesity. Just as there are numerous factors that lead to obesity, there appears to be complexity in what causes neurological and cognitive deficits.

Chapter 4 Summary

It was predicted that BMI would be associated with poorer cognitive performance, such as diminished memory ability. Overall, BMI was not correlated with poorer cognitive performance. When confounding variables were taken into account, there were few associations between BMI and cognitive performance. BMI was correlated with the expected self-report measures (depression, age, self-esteem), and but was not a significant factor when assessing cognitive performance. Age appears to account for a lag in latency, and an Age-BMI interaction may be at play in the area of memory. Previous literature suggested a more direct link between BMI and cognitive performance. These results indicate that the inclusion of more psychological confounds should be considered when investigating cognitive function within obesity.

CHAPTER 5: Studies 4 and 5 - Food Memory: Distraction, Disinhibition, and meal type

5.1 Introduction

Food Memory

The results of Chapter 4 suggest that when controlling for confounding variables the relationship between obesity and cognitive function are very limited although the data suggest that performance on a memory task is impaired as BMI increases. Other research is now closely linking memory to individual food intake (Higgs 2005, Higgs 2008, Higgs and Donohoe 2011). Obesity is related to poorer memory (Benito-Leon et al. 2013), and some research even suggests memory as an important part of appetite regulation (Higgs 2008). However, the relationship between memory and body weight appears to be a scarcely explored topic.

A relevant distinction to address is whether obesity is linked to impaired memory in general or whether specific impairments in processing food memories are important. Francis and Stevenson (2011) point out that intake is both dependant on an individual's memory for previous food and cues indicating hunger, which are regulated by the hippocampus. In their study they found the high fat diets impaired hippocampal function (on general memory tasks) along with increasing food intake and decreasing food recall accuracy (food memory tasks). In their first study, they collected self-reported diet information and found high fat and high sugar diets were associated with worse performance on memory tasks (hippocampal sensitive), but not other cognitive markers. They did not find a relationship with BMI although the participants were young and had mainly normal BMI. In their second study, they repeated their first experiment, while assigning participants to self-reported diet groups (high fat/sugar versus lower high fat/sugar). They found similar results. As part of the same study,

they found high fat/sugar group participants exhibited poorer food memory and more difficulty recognizing hunger and satiation than their lower fat/sugar counterparts. Actual diet was not manipulated by researchers making the nutritional intake uncertain. Additionally, general memory performance may be a relevant factor to consider. Francis and Stevenson (2011) provide support for the idea that diets high in fat/sugar may impair memory, and impaired memory for meals may lead to overeating later on.

Different approaches to food memory research have been taken. Some focus on the manipulation of food memory via distraction (Higgs and Woodward 2009), while others attempt to enhance food memory (Higgs 2002). In both cases, a measure of food memory is then correlated with food intake. In a study by Higgs and Woodward (2009) 16 participants were provided lunch, some of whom were asked to watch television while eating. Later they were offered cookies and asked to recall their memory and vividness of the lunch. Watching television while eating was found to significantly increase cookie intake and decrease meal vividness. Brunstrom and Mitchell (2006) suggested that a distraction while eating may dull an individual's physiological responses to food, thereby making it more difficult to receive cues on fullness. A similar study (Mittal, Stevenson, Oaten, Miller 2011) was conducted on adult females. Participants were first given a snack with or without TV, and later provided with a lunch meal. Subjects in the TV condition ate more during their lunch and had poorer recall for their snack, which supports the idea that that distraction may impair food memory. Additionally, a study by Oldham-Cooper and colleagues (2011) reported that another form of distraction (computer game of solitaire) while consuming lunch made participants in that condition feel less full, eat more biscuits later on, and have poorer memory for the serial order of their lunch items. The influence of distraction may even be seen in childhood, as suggested

by Francis and Birch (2006). In a study on 24 preschool aged children, snack intake was measured after lunch in a TV and no TV condition. Children in the TV condition generally ate less lunch and snacks than children in the no TV condition. However, children who generally watched more TV, and ate more meals while watching TV, were found to eat more lunch. These findings highlight the use of distraction in identifying effects on both food memory and eating behaviour in the lab.

In another study by Higgs (2002), female participants were given a 'lunch cue' which indicated that they must recall what they had eaten for lunch prior to tasting some snacks. When compared to participants who did not received this 'lunch cue', the participants who recalled the lunch they had eaten that day ate significantly fewer snacks. In a second experiment participants were given either a “lunch today cue”, “lunch yesterday” cue or no cue prior to eating. Participants in the “lunch today cue” had the lowest intake out of the three conditions. It is possible that a lunch cue or intake reminder is having the reverse effect of an intake distraction (TV or computer) by enhancing food memory. This supports the idea that varying levels of attention to recent eating behaviour will affect later intake. To test this idea, Higgs and Donohoe (2011) asked female students to eat a fixed lunch mindfully. This entailed focusing on the characteristics of their food as they ate. Their snack intake and lunch vividness was then compared to a control group, and a group that read a newspaper article as they ate (distracted group). They found that eating mindfully not only increased lunch vividness, it decreased snack intake. Therefore, enhancing food memories may be helpful in reducing food intake.

It is likely that factors relating to eating traits and or/obesity may interfere with the processing

of food memories but there is limited evidence on this topic. Eating style has been reported to moderate the effect of food memory on later intake. Higgs, Williamson, and Attwood (2008) offered a popcorn snack ranging in palatability to participants after a fixed lunch. Participants who were asked to recall their lunch from that day ate less of the snack than participants asked to recall lunch from the previous day. The same was seen in a second experiment, but only in women who did not score highly on dietary disinhibition. This demonstrates a link between disinhibition and food memory. Participants also did a cookie taste test, and females who recalled their lunch that day and scored low in disinhibition ate fewer cookies than those participants who recalled the lunch eaten the previous day. Individuals scoring high in disinhibition, do not appear to respond to the memory cue in the way their low disinhibition counterparts do.

Both disinhibition and restraint might be predicated to affect food memories based on other work examining the link between dieting and attention to food cues. Mitchell and Brunstrom (2005) compared female dieters and four groups of non-dieters with varying high/low levels of dietary restraint and disinhibition as measured by the DEBQ and TFEQ. There was no relationship between performance in an attention task and food intake. However, the High-restraint/High-Disinhibited group ate the most out of all groups, and dieters performed better than non-dieters towards the end of the meal in Rapid Visual Information Processing (RVIP) attention tasks. Although this study did not include a measure for memory, attention is required to encode memories. This is may be demonstrating that dieters were more distracted by the RVIP task, which contributed to higher intake. However, the specific link between food memory and eating style has yet to be investigated.

Another factor that might be affecting the formation of food memories is how preoccupied someone is with food, for example: how frequently they think about the food, and if they view the food item in a positive, negative, or neutral light. These 'preoccupations' are made up of food associations (positive vs. negative), which may influence food intake and memory. Soetens and Braet (2010) found that overweight adolescents showed a memory bias for high calorific foods which was not related to negative evaluation of food words. Some researchers believe that the act of dieting may cause an individual to become preoccupied with dieting in the process, and then lose focus in other areas (such as working memory) (Vreugdenburg, Bryan, Kemps 2003). Vreugdenburg, Bryan, Kemps (2003), tested how weight loss may affect memory and found that dieters reported being more preoccupied than non-dieters, and that this mediated the relationship between dieting and working memory (via the central executive and phonological loop). However, the relationship between food memory, eating behaviour, and food preoccupation is yet to be fully investigated. An interesting study by Jones and Rogers (2003) attempted to decipher two causes for memory when dieting; metabolic consequences versus preoccupation. Using cognitive tasks pre- and post-chocolate bar consumption, they found that dieters, when compared to non-dieters, performed more poorly on memory tasks while also exhibiting more food related thoughts. It is possible, that certain types of eaters (restrained, emotional, BED) are experiencing an impaired effect on cognition due to food preoccupations. One study by Pietrowsky and colleagues (2010) found that hungry anorexics did not vary in word recall ability from hungry controls; however, satiated anorexics recalled significantly more words than satiated controls. Perhaps, depending on an individual's eating style, different effects on memory will be observed.

The aim of the present studies was to assess the factors that influence food memories

including BMI, dietary traits and food preoccupation. Two studies are presented and participants are given a lunchtime meal (Study 1) or a snack (Study 2). CANTAB test batteries were used as a general memory measure. The measures of food memory were drawn from the previous literature and include a Visual Analog Scale (VAS) to assess memory vividness and food memory task similar to that used by Koster, Prescott, and Koster (2004). We predicted that higher restraint and tendency towards disinhibition and BMI would be linked with impaired food memory, but this might be moderated by food preoccupation (attentional bias).

5.1.1 STUDY 4 Method

Participants

105 participants were recruited from the Psychology undergraduate population. University recruitment took place through the Research Participation Scheme (RPS), which is designed to enable Psychology students to earn course credits by participating in research. Ethical approval was obtained from the University of Birmingham. Exclusion criteria were regular smoking and being on anti-depressants, as this may interfere with appetite (Chen et al. 2005, Harris, Young, & Hughes 1984). Additionally, 34 participants who did not consume the entire 400 gram pasta lunch were excluded to guarantee uniformity in food intake across participants. This left 71 participants overall. The sample used in this study was university aged females (mean 18.8 years) with a mean BMI of 22.6.

This study was designed to investigate how individual differences affect the relationship between distraction and food memory. This included observing participant food preoccupation scores and eating behaviours (such as restraint), along with disinhibition. Each participant's

snack intake was also measured. We predicted that the distracted group (group 2) would consume more biscuits (snack) and have a poorer memory for the pasta meal they ate at lunch. The food memory taste test used is based on previous food memory research (Koster, Prescott, and Koster 2004). A study by Sulmont-Rosse, Moller, Issanchou, and Koster (2008) used similar taste test methods. Our version of the memory test is designed for recall of one pasta meal consumed for lunch. It was presented 1-2 hours after consumption among a sweeter version, and a bitterer version. The participant was then asked to select which one they had for their meal earlier.

Self-Report Measures

The Dutch Eating Behaviour Questionnaire (DEBQ) (Van Strien et al. 1986) is a validated and internally reliable measure used to assess a participant's eating behaviours (Van Strien et al. 1986). Eating behaviour such as restrained eating has been found to be related to higher BMI (Snoek, Engels, Van Strien, Otten 2013). The DEBQ consists of 33 questions which address a variety of eating attitudes and habits. There are 3 subscales: restrained eating, emotional eating, and external eating. An example of an item from the restrained scale would be ‘When you have put on weight, do you eat less than you usually do?’ An example of an item from the emotional scale would be ‘Do you have a desire to eat when you are irritated?’ An example of an item from the external scale would be ‘If food smells good, do you eat more than usual?’ Responses are in a Likert format ranging from 1 for ‘never’ to 5 for ‘very often’. Emotional and external eating scores were used to calculate a disinhibition score for each participant (Allison and Schlundt 1995).

The Food Preoccupation Questionnaire (FPQ) (Tapper and Pothos 2010) can be used to

assess the frequency of thoughts about food (3 items), along with whether there are positive (9 items), negative (9 items), or neutral (5 items) emotional associations. Four items were reverse scored. Responses are in a Likert format ranging from 1 for ‘completely disagree’ to 5 for ‘completely agree’. Tapper and Pothos (2010) reported good construct validity and reliability (Cronbach’s a > .80). This contains 26 items. (See appendix 4d)

The Barratt Impulsiveness Scale-11 (BIS-11) (Patton, Stanford, Barratt 1995) can be used to measure a participant’s impulsive personality traits. Impulsivity was seen as a relevant measure due to its association with overeating (Guerrierri, Nederkoorn, Schrooten, Martjin, Jansen 2009). This contains 30 questions investigating the patient's thoughts on and behaviours during daily activities and decisions. An example of an item would be ‘I plan tasks carefully.’ with responses in a Likert format ranging from 1 for ‘rarely/never’ to 4 for ‘almost always/always’.

Hunger and Mood Scales There were 11 Visual Analogue Scale (VAS) items that produced a score from 1 (not at all) to 10 (extremely). A hunger example item would be ‘How hungry do you feel right now?’ A mood example item would be ‘How happy do you feel right now?’ This scale was also used to compile overall appetite, negative feeling, positive feeling, and arousal scores. Appetite was calculated by taking inverse scoring fullness ratings and combining them with desire to eat and hunger ratings. Negative feelings were calculated by combining ratings for sadness, stress, irritation, and nervousness. Positive feelings were calculated by combining happy and excited ratings. Arousal measures were calculated by inverse scoring then combining ratings for being relaxed and tired. (See appendix 3d)

Taste Perception Scale (Pasta) In order to assess pasta liking, a 6 item questionnaire was developed. The first three items were Visual Analogue Scale (VAS) scales that produced a score from 1 (not at all) to 10 (extremely) for each item. These questions were about the pasta meal pertaining to taste, such as 'How salty is the pasta?' The last three items were Likert format ranging from 1 for 'strongly disagree' to 5 for 'strongly agree' and were pertaining to liking/palatability. An example of this would be the item 'I like the taste of the pasta'. (See appendix 1d)

Distraction manipulation: A television clip from the popular show 'Friends' was selected as a distraction tool. The clip was saved onto to a USB and may be found at the following link (8 min 31 secs), http://www.youtube.com/watch?v=OnYTl6qre3k

Pasta Memory Test (PMT): The food memory taste test used is based on previous food memory research (Koster, Prescott, and Koster 2004). In Koster and colleagues' experiment, they presented subjects with foods and later used altered versions to test memory of the same previously consumed foods. Alterations to the foods included changes in sweetness, bitterness, and sourness. 12 food samples were presented in total (4 versions of each original food item). Units of change in taste were decided through previous experimentation. Our version of the memory test is designed for recall of one pasta meal consumed for lunch. Units of change in taste were decided through previous experimentation with other subjects, as done by Koster et al. (2004). To prepare the sauce, four 31 gram sample batches were separately prepared. A sweetened one (with 12 ml honey added), a more bitter one (with 6 ml vinegar added), a more sour one (with 6 ml lemon juice added), and the original unaltered pasta sauce. These four samples were then further divided for participants into smaller samples of about 10

grams (as 31 grams was found to be excessive). Before serving the sauce it was heated for 60 seconds (stirring at halfway through). Each sample was labelled A, B, C, or D with only the researcher aware of the actual sample descriptions. The subject was then asked to select which one they had for their meal earlier. This was scored as either correct (1) or incorrect (0). (See Appendix 2d)

*Debriefing questionnaire*s: This measure asks the participant what they recall about their food intake on the day of the experiment and what they suspect the experiment was investigating.

Food: There were two instances of food intake. The first was a plain white bowl filled with Sainsbury's Conchiglie pasta shells (55 g, 362 kcals/100g (1535 kj/100g) with Sainsbury's Tomato & Herb pasta sauce (55 g, 51kcals/100g (216kj/100g). This was served with a glass of water. The second instance of food intake was the snack, which consisted of two types of biscuits in 2 separate plain white bowls labelled, A or B. Both sets of biscuits were broken up to discourage participants from portion controlling. One bowl (randomized) was filled with McVities Digestive biscuits (45g, 478kcal/100g (2003kj/100g). The second bowl was filled with Foxes Crinkle Crunch biscuits (45g, 464kcal/100g (1950kj/100g).

Procedure

The experiment took place at the University of Birmingham. Participants were randomly selected to be in one of two groups. There were two conditions, a non-distracted group and a distracted group. Each participant would have been informed in the Participant Information Sheet (PIS) that they were taking part in a two-part experiment. The study was advertised as a 'mood and meal time' experiment, which complimented the distraction condition of watching

comedy clips. The full cover story can be seen in the PIS in appendix 5d. For the first part of the experiment, they were asked to arrive and answer questionnaires while consuming a light lunch. This lunch consisted of a 400gram pasta meal with drinking water provided. The participants (second group) selected into a 'distraction' group were asked to watch a television programme airing on a computer as they ate. Immediately before and after lunch, both groups completed Visual Analogue Scale (VAS) questionnaires on Hunger and Mood, and pasta meal liking. For the second part of the experiment, they were asked to come back 2 hours later to consume not to snack in-between. On their return, they completed a third Hunger and Mood scale. They were then offered two snacks to eat; Digestive biscuits and chocolate chip cookies. These were placed in two separate bowls labelled A and B and broken up into pieces to disguise portion sizes. Participants were asked the complete a biscuit rating questionnaire to assist in choosing which biscuits to use in an upcoming study. They were informed that they could eat as many as they liked, as they would be thrown out afterwards. They were then told the researcher would return in approximately 10 minutes. When they had finished the snack, they were given the Food Memory Test for the pasta sauce they had for with their lunch, followed by the pasta vividness (VAS). Next, they completed the DEBQ (Van Strien et al. 1986), BIS-11(Patton, Stanford, Barratt 1995), FPQ, and a debriefing questionnaire. Lastly, their height and weight was taken and they were thanked for their participation. They were then asked if they wanted to know what the study was about. If they were interested, they were told which condition they were in and about the study design. Any questions they had were answered, and they were asked to be discreet to others who may participate afterwards. The first part of the experiment lasted approximately 25 minutes, while the second part of the experiment was approximately 20 minutes.

5.1.2 Statistical Analysis

In this between-participants design, questionnaire responses were used to calculate scores for each participant in the categories of eating behaviour, food preoccupation, and disinhibition. The same was done for biscuit (snack) intake, BMI, appetite, negative feelings, and positive feelings. T-tests and ANOVA were run to identify any significant variance between the groups' results (Michael and Proschan 2010), and some participant numbers were reduced when measures were left incomplete. Correlations between measures were also observed. These associations were then investigated through linear regressions, in order to identify significant predictors for variables of interest.

5.1.3 Results

Participant Characteristics

Between groups there were no significant differences in Body Mass Index (BMI), impulsivity, restraint, food preoccupation, eating behaviour, or disinhibition (See Table 1 below).

Table 1. Participant Characteristics and questionnaire measures for Food Memory

Study 1

Means	Not Distracted	Distracted	Overall	t	P-values
Participants	36	37	73		
Age (years)	18.7 (± .9)	19.1 (±.8)	18.9 (±.9)	1.652	.10
Body Mass Index (BMI)	22.5 (±2.9)	22.7 (±4.1)	22.6 (±3.5)	.262	.79
Disinhibition: (Emotional and External DEBQ)	2.8 (±.5)	3.1 (±.6)	2.9 (±.6)	1.513	.14
DEBQ Restraint	2.7 (±.9)	2.4 (±1.0)	2.6 (±.9)	-.891	.99
BIS (1-4)	2.2 (± .4)	2.2 (±.3)	2.2 (±.3)	-.019	.38
FPQ (1-5): Frequency	3.5 (± .8)	3.6 (±.8)	3.6 (±.8)	.033	.97
FPQ : Neutral	2.9 (±.6)	3.0 (±.9)	3.0 (±.7)	.243	.81
FPQ : Negative	2.2 (±.8)	2.3 (±.9)	2.3 (±.8)	.510	.61
FPQ : Positive	3.4 (±.8)	3.4 (±.6)	3.4 (±.7)	-.296	.77

DEBQ: Dutch Eating Behaviour Questionnaire
BIS: Barratt Impulsiveness Scale
FPQ: Food Preoccupation Questionnaire

Appetite and mood between distracted and non-distracted individuals

Appetite and mood between groups were observed throughout the experiment. These measures were collected before lunch, directly after lunch, and prior to biscuit intake. They were compared via 4 ANOVAS for appetite, negative feelings, positive feelings, and arousal. There was a statistically significant effect of time on appetite $F(2, 132) = 202.9$, $p < .001$, negative feelings $F(2, 134) = 7.83$, $p < .001$, positive feelings $F(2, 136) = 3.88$, $p < .001$, and arousal $F(2, 134) = 5.99$, $p < .001$. However, no interactions were found for distraction on appetite $F(1, 66) = 2.7$, $p = .102$, negative feelings $F(1, 67) = .08$, $p = .778$, positive feelings $F(1, 68) = .018$, $p = .894$, or arousal $F(1, 67) = .124$, $p = .726$. Means for appetite, negative feelings, positive feelings, and arousal were then also compared (See Table 2 below). Appetite, negative feelings, and positive feelings varied significantly at each time point ($p < .001$). However, arousal did not vary across time points.

Table 2. Means for Appetite, mood, and arousal for Food Memory Study 1

Variable	Before Lunch	After Lunch	Before Snack
Appetite (0-30)	20.65(±.4.9)	5.0(±.3.8)	11.7(±.6.0)
Negative Feelings (0-40)	11.1 (±.5.9)	9.5 (±.5.9)	9.1 (±.5.7)
Positive Feelings (0-20)	10.2 (±.3.5)	9.7(±.3.2)	10.8(±.3.7)
Arousal (0-20)	9.9 (±.2.2)	8.8 (±.2.6)	9.0 (±.2.4)

Appetite: Hunger (+), Fullness (inverse) and desire to eat (+)

Negative Feelings: Sad (+), stressed (+), irritable (+), and nervous (+)

Positive Feelings: Happy (+) and excited (+)

Arousal: Relaxed (inverse) and tiredness (inverse)

Pasta memory test Results

The Pasta Memory Test did not detect any differences between groups (See Table 2 below). Additionally, pasta vividness did not differ between groups.

Chi Square: Table 3. Pasta Memory Test (PMT) and Pasta Vividness (PV)

Distraction	Pasta Memory Incorrect	Pasta Memory Correct	PMT Totals	Pasta Vividness (Means)
Not Distracted	25	11	36	5.8 (±.2.0)
Distracted	26	9	35	5.7 (±.2.0)
Totals	51	20	71	5.7 (±.2.0)

Correlations between eating behaviour and food memory

Correlational relationships between eating behaviour and outcome measures were observed. These were later used to guide the method of linear regression analysis. Positive food preoccupations had a significant negative correlation with biscuits eaten and significant positive correlation with pasta (meal) vividness. BIS (impulsivity) was negatively correlated with pasta vividness. Restraint was correlated with BMI and negative food preoccupation thoughts. There was no correlation between biscuits eaten and meal vividness (pasta vividness) (See Table 3).

Table 4. Pearsons correlations between eating styles, food preoccupation, BMI, and food memory

	BMI	Res	FPQ+	FPQ-	Dis	BE	PV	BIS
BMI	1	**.31****	-.01	.10	-.06	-.05	.03	.01
Res		1	-.22	**.60****	-.05	-.01	-.13	-.08
FPQ+			1	**-.31****	.22	**-.24***	**.25***	.08
FPQ-				1	.17†	.20	-.06	.10
Dis					1	.10	.13	.09
BE						1	-.06	.11
PV							1	**-.40****
BIS								1

*p <.05, ** p <.01, † trending
BMI: Body Mass Index
FPQ: Food Preoccupation Questionnaire
Restraint (Res): DEBQ Restrained eating measure
Pasta Vividness (PV): Food memory measure
Disinhibition (Dis): DEBQ combined Emotional and External eating measure
Biscuits Eaten (BE): Amount of biscuits eaten
Barratt Impulsiveness Scale-11 (BIS): Impulsivity measure

Linear Regressions predicting meal memory and intake

To test the hypothesis that traits such as disinhibition and restraint predict meal memory (pasta vividness) and later intake (biscuit intake), a multiple linear regression analysis approach was used. Based on the correlations observed, the following predictor variables were chosen; BMI, disinhibition, restraint, negative food preoccupation, and positive food preoccupation. The predictors selected were correlated with the outcome variables for meal memory (pasta vividness) and later intake (biscuit intake). Due to it being a homogenous sample demographics were not included in the models below.

Three linear regressions were carried out to observe the best predictors of pasta vividness and biscuit intake. The first model suggests positive food preoccupation and distraction group are significant predictors of biscuits eaten (See Table 5). The second model revealed that positive food preoccupation (but not distraction group) and BIS (impulsivity) were strong predictors of

pasta vividness (See Table 6). There were no significant interactions with distraction when observing BMI, disinhibition, or restraint. Additionally, a check for interactions between distraction group and FPQ+/FPQ- and BIS and distraction group found no interaction for predicting PV. Plotting was carried out by conducting a median split on positive food preoccupation and running a GLM univariate analysis for predicting pasta vividness and biscuits eaten. The means and standard errors were then used to plot the data. The relationship between distraction and FPQ+ was plotted (Figure 1 below) and demonstrated that individuals that were not distracted and had high FPQ+ scored higher on pasta vividness (food memory measure). Post hoc tests were conducted by splitting the file by condition (distraction vs non-distraction group) and running t-tests comparing high FPQ+ vs low FPQ+ for outcome measures. Results for Figure 1 indicated that when participants were not distracted, higher FPQ+ results in significantly higher pasta vividness (not distracted/low FPQ+ mean= 4.8 (±2.1), (not distracted/higher FPQ+ mean =6.4 (±1.8), p=.022). A trend towards interaction between FPQ+ and distraction when predicting for BE was found which was not significant. This trend was then plotted (Figure 2 below), and indicated that participants with lower FPQ+ scores consumed more biscuits than participants with higher FPQ+ scores, which was exacerbated in the distracted group. Post hoc tests for Figure 2 (run by splitting the file by high and low FPQ+) indicated that lower FPQ+ trended towards significantly increasing biscuit intake for distracted participants (distracted/biscuit intake= 51.2 (±27.9), non-distracted/biscuit intake =36.8(±18.6), p=.096).

