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FACULTY OF MEDICINE, HEALTH AND LIFE SICENCES

School of Psychology

An investigation of the addictive nature of food

Tanya Griffiths BSc (Hons)

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Abstract

The thesis commences with a literature review suggesting common neural circuits mediate food and drug rewards and discusses the application of behavioural and biological theories of addiction to overeating and obesity. Research exploring the relationship between food, reward and overeating is lacking. The current dominant neurobiological theory suggests reward can be separated into psychological components, 'wanting' (incentive salience attribution) and 'liking' (pleasurable/aversive evaluation) mediated by separate neural substrates (Berridge, 1996).

Features of overeating and addiction are discussed with the conclusion that there are many similarities. However presence of tolerance and withdrawal effects, considered central to drug addiction, is weaker in overeating. Psychological processes of restraint, ambivalence and attribution appear to be more applicable to overeating. Although some authors (e.g. Rogers & Smit, 2000) maintain that labelling overeating as an addiction risks trivialising serious addictions, it is argued that the characterisation of overeating as an addiction is important as both issues are associated with serious health complications.

The empirical study investigated if participants who were overweight showed an enhanced attentional or approach bias for food-related stimuli compared to participants of a healthy weight. The relationships between weight, attentional bias and implicit and explicit measures of stimulus valence were explored. Present findings suggest individuals who are overweight have

reduced attentional bias for food cues compared with people of a healthy weight. Evidence of an over-responsive reward system (over-active dopamine system) in response to the sight of food was not present in participants who were overweight. Further research is needed to clarify the issue.

List of Contents

Literature Review

Should overeating be characterised as an addiction?

Empirical Paper

Attentional and approach biases for food related stimuli in individuals who are overweight

List of Tables

List of Figures

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Should overeating be characterised as an addiction?

Literature Review

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Tanya Griffiths

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School of Psychology

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Abstract

Features of addiction include compulsion, loss of control, the discomfort of withdrawal, positive psychoactive effects and harm to health and personal, social and economic functioning. Behavioural and biological theories of addiction attempt to explain these features and may be applicable to feeding behaviour. Palatable food activates the 'reward centres' in the brain leading to prolongation of feeding. Evidence suggests common neural circuits are involved with food and drug rewards. This paper considers the role of opioid, serotoninergic, and dopaminergic pathways. Berridge (1996) suggests reward contains separate psychological components, corresponding roughly to 'wanting' (incentive salience attribution) and 'liking' (pleasurable/aversive evaluation). The 'wanting' process may be mediated by a dopaminergic neural substrate with some evidence for dopaminergic disruption in both dependent drug users, and obese individuals.

Evidence for overeating being characterised as an addiction is equivocal. Features of eating and drug use include psychoactive (mood) effects, external cue (environmental) control of appetites, and cognitive factors of restraint, ambivalence, and attribution. Researchers argue that eating does not produce the powerful neuroadaptive effects (tolerance and withdrawal) central to drug addiction. The utility of characterising overeating as an addiction is briefly considered. Labelling over-consumption of food as an addiction, even if this is associated with high levels of emotional eating and somewhat unstable eating patterns, risks trivialising serious addictions (Rogers & Smit, 2000). This is debatable as the health complications

associated with prolonged drug use are no less serious than those associated with overeating and obesity. Psychological processes of restraint, ambivalence and attribution appear to be more applicable to overeating.

INTRODUCTION

'I have a secret addiction. Often it's one packet a day, sometimes it's more. Occasionally I'll go cold turkey. But whenever I'm depressed or desperate or drunk I'll have a craving. I know all the good places to score. Street corners are best. The relief is immediate - there's a rush, especially in the first few moments. But afterwards I feel guilty. I'll bury the evidence in my bag before I reach home. My name is Louise and I eat crisps'

(http://lifeandhealth.guardian.co.uk/food/story/0,,1614346,00.html, 2004).

Over the past 30 years, one of the most pressing emerging health issues affecting developed countries is the significant increase in numbers of overweight people. The severity of the 'obesity epidemic' is emphasised through recognition by the World Health Organisation as one of the top ten global health problems (Kelner & Helmuth, 2003). The battle with obesity seems headed towards becoming the 'culture war of the new century,' with state legislatures discussing and/or passing bills to ban 'junk food' from schools. Trial lawyers are gearing up to file lawsuits against the food industry, much as they did against the tobacco industry (Brownell & Ludwig, 2002; Egan, 2002). It is likely the scientific community will come under increasing pressure to answer questions about why we eat so much (Del Parigi, Chen, Salbe, Reiman & Tataranni, 2003). Overeating is a maladaptive behaviour and clinical psychology may have much to offer in respect of interventions for obesity.

Overeating is persistent consumption of excess food in relation to energy a person expends, leading to weight gain and often obesity. This may be brief or short-term, such as overindulgence during festivities or holidays or longer-term. While it is known that overeating leads to being overweight and obesity has associated health risks, the aetiology of obesity remains unclear. There are possible neurological, environmental, biological, psychological and genetic influences on obesity that interact in a complex way. Numerous theories attempt explanation of causes of overeating and obesity. A popular biological theory suggests obesity develops from abnormal neuroendocrine processes involved in the control of eating behaviour and energy homeostasis. The hypothalamus is a principal component of the central nervous system for maintaining energy homeostasis (Kalra & Kalra, 2004; Woods & Seeley, 2002). Changes in the hypothalamic response to anorexigenic or orexigenic signals (signals that suppress or stimulate appetite) could result in delayed sensation of satiety.