Table 5. A) Summary of hierarchical linear regression analysis for predicting biscuits eaten (g) and B) Final model of a backwards elimination to predict biscuits eaten			
	B[1](±SD)	ß[2]	P-value

Model 1a			
BMI	-.26(± .8)	-.04	.70
Group	8.8(± 5.3)	.21	.11
Restraint	-3.7(± 3.7)	-.15	.34
FPQ +	-6.7(±4.2)	-.21	.11
FPQ -	5.3(±4.2)	.20	.21
Disinhibition	2.4(±5.0)	.10	.63
Model 2b			
Group	**10.9(± 5.1)**	**.24**	**.04**
FPQ +	**-7.4(± 3.7)**	**-.21**	**.05**

[1]Unstandardized regression coefficient
[2]Standardized regression coefficient
1a R^2=.144
2b R^2= .12
FPQ: Food Preoccupation Questionnaire
Group: immediate v. delay group

Table 6. A) Summary of hierarchical linear regression analysis for disinhibition predicting pasta vividness and B) Final Model of backward elimination regression analysis for variables predicting pasta vividness

	B[1](±SD)	ß[2]	P-value
Model 1a			
BMI	.06(± .06)	.11	.33
Group	.02(± .43)	-.00	.97
Restraint	-.46 (± .31)	-.22	.14

FPQ +	**.74(±.34)**	**.26**	**.03**
FPQ -	.42(±.35)	.18	.23
Disinhibition	.27(±.40)	.08	.50
BIS	**-2.56(±.63)**	**- .46**	**.01**
Model 2b			
BIS	**-2.41(± .61)**	**-.42**	**.01**
FPQ +	**.81(±.31)**	**.28**	**.01**

[1]Unstandardized regression coefficient
[2]Standardized regression coefficient
1a R^2=.07
b R^2= .06
FPQ: Food Preoccupation Questionnaire

Figure 1. Relationship between distraction and positive food preoccupation when predicting Pasta Vividness Food memory (No Interaction)

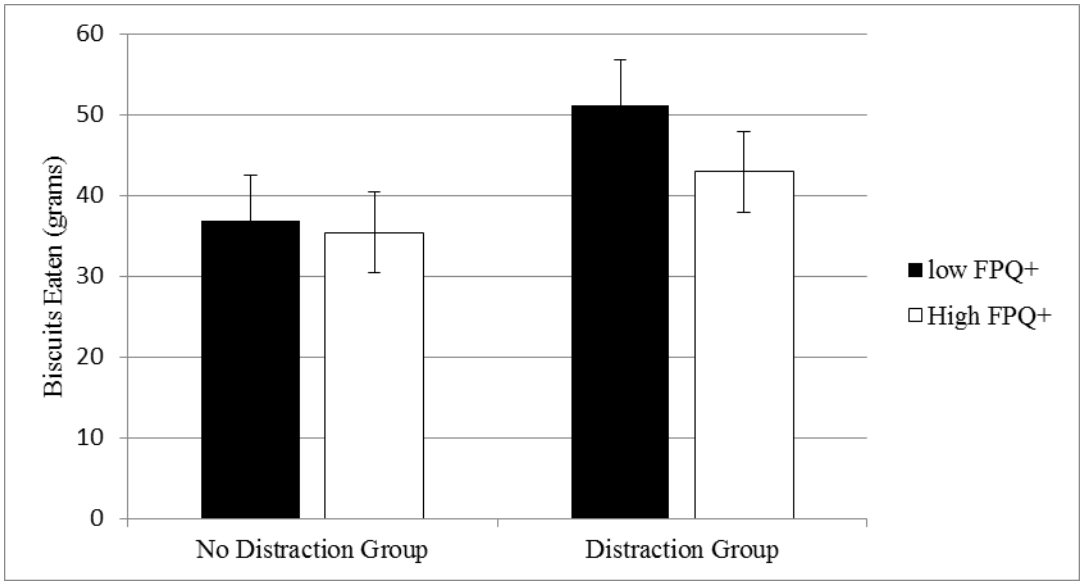
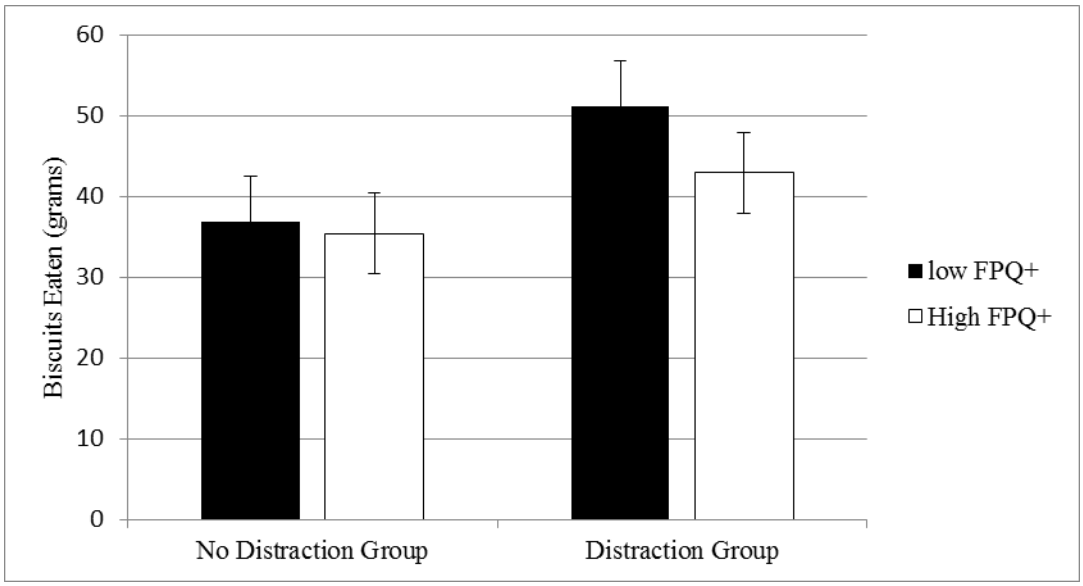


Figure 2. Interaction between distraction and positive food preoccupation when predicting Biscuits eaten (Close to interaction)



5.1.4 Food Memory Study 4: Discussion

The primary aim of the first study was to investigate how distraction may impair food memory and how this might relate to eating behaviour traits and food preoccupation.

Additionally, a new measure for food memory was developed. Linear regressions were carried out to observe the best predictors of pasta memory and biscuit intake. When predicting biscuits eaten, backward elimination and plotting of data suggested positive food preoccupation and distraction group influenced snack intake. No dietary traits were related to biscuit intake, which was not expected. It may be that measures of restraint and disinhibition are not related to actual intake in laboratory taste tests, especially if there is no obvious disinhibiting stimulus.

The food memory measure (pasta sauce taste test) did not detect differences between distracted and non-distracted participants. 20 participants answered the test correctly, 9 of which were in the distracted group (nearly an even split). Unlike Koster's experiment (Koster, Prescott, and Koster 2004), the measure did not appear to detect differences in food memory between participants. This may be due to some key differences. Firstly, Koster's experiment took place at a different meal time (breakfast) which perhaps interacts with food memory encoding differently. Some research has indicated that breakfast consumption boosts memory in general, possibly through an increase in blood glucose (Benton and Parker 1998). The new pasta sauce measure gave the participant one opportunity to select the correct pasta sauce from among other samples. Koster's experiment allowed for several different responses when tasting a food item, such as whether it varied in sweetness, bitterness, or sourness from the original item. This may have increased the number of responses by participants and thereby the likelihood of provided correct information about the food items. Additionally they compared their liking/perceived pleasantness of the original food item at breakfast time and at recall time, reporting that later liking/perceived pleasantness did not vary from what they first reported. This implies subjects had a better memory for certain characteristics of the food item

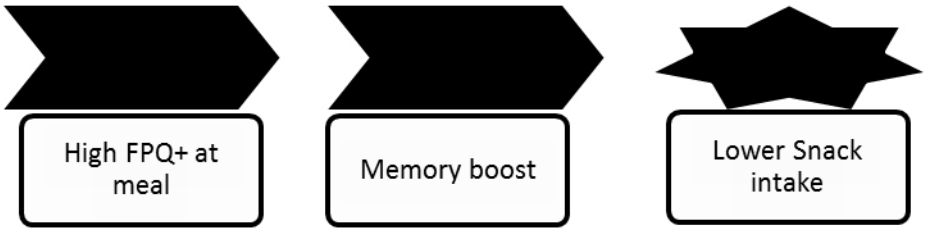
(example: how sour it was), rather than the item in its entirety. In summary, Koster's measure is detecting specific aspects of taste memory for food, not the memory of a food item overall.

Correlations between eating behaviour and outcome measures were generally as predicted. Body Mass Index (BMI) was positively correlated with restraint. The relationship between weight and dietary restraint has been seen before (Verhoef, Camps, Bouwman, Mariman, Westerterp 2014). Restraint was positively correlated with negative food preoccupation, which supports prior research on restraint, negative affect, and eating behaviour (Fay and Finlayson 2011; Jansen, Louwerse, Leemans, Schouten 1998). Disinhibition was not correlated with other measures, although it did trend towards a positive correlation with negative food preoccupation. As there is common ground between restraint and disinhibition (Dykes, Brunner, Martikainen, Wardle 2003), this compliments the relationship restraint also shared with negative food preoccupation. Limitations of using stepwise regression with backward elimination must be considered as generalizability of data may be an issue. However, the sample was representative of the population and so generalizability issues would not be substantial. This method also allows for identifying the most important predictors within this sample.

There was a group effect indicating that distraction increased snack intake, but no significant effect on memory was observed. Plotting demonstrated that individuals that were not distracted and had high FPQ+ scored higher on pasta vividness (food memory measure), and that higher FPQ+ scorers also consumed less biscuits than lower FPQ+ scorers (exacerbated in the distracted group). Overall, distracted participants consumed more biscuits. This is

consistent with previous findings that distraction at lunch inhibits later intake (Higgs and Woodward 2009; Oldham-Cooper et al. 2011; Mittal et al. 2011). No effect of distraction was found when observing appetite, mood, or arousal between the groups which supports the idea that FPQ+ is a distinguishing factor for group food memory/intake. Post hoc tests indicated that when participants were not distracted, higher FPQ+ resulted in significantly higher pasta vividness, and lower FPQ+ trended towards significantly increasing biscuit intake for distracted participants. A potential explanation for this finding is that more positive food focus/association improves memory for the meal event and thereby reduces later snack intake (See Figure 3 below). This may be due to these high FPQ+ individuals paying more attention to food as they eat which increases food memory and decreases appetite later on, which consistent with previously discussed research on attention and food memory (Higgs and Donohoe 2011). Data also highlights that regardless of distraction or intake, FPQ+ is related to better pasta vividness.

Figure 3. How higher positive food preoccupation affects food memory and snack intake



Chapter 5 Study 4 highlights

Disinhibition was not correlated with eating behaviour. It was not a predictor for eating behaviour or food memory. Restraint was correlated with Body Mass Index (BMI) and negative food preoccupation. Distraction group was a predictor for Biscuits Eaten (BE). Positive food preoccupation was negatively correlated with Biscuits Eaten (BE) and positively correlated with the second food memory measure (meal vividness). It was also a predictor for both these measures.

5. 2 Food Memory Follow-up Study 5: Jelly Bean Study

The second study investigates relationships between food memory and general memory, eating behaviour, and food preoccupation. Memory was measured through the use of the CANTAB neuropsychological test battery. This was done in the interest of detecting how overall memory is related to food specific memory and eating behaviour. Additionally, a new food recall task was developed to test snack food memory (in contrast to meal time memory), using Jelly Beans. We predicted that participants with higher BMI, in a delayed group, and higher disinhibition scores would experience poorer food recall ability and lower CANTAB scores than their counterparts. They delay condition was chosen to detect if memory deficits were delay dependent (in contract to distraction dependent). Through the measure of food

preoccupation this study also considers how attentional biases may affect food memory. As a whole, eating style may moderate effects, but food preoccupation (attentional bias) may affect an individual's memory for foods.

5.2.1 Study 5 Method

Participants

100 participants were recruited from the Psychology undergraduate population. Once again, recruitment took place through the Research Participation Scheme (RPS). As in Study 1, exclusion criteria was regular smoking and being on anti-depressants, as this may interfere with appetite. The sample used in this study was university aged females (mean age 18.8 yrs) with a mean BMI of 22.3.

Materials:

CANTAB Memory measures: The Cambridge Neuropsychological Test Automated Battery (CANTAB) has been used extensively in cognitive research, ranging from the study of executive function and cognitive aging, to learning and memory, and to impairment via neurodegenerative disease (Robbins et al. 1994; Fray, Robbins, Sahakian 1996; Robbins et al. 1998; Egerhazi, Berecz, Bartok, Degrell 2007). In this experiment 2 tests will be administered: Delayed Matching to Sample (DMS) and Paired Associates Learning (PAL) (see cantab chapter for test descriptions). Approximately 20 minutes.

Self-Report Measures

The Dutch Eating Behaviour Questionnaire (DEBQ) (Same as study1)

The Food Preoccupation Questionnaire (FPQ) (Same as study1)

The Barratt Impulsiveness Scale-11 (BIS-11) (Same as study1)

A Hunger and Mood Questionnaire (Same as study1)

Jelly Bean Liking Scale to assess Jelly Bean liking. It is a visual analogue scale that produces a score from 1 to 10 for each item. During Jelly Bean consumption, questions were asked about each Jelly Bean pertaining to liking. For example, 'Do you like eating Jelly Bean number 1?' Approximately 2 minutes. (See appendix 6d)

Debriefing questionnaires used to ask the participant what they recall about their food intake on the day of the experiment and what they suspect the experiment was investigating. Approximately 2 minutes.

Jelly Bean Food Recall: Pilot tests were run to determine a quick snack recall task. First M&Ms were tested. These were dismissed as although they varied in colour, they did not distinctly vary in flavour, which was thought would aid in food recall. Next, Jelly Beans were selected as they were similar in size but offered different tastes and colours. Pilots were run with first 10, then 5, then 7 Jelly Beans. The number seven was chosen as it appeared to offer the optimum medium being too difficult and too easy. The Jelly Beans used were orange, red, yellow, pink, black, blue and purple. Different scoring methods were recorded. First, the scoring method of 'all correct' was used. This resulted in only two possible responses: correct versus incorrect. Few participants were able to answer the recall correctly from start to finish, which indicated this scoring method was too difficult to be practical. Another scoring method was finding 'correct segments' within the response. This meant breaking up the 7 jelly bean responses into 3 segments: the first 3, the middle 3, and the final 3. This also meant there was an overlap, with the middle 3 jelly beans being part of the first and second segment. For each

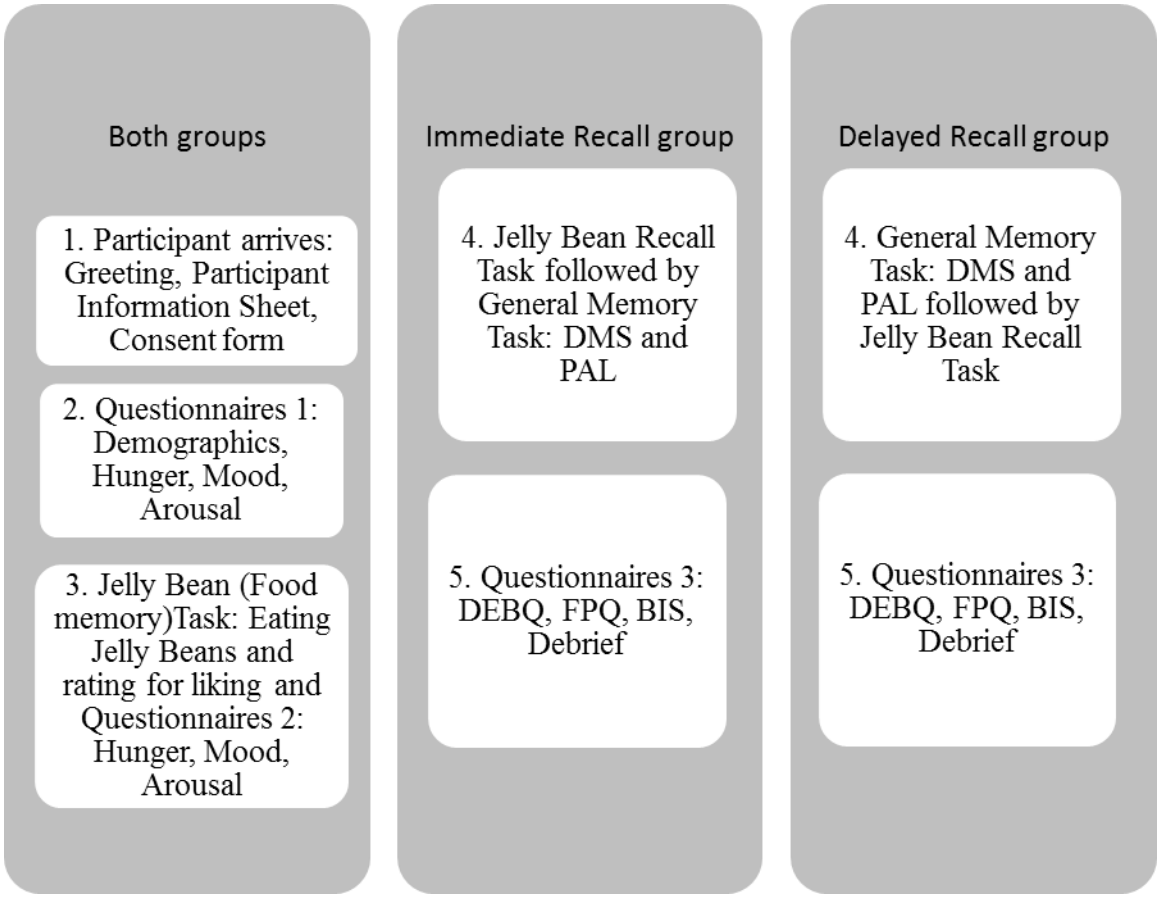
segment the participant answer correctly, they received 1 point. This meant that the minimum score was 0 and the maximum score was 3. Participants did not perform better with this scoring method, and it seemed overly complicated. After analysis, and discussion, the scoring method chosen was ‘correct sequence’. It was chosen as it was straight-forward, and participants scored better on this method for a task that was generally difficult. Better performance was due to not being marked down for a correct jelly bean sequence if it followed an incorrect response. The previous scoring methods deducted points for this. Specifically, this meant that the score they were given was equal to the number of jelly beans they could recall in a sequence, disregarding any incorrect responses provided in-between correct responses. For example, if they correctly recall the first three jelly beans they ate, but misremembered the 4^{th} and 5^{th}, and correctly remembered the 6^{th} and 7^{th}, they will have a score of 5 correct. This method of scoring produced a better range of responses than restricting the measure to completely correct whole sequences. (See appendix 7d)

Procedure

The experiment took place at the University of Birmingham (fourth floor of the Frankland building) and was advertised as a 'mood, chewing, and food' experiment. This enabled researchers to observe the participant eating the jelly beans and note the order of consumption. The cover story can be seen on the PIS in appendix 8d. Each participant was informed of the experiment details via the Research Participation Scheme (RPS), as in Study 1. A diagram of the study procedure can be seen below (Figure 1). They were shown the Participant Information Sheet (PIS) at sign up and instructed to not eat 2 hours prior to coming in for the study. For the first part of the experiment, they completed a hunger and mood questionnaire. They then moved on to the Food task: They were provided with 7 Jelly

Bean candies to eat from a bowl. Participants were instructed to eat all the Jelly Beans, without being aware they would need to remember the order. Participants were then asked to complete the hunger and mood questionnaire for a second time. The individuals randomly selected into Group 1 (no delay group) were then immediately asked to recall the colour order in which they consumed their snack. Group 1 were then moved on to the CANTAB task (approximately 20 minutes). Participants in Group 2 (delayed group) began the experiment the same way as Group 1. For the first part of the experiment, they completed a hunger and mood questionnaire, then moved on to the Food task and consumed 7 Jelly Beans. They were then asked to complete the hunger and mood questionnaire for a second time. Next, they went on to the CANTAB task (instead of recalling the Jelly Beans). After CANTAB, they were then also asked to recall the colour order in which they consumed their jelly bean snacks. They then completed the DEBQ, BIS-11, FPQ, and debriefing questionnaires. Lastly, their height and weight was taken in the kitchen. They were then asked if they wanted to know what the study was about. If they were interested, they were told which condition they were in and about the study design. Any questions they had were answered, and they were asked to be discreet to others who may participate afterwards. They were thanked for their participation. The experiment lasted approximately 40 minutes.

Figure 1. Procedure for Food Memory Study 2



5.2.2 Results

The 2 main outcome measures in this study were food memory (jelly bean recall) and general memory (PAL and DMS). Between groups (Delayed v Immediate) there were no significant differences for BMI, Jelly Bean recall, impulsivity, restraint, external eating, or disinhibition (See Table 1 Below). There were no significant differences between groups. Groups did not vary in CANTAB performance (See Table 2).

Table 1 Participant Characteristics in the delay and no delay conditions for Food Memory Study 2

Means	Delay	No-Delay	Overall	t	P-values
Participants	50	50	73		
Age (years)	18.9 (± .7)	18.9 (±.9)	18.9 (±.8)	-.238	.81
Body Mass Index (BMI)	21.8 (±3.2)	22.9 (±3.5)	22.4 (±3.4)	-1.598	.11
Jelly Bean Recall	4.2 (±2.3)	4.6 (±2.0)	4.7 (±1.9)	-.774	.11
Disinhibition: (Emotional and External DEBQ)	3.1 (±.6)	3.0 (±.5)	3.0 (±.5)	1.201	.23
DEBQ (1-5): DEBQ Restraint	2.4 (±1.0)	2.6 (±.9)	2.5 (±1.0)	-1.215	.23
BIS (1-4)	2.2 (± .4)	2.1 (±.3)	2.2 (±.4)	.478	.63
FPQ (1-5): Frequency	3.5 (± .9)	3.6 (±.7)	3.6 (±.8)	-.756	.45
FPQ : Neutral	3.0 (±.8)	3.0 (±.7)	3.0 (±.7)	.153	.88
FPQ : Negative	2.3 (±.8)	2.2 (±.7)	2.3 (±.7)	.570	.57
FPQ : Positive	3.4 (±.8)	3.5 (±.6)	3.4 (±.7)	-.739	.46

DEBQ: Dutch Eating Behaviour Questionnaire
BIS: Barratt Impulsiveness Scale
FPQ: Food Preoccupation Questionnaire

Table 2 CANTAB: PAL and DMS performance by delay condition

Variables	No delay	Delay	t	P-values
Paired Learning Associates: PAL Mean Latency	1271.33(±405.6)	1205.7 (±219.5)	-1.007	.57
PAL Total errors	5.5 (±6.7)	5.4 (±5.2)	-.117	.91
PAL Total # of trials	10.0 (±2.1)	10.0 (±1.6)	.000	.92
Delayed Match to Sample: DMS Total errors	3.7 (±2.7)	4.2 (±2.5)	1.035	.30
DMS Memory load	-10.8 (±9.5)	-10.5 (±10.1)	.136	.89
DMS mean latency	2896.7(±569.5)	2987.6(±818.4)	.644	.52

Appetite and mood between delay and no-delay groups

Appetite and mood between groups were observed throughout the experiment. These measures were collected before lunch, directly after lunch, and prior to biscuit intake. They were compared via 4 ANOVAS for appetite, negative feelings, positive feelings, and arousal. There was no effect of time on negative feelings $F(1, 98) = .724$, $p = .397$ or positive feelings $F(1, 98) = .004$, $p = .951$. There was a statistically significant effect of time on appetite $F(1, 98) = 235.7$, $p < .001$ and arousal $F(1, 98) = 9.6$, $p = .003$. No interactions were found for delay condition on appetite $F(1, 98) = 1.3$, $p = .265$, positive feelings $F(1, 98) = .221$, $p = .639$, or arousal $F(1, 98) = .038$, $p = .846$. However, for negative feelings there was a delay

condition interaction $F(1, 98) = 5.3$, $p = .023$. Means for appetite, negative feelings, positive feelings, and arousal were then also compared (See Table 3 below). Generally, appetite, negative feelings, and positive feelings did not vary significantly between conditions or before and after Jelly Bean consumption. However, negative feelings did vary significantly between conditions before Jelly Bean recall, with the delay group experiencing higher negative feelings.

Table 3. Means for Appetite mood, and arousal for Food Memory Study 2

Variable	Before JB (D)	Before JB (I)	After JB (D)	After JB (I)
Appetite (0-20)	18.7 (±.5.1)	18.3 (±.5.3)	10.9 (±.3.9)	9.7(±.3.7)
Negative Feelings (0-40)	**11.0 (±.6.2)***	**7.9 (±.4.4)***	10.8 (±.9.2)	9.2 (±.6.4)
Positive Feelings (0-20)	10.3 (±.2.9)	9.9 (±.2.9)	10.2(±.3.7)	9.9 (±.3.8)
Arousal (0-20)	8.3 (±.2.5)	8.3 (±.2.3)	9.5 (±.3.1)	9.3 (±.3.7)

Appetite: Fullness (inverse) and desire to eat (+)

Negative Feelings: Sad (+), stressed (+), irritable (+), and nervous (+)

Positive Feelings: Happy (+) and excited (+)

Arousal: Relaxed (inverse) and tiredness (inverse)

JB: Jelly Bean

D: Delayed Recall Condition, I: Immediate Recall Condition

*Significant at the .05 level

Linear Regressions predicting memory performance

A multiple linear regression analysis approach was used to test the hypothesis that traits such as disinhibition and restraint predict food memory (jelly bean recall) and general memory (PAL and DMS). Based on the correlations observed, the following predictor variables were chosen; BMI, delay group, disinhibition, restraint, negative food preoccupation, and positive food preoccupation. The predictors selected were correlated with the outcome variables for cognitive performance, which PAL and DMS task measures. Due to it being a homogenous sample demographics were not included in the models below. When predicting Jelly Bean Recall, no significant factors were found (See Table 4). Backward elimination resulted in a model suggesting that both negative food preoccupation and restraint are significant predictors (See Table 4). An interaction was found between positive food preoccupation and delay condition when predicting Jelly Bean Recall (Table 4). This interaction was then plotted (Figure 2) and indicated that participants who score lower on FPQ+ had better Jelly Bean Recall in the immediate condition than participants who had higher scores in FPQ+. However, in the delayed condition, participants with higher FPQ+ had better Jelly Bean Recall. This interaction was not found with negative food preoccupation. No distraction interactions were found when observing BMI or disinhibition. Post hoc tests were conducted by splitting the file by condition (delay vs immediate group) and running t-tests comparing high FPQ+ vs low PFQ+ for outcome measures. For Figure 2, it indicated that participants low on FPQ+ had significantly lower Jelly Bean recall when in the delay condition (delay/low FPQ+ mean recall= 4.0 (±1.9), (immediate/higher FPQ+ mean recall=5.2 (±2.0), p=.026). An interaction was not found between restraint and delay condition, but as it was a significant predictor, the relationship was plotted (Figure 3). The relationship (Figure 3) indicates that restrained eaters had better Jelly Bean Recall in both conditions. Post hoc tests for Figure 3 indicated that

participants did not vary significantly on Jelly Bean recall, regardless of restraint level or group condition. When predicting PAL mean latency, PAL total errors, and PAL total trials, none of the measures were significant predictors and backward elimination did not indicate otherwise. When predicting DMS total errors and DMS mean latency, none of the measures were significant predictors and backward elimination did not indicate otherwise. When using a hierarchical linear regression to predict DMS Memory load (the total number of items which can be remembered), BMI was a significant factor (See Table 5).

Table 4. Summary of linear regression analysis (1a) and backward elimination(2b) for JB Recall and the interaction between FPQ+ and group condition predicting Jelly Bean Recall

	B[1](±SD)	ß[2]	P-value
Model 1a			
BMI	-.05(± .07)	-.09	.39
Delay	-.66(± .50)	-.15	.15
Restraint	-.65(± .28)	-.28	.03
FPQ +	-.38(±.34)	-.12	.28
FPQ -	.70(±.37)	.24	.06

Disinhibition	-.11(±.41)	-.03	.79
Model 2b			
Group	-.55(±.44)	-.13	.22
Restraint	**-.73(±.28)**	**-.31**	**< .01**
FPQ -	.75(±.35)	.26	.03
Model 3c			
Group	-.42 (± .42)	-.09	.33
FPQ +	**-.73 (±.32)**	**-.23**	**.02**
FPQ+xGroup	**2.2 (±.6)**	**.34**	**<.001**

[1]Unstandardized regression coefficient
[2]Standardized regression coefficient
1a R^2=.10
2b R^2=.08
3c R^2=.14
FPQ: Food Preoccupation Questionnaire

Figure 2. Plotting of interaction between positive food preoccupation and group condition when predicting Jelly Bean Recall

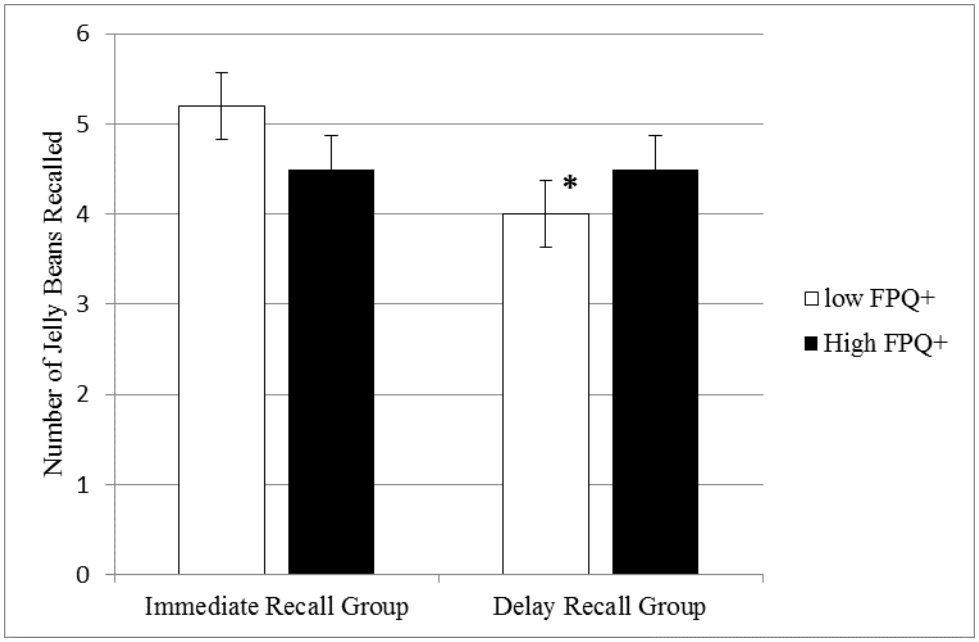


Figure 3. Plotting of interaction between restraint and group condition when predicting Jelly Bean Recall

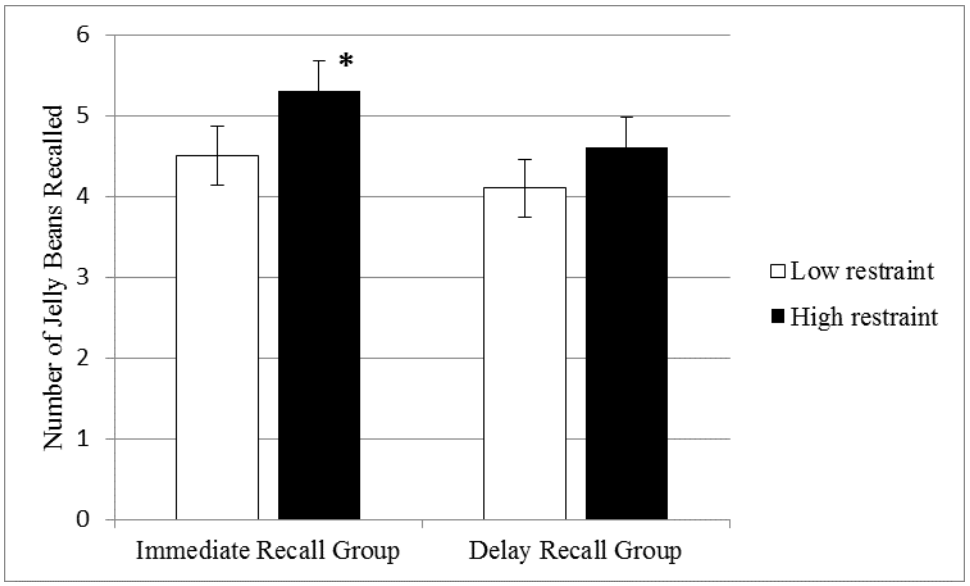


Table 5. Summary of linear regression analysis for traits predicting general

memory (DMS Memory Load)			
	B[1](±SD)	ß[2]	P-value
Group	-.92(± 2.1)	-.05	.65
Restraint	.60(± 1.3)	.06	.66
FPQ +	-1.3 (±1.6)	-.09	.40
FPQ -	.76(±1.7)	.06	.66
Disinhibition	1.3(±1.9)	.07	.51
BMI	**-.70(±5.0)**	**-.24**	**.03**

[1]Unstandardized regression coefficient
[2]Standardized regression coefficient
R^2=.07
FPQ: Food Preoccupation Questionnaire

5.2.3 Food Memory Study 5 Discussion

The primary aim of the second study was to observe how food memory is related to general memory, eating behaviour, and food preoccupation. A food recall task was also developed to test snack food memory (in contrast to meal time memory), using Jelly Beans. The element of distraction was removed and replaced with Delayed versus Immediate recall group. This was done to detect if a delay in recollection along with distraction via a cognitive task was enough to impair food memory. The 2 main outcome measures in this study were food memory (Jelly Bean recall) and general memory (via CANTAB tasks PAL and DMS). Between groups (Delayed v Immediate), there was a significantly lower Jelly Bean recall for low FPQ+ participants in the delay condition. There was no significant effect of restraint on recall in either condition. Generally, groups did not differ in PAL or DMS. However, the Delay group had a slight lag in DMS latency.

Linear regressions were carried out to observe the best predictors of food memory (jelly bean recall) and general memory (PAL and DMS). When predicting Jelly Bean Recall, backward elimination showed negative food preoccupation and restraint were significant predictors. Limitations of using stepwise regression with backward elimination must be considered as generalizability of data may be an issue. However, the sample was representative of the population and so generalizability issues would not be substantial. This method also allows for identifying the most important predictors within this sample. The relationship with restraint was explored and indicated that in both conditions, more highly restrained participants demonstrated better Jelly Bean recall. It has been suggested that restrained eaters pay more attention to food cues (Hollitt, Kemps, Tiggemann, Smeets, Mills 2010), and this may lead to better food memory. Soetens and Braet (2007) reported that even in overweight adolescents, a memory bias for high calorie food could be found. When observing food memory in particular, focusing on food has also been seen to assist with food recall (Higgs and Donohoe 2011). This implies that restrained eaters more accurate memory may have more to do with the attention they pay to food than cognitive ability. When predicting PAL mean latency, PAL total errors, PAL total trials, DMS total errors, and DMS mean latency, no significant predictors were found. However, when using a hierarchical linear regression to predict DMS Memory load, BMI was a significant factor. As has been discussed throughout this thesis, a link between BMI and memory has been repeatedly suggested (Gunstad et al. 2006). However, this study has only found one memory variable to be associated. BMI was a predictor specifically for the DMS memory load (the amount of items which can be remembered), one measure for overall memory. Specifically, it indicates deficits in the medial

temporal lobe. This was the only CANTAB measure that was associated with BMI and perhaps it should be continue to be considered in obesity-cognition research. Due to BMI not being related to food memory, data suggests that there may be a difference between general memory and food memory. However, a female university aged population was used, which may make memory deficits less pronounced. This is the first study to investigate these differences and further supporting studies in future would be beneficial.

An interaction was found between positive food preoccupation and group condition when predicting Jelly Bean Recall. This supports the positive food preoccupation findings in Study 1, in which it served as a predictor for meal vividness. Plotting indicated that participants who score higher on positive food preoccupation had better recall than those with lower FPQ+ in the delay condition. However, participants with lower FPQ+ had better Jelly Bean recall in the immediate recall condition. Post hoc tests indicated that participants low on FPQ+ had significantly lower Jelly Bean recall when in the delay condition. Otherwise, participants did not vary significantly on Jelly Bean recall, regardless of restraint level or group condition. This effect on memory appears to be associated with the delay, which consisted of cognitive tasks before recall. Perhaps the cognitive tasks served as a form of distraction or preoccupation, which would supports the literature that suggests that distraction and/or preoccupation impairs memory (Higgs and Woodward 2009; Jones and Rogers 2003).
As this was a snack condition, in contrast to a meal condition, it is possible that different attentional cues are being triggered. This adds to the idea that as snacks are not seen as positive eating behaviour, they do not produce positive food associations. Perhaps participants with higher positive food preoccupation are not having their positive biases activated, in contrast to those with lower positive food preoccupation. Simultaneously, individuals with

lower positive food preoccupation might be focusing on negative food cues such as ‘This snack is high in sugar’ which are boosting short term memory. These participants are then distracted by the cognitive memory task, decreasing their memory for the jelly bean snack in the delay condition. This idea supported by that data which indicates negative food preoccupation as a predictor for jelly bean recall. These results suggest that preoccupation positive or negative increases memory for snack foods.

Jelly Bean recall, the food memory measure, was not correlated with CANTAB memory measures and neither was disinhibition. As the Jelly Bean Recall measure was unique to this study, there is little food memory research that is directly comparable. Additionally, there is a lack of studies which combine food memory and general memory, making this relationship difficult to predict. As in Study 1, restraint was positively correlated with BMI and negative food preoccupation.

5.2.4 Food Memory Study 4 and 5 discussion

Overall, the data from Study 4 and 5 suggest that food preoccupation may be an important factor affecting foods memory. Food preoccupation generally appears to play an important role in food memory and food intake. Positive food preoccupation was found to predict meal memory along with snack intake and snack memory while negative food preoccupation predicted snack (Jelly Bean) memory. It is difficult to identify the differences positive and negative food preoccupations may have on memory, but it may rely on food type. The pasta meal may be considered a more wholesome eating event (triggering positive food associations/attentional bias), while the Jelly Bean snack is associated with sweets and indulgence (triggering negative food associations/attentional bias). Having found no effects of

BMI or eating traits on food memory may indicate that food preoccupation (positive or negative) is accounting for variance in food memory not adiposity per se. This is supported by the fact that restraint correlated with BMI and with positive/negative food preoccupation across both studies. These studies confirm prior reports on the association between BMI and restrained eating. Interestingly, restraint (and BMI in Study 4) also appear to be associated with negative food preoccupation. Restraint was also a predictor for food memory in Study 4. This may suggest that restrained overeaters either develop, or already have, a kind of negative bias for certain foods. Perhaps this bias serves as a form of attention and contributes to better food memory.

Some strengths and limitations exist when considering the sample in both studies. University aged females were readily available for testing, while a more diverse range of participants would have been desirable. This means that results might not be applicable across the population. However, these findings may be very useful when considering female populations. ANOVAS for appetite, negative feelings, positive feelings, and arousal were as expected. No interactions between distraction and these measures were found. When observing appetite there was an overall difference in appetite before and after eating. This indicates that the measure for hunger/appetite detected the desired variable. Additionally, distraction while eating was, as in prior research, found to increase snack intake. This is proving to be a useful condition when conducting food memory research. A distraction condition is recommended for future experiments. Contrary to some literature, disinhibition was not found to relate to eating behaviour such as restraint. This relationship has been found before (Bellisle, Clément, Barzic, Gall, Guy-Grand, Basdevant 2004) and may be seen as a limitation. However, the DEBQ (Van Strien et al. 1986) which was used to calculate both restraint and disinhibition is well validated and often cited.

Two food recall measures were produced for these studies. For Study 4, the pasta sauce taste test, did not detect any differences between distraction groups. This suggests the task may not have successfully measured food memory. However, an additional food memory measure (meal vividness) was included. This made it possible to still have data using a more reliable measure food memory. For Study 5, the Jelly Bean recall task was developed. Several methods of scoring the task were discussed amongst researchers, and observed after data collection. However, this measure may not be accurate. Predictors of memory, such as restraint, were found for Jelly Bean Recall. This may support the argument that food memory was being detected imply that food memory was being accurately measured. Further investigation of this may measure may prove useful in food memory research.

Findings suggest the importance of food preoccupation as a measure in future food memory research. Its potential role in not only food memory, but later food intake, makes FPQ a significant predictor of eating behaviour as a whole. Specifically, investigations into positive food preoccupation and eating behaviour may reveal intricacies within the obesity-cognition process.

Chapter 5 Study 5 Summary

The study aimed to assess factors that influence food memories, such as BMI, dietary traits, and food preoccupation. We predicted that higher restraint and tendency towards disinhibition and higher BMI would be linked with impaired food memory, but this might be moderated by food preoccupation (attentional bias). Food memory was instigated via a lunchtime meal (Study 1), a snack (Study 2), and general memory measured via CANTAB test batteries (Study 2). Disinhibition was not correlated with food memory or general memory. Restraint

was positively correlated with BMI and negative food preoccupation. BMI was positively correlated with negative food preoccupation and negatively correlated with one of the general memory measures (DMS memory load). It was also a significant predictor for DMS memory load. Negative food preoccupation and restraint were predictors for food memory, while positive food preoccupation predicted food intake. An interaction between positive food preoccupation and group was found to predict food memory in both studies. Findings suggest that an individual's preoccupation with food may be associated with their recollection of an eating episode later on, and thereby later food intake.

CHAPTER 6: General Discussion of thesis

6.1 Summary of findings

The aim of this thesis was to explore how obesity interacts with various aspects of emotion and cognition. Eating behaviour through the DEBQ (Van Strien et al. 1986) and food preoccupation through the FPQ (Tapper and Pothos 2010) were used to measure cognitive processes in relation to food intake. Emotional mechanisms were observed through alexithymia (Rieffe, Oosterveld, and Terwogt 2006), anxiety and depression (Zigmond & Snaith 1983; Beck et al. 1961) scores. Traditional cognitive aspects such as memory, attention, and executive function were assessed thought the Cambridge Neuropsychological Test Automated Battery (CANTAB) (Robbins et al. 1994). A significant aspect of the study design is the incorporation of factors that may have, in past literature, contributed towards effects. Along with age, gender, and BMI, variables such as education, ethnicity, self-esteem (SE) (Rosenberg 1965), impulsivity (BIS) (Patton, Stanford, Barratt 1995), and restraint via the DEBQ were controlled for when appropriate. In addition to this, where appropriate, food preoccupation and depression were controlled for when not included in the outcome measures. It was predicted that higher BMI would be correlated with cognitive impairment, negative emotions, higher scores on the DEBQ, and less healthy lifestyle habits. Due to the variation in sample BMI across studies, some of these answers remain unclear.

Study 1: Emotional processing and quality of life in severe obesity

Clinically obese individuals were found to be at higher risk for anxiety and depression. Anxious and depressed patients reported significantly poorer quality of life (such as physical function and health score) and sleep efficiency scores when compared to their non-

depressed/anxious counterparts. There were no significant differences in BMI between groups, which indicate psychological perspective has significantly influence their views on health status. In addition, quality of life was found to be the best predictor of depression in this sample. These findings add to the idea that the obese experience is variable and complicated by comorbidities. On average, super-morbidly obese patients who participated in a 12 month weight management programme lost weight. Body Mass Index (BMI) was positively correlated with weight-loss, and total number of comorbidities, which suggests that as weight increases so do health complications. Weight change was correlated with programme attendance, but not depression and anxiety.

Study 2: Alexithymia

Overall Alexithymia score was associated with lower fruit and vegetable intake. The alexithymia sub-category Difficulty Describing Feelings (DDF) emerged as the most relevant measure. DDF was associated with lower fruit and vegetable intake, lower exercise frequency, and trended toward higher BMI z-score and unhealthy snacking. Psychological Distress (PD) was negatively correlated with exercise frequency and DDF. This highlighted that although emotional processing influences lifestyle, it is not necessarily one and the same as PD.

Study 3: CANTAB

Participants with higher BMI did not perform more poorly on CANTAB cognitive tasks. BMI was positively associated with emotional eating, depression, impulsivity, and age. It was negatively associated with self-esteem and education. Restrained eating was correlated with emotional eating, depression, and self-esteem. BMI was not consistently correlated with cognitive task performance. BMI was correlated with: PAL Total Errors, PAL number of

trials to complete all stages, and AGN total misses for sad words. Additionally, it was related to some measures of latency: DMS overall latency, PAL overall latency, AGN latency for sad words, IED latency stages 8-9, and SOC mean thinking time. When controlling for the confounds of ethnicity, education, BIS (impulsivity), BDI (depression), SE (self-esteem), age, and gender, BMI was not the strongest predictor for cognitive performance. Age and gender were the top predictors for performance overall. No interactions were found between BMI and gender. These findings suggest obesity-cognition literature may find other factors are contributing towards cognitive decline, not obesity alone.

Food Memory Studies (4 and 5)

The relationship between distraction and food memory was observed, along with differences in eating behaviour and food preoccupation scores. Study 5 investigated how food memory is related to general memory, eating behaviour, and food preoccupation. Two new measures for food memory were developed. The first, the pasta sauce taste test, did not detect differences between distracted and non-distracted participants. The second food memory measure was developed to test snack food memory, using Jelly Beans. This measure did not detect differences between groups, but appeared sensitive to food memory with further analysis. In Study 4, restraint was correlated with BMI and negative food preoccupations. Positive food preoccupations had a significant negative correlation with biscuits eaten, and positive correlation with the second food memory measure (meal vividness). Positive food preoccupation also served as a predictor for both these measures. Additionally, distraction group predicted biscuits eaten. In study 5, between groups (Delayed v Immediate) there were no significant differences for Jelly Bean recall (food memory) or PAL and DMS performance (general memory measure), except for a slight lag in DMS latency for the delay group.

Overall, disinhibition was not correlated with food preoccupation, food memory or general memory. Also, positive food preoccupation was found to predict food memory in both studies. These studies suggest that food memory may be different from overall memory. Findings imply that different sub-types of preoccupation serve as forms of attention bias, which may actually aid in food memory.

6.2 Critical overview

One of the primary aims of this thesis was to come closer to deciphering how the mechanisms behind obesity interact. One way to observe this is by considering how BMI interacted with measures across studies. In a super-morbidly obese weight management group sample, BMI was negatively correlated with weight change (as BMI increases, weight-loss increases), and positively correlated with some quality of life measures (physical function and public distress). Participants were found to generally be very anxious and depressed, and BMI was also a strong predictor of quality of life overall. In an adolescent sample, BMI z-score was positively correlated with unhealthy snacking and the Alexithymia subscale DDF. In a diverse sample, BMI was positively correlated with age, emotional eating, and depression, while being negatively correlated to self-esteem and education level. It was also a strong predictor for latency in the cognitive Delayed Match to Sample (DMS) memory task. Lastly, in a university sample, BMI was positively correlated with restraint, negative food preoccupation, and negatively correlated with the memory load (the number of items which can be remembered) measure in the Delayed Match to Sample (DMS) task. When combined, these findings suggest that higher BMI is related to poorer eating habits, anxiety and depression, poorer perceived quality of life, aging, low self-esteem, and difficulties with memory.

A potential view of the relationships investigated is that of an obesity cycle. In this cycle the comorbidities/factors which repeatedly appear in the literature contribute towards the perpetuation of obesity and cognitive disruptions. These cognitive disruptions may range from preoccupations or distractions that prevent an individual from functioning at their intellectual best, to emotional fluctuations which cause an individual to use old coping mechanisms (such as emotional eating). Figure 1 demonstrates how this cycle may operate.

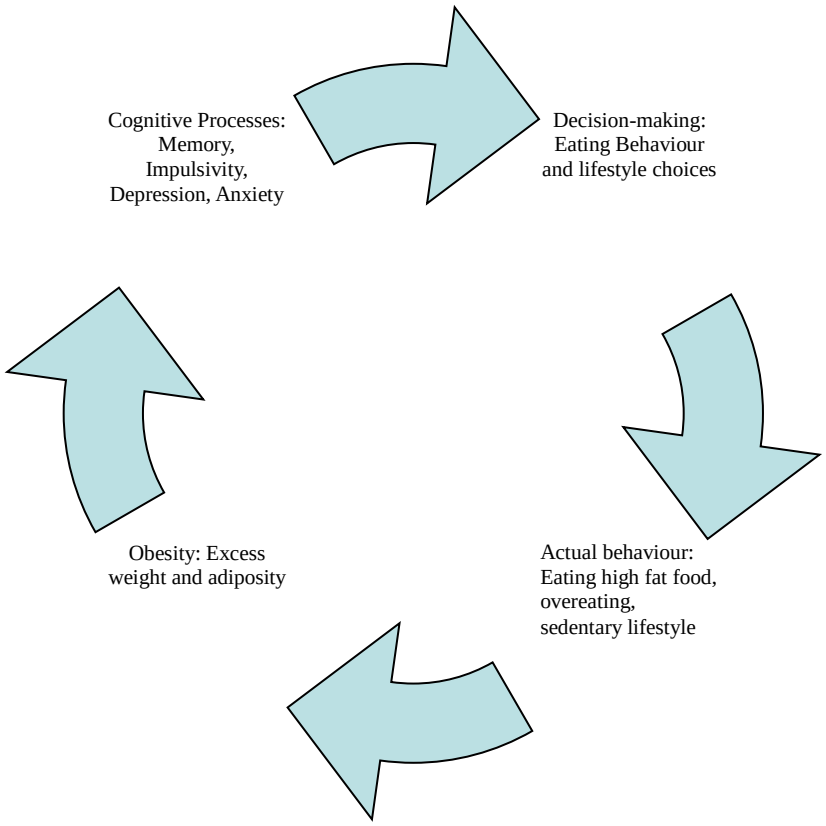


Figure 1: Obesity Cycle Model

When observing this model it may be of use to discuss how examples from study findings fit into each phase. Starting at the cognitive processes, higher BMI was found to be associated with anxiety and depression, low self-esteem, and problems with working memory. The emotional dysregulation may create a need for coping mechanisms, while memory

impairment may make it difficult for an individual to recall what they have eaten. In the next two phases, Decision-making and Actual behaviour, coping mechanisms or behaviours used to deal with emotional dysregulation may develop. For instance, emotional eating, which was found to be positively correlated with BMI, or less active lifestyle (which was related to alexithymia subscale Difficulty Describing Feelings). The type of food selected may be unhealthy, as BMI was also related to unhealthy food choices. This leads the obesity phase, where a poorer perceived quality of life takes place, and individuals become more at risk for these complications as they age. However, it seems as though individuals may begin the obesity cycle at different phases, most likely depending on their emotional states.

6.3 Implications

It is a relatively novel approach to view cognition as both a performance based ability (memory for example) and an emotional measure (depression for example) at once. As these traits are so closely linked, it has proved insightful to combine them in obesity research. Additionally, although prior study designs have used computerized assessments of neuropsychological ability, they have not included emotional well-being or eating behaviour. In this thesis, these measures have been used jointly due to the importance of considering an individual's complex cognitive profile, and how it may result in a more complete picture of their relationship with obesity. For instance, previous studies that have assessed an individual's performance on a cognitive task (Saczynski et al. 2008; Lasselin et al. 2012; Kaufmann et al. 2012), have not included consideration of depression or food preoccupation, which may result in findings about cognition that are not as accurate as they can be.

This thesis highlights that a major mechanism behind obesity is the cognitive process experienced by an individual. This process may include emotions, decision making, trends in behaviour (such as binge eating), and memory for previous meals. These findings are individually supported by prior studies linking obesity to memory impairment (Lasikiewicz, Hendrickx, Talbot, and Dye 2013; Winocur and Greenwood 2005), emotional well-being (Jorm et al. 2007), and anxiety and depression (Strine et al. 2007). Although there may be an association between certain cognitive characteristics and obesity, it is not clear what type of cognitive traits play a larger role. Furthermore, research lacks a consideration of what cognitive traits are related to obesity in unison. For example, it is not known how significant cognitive ability (in areas such as memory and executive function) is in comparison to an individual's emotional state when making lifestyle choices. As discussed earlier in the obesity cycle model, emotional dysregulation may cause an individual to turn to unhealthy coping mechanisms. However, memory impairment in any situation will make it difficult to regulate food intake. Deciphering if memory or emotion plays a more important role may aid in developing more successful intervention approaches. Additionally, when using a cognitive approach to investigate obesity, it should be remembered that being unaware of how to make healthy lifestyle choices may lead an individual without cognitive impairments or emotional dysregulation into the obesity cycle.

Results for this thesis are of particular interest due to the uniqueness of samples, measures, and findings. Overall, emotions play a larger than expected role in eating behaviour and obesity. Age and gender, appear to affect cognitive function more so than weight, when controlling for impulsivity, emotions, and demographics. The question is, are some of these

factors inherent to the obese condition? Perhaps. However, narrowing the precise mechanisms behind cognitive decline and eating behaviour will assist in better research strategies, and more promising interventions.

As these results indicate, being very overweight increases an individual's chances of being anxious and depressed. Being more anxious and depressed, may then distort your view on your quality of life and physical function. The study on food memory pointed out how food preoccupations, in particular positive ones (or lack thereof), may determine later food intake. Perhaps one aspect of the obesity cycle is this initial emotional perspective, on life and/or food, which guides our lifestyle decisions. This process may begin in adolescence. Although psychological distress in itself may not predict lifestyle choices in youth, Difficulty Describing Feelings (DDF), which did predict healthy lifestyle choices, may be an initial marker of emotional processing dysfunction. This dysfunction may also be related to emotional eating, which was positively correlated with BMI. It is also of interest that age and gender may account for cognitive impairments following weight gain, as they are traits that will not be addressed in interventions. They are, however, significantly correlated with depression, which can be treated. As mentioned in Chapter 4, emotional and behavioural changes are among the more efficient tactics when attempting lifestyle change, and should perhaps gain more recognition in this obesity-cognition research area. On the other hand, simple techniques such as avoiding distraction while eating may also help with overweight as well.

Findings in this data have aided in understanding previous obesity literature. This data has indicated that there is a relatively unstudied complexity behind the relationships between

obesity, eating behaviour, emotions, and cognition. Using these findings, and continuing research in this vein, may improve future weight management interventions. Changes may include training staff in clinical psychology, such as how to identify signs of anxiety and depression. Building awareness within health professionals about the influence of emotional well-being on physical well-being would be beneficial. It is a common perception that weight-loss and obesity is simply the result of overeating and a sedentary nature; however, this data suggest emotion/cognitive health are serious roadblocks to weight-loss. Weight-loss aside, treatment of these emotional issues may lead to improved quality of life. As stated previously, improved quality of life may aid in weight-loss, which should be stressed to professionals aiming to hit weight-loss targets. Interventions aimed at improving self-esteem and increasing positive feelings may become more common.

6.4 Strengths and Limitations

A potential limitation of this thesis was the fluctuation in sample types. Study 1 had a clinically super-morbidly obese sample, the alexithymia study had healthy adolescents (Study 2), and Studies 4 and 5 tested university aged females, while Study 3 recruited a more diverse mixture of participants. These sample choices were necessary to ensure strong participant numbers in each study. These differences may appear to make the results more difficult to apply to the general population. However, each sample is actually representative of populations which are in need of further investigation. There are certainly gaps in the literature for studies on the super-morbidly obese, the mechanisms behind healthy lifestyle for adolescents, food memory investigation overall, and cognitive influences on weight in a more

'general' sample. When considering the CANTAB study it is important to remember that the sample used was more educated than the general population, with 45.7% of participants being at degree level or above. As education was controlled for this should not have significantly affected findings. Overall, there were significantly more females in the studies, which may have affected the results. However, gender was always controlled for and considered.

A relevant debate in obesity research is the use of BMI in contrast to other measures such as waist circumference, which has been reported as a better indicator for comorbidities (Savva et al. 2000). However, no significant differences have also been suggested between these measures (Reilly et al. 2010; Garnett et al. 2007). Overall, the conclusion appears to be that BMI is a good predictor for health outcomes, while waist circumference may occasionally be more sensitive. In future it may be of use to, in addition to BMI, include measures of waist circumference along with body fat composition. This would enable researchers to determine which obesity measures are more appropriate for their area of interest. For instance, within quality of life and emotion based research, BMI may be an accurate and cost effective measure. But perhaps in cognition research, body fat composition measurements would be more sensitive.

Questionnaires were used throughout the studies, making much of the data self-report. All the questionnaire measures have been validated and widely used. Self-report measures are popular in research as researchers can collect large amounts of data easily (Boca and Noll 2002). In the current studies, a researcher was always readily available provide hands-off supervision and assistance during administration. However, this is also a strength of the study, as it enabled researchers to include the variety of measures recorded (ranging from food

preoccupation to sex life, to anxiety, and sleep) and finding a large population of severely overweight participants to observe. Some alternatives to self-report measures may be to request consent and ethical approval to access participant medical records, which would enable accurate reporting of mental (such as anxiety and depression) and physical health (such as smoking status and diabetes) . However, compared to anonymous questionnaires, this would be more invasive and may deter participation in studies. Individually obtaining medical documentation may also prove time consuming while increasing study costs (for labour) and length. In terms of eating behaviour traits, it would be very difficult to measure without a questionnaire. Observing a participant in a food laboratory for traits such as emotional would be complex, along with being a measure of only one (or a few) instances, which may be an inaccurate representation of the participant. Once again, this would be much more time consuming and likely produces less data at a higher cost.

Some measures were developed for the studies they were used in. For example, in the alexithymia study, Psychological distress (PD) was a created by combining four items. These items were straightforward, such as ‘Do you feel unhappy, sad, or depressed?’ The items used were also all highly correlated and fit previous literature expectations such as being related to higher BMI (Sjoberg, Nilsson, Leppert 2005) and less exercise (Field, Diego, Sanders 2001). The reason for this was due to a need for assessments that are not yet available. This simultaneously serves as a benefit as trial and error in testing new tools increases current knowledge in the area, and assists in the development of more sensitive measures for the future. For instance, there were mixed findings for the two food recall measures produced in the food memory studies. For Study 4, the pasta sauce taste test did not appear to measure food memory. This may be due to the task being too difficult. Fortunately, an additional

Visual Analogue Scale (VAS) for food memory measure (meal vividness) was included as a more reliable measure food memory. This enabled researchers to run more extensive pilots when developing the food memory measure (Jelly Bean Recall) for Study 5, which did appear to be sensitive to food memory.

There were some expected relationships that were not found. In Chapter 2, more anxious individuals were expected to lose less weight (Anderson, Cohen, Naumova, Must 2007). It should, however, be considered that this previous finding may not apply towards super-morbidly obese populations with higher levels of anxiety/depression. In the alexithymia study, the finding that DDF was the best predictor for lifestyle habits in adolescents was also unexpected. This may prove useful in future health-alexithymia research. In the food memory studies, disinhibition was not found to play a significant role in cognitive or eating processes. However, it was derived from a highly used and validated questionnaire (DEBQ) (Van Strien et al. 1986). The addition of novel measures such as food preoccupation may have diluted the previously disinhibition affect. Although disinhibition was not related to meal memory, it was tending towards a positive relationship with negative food preoccupation in Study 4, which was a significant predictor of snack recall in Study 5. Additionally, positive food preoccupation had a negative correlation to biscuits eaten, which would have been closely linked to disinhibited behaviour.

There are several novel aspects to this thesis. It included the first study of its kind to collect alexithymia and lifestyle and eating behaviour in adolescents. The weight management study provided a unique insight into a super-morbidly obese sample. The food memory studies combined food preoccupation, unique food memory specific measures, and observed eating

behaviour. In the CANTAB study, correlations are supported by prior obesity research findings. However, other results are not directly comparable. As this study included controlling for factors unique to obesity-cognition research, such as depression and impulsivity, the results may be seen as an extension of the prior literature.

6.5 Future studies

Previous literature in the area of obesity is vast, and future studies which incorporate an array of obesity-cognition factors are recommended. In the past, the literature has lacked integration of the emotional (anxiety, depression, self-esteem), cognitive (memory, impulsivity, mental flexibility), and behavioural (disinhibition, actual food intake) areas of obesity. Research can be found within each of these areas, but more collaborations would reflect the workings of a more complete depiction of the obese experience on a daily basis. As depicted in the obese cycle model (above), there are likely indicators or catalysts for behaviour which leads an individual to obesity and keeps them there. This thesis approaches obesity as more than a BMI, but as a complex routine that individuals may have entered recently, or as early as adolescence. It highlights that investigations which measure not only food intake, but personality traits and emotional states are essential in the future.

Depending on the specific area of focus, particular factors are advised. When investigating weight interventions in the severely obese, researchers who are familiar with areas in clinical

psychology, such as anxiety, depression, and self-esteem, would be useful in building models which incorporate the role of emotions in eating behaviour. For instance, studies which measure negative emotions prior to lifestyle change, and then include a group that receives cognitive therapy, may be able to report on whether therapy can improve weight change outcomes. This may also improve quality of life, which is related to anxiety and depression in the severely obese. In quality of life research, controlling for age, gender, and depression/anxiety is also important. However, it would be very interesting to further investigate the relationship between emotions and perceptions in the obese. Of particular curiosity may be observing any other differences (such as personal family history or medical history) between non-anxious/non-depressed obese individuals compared to the anxious/depressed obese. Perhaps if researchers could pinpoint how the non-anxious/non-depressed obese has avoided emotional dysregulation, they will be able to devise interventions to assist their anxious/depressed counterparts. 'For example, it may be insightful to design a qualitative study which interviews severely obese individuals who have reported good quality of life. This may demonstrate whether interventions aimed at increasing positive feelings may be of use when creating lifestyle change. In neurocognitive research, controlling for age, gender, and depression/anxiety is important. In addition to this, traits such as impulsivity, self-esteem, education level, and ethnicity may also play a significant role. Further studies on different forms of memory would be beneficial in this area. Specifically, studies examining several kinds of memory (food memory, visuospatial memory, working memory) and how different kinds of people may experience deficits as their weight increases. This can be across different ethnicities, genders, athletes and non-athletes, and age ranges. It would be interesting to see the commonalities and differences that appear across the population and this may help in developing interventions aimed at specific subgroups. For

example, a large study comparing men and women of varying BMI's and ethnicities, which measures food memory, later food intake, and general may prove insightful. Another interesting study would be one that follows adolescents who exhibit emotional dysfunction, into adulthood. This may help in pinpointing lifestyle destructive behaviours.

The previous literature has shown associations between cognition and obesity (Volkow et al. 2008; Brooks et al. 2013). Specifically, cognitive deficits in memory (Lasikiewicz, Hendrickx, Talbot, and Dye 2013; Winocur and Greenwood 2005), executive function (Wang et al. 2013; Gunstad, Paul, Cohen, Tate, Spitznagel, Gordon 2007), and even higher rates in impulsivity (Mobbs, Crepin, Thiery, Golay, Linden 2010; Nederkoorn, Jansen, Mulkens, Jansen 2007) have been reported. However, the literature does not make clear how this relationship originates or progresses. This thesis has found that although those cognition-obesity associations due exist, they are complicated by other factors. For example, prior studies highlight that emotional disturbances, such as anxiety (Strine et al. 2008), depression (Jorm et al. 2007), and alexithymia (Zak-Golab et al. 2013) have been found to correlate with BMI, yet the role of emotions in the obese-cognition cycle has remained relatively unexplored in past literature. Additionally, previous studies (van den Berg et al. 2009; Saczynski et al. 2008), consider confounds such as age and gender, but exclude other relevant factors in obesity, such as food preoccupations and eating attitudes. This thesis suggests that when including these potentially underlying mechanisms, there is a less direct relationship between obesity and cognition than the literature implies. For instance, study 3 found that BMI was not the best predictor for cognitive performance, while studies 4 and 5 indicated that positive food preoccupation predicted memory for food, but not general memory. This thesis suggests the importance of incorporating additional relevant factors when investigating how the cognition,

emotion, and obesity association develops.

There are an abundance of factors that may lead to obesity, and it is unclear which ones have the most significant influence. The findings of this thesis bring to attention the need for more comprehensive approaches to obesity research. Due to the interactions between psychological, biological, and neurological traits, future studies should be inclusive of these areas. Generally speaking, more research designs which consider the entire obesity-cognition cycle, within a diverse sample, may result in findings that significantly advance this area.

References

Adami, G. F., Campostano, A., Ravera, G., Leggieri, M., & Scopinaro, N. (2001). Alexithymia and Body Weight in Obese Patients. *Behavioral Medicine*, *27*(3), 121-126.

Ahima, R. S. (2009). Connecting obesity, aging and diabetes. *Nature Medicine*, *15*(9), 996-997.

Algul, A., Ates, M. A., Semiz, U. B., et al., Evaluation of general psychopathology, subjective sleep quality, and health-related quality of life in patients with obesity. *International Journal of Psychiatry in Medicine*, vol. 39, no. 3, pp. 297–312, 2009.

Alhola, P., & Polo-Kantola, P. (2007). Sleep deprivation: Impact on cognitive performance. *Neuropsychiatric disease and treatment*, *3*(5), 553-567.

Allison, D. B., & Schlundt, D. G. (1995). Assessment of specific eating behaviors and eating style. *Handbook of assessment methods for eating behaviors and weight related problems: measures, theory, and research* (pp. 241-302). Thousand Oaks, Calif.: Sage Publications.

Alosco, M. L., Spitznagel, M. B., Strain, G., Devlin, M., Cohen, R., Paul, R., et al. (2013). Improved memory function two years after bariatric surgery. *Obesity*, *22*(1), 32-38.

Alvarez-Castro, P., Pena, L., & Cordido, F. (2013). Ghrelin in Obesity, Physiological and Pharmacological Considerations. *Mini-Reviews in Medicinal Chemistry*, *13*(4), 541-552.

Anderson, S.E., Cohen, P., Naumova, E. N., Jacques, P. F., Must, A. Adolescent obesity and risk for subsequent major depressive disorder and anxiety disorder: prospective evidence. *Psychosom Med* 2007;69 (8) 740- 747.

Anderson, S. E., Cohen, P., Naumova, E. N., Must, A. Association of depression and

anxiety disorders with weight change in a prospective community-based study of children followed up into adulthood. *Arch Pediatr Adolesc Med* 2006;160:285– 91.

Apovian, C. M., Frey, C. M., Wood, G. C., Rogers, J. Z., Still, C. D., Jensen, G. L. Body mass index and physical function in older women. *Obes Res*, 2002;10:740–7.

Arble, D. M., Bass, J., Laposky, A. D., Vitaterna, M. H., & Turek, F. W. (2009). Circadian Timing of Food Intake Contributes to Weight Gain. *Obesity*, *17*(11), 2100-2102.

Astrup, A. and Finer, N. (2000), Redefining Type 2 diabetes: 'Diabesity' or 'Obesity Dependent Diabetes Mellitus'?. *Obesity Reviews*

Atlantis E, Baker M (2008) Obesity effects on depression: Systematic review of epidemiological studies. International Journal of Obesity, 32: 881–891.

Averbukh, Y., Heshka, S., El-Shoreya, H., et al. (2003) Depression score predicts weight loss following Roux-en-Y gastric bypass. *Obes Surg.* 13: 833–836.

Barnham, K. J., Masters, C. L., & Bush, A. I. (2004). Neurodegenerative Diseases And Oxidative Stress. *Nature Reviews Drug Discovery*, *3*(3), 205-214.

Batsis, J. A., Romero-Corral, A., Collazo-Clavell, M. L., Sarr, M. G., Somers, V. K., & Lopez-Jimenez, F. (2008). Effect of Bariatric Surgery on the Metabolic Syndrome: A Population-Based, Long-term Controlled Study. *Mayo Clinic Proceedings*, *83*(8), 897-906.

Beccuti, G., & Pannain, S. (2011). Sleep and obesity. *Current Opinion in Clinical Nutrition and Metabolic Care* , *14*(4), 402-412.

Beebe, D. W., Lewin, D., Zeller, M., McCabe, M,. MacLeod, K., Daniels, A. R., Amin, R. Sleep in Overweight Adolescents: Shorter Sleep, Poorer Sleep Quality, Sleepiness, and Sleep-Disordered Breathing *J. Pediatr. Psychol*. (2007) 32(1): 69- 79.

Bellisle, F., ClÃ©ment, K., Barzic, M. L., Gall, A. L., Guy-Grand, B., & Basdevant, A. (2004). The Eating Inventory and Body Adiposity from Leanness to Massive Obesity: a Study of 2509 Adults**. *Obesity*, *12*(12), 2023-2030.

Belsky, D. W., Caspi, A., Goldman-Mellor, S., Meier, M. H., Ramrakha, S., Poulton, R., et al. (2013). Is Obesity Associated With a Decline in Intelligence Quotient During the First Half of the Life Course?. *American Journal of Epidemiology*, *178*(9), 1461-1468.

Benito-LeÃ³n, J., Mitchell, A. J., HernÃ¡ndez-Gallego, J., & Bermejo-Pareja, F. (2013). Obesity and impaired cognitive functioning in the elderly: a population-based cross-sectional study (NEDICES). *European Journal of Neurology*, *20*(6), 899-e77.

Benton, D., & Parker, P. Y. (1998). Breakfast, blood glucose, and cognition. *The American Society for Clinical Nutrition*, *67*, 7725-7785.

Betteridge, D. (2000). What Is Oxidative Stress?. *Metabolism*, *49*(2), 3-8.

Biessels, G. J., & Gispen, W. H. (2005). The impact of diabetes on cognition: What can be learned from rodent models?. *Neurobiology of Aging*, *26*(1), 36-41.

Bixler, E. O., Vgontzas, A. N., Lin, H. M. Association of hypertension and sleep-disordered breathing. *Arch Intern Med.* 2000;160:2289–95.

Blakemore, A. I., & Buxton, J. L. (2014). Obesity, genetic risk, and environment. *BMJ*, *348*(mar19 3), g1900-g1900.

Boehmer, T. K., Lovegreen, S. L., Haire-Joshu, D., & Brownson, R. C. (2006). What Constitutes an Obesogenic Environment in Rural Communities?. *American Journal of Health Promotion*, *20*(6), 411-421.

Bondia-Pons, I., Ryan, L., & Martinez, J. A. (2012). Oxidative stress and inflammation interactions in human obesity. *Journal of physiology and biochemistry*, *68*(4), 701-

711.

Bonnet, A., Brejard, V., Pasquier, A., & Pedinielli, J. (2012). Affectivity and alexithymia: Two dimensions explicative of the relationship between anxiety and depressive symptoms. *Encephale-Revue De Psychiatrie Clinique Biologique et Therapeutiqu*, *38*(3), 187-193.

Bouchard, D. R., Janssen, I. Dynapenic-obesity and physical function in older adults. *J Gerontol A Biol Sci Med Sci*. 2010;65(1):71–77.

Bourke, C., Porter, R. J., Carter, J. D., Mcintosh, V. V., Jordan, J., Bell, C., et al. (2012). Comparison of neuropsychological functioning and emotional processing in major depression and social anxiety disorder subjects, and matched healthy controls. *Australian and New Zealand Journal of Psychiatry*, *46*(10), 972-981.

Bourke, M. P., Taylor, G. J., Parker, J. D., & Bagby, R. M. (1992). Alexithymia in women with anorexia nervosa. A preliminary investigation. *The British Journal of Psychiatry*, *161*(2), 240-243.

Boynton-Jarrett, R., Thomas, T. N., Peterson, K. E., Wiecha, J., Sobol, A. M., & Gortmaker, S. L. (2003). Impact Of Television Viewing Patterns On Fruit And Vegetable Consumption Among Adolescents. *Pediatrics*, *112*(6), 1321-1326.

Brunstrom, J. M., Davison, C. J., & Mitchell, G. L. (2005). Dietary restraint and cognitive performance in children. *Appetite*, *45*(3), 235-241.

Brunstrom, J., & Mitchell, G. (2006). Effects of distraction on the development of satiety. *British Journal of Nutrition*, *96*(4), 761-769.

Burger, K., & Stice, E. (2014). Greater striatopallidal adaptive coding during cueâ€“reward learning and food reward habituation predict future weight gain. *NeuroImage*, *99*(1), 122-128.

Butt, H., Atturu, G., London, N., Sayers, R., & Bown, M. (2010). Telomere Length Dynamics in Vascular Disease: A Review. *European Journal of Vascular and Endovascular Surgery*, *40*(1), 17-26.

Buysse, D.J., Reynolds III, C.F., Monk, T.H., Berman, S.R., & Kupfer, D.J. (1989). The Pittsburgh Sleep Quality Index: A new instrument for psychiatric practice and research. *Journal of Psychiatric Research, 28*(2), 193-213.

Carano, A., Salerno, R. M., Gambi, F., Berardis, D. D., Ferro, F. M., Cotellessa, C., et al. (2006). Alexithymia and body image in adult outpatients with binge eating disorder. *International Journal of Eating Disorders*, *39*(4), 332-340.

Casto, S. D., Defries, J. C., & Fulker, D. W. (1995). Multivariate genetic analysis of Wechsler Intelligence Scale for Childrenâ€"Revised (WISC-R) factors. *Behavior Genetics*, *25*(1), 25-32.

Chaput, J., Klingenberg, L., Astrup, A., & SjÃ¶din, A. M. (2011). Modern sedentary activities promote overconsumption of food in our current obesogenic environment. *Obesity Reviews*, *12*(5), e12-e20.

Chen, H., Vlahos, R., Bozinovski, S., Jones, J., Anderson, G. P., & Morris, M. J. (2005). Effect of Short-Term Cigarette Smoke Exposure on Body Weight, Appetite and Brain Neuropeptide Y in Mice. *Neuropsychopharmacology*, *30*, 713-719.

Church, T. S., Bouchard, C., Blair, S. N., Martin, C. K., Rodarte, R. Q., Earnest, C. P., et al. (2011). Trends over 5 Decades in U.S. Occupation-Related Physical Activity and Their Associations with Obesity. *PLoS ONE, 6*(5), e19657.

Clerici, M., Albonetti, S., Papa, R., Penati, G., & Invernizzi, G. (1992). Alexithymia and Obesity. *Psychotherapy and Psychosomatics*, *57*(3), 88-93.

Cochrane, C. E., Brewerton, T. D., Wilson, D. B., & Hodges, E. L. (1993). Alexithymia in the eating disorders. *International Journal of Eating Disorders*, *14*(2), 219-222.

Collins, S., Daniel, K., & Rohlfs, E. (1999). Depressed expression of adipocyte beta-adrenergic receptors is a common feature of congenital and diet-induced obesity in rodent. *International Journal of Obesity and Related Metabolic Disorders*, *23*(7), 669-677.

Corcos, M., Guilbaud, O., Speranza, M., Paterniti, S., Loas, G., Stephan, P., et al. (2000). Alexithymia and depression in eating disorders. *Psychiatry Research*, *93*(3), 263-266.

Correia, M. L., & Haynes, W. G. (2004). Leptin, obesity and cardiovascular disease. *Current opinion in nephrology and hypertension*, *13*(2), 215-223.

Crisp, A.H., McGuinness, B. (1976) Jolly fat: relation between obesity and psychoneurosis in general population. *Br Med J*, 1 , 7–9

Davis, C., Strachan, S., & Berkson, M. (2004). Sensitivity to reward: implications for overeating and overweight. *Appetite*, *42*(2), 131-138.

Deborde, A., Berthoz, S., Godart, N., Perdereau, F., Corcos, M., & Jeammet, P. (2006). Relations between alexithyrnia and anhedonia : a study in eating disordered and control subjects. *Encephale-Revue De Psychiatrie Clinique Biologique Et Therapeutique*, *33*(1), 83-91.

den Berg, E. v., Kloppenborg, R. P., Kessels, R. P., Kappelle, L. J., & Biessels, G. J. (2009). Type 2 Diabetes Mellitus, Hypertension, Dyslipidemia And Obesity: A Systematic Comparison Of Their Impact On Cognition. *Biochimica et Biophysica Acta (BBA) - Molecular Basis of Disease*, *1792*(5), 470-481.

Diano, S., Tschap, M. H., Nonaka, N., Gaskin, F. S., Horvath, B., Silva, I. d., et al. (2006). Ghrelin Controls Hippocampal Spine Synapse Density and Memory Performance.

Nature Neuroscience, *9*(3), 381-388.

Dolan, M. (2012). The neuropsychology of prefrontal function in antisocial personality disordered offenders with varying degrees of psychopathy. *Psychological Medicine, 42(8)*, 1715-1725.

Dykes, J., Brunner, E. J., Martikainen, P. T., & Wardle, J. (2003). Socioeconomic gradient in body size and obesity among women: the role of dietary restraint, disinhibition and hunger in the Whitehall II study. *International Journal of Obesity*, *28*(2), 262-268.

Ebbeling, C., Pawlak, D., & Ludwig, D. (2002). Childhood Obesity: Public-health Crisis, Common Sense Cure. *The Lancet*, *360*(9331), 473-482.

Edwards, C., Nicholls, D., Croker, H., Zyl, S. V., Viner, R., & Wardle, J. (2005). Family-based behavioural treatment of obesity: acceptability and effectiveness in the UK. *European Journal of Clinical Nutrition*, *60*(5), 587-592.

Egerhazi, A., Balla, P., Ritzl, A., Varga, Z., Frecska, E., & Berecz, R. (2013). Automated Neuropsychological Test Battery in depression –preliminary data, 5-11.

Elfhag, K., & Lundh, L. (2007). TAS-20 alexithymia in obesity, and its links to personality. *Scandinavian Journal of Psychology*, *48*(5), 391-398.

Elvsashagen, T., Vera, E., Baen, E., Bratlie, J., Andreassen, O. A., Josefsen, D., et al. (2011). The load of short telomeres is increased and associated with lifetime number of depressive episodes in bipolar II disorder. *Journal of Affective Disorders*, *135*(1-3), 43-50.

Epel, E. S., Blackburn, E., Lin, J., Dhabhar, F., Adler, N., Marrow, J., et al. (2004). Accelerated Telomere Shortening In Response To Life Stress. *Proceedings of the National Academy of Sciences*, *101*(49), 17312-17315.

Epel, E. (2009). Psychological and metabolic stress: A recipe for accelerated cellular aging?. *Hormones*, *8*(1), 7-22.

Epel, E., Daubenmier, J., Moskowitz, J. T., Folkman, S., & Blackburn, E. (2009). Can Meditation Slow Rate Of Cellular Aging? Cognitive Stress, Mindfulness, And Telomeres. *Annals of the New York Academy of Sciences*, *1172*(1), 34-53.

Faulconbridge, L., Wadden, T., Sarwer, D., Womble, L., Hesson, L., Stunkard, A., & Fabricatore, A. (2009). Changes in symptoms of depression with weight loss: Results of a randomized trial. *Obesity*, 17(5), 1009-1016.

Fay, S. H., & Finlayson, G. (2011). Negative affect-induced food intake in non-dieting women is reward driven and associated with restrainedâ€“disinhibited eating subtype. *Appetite*, *56*(3), 682-688.

Fedor, A., & Gunstad, J. (2013). Higher BMI Is Associated with Reduced Cognitive Performance in Division I Athletes. *Obesity Facts*, *6*(2), 185-192.

Ferrante, A. W. (2007). Obesity-induced inflammation: a metabolic dialogue in the language of inflammation. *Journal of Internal Medicine*, *262*(4), 408-414.

Flegal, K., Kit, B., Orpana, H., & Graubard, B. (2013). Association of All-Cause Mortality With Overweight and Obesity Using Standard Body Mass Index Categories. *The Journal of the American Medical Association*, *309*(1), 71-82.

Flook, L., Goldberg, S. B., Pinger, L., Bonus, K., & Davidson, R. J. (2013). Mindfulness for teachers: A pilot study to assess effects on stress, burnout and teaching efficacy. *Mind, Brain, and Education, 7(3),* 182–195.

Francis, H., & Stevenson, R. (2011). Higher reported saturated fat and refined sugar intake is associated with reduced hippocampal-dependent memory and sensitivity to interoceptive signal. *Behavioral Neuroscience*, *125*(6), 943-955.

Fredriksson, R., Hagglund, M., Olszewski, P. K., Stephansson, O., Jacobsson, J. A., Olszewska, A. M., et al. (2008). The Obesity Gene, FTO, Is of Ancient Origin, Up-Regulated during Food Deprivation and Expressed in Neurons of Feeding-Related Nuclei of the Brain. *Endocrinology, 149*(5), 2062-2071.

Friedman, M.A., Brownell, K.D. (1995). Psychological correlates of obesity: moving to the next research generation. *Psychol Bull*, 117, pp. 3–20

Friedman, J., & Halaas, J. (1998). Leptin and the regulation of body weight in mammals. *Nature, 395,* 763-770.

Fukunishi, I., & Kaji, N. (1997). Externally Oriented Thinking Of Obese Men And Women. *Psychological Reports, 80*(1), 219-224.

Galioto, R. M., Alosco, M. L., Spitznagel, M. B., Stanek, K. M., & Gunstad, J. (2013). Cognitive reserve preserves cognitive function in obese individuals. *Aging, Neuropsychology, and Cognition*, *20*(6), 684-699.

Galioto, R., Garcia, S., Spitznagel, M. B., Strain, G., Devlin, M., Crosby, R. D., et al. (2013). The Mini-Mental State Exam (MMSE) is not sensitive to cognitive impairment in bariatric surgery candidates. *Surgery for Obesity and Related Diseases, 10*(3), 553-557.

Galioto, R., Gunstad, J., Heinberg, L. J., & Spitznagel, M. B. (2013). Adherence and Weight Loss Outcomes in Bariatric Surgery: Does Cognitive Function Play a Role?. *Obesity Surgery*, *23*(10), 1703-1710.

Geliebter, A. (2003). Emotional Eating In Overweight, Normal Weight, And Underweight Individuals. *Eating Behaviors*, *3*(4), 341-347.

Gennaro, L. D., Martina, M., Curcio, G., & Ferrara, M. (2004). The relationship between alexithymia, depression, and sleep complaints. *Psychiatry Research*, *128*(3), 253-258.

Gibson, E. L., & Green, M. W. (2002). Nutritional influences on cognitive function: mechanisms of susceptibility. *Nutrition Research Reviews*, *15*(01), 169.

Gluck, M. E., Geliebter, A., Satov, T. Night eating syndrome is associated with depression, low self-esteem, reduced daytime hunger, and less weight loss in obese outpatients. *Obes Res*, 9 :264– 267,2001.

Goldney, R. D., Phillips, P. J., Fisher, L. J., Wilson, D. H. Diabetes, depression, and quality of life: a population study. *Diabetes Care* 2004;271066- 1070.

Gomez-Pinilla, F., Vaynman, S., & Ying, Z. (2008). Brain-derived neurotrophic factor functions as a metabotrophin to mediate the effects of exercise on cognition. *European Journal of Neuroscience*, *28*(11), 2278-2287.

Grant, J. E., Odlaug, B. L., Chamberlain, S. R., & Schreiber, L. R. N. (2012). Neurocognitive dysfunction in strategic and non-strategic gamblers. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, *38(2),* 336-340.

Green, M. W., Rogers, P. J., Elliman, N. A., & Gatenby, S. J. (1994). Impairment of cognitive performance associated with dieting and high levels of dietary restraint. *Physiology & Behavior*, *55*(3), 447-452.

Griffith, P. S., Birch, D., Sharma, A., & Karmali, S. (2012). Managing complications associated with laparoscopic Roux-en-Y gastric bypass for morbid obesity. *Canadian Journal of Surgery*, *55*(5), 329-336.

Guerrieri, R., Nederkoorn, C., Schrooten, M., Martijn, C., & Jansen, A. (2009). Inducing impulsivity leads high and low restrained eaters into overeating, whereas current dieters stick to their diet. *Appetite*, *53*(1), 93-100.

Gunstad, J., Paul, R. H., Cohen, R. A., Tate, D. F., & Gordon, E. (2006). Obesity is associated with memory deficits in young and middle-aged adults. *Eating and Weight Disorders*

- *Studies on Anorexia, Bulimia and Obesity*, *11*(1), e15-e19.

Gunstad, J., Paul, R., Cohen, R., Tate, D., Spitznagel, M., & Gordon, E. (2007). Elevated body mass index is associated with executive dysfunction in otherwise healthy adults. *Comprehensive Psychiatry*, *48*(1), 57-61.

Hannon, T. S., Rofey, D. L., Ryan, C. M., Clapper, D. A., Chakravorty, S., & Arslanian, S. A. (2012). Relationships among Obstructive Sleep Apnea, Anthropometric Measures, and Neurocognitive Functioning in Adolescents with Severe Obesity. *The Journal of Pediatrics*, *160*(5), 732-735.

Harris, B., Young, J., & Hughes, B. (1984). Changes occurring in appetite and weight during short-term antidepressant treatment. *The British Journal of Psychiatry*, *145*(6), 645-648.

Hartman, D. (2009). Wechsler Adult Intelligence Scale IV (WAIS IV): Return Of The Gold Standard. *Applied Neuropsychology*, *16*(1), 85-87.

Haviland, M. G., Warren, W. L., & Riggs, M. L. (2000). An Observer Scale to Measure Alexithymia. *Psychosomatics*, *41*(5), 385-392.

Haworth, C. M., Carnell, S., Meaburn, E. L., Davis, O. S., Plomin, R., & Wardle, J. (2008). Increasing heritability of bmi and stronger associations with the fto gene over childhood. *Obesity*, *16*(12), 2663-2668.

Hendrickx, H., Mcewen, B. S., & Ouderaa, F. V. (2005). Metabolism, mood and cognition in aging: The importance of lifestyle and dietary intervention. *Neurobiology of Aging*, *26*(1), 1-5.

Higgs, S. (2002). Memory for recent eating and its influence on subsequent food intake. *Appetite*, *39*(2), 159-166.

Higgs, S. (2005). Memory and its role in appetite regulation. *Physiology & Behavior*, *85*(1),

67-72.

Higgs, S. (2008). Cognitive influences on food intake: The effects of manipulating memory for recent eating. *Physiology & Behavior*, *94*(5), 734-739.

Higgs, S., & Donohoe, J. E. (2011). Focusing on food during lunch enhances lunch memory and decreases later snack intake. *Appetite*, *57*(1), 202-206.

Higgs, S., Williamson, A., & Attwood, A. (2008). Recall of recent lunch and its effect on subsequent snack intake. *Physiology & Behavior*, *94*(3), 454-462.

Higgs, S., & Woodward, M. (2009). Television watching during lunch increases afternoon snack intake of young women. *Appetite*, *52*(1), 39-43.

Hollitt, S., Kemps, E., Tiggemann, M., Smeets, E., & Mills, J. S. (2010). Components of attentional bias for food cues among restrained eaters. *Appetite*, *54*(2), 309-313.

Holub, S. C., Haney, A. M., & Roelse, H. (2012). Deconstructing the concept of the healthy eater self-schematic: Relations to dietary intake, weight and eating cognitions. *Eating Behaviors*, *13*(2), 106-111.

Houben, J. M., Moonen, H. J., Schooten, F. J., & Hageman, G. J. (2008). Telomere length assessment: Biomarker of chronic oxidative stress?. *Free Radical Biology and Medicine*, *44*(3), 235-246.

Hurst, N., Kind, P., Ruta, D., Hunter, M., & Stubbings, A. (1997). Measuring health-related quality of life in rheumatoid arthritis: validity, responsiveness and reliability of EuroQol (EQ-5D). *Rheumatology*, *36*(5), 551-559.

Hwang, L., Yang, W., Chiou, L., Ho, I., Liang, K., Chen, C., et al. (2009). Sex Differences in High-fat Diet-induced Obesity, Metabolic Alterations and Learning, and Synaptic Plasticity Deficits in Mice. *Obesity*, *18*(3), 463-469.

Inelmen, E. M., Toffanello, E. D., Enzi, G., Gasparini, G., Miotto, F., Sergi, G. et al.

Predictors of drop-out in overweight and obese outpatients. *Int J Obes Relat Metab Disord* 2005; 29: 122–128.

Isreal, A. C., Stolmaker, L., & Adrian, C. A. G. (2006). Thoughts about food and their: Relationship to obesity and weight control. *International Journal of Eating Disorders*, *4(4)*, 549–558.

Jansen, A., Louwerse, E., Leemans, N., & Schouten, E. (1998). Self-esteem as a better predictor of restrained eaters' food intake than attributional style and disinhibition tendency. *European Journal of Personality*, *12*(1), 43-56.

Jemal, A., Bray, F., Center, M. M., Ferlay, J., Ward, E. and Forman, D. (2011), Global cancer statistics. *CA: A Cancer Journal for Clinicians*, 61: 69–90.

Johns, M. W. A new method for measuring daytime sleepiness: the Epworth Sleepiness Scale. *Sleep* 1991; 50-55.

Johnson, F., Pratt, M., & Wardle, J. (2011). Dietary restraint and self-regulation in eating behavior. *International Journal of Obesity*, *36*(5), 665-674.

Jones, N., & Rogers, P. J. (2003). Preoccupation, food, and failure: An investigation of cognitive performance deficits in dieters. *International Journal of Eating Disorders*, *33*(2), 185-192.

Jorm, A. F., Korten, A. E., Christensen, H., Jacomb, P. A., Rodgers, B., & Parslow, R. A. (2003). Association of obesity with anxiety,depression, and emotional well-being: A community survey. *Australia and New Zealand Journal of Public Health*, 27, 434–440.

Joukamaa, M., Taanila, A., Miettunen, J., Karvonen, J. T., Koskinen, M., & Veijola, J. (2007). Epidemiology of alexithymia among adolescents. *Journal of Psychosomatic Research*, *63*(4), 373-376.

Kanaya, A. M., Vittinghoff, E., Shlipak, M., Resnick, H., Visser, M., Grady, D., et al. (2003). Association of Total and Central Obesity with Mortality in Postmenopausal Women with Coronary Heart Disease. *American Journal of Epidemiology*, *158*(12), 1161-1170.

Kanazawa, S. (2014). Intelligence and obesity: which way does the causal direction go?. *Current opinion in endocrinology, diabetes, and obesity*, *Published ahead of print*, unknown.

Kanoski, S., Hayes, M., Greenwald, H., Gianess, S., Fortin, C., Gilbert, J., et al. (2011). Hippocampal Leptin Signaling Reduces Food Intake and Modulates Food-Related Memory Processing. *Neuropsychopharmacology*, *36*, 1859â€“1870.

Kanoski, S., Meisel, R., Mullins, A., & Davidson, T. (2007). The effects of energy-rich diets on discrimination reversal learning and on BDNF in the hippocampus and prefrontal cortex of the rat. *Behavioural Brain Research*, *182*(1), 57-66.

Karlsson, J., Taft, C., Ryden, A., Sjostrom, L., Sullivan, M. Ten-year trends in health-related quality of life after surgical and conventional treatment for severe obesity: the SOS intervention study. *Int J Obes* 2007;31:1248–61.

Karukivi, M., Hautala, L., Kaleva, O., Haapasalo-Pesu, K., Liuksila, P., Joukamaa, M., et al. (2010). Alexithymia is associated with anxiety among adolescents. *Journal of Affective Disorders*, *125*(1-3), 383-387.

Karukivi, M., Hautala, L., Korpelainen, J., Haapasalo-Pesu, K., Liuksila, P., Joukamaa, M., et al. (2010). Alexithymia and Eating Disorder Symptoms in Adolescents. *Eating Disorders*, *18*(3), 226-238.

Katschnig, H., Jaidhauser, K., Subasi, B., Zoghlami, A., & Serim, M. (1997). Quality of life in depression. *European Psychiatry*, *12*(1002), 116s-116s.

Kaufmann, L., Pixner, S., Starke, M., Zotter, S., KÃ¶hle, J., Meraner, D., et al. (2012). Neurocognition and brain structure in pediatric patients with type 1 Diabetes. *Journal of Pediatric Neuroradiology*, *1*(1), 25-35.

Kemps, E., & Tiggemann, M. (2005). Working memory performance and preoccupying thoughts in female dieters: Evidence for a selective central executive impairment. *British Journal of Clinical Psychology*, *44*(3), 357-366.

Kemps, E., Tiggemann, M., Wade, T., Ben-Tovim, D., & Breyer, R. (2006). Selective Working Memory Deficits In Anorexia Nervosa. *European Eating Disorders Review*, *14*(2), 97-103.

Khan, M. A., St Peter, J. V., Breen, G. A., Hartley, G. G., Vessey, J. T. Diabetes disease stage predicts weight loss outcomes with long-term appetite suppressants. *Obes Res*. 2000;8(1):43-48.

Killgore, W. (2010). Effects of sleep deprivation on cognition.. *Progress in Brain Research*, *185*, 105-129.

Killgore, W. D., Kahn-Greene, E. T., Lipizzi, E. L., Newman, R. A., Kamimori, G. H., & Balkin, T. J. (2008). Sleep Deprivation Reduces Perceived Emotional Intelligence And Constructive Thinking Skills. *Sleep Medicine*, *9*(5), 517-526.

Kolotkin, R., & Crobsy, R. (2002). Psychometric evaluation of the Impact Of Weight On Quality Of Life-Lite Questionnaire (IWQOL-Lite) in a community sample. *Quality of Life Research*, *11*(2), 157-171.

Kolotkin, R. L., Crosby, R .D., Kosloski, K. D., Williams, G. R. Development of a brief measure to assess quality of life in obesity. *Obesity Research*. 2001; 9: 102-111

Kolotkin, R. L., Crosby, R. D., Williams, G. R. Health-related quality of life varies among obese subgroups. *Obes Res.*2002;10:748-756.

Kolotkin, R. L., Meter, K. and Williams, G. R. (2001), Quality of life and obesity. *Obesity Reviews*, 2: 219–229.

Koster, M., Prescott, J., & Koster, E. (2004). Incidental Learning and Memory for Three Basic Tastes in Food. *Chemical Senses*, *29*(5), 441-453.

Lang, I., Llewellyn, D., Alexander, K., Melzer, D. Obesity, physical function, and mortality in older adults. *J Am Geriatr Soc*. 2008;56:1474–1478.

Laquatra, T. A., & Clopton, J. R. (1994). Characteristics of alexithymia and eating disorders in college women. *Addictive Behaviors*, *19*(4), 373-380.

Larsen, J., Vanstrien, T., Eisinga, R., & Engels, R. (2006). Gender Differences In The Association Between Alexithymia And Emotional Eating In Obese Individuals. *Journal of Psychosomatic Research*, *60*(3), 237-243.

Larson, D., Neumark-Sztainer, M., Hannan, C., & Story, M. (2007). Trends in Adolescent Fruit and Vegetable Consumption, 1999â€“2004: Project EAT. *American Journal Preventive Medicine*, *32*(2), 198â€“208.

Lasikiewicz, N., Hendrickx, H., Talbot, D., & Dye, L. (2013). Exploring stress-induced cognitive impairment in middle aged, centrally obese adults. *Stress*, *16*(1), 44-53.

Lean, M. E. J., Hans, T. S. & Seidell, J. C. Impairment of health and quality life in people with large waist circumference. *Lancet* **351,** 853–856 (1998).

Lehnert, T., Sonntag, D., Konnopka, A., Riedel-Heller, S. and König, H.-H. (2012), The long-term cost-effectiveness of obesity prevention interventions: systematic literature review. *Obesity Reviews*, 13: 537–553.

Lemogne, C., Piolino, P., Friszer, S., Claret, A., Girault, N., Jouvent, R., et al. (2006). Episodic autobiographical memory in depression: Specificity, autonoetic consciousness, and self-perspective. *Consciousness and Cognition*, *15*(2), 258-268.

Lenclave, M. D., Florequin, C., & Bailly, D. (2001). Obesity, alexithymia, psychopathological disorders, and binge eating: a comparative study between 40 obese subjects and 32 controls. *Encephale-Revue De Psychiatrie Clinique Biologique Et Therapeutique* , *27*(4), 343-350.

Lewis, S., & Puymbroeck, M. V. (2008). Obesity-Stigma as a Multifaceted Constraint to Leisure. *Journal of Leisure Research*, *40*(4), 574-588.

Li, E., Kim, Y., Kim, S., & Park, S. (2013). Ghrelin-induced hippocampal neurogenesis and enhancement of cognitive function are mediated independently of GH/IGF-1 axis: lessons from the spontaneous dwarf rats. *Endocrine Journal*, *60*(9), 1065-1075.

Li, S., Zhao, J. H., Luan, J., Ekelund, U., Luben, R. N., Khaw, K., et al. (2010). Physical Activity Attenuates the Genetic Predisposition to Obesity in 20,000 Men and Women from EPIC-Norfolk Prospective Population Study. *PLoS Medicine*, *7*(8), e1000332.

Li, S., Zhao, J. H., Luan, J., Langenberg, C., Luben, R. N., Khaw, K. T., et al. (2011). Genetic predisposition to obesity leads to increased risk of type 2 diabetes. *Diabetologia*, *54*(4), 776-782.

Lin, S., Thomas, T., Storlien, L., & Huang, X. (2000). Development of high fat diet-induced obesity and leptin resistance in C57BI/6J mic. *International Journal of Obesity*, 24(5), 639-646.

Lindenberger, U., & Potter, U. (1998). The complex nature of unique and shared effects in hierarchical linear regression: Implications for developmental psychology. *Psychological Methods*, *3*(2), 218-230.

Linke, J., King, A. V., Poupon, C., Hennerici, M. G., Gass, A., & Wessa, M. (2013). Impaired Anatomical Connectivity and Related Executive Functions: Differentiating Vulnerability and Disease Marker in Bipolar Disorder. *Biological Psychiatry, 74(12),*

870-871.

Loos, R. J., & Bouchard, C. (2003). Obesity - is it a genetic disorder?. *Journal of Internal Medicine*, *254*(5), 401-425.

Lucassen, E. A., Cizza, G., Reddy, H., Csako, G., Gershengorn, J., Ramer, A., et al. (2014). Sleep Extension Improves Neurocognitive Functions in Chronically Sleep-Deprived Obese Individuals. *PLoS ONE*, *9*(1), e84832.

Ludwig, D. S., Majzoub, J. A., Al-Zahrani, A., Dallal, G. E., Blanco, I., & Roberts, S. B. (1999). High Glycemic Index Foods, Overeating, And Obesity. *Pediatrics*, *103*(3), e26-e26.

Lumley, M. A., Ovies, T., Stettner, L., Wehmer, F., & Lakey, B. (1996). Alexithymia, social support and health problems. *Journal of Psychosomatic Research*, *41*(6), 519-530.

Luppino, F. S., de Wit, L. M., Bouvy, P. F., et al. Overweight, Obesity, and Depression: A Systematic Review and Meta-analysis of Longitudinal Studies. *Arch Gen Psychiatry*. 2010;67(3):220-229.

Luppino, F. S., de Wit, L. M., Bouvy, P. F., Stijnen, T., Cuijpers, P., Penninx, B. W., et al. (2010). Overweight, Obesity, And Depression: A Systematic Review And Meta-analysis Of Longitudinal Studies. *Archives of General Psychiatry*, *67*(3), 220-229.

Mainous, A. G., & Diaz, V. A. (2010). Telomere length as a risk marker for cardiovascular disease: the next big thing?. *Expert Review of Molecular Diagnostics*, *10*(8), 969-971.

Martin, S., Qasim, A., & Reilly, M. (2008). Leptin resistance - A possible interface of inflammation and metabolism in obesity-related cardiovascular disease. *Journal of the American College of Cardiology*, *52*(15), 1201-1210.

McCarthy, M. I., Wareham, N. J., Owen, K. R., Hattersley, A. T., Smith, G. D., Morris, A. D., et al. (2007). A Common Variant in the FTO Gene Is Associated with Body Mass

Index and Predisposes to Childhood and Adult Obesity. *Science*, *316*(5826), 889-894.

McDonald, R. J., Zelinski, E. L., Keeley, R. J., Sutherland, D., Fehr, L., & Hong, N. S. (2013). Multiple effects of circadian dysfunction induced by photoperiod shifts: Alterations in context memory and food metabolism in the same subjects. *Physiology & Behavior*, *118*, 14-24.

McIntyre, R. S., Cha D. S., Soczynska, J. K., Woldeyohannes, H. O., Gallaugher, L. A., Kudlow, P., Alsuwaidan, M., & Baskaran, A. (2013). Cognitive deficits and functional outcomes in major depressive disorder: determinants, substrates, and treatment interventions. 515-527.

Mcternan, C., Mcternan, P., Harte, A., Levick, P., Barnett, A., & Kumar, S. (2002). Resistin, central obesity, and type 2 diabetes. *The Lancet*, *359*(9300), 46-47.

Meisel, S., & Wardle, J. (2014). 'Battling my Biology': Psychological Effects of Genetic Testing for Risk of Weight Gain. *Journal of Genetic Counseling*, *23*(2), 179-186.

Mitchell, G. L., & Brunstrom, J. M. (2005). Everyday dietary behaviour and the relationship between attention and meal size. *Appetite*, *45*(3), 344-355.

Mitchell, M. T., Pizzitola, V. J., Knuttinen, M., Robinson, T., & Gasparaitis, A. E. (2005). Atypical complications of gastric bypass surgery. *European Journal of Radiology*, *53*(3), 366-373.

Mittal, D., Stevenson, R. J., Oaten, M. J., & Miller, L. A. (2011). Snacking while watching TV impairs food recall and promotes food intake on a later TV free test meal. *Applied Cognitive Psychology*, *25*(6), 871-877.

Mobbs, O., Crepin, C., Thiery, C., Golay, A., & Linden, M. V. (2010). Obesity and the four facets of impulsivity. *Patient Education and Counseling*, *79*(3), 372-377.

Mobley, L., Root, E., Finkelstein, E., Khavjou, O., Farris, R., & Will, J. (2006). Environment,

Obesity, And Cardiovascular Disease Risk In Low-Income Women. *American Journal of Preventive Medicine, 30*(4), 327-327.

Mogg, K., Bradley, B. P. (2005). Attentional Bias in Generalized Anxiety Disorder Versus Depressive Disorder. *Cognitive Therapy and Research, 29(1),* 29-45.

Morosin, A., & Riva, G. (1997). Alexithymia In A Clinical Sample Of Obese Women. *Psychological Reports, 80*(2), 387-394.

Morrison, C. D., Bruce-Keller, A. J., Keller, J. N., Uranga, R. M., Purpera, M. N., White, C. L., et al. (2010). High fat diet increases hippocampal oxidative stress and cognitive impairment in aged mice: implications for decreased Nrf2 signaling. *Journal of Neurochemistry*, *114*(6), 1581-1589.

Msika, S. (2003). Surgery for morbid obesity: 2. Complications. Results of a technologic evaluation by the ANAES. *Journal de chirurgie, 140*(1), 4-21.

Nederkoorn, C., Jansen, E., Mulkens, S., & Jansen, A. (2007). Impulsivity predicts treatment outcome in obese children. *Behaviour Research and Therapy, 45*(5), 1071-1075.

Netzer, N., Stoohs, R., Netzer, C., Clark, K., & Strohl, K. (1999). Using the Berlin Questionnaire to identify patients at risk for the sleep apnea syndrome. *Annals of Internal Medicine, 131*(7), 485-491.

Noli, G., Cornicelli, M., Marinari, G. M., Carlini, F., Scopinaro, N., & Adami, G. F. (2010). Alexithymia and eating behaviour in severely obese patients. *Journal of Human Nutrition and Dietetics*, *23*(6), 616-619.

Nowakowski, M. E., Mcfarlane, T., & Cassin, S. (2013). Alexithymia and eating disorders: a critical review of the literature. *journal of eating disorders*, *1*(1), 21.

Olde Dubbelink, K. T., Hillebrand, A., Twisk, J. W. R., Deijen, J. B., Stoffers, D., Schmand, B. A., Stam, C. J., & Berendse, H. W. (2014). Predicting dementia in Parkinson

disease by combining neurophysiologic and cognitive markers. 263-270.

Oldham-Cooper, R. E., Hardman, C. A., Nicoll, C. E., Rogers, P. J., & Brunstrom, J. M. (2011). Playing a computer game during lunch affects fullness, memory for lunch, and later snack intake. *American Journal of Clinical Nutrition*, *93*(2), 308-313.

Orth, U., Robins, R. W., & Roberts, B. W. (2008). Low Self-esteem Prospectively Predicts Depression In Adolescence And Young Adulthood.. *Journal of Personality and Social Psychology*, *95*(3), 695-708.

Ouwens, M. A., Strien, T. V., & Leeuwe, J. F. (2009). Possible pathways between depression, emotional and external eating. A structural equation model. *Appetite*, *53*(2), 245-248.

Ouwens, M. A., Strien, T. V., & Staak, C. P. (2003). Tendency toward overeating and restraint as predictors of food consumption. *Appetite*, *40*(3), 291-298.

Pagoto, S., Bodenlos, J. S., Kantor, L., Gitkind, M., Curtin, C., & Ma, Y. (2007). Association of major depression and binge eating disorder with weight loss in a clinical setting. *Obesity*, *15*(11), 2557-2559

Patel, S. R., & Hu, F. B. (2008). Short Sleep Duration And Weight Gain: A Systematic Review. *Obesity*, *16*(3), 643-653.

Patterson, M., Bloom, S. R., & Gardiner, J. V. (2011). Ghrelin and appetite control in humansâ€"Potential application in the treatment of obesity. *Peptides*, *32*(11), 2290-2294.

Pelone, F., Specchia, M. L., Veneziano, M. A., Capizzi, S., Bucci, S., Mancuso, A., Ricciardi, W. and de Belvis, A. G. (2012), Economic impact of childhood obesity on health systems: a systematic review. *Obesity Reviews*, 13: 431–440.

Pereira, M., Kartashov, A., Ebbeling, C., Vanhorn, L., Slattery, M., Jacobsjr, D., et al. (2005). Fast-food Habits, Weight Gain, And Insulin Resistance (the CARDIA Study): 15-year

Prospective Analysis. *The Lancet, 365*(9453), 36-42.

Pergola, G. D., & Silvestris, F. (2013). Obesity as a Major Risk Factor for Cancer. *Journal of Obesity, 2013*. Retrieved November 18, 2013, from http://dx.doi.org/10.1155/2013/291546

Pinaquy, S., Chabrol, H., Simon, C., Louvet, J., & Barbe, P. (2003). Emotional Eating, Alexithymia, and Binge-Eating Disorder in Obese Women. *Obesity, 11*(2), 195-201.

Pinna, F., Lai, L., Orru, S., Velluzzi, W., Loviselli, F., Carpiniello, A., et al. (2011). Obesity, alexithymia and psychopathology: A case-control study. *Eating and Weight Disorders - Studies on Anorexia, Bulimia and Obesity, 16*(3), e164-e170.

Putte, E. M., Engelbert, R. H., Kuis, W., Kimpen, J. L., & Uiterwaal, C. S. (2007). Alexithymia in adolescents with chronic fatigue syndrome. *Journal of Psychosomatic Research, 63*(4), 377-380.

Rabkin, S. W. (2007), Epicardial fat: properties, function and relationship to obesity. *Obesity Reviews*, 8: 253–261.

Ravussin, E., & Bogardus, C. (2000). Energy balance and weight regulation: genetics versus environment. *British Journal of Nutrition, 83*(S1), S17-S20.

Rhodes, S. K., Shimoda, K. C., Waid, L., O'neil, P. M., Oexmann, M. J., Collop, N. A., et al. (1995). Neurocognitive deficits in morbidly obese children with obstructive sleep apnea. *The Journal of Pediatrics, 127*(5), 741-744.

Rieffe, C., Oosterveld, P., & Terwogt, M. M. (2006). An alexithymia questionnaire for children: Factorial and concurrent validation results. *Personality and Individual Differences, 40*(1), 123-133.

Robinson, E., Aveyard, P., Daley, A., Jolly, K., Lewis, A., Lycett, D., et al. (2013). Eating attentively: a systematic review and meta-analysis of the effect of food intake

memory and awareness on eating. *American Journal of Clinical Nutrition, 97*(4), 728-742.

Rode, L., Nordestgaard, B., Weischer, M., & Bojesen, S. (2014). Increased Body Mass Index, Elevated C-reactive Protein, and Short Telomere Length. *The Journal of Clinical Endocrinology and Metabolism*, *99*(9), E1671-5.

Rolland, Y., Lauwers-Cances, V., Cristini, C., et al. Difficulties with physical function associated with obesity, sarcopenia, and sarcopenic-obesity in community-dwelling elderly women: the EPIDOS (EPIDemiologie de l'OSteoporose) Study. *Am J Clin Nutr* 2009;89:1895–900.

Rude, S. S., Wenzlaff, R. M., Gibbs, B., Vane, J., & Whitney, T. (2002). Negative processing biases predict subsequent depressive symptoms. *Cognition & Emotion, 16(3),* 423-440.

Ryan, C. M. (2006). Improving Metabolic Control Leads to Better Working Memory in Adults With Type 2 Diabetes. *Diabetes Care*, *29*(2), 345-351.Sabia, S., Kivimaki, M., Shipley, M. J., Marmot, M. G., & Singh-Manoux, A. (2009). Body mass index over the adult life course and cognition in late midlife: the Whitehall II Cohort Study. *American Journal of Clinical Nutrition*, *89*(2), 601-607.

Sach, T. H., Barton, G. R., Doherty, M., Muir, K. R., Jenkinson, C., Avery, A. J. The relationship between body mass index and health-related quality of life: comparing the EQ-5D, EuroQol VAS and SF-6D. *Int J Obes (Lond)*. 2007; 31: 189–196.

Saczynski, J. S., Gudnason, V., Harris, T. B., Olafsdottir, E., Eiriksdottir, G., Peila, R., et al. (2008). Cognitive Impairment: An Increasingly Important Complication of Type 2 Diabetes: The Age, Gene/Environment Susceptibility-Reykjavik Study. *American Journal of Epidemiology*, *168*(10), 1132-1139.

Sakkinen, P., Kaltiala-Heino, R., Ranta, K., Haataja, R., & Joukamaa, M. (2007). Psychometric Properties of the 20-Item Toronto Alexithymia Scale and Prevalence of Alexithymia in a Finnish Adolescent Population. *Psychosomatics, 48*(2), 154-161.

Sarisoy, G., Atmaca, A., Ecemis, G., Gumus, K., & Pazvantoglu, O. (2013). Impulsivity in patients with obesity and correlations with body perception and self-esteem. *Anatolian Journal of Psychiatry , 14*(1), 53-61.

Sasai, K., Tanaka, K., & Hishimoto, A. (2010). Alexithymia and its relationships with eating behavior, self esteem, and body esteem in college women. *Kobe Journal of Medical Sciences, 56*(6), E231-E238.

Scaife, J. C., & Duka, T. (2009). Behavioural measures of frontal lobe function in a population of young social drinkers with binge drinking pattern. *Pharmacology, Biochemistry, and Behaviour, 93(3),* 354-362.

Scarpace, P. J., & Zhang, Y. (2008). Leptin resistance: a prediposing factor for diet-induced obesity. *AJP: Regulatory, Integrative and Comparative Physiology*, *296*(3), R493-R500.

Schag, K., SchÃ¶nleber, J., Teufel, M., Zipfel, S., & Giel, K. E. (2013). Food-related impulsivity in obesity and Binge Eating Disorder - a systematic review. *Obesity Reviews*, *14*(6), 477-495.

Schmidt, U., Jiwany, A., & Treasure, J. (1993). A controlled study of alexithymia in eating disorders. *Comprehensive Psychiatry*, *34*(1), 54-58.

Schousboe, K., Luciano, M., Kaprio, J., Willemsen, G., Kyvik, K. O., Mortensen, J., et al. (2003). Sex Differences in Heritability of BMI: A Comparative Study of Results from Twin Studies in Eight Countries. *Twin Research*, *6*(05), 409-421.

Scuteri, A., Mulas, A., Lakatta, E., Busonero, F., Maschio, A., Lai, S., et al. (2007). Genome-

Wide Association Scan Shows Genetic Variants in the FTO Gene Are Associated with Obesity-Related Traits. *PLoS Genetics*, *3*(7), e115.

Shallice, T. (1982). Specific Impairments of Planning. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *298*(1089), 199-209.

Shefer, G., Marcus, Y., & Stern, N. (2013). Is obesity a brain disease?. *Neuroscience & Biobehavioral Reviews*, *37*(10), 2489-2503.

Silventoinen, K., Rokholm, B., Kaprio, J., & SÃ¸rensen, T. I. (2009). The Genetic And Environmental Influences On Childhood Obesity: A Systematic Review Of Twin And Adoption Studies. *International Journal of Obesity*, *34*, 29â€“40.

Simon, N. M., Smoller, J. W., McNamara, K. L., Maser, R. S., McNamara, A. K., Pollack, M. H., et al. (2006). Telomere Shortening And Mood Disorders: Preliminary Support For A Chronic Stress Model Of Accelerated Aging. *Biological Psychiatry*, *60*(5), 432-435.

Soetens, B., & Braet, C. (2007). Information processing of food cues in overweight and normal weight adolescents. *British Journal of Health Psychology*, *12*(2), 285-304.

Spitznagel, M. B., Alosco, M., Strain, G., Devlin, M., Cohen, R., Paul, R., et al. (2013). Cognitive function predicts 24-month weight loss success after bariatric surgery. *Surgery for Obesity and Related Diseases*, *9*(5), 765-770.

Stanek, K. M., & Gunstad, J. (2013). Can bariatric surgery reduce risk of Alzheimer's disease?. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, *47*, 135-139.

Stanek, K. M., Strain, G., Devlin, M., Cohen, R., Paul, R., Crosby, R. D., et al. (2013). Body mass index and neurocognitive functioning across the adult lifespan.. *Neuropsychology*, *27*(2), 141-151.

Statistics on Obesity, Physical Activity, and Diet: England 2014. *Health and Social Care Information Centre* , *PUB13648*. Retrieved August 19, 2014, from http://www.hscic.gov.uk/catalogue/PUB13648/Obes-phys-acti-diet-eng-2014-rep.pdf

Steffens, D.C., Otey, E., Alexopoulos, G.S., Butters, M.A., Cuthbert, B.,Ganguli, M., Geda, Y.E., Hendrie, H.C., Krishnan, R.R., Kumar, A.,Lopez, O.L., Lyketsos, C.G., Mast, B.T., Morris, J.C., Norton, M.C.,Peavy, G.M., Petersen, R.C., Reynolds, C.F., Salloway, S., Welsh-Bohmer, K.A., Yesavage, J., 2006. Perspectives on depression, mildcognitive impairment, and cognitive decline. *Arch. Gen. Psychiatry* 63, 130–138.

Stice, E., Spoor, S., Bohon, C., & Small, D. M. (2008). Relation Between Obesity And Blunted Striatal Response To Food Is Moderated By TaqIA A1 Allele. *Science*, *322*(5900), 449-452.

Stordal, E., Mykletun, A., & Dahl, A. A. (2003). The association between age and depression in the general population: a multivariate examination. *Acta Psychiatrica Scandinavica*, *107*(2), 132-141.

Strien, T. V., Frijters, J. E., Bergers, G. P., & Defares, P. B. (1986). The Dutch Eating Behavior Questionnaire (DEBQ) for assessment of restrained, emotional, and external eating behavior. *International Journal of Eating Disorders*, *5*(2), 295-315.

Strine, T. V., Mokdad, A., Dube, S., Balluz, L., Gonzalez, O., Berry, J. T., et al. (2008). The association of depression and anxiety with obesity and unhealthy behaviors among community-dwelling US adult. *General Hospital Psychiatry*, *30*(2), 127-137.

Strien, T. V., & Ouwens, M. A. (2007). Effects of distress, alexithymia and impulsivity on eating. *Eating Behaviors*, *8*(2), 251-257.

Stroebe, W., Koningsbruggen, G. M., Papies, E. K., & Aarts, H. (2013). Why most

dieters fail but some succeed: A goal conflict model of eating behavior.. *Psychological Review*, *120*(1), 110-138.

Stunkard, A. J., Foch, T., & Hrubec, Z. (1986). A twin study of human obesity. *JAMA: The Journal of the American Medical Association*, *256*(1), 51-54.

Surcinelli, P., Baldaro, B., Balsamo, A., Bolzani, R., Gennari, M., & Rossi, N. C. (2007). Emotion Recognition And Expression In Young Obese Participants: Preliminary Study. *Perceptual And Motor Skills*, *105*(2), 477-482.

Surman, C., Hammerness, P., Petty, C., Spencer, T. J., Doyle, R., Napolean, S., Chu, N., Yorks, D., & Biederman J. (2013). A pilot open label prospective study of memantine monotherapy in adults with ADHD. *The World Journal of Biological Psychiatry*, 291-298.

Swinburn, B. (1999). Dissecting Obesogenic Environments: The Development And Application Of A Framework For Identifying And Prioritizing Environmental Interventions For Obesity. *Preventive Medicine*, *29*(6), 563-570.

Swinburn, B. (2009). Obesity Prevention In Children And Adolescents. *Child and Adolescent Psychiatric Clinics of North America*, *18*(1), 209-223.

Swinburn, B., Egger, G., (2004). The runaway weight gain train: too many accelerators, not enough brakes. *British Medical Journal (International Edition)* Vol 329, Issue 7468, p 736.

Swinburn, B. A., Sacks, G., Hall, K. D., McPherson, K., Finegood, D. T., Moodie, M. L., et al. (2011). The Global Obesity Pandemic: Shaped By Global Drivers And Local Environments. *The Lancet*, *378*(9793), 804-814.

Takeda, S., Sato, N., Uchio-Yamada, K., Sawada, K., Kunieda, T., Takeuchi, D., et al. (2010). Diabetes-accelerated memory dysfunction via cerebrovascular inflammation and AÎ²

deposition in an Alzheimer mouse model with diabetes. *Proceedings of the National Academy of Sciences of the United States of America*, *107*(15), 7036-7041.

Tamura, Y., Izumiyama-Shimomura, N., Araki, A., Takubo, K., Ito, H., Kimbara, Y., et al. (2014). beta-Cell Telomere Attrition in Diabetes: Inverse Correlation Between HbA1c and Telomere Length. *The Journal of Clinical Endocrinology and Metabolism*, *99*(8), 2771-7.

Tapper, K., & Pothos, E. M. (2010). Development and validation of a Food Preoccupation Questionnaire. *Eating Behaviors*, *11*(1), 45-53.

Tattar, P. N. (2013). R Statistical Application Development by Example Beginner's Guide.

Taylor, G. J., Parker, J. D., Bagby, R., & Bourke, M. P. (1996). Relationships between alexithymia and psychological characteristics associated with eating disorders. *Journal of Psychosomatic Research*, *41*(6), 561-568.

Ted, H., & Scott, K. (2014). Blood-brain barrier disruption: mechanistic links between Western diet consumption and dementia. *Frontiers in Aging Neuroscience*, *6*(88), unknown.

Teixeira, P. J., Going, S. B., Sardinha, L. B. and Lohman, T. G. (2005), A review of psychosocial pre-treatment predictors of weight control. *Obesity Reviews*, 6: 43– 65.

Thamotharan, S., Lange, K., Zale, E. L., Huffhines, L., & Fields, S. (2013). The role of impulsivity in pediatric obesity and weight status: A meta-analytic review. *Clinical Psychology Review*, *33*(2), 253-262.

The EuroQol Group (1990). EuroQol-a new facility for the measurement of health-related quality of life. *Health Policy* 16(3):199-208.

Tiggemann, M., Kemps, E., & Parnell, J. (2010). The selective impact of chocolate craving on visuospatial working memory. *Appetite*, *55*(1), 44-48.

Tozuka, Y., Kumon, M., Wada, E., Onodera, M., Mochizuki, H., & Wada, K. (2010). Maternal obesity impairs hippocampal BDNF production and spatial learning performance in young mouse offspring. *Neurochemistry international*, *57*(3), 235-247.

Tozzi, E., Andrisani, G., Maioran, D., Pezzi, L., Leopardi, R., Fiorentini, N., et al. (2013). Alexithymia in headache sufferers: A psychosomatic. *Educational Research*, *4*(3), 286-293.

Tucsek, Z., Toth, P., Sosnowska, D., Gautam, T., Koller, A., Sonntag, W., et al. (2014). Obesity in aging exacerbates blood brain barrier disruption, neuroinflammation and oxidative stress in the mouse hippocampus: effects on expression of genes involved in beta-amyloid generation and Alzheimerâ€™s disease. *The Federation of American Societies for Experimental Biology*, *28*(1), 665.

Tyrka, A. R., Price, L. H., Kao, H., Porton, B., Marsella, S. A., & Carpenter, L. L. (2010). Childhood Maltreatment And Telomere Shortening: Preliminary Support For An Effect Of Early Stress On Cellular Aging. *Biological Psychiatry*, *67*(6), 531-534.

Tzanetakou, I., Katsilambros, N., Benetos, A., Mikhailidis, D., & Perrea, D. (2012). "Is obesity linked to aging?": adipose tissue and the role of telomeres. *Ageing Research Reviews*, *11*(2), 220-229.

Valdes, A., Andrew, T., Gardner, J., Kimura, M., Oelsner, E., Cherkas, L., et al. (2005). Obesity, cigarette smoking, and telomere length in women. *The Lancet*, *366*(9486), 662-664.

van Tol, M. J., Van Der Wee, N. J., Demenescu, L. R., Nielen, M. M., Aleman, A., Renken, R., et al. (2011). Functional MRI correlates of visuospatial planning in out-patient depression and anxiety. *Acta Psychiatrica Scandinavica*, *124*(4), 273-284.

Verhoef, S. P., Camps, S. G., Bouwman, F. G., Mariman, E. C., & Westerterp, K. R. (2014).

Genetic predisposition, dietary restraint and disinhibition in relation to short and long-term weight loss. *Physiology & Behavior*, *128*, 247-251.

Villareal, D. T., Banks, M., Siener, C., Sinacore, D. R., Klein, S. Physical frailty and body composition in obese elderly men and women. *Obes Res* 2004;12913- 920.

Vincent, H. K., Innes, K. E., & Vincent, K. R. (2007). Oxidative stress and potential interventions to reduce oxidative stress in overweight and obesity. *Diabetes, Obesity and Metabolism*, *9*(6), 813-839.

Vreugdenburg, L., Bryan, J., & Kemps, E. (2003). The effect of self-initiated weight-loss dieting on working memory: the role of preoccupying cognitions. *Appetite*, *41*(3), 291-300.

Wadden, T. A., Butryn, M. L., & Byrne, K. J. (2004). Efficacy Of Lifestyle Modification For Long-Term Weight Control. *Obesity*, *12*, 151S-162S.

Wadden, T. A., Butryn, M. L., & Wilson, C. (2007). Lifestyle Modification for the Management of Obesity. *Gastroenterology*, *132*(6), 2226-2238.

Wadden, T. A., Kaplan, R., Osei, S. Y., Hesson, L. A., Cato, R. K., Phelan, S., et al. (2005). Randomized Trial of Lifestyle Modification and Pharmacotherapy for Obesity. *New England Journal of Medicine*, *353*(20), 2111-2120.

Wang, C. S., Burke, J. R., Steffens, D. C., Hulette, C. M., Breitner, J. C., & Plassman, B. L. (2009). Twin pairs discordant for neuropathologically confirmed Lewy body dementia. *Journal of Neurology, Neurosurgery & Psychiatry*, *80*(5), 562-565.

Wang, J., Chen, R., Peng, W., Zhang, Y., Shen, J., Li, J., et al. (2013). Association between obesity and cognition impairment in patients with moderate-to-severe obstructive sleep apnea-hypopnea syndrome. *Zhonghua Yi Xue Za Zhi*, *93*(48), 3817-3821.

Wardle, J., Carnell, S., Haworth, C. M., Farooqi, I. S., O'rahilly, S., & Plomin, R. (2008).

Obesity Associated Genetic Variation in FTO Is Associated with Diminished Satiety. *Journal of Clinical Endocrinology & metabolism* , *93*(9), 3640-3643.

Wardle, J., Carnell, S., Haworth, C., & Plomin, R. (2008). Evidence for a strong genetic influence on childhood adiposity despite the force of the obesogenic environment. *American Journal of Clinical Nutrition*, *87*(2), 398-404.

Wardle, J., Guthrie, C., Sanderson, S., Birch, L., & Plomin, R. (2001). Food and activity preferences in children of lean and obese parent. *International Journal of Obesity*, *25*(7), 971-977.

Wardle, J., Sanderson, S., Guthrie, C. A., Rapoport, L., & Plomin, R. (2002). Parental Feeding Style And The Inter-generational Transmission Of Obesity Risk. *Obesity*, *10*(6), 453-462.

Werthmann, J., Roefs, A., Nederkoorn, C., Mogg, K., Bradley, B. P., & Jansen, A. (2011). Can(not) take my eyes off it: Attention bias for food in overweight participants.. *Health Psychology*, *30*(5), 561-569.

Westenhoefer, J., Engel, D., Holst, C., Lorenz, J., Peacock, M., Stubbs, J., et al. (2013). Cognitive and weight-related correlates of flexible and rigid restrained eating behaviour. *Eating Behaviors*, *14*(1), 69-72.

Westerterp, K. R., & Speakman, J. R. (2008). Physical Activity Energy Expenditure Has Not Declined Since The 1980s And Matches Energy Expenditures Of Wild Mammals. *International Journal of Obesity*, *32*(8), 1256-1263.

Wheeler, K., Greiner, P., & Boulton, M. (2005). Exploring Alexithymia, Depression, and

Binge Eating in Self-Reported Eating Disorders in Women. *Perspectives in Psychiatric Care*, *41*(3), 114-123.

White, C. L., Morrison, C. D., Ingram, D. K., Keller, J. N., Hise, T. L., Fernandez-Kim, S., et al. (2009). Effects of high fat diet on Morris maze performance, oxidative stress, and inflammation in rats: Contributions of maternal diet. *Neurobiology of Disease*, *35*(1), 3-13.

Whitmer, R. A., Gustafson, D. R., Barrett-Connor, E., Haan, M. N., Gunderson, E. P., & Yaffe, K. (2008). Central Obesity And Increased Risk Of Dementia More Than Three Decades Later. *Neurology*, *71*(14), 1057-1064.

Williamson, D. A., Regeski, J., Lang, W., et al. Impact of a Weight Management Program on Health Related Quality of Life in Overweight Adults with Type 2 Diabetes. *Arch Intern Medicine*. 2009; 169 (2): 163-171.

Winocur, G., Greenwood, C. E., Piroli, G. G., Grillo, C. A., Reznikov, L. R., Reagan, L. P., et al. (2005). Memory impairment in obese Zucker rats: An investigation of cognitive function in an animal model of insulin resistance and obesity. *Behavioral Neuroscience*, *119*(5), 1389-1395.

Wisniewski, A. B., & Chernausek, S. D. (2009). Gender in childhood obesity: Family environment, hormones, and genes. *Gender Medicine*, *6*, 76-85.

Wu, B., Fukuo, K., Suzuki, K., Yoshino, G., & Kazumi, T. (2009). Relationships of Systemic Oxidative Stress to Body Fat Distribution, Adipokines and Inflammatory Markers in Healthy Middle-aged Women. *Endocrine Journal*, *56*(6), 773-782.

Xu, H., Tartaglia, L. A., Chen, H., Ross, J. S., Nichols, A., Sole, J., et al. (2003). Chronic inflammation in fat plays a crucial role in the development of obesity-related insulin resistance. *Journal of Clinical Investigation*, *112*(12), 1821-1830.

Xu, W. L., Atti, A. R., Gatz, M., Pedersen, N. L., Johansson, B., & Fratiglioni, L. (2011). Midlife overweight and obesity increase late-life dementia risk: A population-based twin study. *Neurology*, *76*(18), 1568-1574.

Yesavage, J., Kinoshita, L., O'Hara, R., Noda, A., Lazzeroni, L., Fairchild, K., et al. (2014). Effects of body mass index-related disorders on cognition: preliminary results. *Diabetes, Metabolic Syndrome and Obesity : Targets and Therapy* , *7*, 145-151.

Young, L. R., & Nestle, M. (2007). Portion Sizes And Obesity: Responses Of Fast-Food Companies. *Journal of Public Health Policy*, *28*(2), 238-248.

Zabena, C., GonzÃ¡lez-SÃ¡nchez, J. L., MartÃ nez-Larrad, M. T., Torres-GarcÃ a, A., Alvarez-FernÃ¡ndez-Represa, J., CorbatÃ³n-Anchuelo, A., et al. (2009). The FTO Obesity Gene. Genotyping and Gene Expression Analysis in Morbidly Obese Patients. *Obesity Surgery*, *19*(1), 87-95.

Zak-Golab, A., Tomalski, R., Bak-Sosnowska, M., Holeck, M., Kocelak, P., Olszanecka-Glinianowicz, M. (2013). Alexithymia, depression, anxiety and binge eating in obese women. *European Journal of Psychiatry* , *27*(3), 149-159.

Zigmond, A. S., Snaith, R. P. (1983). "The hospital anxiety and depression scale". *Acta Psychiatrica Scandinavica* **67** (6): 361–370.

Zonnevijlle-Bendek, M., Goozen, S. v., Cohen-Kettenis, P., Elburg, A. v., & Engeland, H. v. (2002). Do adolescent anorexia nervosa patients have deficits in emotional functioning. *European Child & Adolescent Psychiatry*, *11*(1), 38-42.

Zwaan, M. D., Bach, M., Mitchell, J. E., Ackard, D., Specker, S. M., Pyle, R. L., et al. (1995). Alexithymia, obesity, and binge eating disorder. *International Journal of Eating Disorders*, *17*(2), 135-140.

Appendices

A)

Questionnaires collected as part of routine data collection in Weight Management Service (*Chapter 2*)

Appendix 1a: Pittsburgh Sleep Quality Index (PSQI)

Appendix 2a: Epworth Sleepiness Scale (ESS)

Appendix 3a: Hospital Anxiety and Depression Scale (HADS)

Appendix 4a: Impact of Weight on Quality of Life-Lite (IWQOL-Lite)

B)

Questionnaires collected as part of Alexithymia Study (*Chapter 3*)

Appendix 1b: The Alexithymia Questionnaire for children (Rieffe, Oosterveld, and Terwogt 2006)

Appendix 2b: Healthy Lifestyle habits Questionnaire

Appendix 3b: Psychological Distress Questionnaire

C)

Questionnaires collected as part of CANTAB study (*Chapter 4)*

Appendix 1c: The Beck Depression Inventory (BDI) (Beck et al. 1961)

Appendix 2c: The Barratt Impulsiveness Scale-11 (BIS-11) (Patton, Stanford, Barratt 1995)

Appendix 3c: The Dutch Eating Behaviour Questionnaire (DEBQ) (Van Strien et al. 1986)

Appendix 4c: Lifestyle Questionnaire

Appendix 5c: The Self-Esteem Scale (SE) (Rosenberg 1965)

Appendix 6c: CANTAB Participant Information Sheet (PIS) and consent form

D)

Questionnaires collected as part of Food Memory Study 1 and 2 (*Chapter 5)*

Appendix 1d: Taste Perception Scale (Pasta)

Appendix 2d: Pasta Memory Test (PMT)

Appendix 3d: Hunger and Mood Scales

Appendix 4d: The Food Preoccupation Questionnaire (FPQ) (Tapper and Pothos 2010)

Appendix 5d: Pasta Participant Information Sheet (PIS) and consent form Study 1

Appendix 6d: Jelly Bean Liking Scale

Appendix 7d: Jelly Bean Food Recall:

Appendix 8d: Jelly Bean Participant Information Sheet (PIS) and consent form Study 2

Appendix 1a

Pittsburgh Sleep Quality Index (PSQI)

We would like to ask you to provide us information about your sleep. The following questions relate to your usual sleep habits during the past month only. Your answers should indicate the most accurate reply for the majority of days and nights in the past month. Please answer all questions.

1) During the past month, what time have you usually gone to bed at night?

Bed Time | h | h | : | m | m | Am/Pm

2) During the past month, how long (in minutes) has it usually taken you to fall asleep each night?

Number of Minutes | | | |

3) During the past month, what time have you usually got up in the morning?

Getting Up Time | h | h | : | m | m |

4) During the past month, how many hours of actual sleep did you get at night? (This may be different than the number of hours you spent in bed.)

Hours of Sleep per Night | | |

For each of the remaining questions, check the one best response. Please answer all questions.

5) During the past month, how often have you had trouble sleeping because you . . .

5a) Cannot get to sleep within 30 minutes

- [1] Not during the past month
- [2] Less than once a week
- [3] Once or twice a week
- [4] Three or more times a week

5b) Wake up in the middle of the night or early morning

- [1] Not during the past month
- [2] Less than once a week
- [3] Once or twice a week
- [4] Three or more times a week

5c) Have to get up to use the bathroom

- [1] Not during the past month
- [2] Less than once a week
- [3] Once or twice a week
- [4] Three or more times a week

5d) Cannot breathe comfortably

- [1] Not during the past month
- [2] Less than once a week
- [3] Once or twice a week
- [4] Three or more times a week

5e) Cough or snore loudly

1 Not during the past month
2 Less than once a week
3 Once or twice a week
4 Three or more times a week

5f) Feel too cold

1 Not during the past month
2 Less than once a week
3 Once or twice a week
4 Three or more times a week

5g) Feel too hot

1 Not during the past month
2 Less than once a week
3 Once or twice a week
4 Three or more times a week

5h) Had bad dreams

1 Not during the past month
2 Less than once a week
3 Once or twice a week
4 Three or more times a week

5i) Have pain

1 Not during the past month
2 Less than once a week
3 Once or twice a week
4 Three or more times a week

Other reason(s), please describe _____

5j) How often during the past month have you had trouble sleeping because of this?

1 Not during the past month
2 Less than once a week
3 Once or twice a week
4 Three or more times a week

6) During the past month, how often have you taken medicine to help you sleep (prescribed or "over the counter")?

1 Not during the past month
2 Less than once a week
3 Once or twice a week
4 Three or more times a week

7) During the past month, how often have you had trouble staying awake while driving,eating meals, or engaging in social activity?

1 Not during the past month
2 Less than once a week
3 Once or twice a week
4 Three or more times a week

8) During the past month, how much of a problem has it been for you to keep up enough enthusiasm to get things done?

1 No problem at all
2 Only a very slight problem
3 Somewhat of a problem
4 A very big problem

9) During the past month, how would you rate your sleep quality overall?

1 Very good
2 Fairly good
3 Fairly bad
4 Very bad

Appendix 2a

Epworth Sleepiness Scale

Please use following scale to indicate the chance of falling asleep during different situations:

0= would never fall asleep in that situation,
1= there is a slight chance of falling asleep in that situation,
2= there is a medium chance of falling sleep in that situation,
3= there is a high chance of falling asleep in that situation

1) Sitting and reading ☐

2) Watching TV ☐

3) Sitting inactive in a public place (ie. theatre or meeting) ☐

4) As a passenger in a car for an hour without a break ☐

5) Lying down to rest in the afternoon when circumferences permit ☐

6) Sitting and talking to someone ☐

7) Sitting quietly after a lunch without alcohol ☐

8) In a car while stopped for a few minutes in traffic ☐

Appendix 3a

HADS

Your nurse is aware that emotions play an important part in your illness and treatment. If your nurse knows about these feelings he will be able to help you more.

This questionnaire is designed to help your nurse to know how you feel. Read each item and place a firm tick in the box opposite the reply, which comes closest to how you have been feeling in the past week.

Don't take too long over your replies; your immediate reaction to each item will probably be more accurate than a long thought-out response. Tick one box only in each section

1. I feel tense or wound up:

- Most of the time ☐
- A lot of the time ☐
- Time to time, occasionally ☐
- Not at all ☐

2. I still enjoy the things I used to enjoy:

- Definitely as much ☐
- Not quite so much ☐
- Only a little ☐
- Hardly at all ☐

3. I get a sort of frightened feeling as if something awful is about to happen:

- Very definitely and quite badly ☐
- Yes, but not too badly ☐
- A little, but it doesn't worry me ☐
- Not at all ☐

4. I can laugh and see the funny sides of things:

- As much as I always could ☐
- Not quite so much now ☐
- Definitely not so much now ☐
- Not at all ☐

5. Worrying thoughts go through my mind:

- A great deal of the time ☐
- A lot of the time ☐
- From time to time but not too often ☐
- Only occasionally ☐

6. I feel cheerful:

- Not at all ☐
- Not often ☐
- Sometimes ☐
- Most of the times ☐

7. I can sit at ease and feel relaxed:

- Definitely ☐
- Usually ☐
- Not often ☐
- Not at all ☐

8. I feel as if I am slowed down:

- Nearly all of the time ☐
- Very often ☐
- Sometimes ☐
- Not at all ☐

9. I get a sort of frightened feeling like 'butterflies' in the stomach:

- Definitely as much ☐
- Not quite so much ☐
- Only a little ☐
- Hardly at all ☐

10. I have lost interest in my appearance:

- Definitely ☐
- I don't take so much care as I should ☐
- I may not take quite as much care ☐
- I take just as much care as ever ☐

11. I feel restless as if I have to be on the move:

- Very much indeed ☐
- Quite a lot ☐
- Not very much ☐
- Not at all ☐

12. I look forward with enjoyment to things:

- As much as I ever did ☐
- Rather less that I used to ☐
- Definitely less that I used to ☐
- Hardly at all ☐

13. I get sudden feelings of panic:

- Very often indeed ☐
- Quite often ☐
- Not very often ☐
- Not at all ☐

14. I can enjoy a good book or radio or TV programme:

- Definitely ☐
- Usually ☐
- Not often ☐
- Very seldom ☐

Appendix 4a

Impact of Weight on Quality of life Questionnaire (IWQOI-Lite)

Please answer the following statements by choosing the best answer applies to you **in the past week**.

Physical Function	Always True	Usually True	Some-times True	Rarely True	Never True
1.Because of my weight I have trouble picking up objects	☐	☐	☐	☐	☐
2. Because of my weight I have trouble tying my shoes	☐	☐	☐	☐	☐
3. Becasue of my weight I have difficulty getting up from chairs	☐	☐	☐	☐	☐
4. Becasue of my weight I have trouble using stairs	☐	☐	☐	☐	☐
5. Becasue of my weight I have difficulty putting on or taking off my clothing	☐	☐	☐	☐	☐
6. Becasue of my weight I have trouble with mobility	☐	☐	☐	☐	☐
7. Becasue of my weight I have trouble my legs	☐	☐	☐	☐	☐
8. I feel short of breath with only mild exertion	☐	☐	☐	☐	☐
9. I am troubled by painful or stiff joints	☐	☐	☐	☐	☐
10. My ankles and lower legs are swollen at the end of the day	☐	☐	☐	☐	☐
11. I am worried about my health	☐	☐	☐	☐	☐
Self esteem	Always True	Usually True	Some-times True	Rarely True	Never True
1.Because of my weight I am self-conscious	☐	☐	☐	☐	☐
2. Because of my weight my self-seteem is not what it could be	☐	☐	☐	☐	☐
3. Becasue of my weight I feel unsure of myself	☐	☐	☐	☐	☐
4. Because of my weight I don't like myself	☐	☐	☐	☐	☐
5. Becasue of my weight I am afraid of being rejected	☐	☐	☐	☐	☐

Self esteem	Always True	Usually True	Some-times True	Rarely True	Never True
6. Because of my weight I avoid looking in mirrors or seeing myself in photographs	☐	☐	☐	☐	☐
7. Because of my weight I am embarressed to be seen in public places	☐	☐	☐	☐	☐
Sexual Life	Always True	Usually True	Some-times True	Rarely True	Never True
1. Because of my weight I do not enjoy sexual activity	☐	☐	☐	☐	☐
2. Because of my weight I have little or no sexual desire	☐	☐	☐	☐	☐
3. Becasue of my weight I have difficulty with sexual performance	☐	☐	☐	☐	☐
4. Becasue of my weight I avoid sexual encounters whenever possible	☐	☐	☐	☐	☐
Public Distress	Always True	Usually True	Some-times True	Rarely True	Never True
1. Because of my weight I experience ridicule, teasing, or unwanted attention	☐	☐	☐	☐	☐
2. Because of my weight I worry about fitting into seats in public places (e.g. theatres, resturants, cars)	☐	☐	☐	☐	☐
3. Becasue of my weight I worry about fitting through aisles or turnstiles	☐	☐	☐	☐	☐
4. Becasue of my weight I worry about finding chairs that are strong enough to hold my weight	☐	☐	☐	☐	☐
5. Because of my weight I experience discrimination by others	☐	☐	☐	☐	☐
Work*	Always True	Usually True	Some-times True	Rarely True	Never True
1. Because of my weight I have trouble getting things accomplished or meeting my responsibilities	☐	☐	☐	☐	☐
2. Because of my weight I am less productive than I could be	☐	☐	☐	☐	☐

Appendix 1b (Adapted from online format)

The way I feel…

Please fill out your first name .....
And your date of birth .....

On the next pages, you will find 20 short sentences. Every sentence is a statement about how people can feel or think about their feelings. You can mark each sentence if this is often true, sometimes true or not true for you. Choose the answer that best fits you. You can only mark one answer.

If you find that difficult, choose the answer that fits you most of the time. Different people have different feelings and ideas about their feelings. Therefore, there are no right or wrong answers, because it is just about what you think.

(The online answer options were: Not true, Sometimes True, Often True)

1	**I am often confused about the way I am feeling inside**	_	_	_
2	**I find it difficult to say how I feel inside**	_	_	_
3	**I feel things in my body that even doctors don't understand**	_	_	_
4	**I can easily say how I feel inside**	_	_	_
5	**When I have a problem, I want to know where it comes from and not just talk about it**	_	_	_
6	**When I am upset, I don't know if I am sad, scared, or angry**	_	_	_
7	**I am often puzzled by things that I feel in my body**	_	_	_
8	**I'd rather wait and see what happens, instead of thinking about why things happen**	_	_	_
9	**Sometimes I can't find the words to say how I feel inside**	_	_	_
10	**It is important to understand how you feel inside**	_	_	_
11	**I find it hard to say how I feel about other people**	_	_	_
12	**Other people tell me that I should talk more about how I feel inside**	_	_	_
13	**I don't know what's going on inside me**	_	_	_
14	**I often don't know why I am angry**	_	_	_
15	**I prefer talking to people about everyday things,**			

	rather than about how they feel	_	_	_
16	**I prefer watching funny television programmes, rather than films that tell a story about other people's problems**			
17	**It is difficult for me to say how I really feel inside, even to my best friend**	_	_	_
18	**I can feel close to someone, even when we are sitting still and not saying anything**	_	_	_
19	**Thinking about how I feel, helps me when I want to do something about my problems**	_	_	_
20	**When I have to concentrate on a film to understand the story, I enjoy the film much less**	_	_	_

Please check
that you have marked all of the sentences.

Thank you!

Appendix 2b

Healthy lifestyle habits (adapted from online questionnaire)

How many fruits and vegetables do you eat every day?

None One to two three to four four or more

How often do you exercise (run, play sports, or other activity) outside of school?

Never rarely sometimes often very often

How often do you eat unhealthy snacks (e.g. crisps, chocolate, cake)?

Daily almost daily sometimes rarely

Appendix 3b

Psychological distress (adapted from online questionnaire)

*Each item was on a 3 point Likert scale; 1= 'a lot', 2= 'somewhat', 3= 'not at all'.

Do you feel hopeless about the future (in last two weeks)? A lot somewhat Not at all

Do you feel unhappy; sad, or depressed (in last two weeks)? A lot somewhat Not at all

Do you worry too much about things (in last two weeks)? A lot somewhat Not at all

Do you feel nervous or tense (in last two weeks)? A lot somewhat Not at all

Appendix 1c

Participant number

Fill out the following questionnaire, read each item carefully and circle the number next to the answer that best reflects how you have been feeling during
the past few days. Circle one answer for each of the twenty-one questions. ... If more than one answer applies to how you have been feeling, circle the higher
number. If in doubt, make your best guess. Do not leave any question unanswered.

1.
0 I do not feel sad.
1 I feel sad.
2 I am sad all the time and I can't snap out of it.
3 I am so sad or unhappy that I can't stand it.
2.
0 I am not particularly discouraged about the future.
1 I feel discouraged about the future.
2 I feel I have nothing to look forward to.
3 I feel that the future is hopeless and that things cannot improve.
3.
0 I do not feel like a failure.
1 I feel I have failed more than the average person.
2 As I look back on my life, all I can see is a lot of failures.
3 I feel I am a complete failure as a person.
4.
0 I get as much satisfaction out of things as I used to.
1 I don't enjoy things the way I used to.
2 I don't get real satisfaction out of anything anymore.
3 I am dissatisfied or bored with everything.
5.
0 I don't feel particularly guilty.
1 I feel guilty a good part of the time.
2 I feel quite guilty most of the time.
3 I feel guilty all of the time.
6
0 I don't feel I am being punished.
1 I feel I may be punished.
2 I expect to be punished.
3 I feel I am being punished.
7.
0 I don't feel disappointed in myself.
1 I am disappointed in myself.
2 I am disgusted with myself.

3 I hate myself.
8.
0 I don't feel I am any worse than anybody else.
1 I am critical of myself for my weaknesses or mistakes.
2 I blame myself all the time for my faults.
3 I blame myself for everything bad that happens.

9.
0 I don't have any thoughts of killing myself.
1 I have thoughts of killing myself, but I would not carry them out.
2 I would like to kill myself.
3 I would kill myself if I had the chance.
10.
0 I don't cry any more than usual.
1 I cry more than I used to.
2 I cry all the time now.
3 I used to be able to cry, but now I can't cry even though I want to.
11.
0 I am no more irritated by things than I ever am.
1 I am slightly more irritated now than usual.
2 I am quite annoyed or irritated a good deal of the time.
3 I feel irritated all the time now.
12.
0 I have not lost interest in other people.
1 I am less interested in other people than I used to be.
2 I have lost most of my interest in other people.
3 I have lost all my interest in other people.
13.
0 I make decisions about as well as I ever could.
1 I put off making decisions more than I used to.
2 I have greater difficulty in making decisions than before.
3 I can't make decisions at all anymore.
14.
0 I don't feel that I look any worse than I used to.
1 I am worried that I am looking old or unattractive.
2 I feel that there are permanent changes in my appearance that make me look
unattractive.
3 I believe I look ugly.
15.
0 I can work about as well as before.
1 It takes an extra effort to get started at doing something.
2 I have to push myself very hard to do anything.
3 I can't do any work at all.
16.
0 I can sleep as well as usual.
1 I don't sleep as well as I used to.

2 I wake up 1 – 2 hours earlier than usual and find it hard to get back to sleep.
3 I wake up several hours earlier than I used to and cannot get back to sleep.
17.
0 I don't get more tired than usual.
1 I get tired more easily than I used to.
2 I get tired from doing almost anything.
3 I am too tired to do anything.

18.
0 My appetite is no worse than usual.
1 My appetite is not as good as it used to be.
2 My appetite is much worse now.
3 I have no appetite at all anymore.
19.
0 I haven't lost much weight, if any, lately.
1 I have lost more than five pounds.
2 I have lost more than ten pounds.
3 I have lost more than fifteen pounds.
20.
0 I am no more worried about my health than usual.
1 I am worried about physical problems such as aches and pains, or upset stomach, or constipation
2 I am very worried about my physical problems that I cannot think about anything else.
3 I am so worried about my physical problems that I cannot think about anything
else.
21.
0 I have not noticed any recent changes in my interest in sex.
1 I am less interested in sex than I used to be.
2 I am much less interested in sex now.
3 I have lost interest in sex completely.

Appendix 2c

Participant number

DIRECTIONS: People differ in the ways they act and think in different situations. This is a test to measure some of the ways in which you act and think. Read each statement and put an X on the appropriate circle on the right side of this page. Do not spend too much time on any statement. Answer quickly and honestly.

	O	O	O	O
	Rarely/Never	Occasionally	Often	Almost Always/Always
1 I plan tasks carefully.	O	O	O	O
2 I do things without thinking.	O	O	O	O
3 I make-up my mind quickly.	O	O	O	O
4 I am happy-go-lucky.	O	O	O	O
5 I don't "pay attention."	O	O	O	O
6 I have "racing" thoughts.	O	O	O	O
7 I plan trips well ahead of time.	O	O	O	O
8 I am self controlled.	O	O	O	O
9 I concentrate easily.	O	O	O	O
10 I save regularly.	O	O	O	O
11 I "squirm" at plays or lectures.	O	O	O	O
12 I am a careful thinker.	O	O	O	O
13 I plan for job security.	O	O	O	O
14 I say things without thinking.	O	O	O	O
15 I like to think about complex problems.	O	O	O	O
16 I change jobs.	O	O	O	O
17 I act "on impulse."	O	O	O	O
18 I get easily bored when solving thought problems.	O	O	O	O
19 I act on the spur of the moment.	O	O	O	O
20 I am a steady thinker.	O	O	O	O
21 I change residences.	O	O	O	O
22 I buy things on impulse.	O	O	O	O
23 I can only think about one thing at a time.	O	O	O	O
24 I change hobbies.	O	O	O	O
25 I spend or charge more than I earn.	O	O	O	O
26 I often have extraneous thoughts when thinking.	O	O	O	O
27 I am more interested in the present than the future.	O	O	O	O
28 I am restless at the heatre or lectures.	O	O	O	O
29 I like puzzles.	O	O	O	O

30 I am future oriented.	O O O O

Appendix 3c

Participant number

Please answer ALL questions. Circle the appropriate response.

When you have put on weight do you eat less than you usually do?	**Not relevant**	**never**	**seldom**	**sometimes**	**often**	**very often**
Do you try to eat less at mealtimes than you would like to eat?		**Never**	**seldom**	**sometimes**	**often**	**very often**
How often do you refuse food or drink offered to you because you are concerned about your weight?		**Never**	**seldom**	**sometimes**	**often**	**very often**
Do you watch exactly what you eat?		**Never**	**seldom**	**sometimes**	**often**	**very often**
Do you deliberately eat foods that are slimming?		**Never**	**seldom**	**sometimes**	**often**	**very often**
When you have eaten too much, do you eat less than usual the following day?	**Not relevant**	**never**	**seldom**	**sometimes**	**often**	**very often**
Do you deliberately eat less in order not to become heavier?		**Never**	**seldom**	**sometimes**	**often**	**very often**
How often do you try not to eat between meals because you are watching your weight?		**Never**	**seldom**	**sometimes**	**often**	**very often**
How often in the evenings do you try not to eat because you are watching your weight?		**Never**	**seldom**	**sometimes**	**often**	**very often**
Do you take your weight into account with what you eat?		**Never**	**seldom**	**sometimes**	**often**	**very often**

If food tastes good to you, do you eat more than usual?		**Never**	**seldom**	**sometimes**	**often**	**very often**
If food smells good, do you eat more than usual?		**Never**	**seldom**	**sometimes**	**often**	**very often**
If you smell something delicious, do you have a desire to eat it?		**Never**	**seldom**	**sometimes**	**often**	**very often**
If you have something delicious to eat, do you eat it straight away?		**Never**	**seldom**	**sometimes**	**often**	**very often**
If you walk past a baker, do you have a desire to buy something delicious?		**Never**	**seldom**	**sometimes**	**often**	**very often**
If you walk past a snackbar or café, do you have a desire to buy something delicious?		**Never**	**seldom**	**sometimes**	**often**	**very often**
If you see others eating, do you also have a desire to eat?		**Never**	**seldom**	**sometimes**	**often**	**very often**
Can you resist eating delicious foods?		**Never**	**seldom**	**sometimes**	**often**	**very often**

Do you eat more than usual, when you see others eating?		**Never**	**seldom**	**sometimes**	**often**	**very often**
When preparing a meal, are you inclined to eat something?		**Never**	**seldom**	**sometimes**	**often**	**very often**

Do you have a desire to eat when you are irritated?	**Not relevant**	**never**	**seldom**	**sometimes**	**often**	**very often**
Do you have a desire to eat when you have nothing to do?	**Not relevant**	**never**	**seldom**	**sometimes**	**often**	**very often**
Do you have a desire to eat when you are depressed or discouraged?	**Not relevant**	**never**	**seldom**	**sometimes**	**often**	**very often**
Do you have a desire to eat when you are feeling lonely?	**Not relevant**	**never**	**seldom**	**sometimes**	**often**	**very often**
Do you have a desire to eat when you somebody lets you down?	**Not relevant**	**never**	**seldom**	**sometimes**	**often**	**very often**
Do you have a desire to eat when you are cross?	**Not relevant**	**never**	**seldom**	**sometimes**	**often**	**very often**
Do you have a desire to eat when you are something unpleasant is about to happen?		**Never**	**seldom**	**sometimes**	**often**	**very often**
Do you get the desire to eat when you are anxious, worried or tense?		**Never**	**seldom**	**sometimes**	**often**	**very often**
Do you have a desire to eat when things are going against you and when things have gone wrong?		**Never**	**seldom**	**sometimes**	**often**	**very often**
Do you have a desire to eat when you are frightened?	**Not relevant**	**never**	**seldom**	**sometimes**	**often**	**very often**
Do you have a desire to eat when you are disappointed?	**Not relevant**	**never**	**seldom**	**sometimes**	**often**	**very often**
Do you have a desire to eat when you are emotionally upset?	**Not relevant**	**never**	**seldom**	**sometimes**	**often**	**very often**
Do you have a desire to eat when you are bored or restless?	**Not relevant**	**never**	**seldom**	**sometimes**	**often**	**very often**

Appendix 4c

Participant number

Lifestyle Questionnaire

Please answer <u>all</u> of the following questions. Please circle the appropriate response.

1) What is your age?

_____

2) Please state your gender

Male Female

3) What is your occupation?

_____

4) What is your highest level of education?

_____

5) Do you regularly eat breakfast in the morning?

Yes No

6) Where do you normally eat your main meal of the day?

_____

7) Do you regularly eat lunch?

Yes No

8) Do you ever leave food on your plate because you feel full before the end of the meal?

Never Occasionally Often Always

9) Do you smoke?

Yes No

If yes, please indicate how many cigarettes you smoke per day, on average.

1-5 5-10 10-15 15-20 20-25 25+

10) Do you drink alcohol?

Yes No

If yes, please indicate how many units of alcohol you drink per week, on average.

1-5 5-10 10-15 15-20 20-25 25-30 30-35 35-40 40+

(1 unit = ½ pint beer, lager or cider,1 small glass of wine or 1 single measure of spirits)

11) Are you vegetarian or vegan?

Yes No

10) Do you have any food related allergies? (please describe)

---

10) **Are you on a restricted diet?**

Yes No

If yes, please describe

_____

---

10) **What has been your highest weight?**

_____
_____

10) **What has been your lowest weight?**

_____
_____

14) Do you suffer from any medical illnesses e.g. diabetes or epilepsy?

Yes No

If yes, please state

_____
_____

15) Are you currently taking any medication?

Yes No

If yes, please state the name of the medication and the condition it is used to treat *(optional)*

_____

16)	**Which of the following best describes you (*please complete one box in both parts):***							
a)	Asian	☐	White	☐	Black	☐	Other	☐
b)	African	☐	Caribbean	☐	European	☐	Pakistani	☐
	Indian	☐	Bangladeshi	☐	Chinese	☐	Other	☐

If 'other' please specify:

Thank you

Appendix 5c

Participant number

Rosenberg (SES)

Instructions: Below is a list of statements dealing with your general feelings about yourself. If you strongly agree, circle **SA**. If you agree with the statement, circle **A**. If you disagree, circle **D**. If you strongly disagree, circle **SD**.

1.	On the whole, I am satisfied with myself.	SA	A	D	SD
2.	At times, I think I am no good at all.	SA	A	D	SD
3.	I feel that I have a number of good qualities.	SA	A	D	SD
4.	I am able to do things as well as most other people.	SA	A	D	SD
5.	I feel I do not have much to be proud of.	SA	A	D	SD
6.	I certainly feel useless at times.	SA	A	D	SD
7.	I feel that I'm a person of worth, at least on an equal plane with others.	SA	A	D	SD
8.	I wish I could have more respect for myself.	SA	A	D	SD
9.	All in all, I am inclined to feel that I am a failure.	SA	A	D	SD
10.	I take a positive attitude toward myself.	SA	A	D	SD

Appendix 6c

THE UNIVERSITY
OF BIRMINGHAM

Participant Information Sheet

Cognitive Investigations and Health

The Study:

In this study, neuropsychological processes will be measured using computerized tests. When looking at cognitive function, participants' general health will also be considered. This will involve collecting information on areas such as behaviours and eating habits.

What will I be asked to do if I take part?

First, you will be asked to answer questions about yourself through questionnaires. You will then complete multiple short tests on a computer. This will take approximately 2 hours.

Will my data be anonymous?

Yes, the information you provide will be anonymous. Responses and consent forms will be stored within a lockable file. Participants' data will be coded with numbers and documents matching codes to participants' names will be stored separately. No personally identifiable info will be published.

Do I have to take part?

You do not have to take part in the study. If you decide to take part and later change your mind, either before you start the study or during it, you can withdraw without giving your reasons, and if you wish, your data will be destroyed. After completion of the study all data will be stored in a completely anonymous format and therefore it will not be possible to identify of delete specific participant data.

Where can I obtain further information if I need it?

If you would like to take part in this study or would like more information, please contact Iraida Neira at Ixn082@bham.ac.uk . To speak to the supervisor of this study, contact Suzanne Higgs at s.higgs.1@bham.ac.uk .

THE UNIVERSITY
OF BIRMINGHAM

Cognitive Investigations and Health

Consent form

The study is designed to investigate potential links between cognitive function and physical health.

In this study you will be required to attend one session that may last up to 2 hours. The session will involve you answering questionnaires and completing computer based cognitive tests.

This process is completely confidential. You may contact the researcher at any point in the future to request a copy of any reports resulting from this data.

Please note that participation is voluntary and you are free to withdraw from the experiment at any time and for any reason. All data collected will be anonymous.

Please sign the form to indicate that you have read and understand these instructions.

- I agree to participate in this experiment and understand that I am free to withdraw at any time.
- I agree to having my participant ID saved so that I may be contacted for future studies by this researcher.
- I have received and read the above information about the study and understand what is involved from me as a participant
- I know that the information collected about me is and will remain completely confidential
- I understand that I am free to withdraw at any time

Signed ----------

Name ----------
(printed)

Date ----------

Appendix 1d

Pasta Sauce Taste Scale
Please mark your response with an X on each line.

How pleasant was your pasta meal?

Low_____High

How salty was your pasta sauce?

Low_____High

How sweet was your pasta sauce?

Low_____High

How sour was your pasta sauce?

Low_____High

Appendix 2d

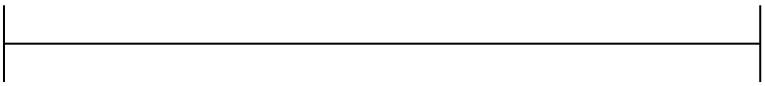
Pasta Taste Test

Which pasta sauce did you eat for lunch? A, B, C, or D?

How vividly (or well) to you remember your pasta lunch right now?

NOT AT ALL Vivid

Hungry



Appendix 3d

Participant number:

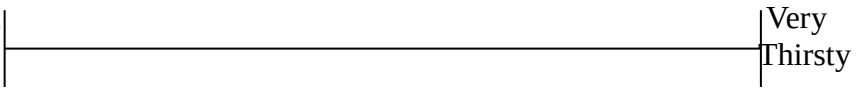
Hunger and Mood Scale

The following rating scales consist of a line with two end points. The line represents a continuum of possibilities between two statements. Above the line will be a rating question e.g. How hungry are you? Please mark a single vertical line on the scale at the place that best describes your answer.

Example:

How **THIRSTY** do you feel right now?

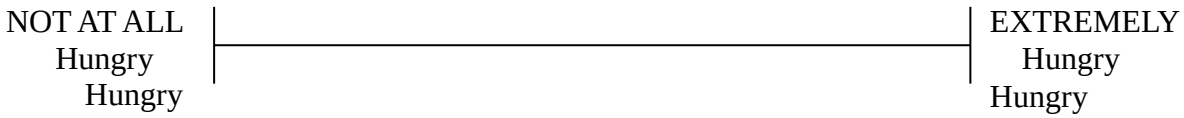
Not at all **Thirsty**



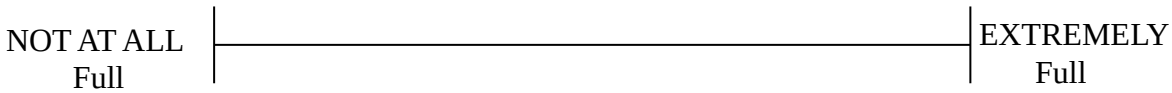
Very Thirsty

Please answer the following:

How HUNGRY do you feel right now?

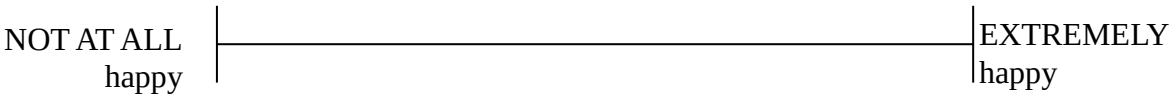


How FULL do you feel right now?

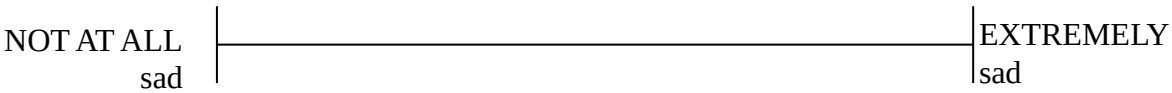


How strong is your DESIRE to eat right now?

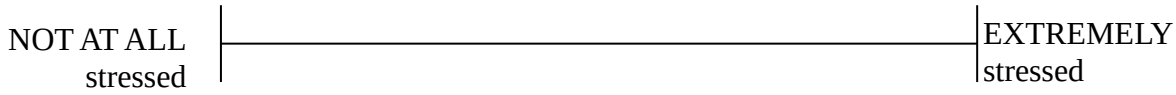
How HAPPY do you feel right now?



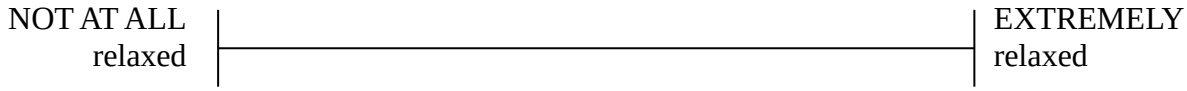
How SAD do you feel right now?



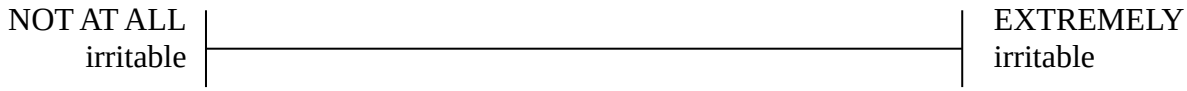
How STRESSED do you feel right now?



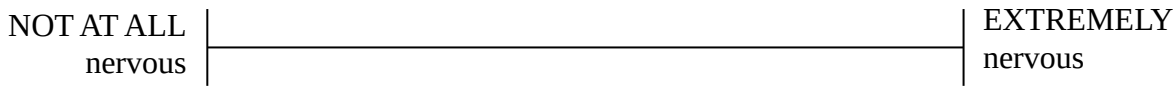
How RELAXED do you feel right now?



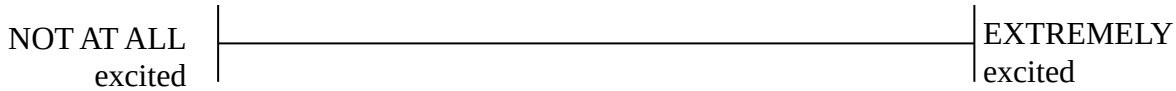
How IRRITABLE do you feel right now?



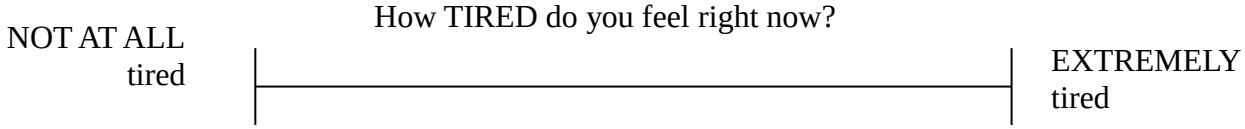
How NERVOUS do you feel right now?



How EXCITED do you feel right now?



How TIRED do you feel right now?



Thank you

Appendix 4d

Please rate the extent to which you agree or disagree with the following statements, by placing a tick in the appropriate box. Some of the questions may look as if they are 'opposite' to one another. However, **please don't worry about being consistent in your responses**. It is often the case that we feel one way in some situations but a completely different way in other situations. As such, you should answer each question as if it were the only question and avoid looking back at your previous answers.

Participant number ……..……

		1 Completely disagree	2 Disagree a bit	3 Neither agree nor disagree	4 Agree a bit	5 Completely agree
1	I spend a lot of time thinking about food	☐	☐	☐	☐	☐
2	Planning meals can be quite stressful	☐	☐	☐	☐	☐
3	I often find myself thinking about food	☐	☐	☐	☐	☐
4	My thoughts about food don't tend to me particularly pleasant or unpleasant	☐	☐	☐	☐	☐
5	I really enjoy myself thinking about food	☐	☐	☐	☐	☐
6	I can get quite stressed if I start to think about food	☐	☐	☐	☐	☐
7	I often struggle with thoughts about food	☐	☐	☐	☐	☐
8	I like thinking about my favourite food	☐	☐	☐	☐	☐
9	When I think about food it's not usually linked to any particular emotion	☐	☐	☐	☐	☐
10	I often look forward to my next meal	☐	☐	☐	☐	☐
11	I hate being distracted with thoughts about food	☐	☐	☐	☐	☐
12	I don't particularly enjoy or dislike thinking about food	☐	☐	☐	☐	☐
13	I worry I spend too much time thinking about food	☐	☐	☐	☐	☐
14	I love thinking about food	☐	☐	☐	☐	☐
15	Thinking about food can put me in a bad mood	☐	☐	☐	☐	☐
16	Sometimes I think about food just for the fun of it	☐	☐	☐	☐	☐
17	I don't think about food all that much	☐	☐	☐	☐	☐
18	Deciding what to eat can be quite stressful	☐	☐	☐	☐	☐
19	I can get really excited thinking about food	☐	☐	☐	☐	☐
20	I don't pay much attention to thoughts about food	☐	☐	☐	☐	☐
21	Thinking about food can put me in a good mood	☐	☐	☐	☐	☐
22	I hate thinking about food	☐	☐	☐	☐	☐
23	Thinking about food doesn't really excite or depress me	☐	☐	☐	☐	☐
24	I enjoy deciding what to eat in a restaurant	☐	☐	☐	☐	☐
25	Thinking about food can make me quite miserable	☐	☐	☐	☐	☐
26	I enjoy planning what I'm going to eat	☐	☐	☐	☐	☐

5 Completely agree
4 Agree a bit
3 Neither agree nor disagree
2 Disagree a bit
1 Completely disagree

Appendix 5d

THE UNIVERSITY
OF BIRMINGHAM

Participant Information Sheet

Mood and Meal time Study

The Study:

We are investigating the relationship between meal consumption and mood. This study will involve coming in for a meal and snack.

What will I be asked to do if I take part?

During the meal you will be asked to fill out questionnaires about your mood and behaviours. To take part in this study you must not have any food allergies, and be happy to consume biscuits and pasta. All participation is voluntary.

Will my data be anonymous?

Yes, the information you provide will be anonymous. Responses and consent forms will be stored within a lockable file. Participants' data will be coded with numbers and documents matching codes to participants' names will be stored separately. No personally identifiable info will be published.

Do I have to take part?

You do not have to take part in the study. If you decide to take part and later change your mind, either before you start the study or during it, you can withdraw without giving your reasons, and if you wish, your data will be destroyed. After completion of the study all data will be stored in a completely anonymous format and therefore it will not be possible to identify or delete specific participant data.

Where can I obtain further information if I need it?

If you would like to take part in this study or would like more information, please contact Iraida Neira at Ixn082@bham.ac.uk . To speak to the supervisor of this study, contact Suzanne Higgs at s.higgs.1@bham.ac.uk .

THE UNIVERSITY
OF BIRMINGHAM

Mood and Meal time Study

Consent form

The study is designed to investigate eating behaviours.

In this study you will be required to attend two sessions that may last total up to 1 hour total. The session will involve you answering questionnaires eating a light lunch and snack.

This process is completely confidential. You may contact the researcher at any point in the future to request a copy of any reports resulting from this data.

Please note that participation is voluntary and you are free to withdraw from the experiment at any time and for any reason.

All data collected will be anonymous.

Please sign the form to indicate that you have read and understand these instructions.

- I agree to participate in this experiment and understand that I am free to withdraw at any time.
- I have received and read the above information about the study and understand what is involved from me as a participant
- I know that the information collected about me is and will remain completely confidential
- I understand that I am free to withdraw at any time

Signed ----------

Name ----------
(printed)

Date ----------

Appendix 6d

Participant number:

Jelly Bean Scale

The following rating scales consist of a line with two end points. The line represents a continuum of possibilities between two statements. Above the line will be a rating question e.g. How hungry are you? Please mark a single vertical line on the scale at the place that best describes your answer.

Example:

How **THIRSTY** do you feel right now?

Not at all **Thirsty** ——— Very **Thirsty**

Please answer the following:

1. Do you like eating Jelly Bean number 1?

DO NOT LIKE AT ALL ——— LIKE VERY MUCH

Hungry

Hungry

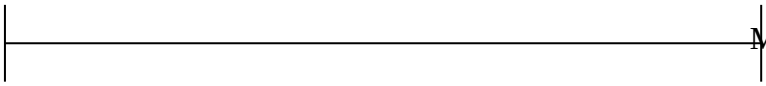
Move on to NEXT BOWL

2. Do you like eating Jelly Bean number 2?

DO NOT LIKE AT ALL ——— LIKE VERY MUCH

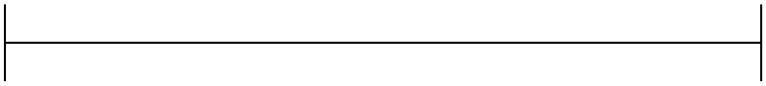
Move on to NEXT BOWL

3. Do you like eating Jelly Bean number 3?

DO NOT LIKE AT ALL  LIKE VERY MUCH

Move on to NEXT BOWL

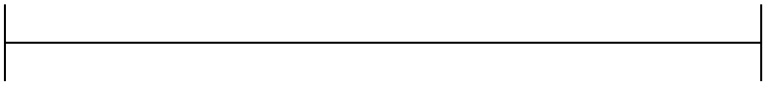
4. Do you like eating Jelly Bean number 4?

DO NOT LIKE AT ALL  LIKE VERY MUCH

Liv

Move on to NEXT BOWL

5. Do you like eating Jelly Bean number 5?

DO NOT LIKE AT ALL  LIKE VERY MUCH

Move on to NEXT BOWL

6. Do you like eating Jelly Bean number 6?

DO NOT LIKE AT ALL — LIKE VERY MUCH

7. Do you like eating Jelly Bean number 7?

DO NOT LIKE AT ALL — LIKE VERY MUCH

8. Do you like eating Jelly Bean number 8?

DO NOT LIKE AT ALL — LIKE VERY MUCH

9. Do you like eating Jelly Bean number 10?

DO NOT LIKE AT ALL — LIKE VERY MUCH

Move on to NEXT BOWL

10. Do you like eating Jelly Bean number 10?

DO NOT LIKE AT ALL |——————————————————————| LIKE VERY MUCH

Thank you

Appendix 7d

Jelly Bean Recall:

What did you eat?
Please write down colour and flavour Jelly Beans next to the appropriate number order:

1. _____
2. _____
3. _____
4. _____
5. _____
6. _____
7. _____

Appendix 8d

THE UNIVERSITY
OF BIRMINGHAM

Participant Information Sheet

Mood, Chewing, and Food Study

We are investigating the relationship between food consumption and other factors, such as mood and chewing style. This study will involve coming in for a snack and completing questionnaires.

What will I be asked to do if I take part?

You will be asked to fill out questionnaires about your mood and behaviours and to eat Jelly Beans. To take part in this study you must not have any food allergies. All participation is voluntary.

Will my data be anonymous?

Yes, the information you provide will be anonymous. Responses and consent forms will be stored within a lockable file. Participants' data will be coded with numbers and documents matching codes to participants' names will be stored separately. No personally identifiable info will be published.

Do I have to take part?

You do not have to take part in the study. If you decide to take part and later change your mind, either before you start the study or during it, you can withdraw without giving your reasons, and if you wish, your data will be destroyed. After completion of the study all data will be stored in a completely anonymous format and therefore it will not be possible to identify or delete specific participant data.

Where can I obtain further information if I need it?

If you would like to take part in this study or would like more information, please contact Iraida Neira at Ixn082@bham.ac.uk . To speak to the supervisor of this study, contact Suzanne Higgs at s.higgs.1@bham.ac.uk .

THE UNIVERSITY
OF BIRMINGHAM

Mood, Chewing, and Food Study

Consent form

The study is designed to investigate thoughts and behaviours when consuming food.

In this study you will be required to one session that may up to 90 minutes total. The session will involve you answering questionnaires eating a snack.

This process is completely confidential. You may contact the researcher at any point in the future to request a copy of any reports resulting from this data.

Please note that participation is voluntary and you are free to withdraw from the experiment at any time and for any reason.

All data collected will be anonymous.

Please sign the form to indicate that you have read and understand these instructions.

- I agree to participate in this experiment and understand that I am free to withdraw at any time.
- I have received and read the above information about the study and understand what is involved from me as a participant
- I agree to be contacted for future studies by the researcher
- I know that the information collected about me is and will remain completely confidential
- I understand that I am free to withdraw at any time

Signed ----------

Name ----------
(printed)

Date ----------