A more psychological approach towards obesity focuses upon response to food as a reward, for example, citing the social implications of food as reward (e.g. having to clean one's plate before earning dessert) (Shizgal, Fulton, & Woodside, 2001). Blum, Cull, Braverman and Comings (1996) suggest obesity is an outcome of 'reward deficiency syndrome', similar to other reward disorders such as drug addiction, thought to be due in part to a reduction in dopamine receptors. While these psychological approaches may seem incompatible, hunger and satiation regulation probably stems from interaction between these endocrine and behavioural processes (Saper, Chou, & Elmquist, 2002).

The parallel between drug dependence, a condition more conventionally thought of as addiction, and overeating leading to obesity, may not seem immediately apparent. Addiction is a chronically relapsing disorder characterised by compulsion to seek and take addictive substances; loss of control in limiting intake of the addictive substances; and onset of a negative emotional state (e.g., anxiety, irritability) when access to the addictive substance is prevented (i.e., dependence) (Koob, Sanna, & Bloom, 1998). Individuals who are dependent on addictive substances may relapse when cravings are elicited by exposure to drug-related cues (Del Parigi et al., 2003).

Similar behaviour characterises craving for food in over-eaters (Grigson, 2002). Eating disorders tend to cluster with drug and alcohol abuse in individuals and families (Grigson, 2002). Evidence suggests common neural circuits with opioid (Kelley et al., 2002; Pelchat, 2002; Saper et al., 2002), serotoninergic (Pelchat, 2002; Saper et al., 2002) and dopaminergic (Pelchat, 2002; Saper et al., 2002, Schultz, 2002) pathways trigger food and drug rewards. It has been proposed that the dopaminergic brain circuitry underlying addictions originally developed to support eating behaviour (Wise, 1997).

Psychological theories attempt to explain overeating in various ways e.g. *psychosomatic theory* (Kaplan & Kaplan, 1957), describes eating in response to different emotional states while neglecting internal states of hunger and satiety); *externality theory* (Schacter, 1968) describes eating in response to external food cues regardless of internal states of hunger and satiety); and *restraint theory* (Herman & Polivy, 1975) concentrates on dieting as the cause of overeating. Other factors

shared by overeating and addictive disorders include denial, loss of control, preoccupation with the addictive substance, secrecy around ingestion, continuation of same behaviours despite adverse consequences, altered mood and withdrawal symptoms when addictive substances are absent or inaccessible, leading to depression, anxiety and distress.

The concept of food as an addictive substance is widely used in popular media and casual conversation and has been the subject of scientific research (e.g. Colantuoni et al., 2002; Rogers & Smit, 2000). However views on overeating and being overweight as the result of an addiction are still the subject of debate, with counterarguments that food and eating problems are illnesses, disorders or compulsions (Power, 2005). Previous research suggests some theoretical psychological models that were used to understand drug taking in addiction may also apply to overeating and obesity.

Another important point to highlight is that food is not a unitary stimulus but comprised of many different, albeit related stimuli and there may be significant variation in the salience of these stimuli between individuals. In a similar way there may be significant variation in the salience of different drugs between those who are addicted to drugs. The present study is interested in the class of stimuli comprising of palatable foods (high in sugar and/or fats), the salience of which may also vary between individuals.

Obesity and addiction are multifactorial disorders with a genetic component (Volkow & Wise, 2005) where vulnerability to addiction and variability in body mass index

(BMI) are attributed to genetic differences (Baessler et al., 2005; Kendler, Thornton & Pedersen, 2000; Uhl, Liu & Naiman, 2002). In addiction various stressors in the environment are associated with differing sensitivity to addiction-prone and addiction-resistant phenotypes (Kosten et al., 1997; Ranaldi, Bauco, McCormick, Cools & Wise, 2001). Stress may be a factor involved in obesity (Dallman, Pecoraro, & la Fleur, in press) and addiction (Kreek & Koob, 1998). Environmental factors affecting obesity may include increased variety and food supply (Raynor & Epstein, 2001); palatability, larger portions and higher energy density (Kral and Rolls, 2004); greater accessibility and reduced cost (Jeffery & Utter, 2003); decreases in manual work in the home and industry leading to a decrease in physical activity and more sedentary lifestyle (Wardle, 2005). There are multiple factors involved in development and maintenance of obesity and addiction. However detailed commentary on the factors outlined above is outside the scope of this literature review.

The first part of the review briefly outlines addiction theories, describes the neuroanatomy of reward circuitry which may be involved with addiction and overeating. Psychobiological processes of overeating will be discussed in relation to the opioid, serotonin, and dopamine systems with the aim of evaluating the link between addiction, overeating and obesity in relation to brain reward systems. Implications of this reward circuitry will be discussed with regard to addictive substances and food. The second part of the review considers whether overeating and addiction have similar psychological consequences and explores implications for treatment.

Addiction Theories

The concept of addiction has evolved in light of new scientific knowledge and political and social pressures (Akers, 1991; Orford, 1992). There are various definitions or criteria for addiction. Almost all emphasise compulsion or loss of control, discomfort of withdrawal from an addictive substance, positive psychoactive effects, tolerance and harm caused to health and personal, social and economic functioning (Akers, 1991; Altman et al., 1996; Gold, Johnson & Stennie, 1997; Gossop, 1989; Heather, 1998; James, 1997; Koob & La Moal, 1997; Robinson & Berridge, 1993; Stolerman & Jarvis, 1995). West (2001) argues medical, psychological and social harm can be caused by addiction. These factors combined with the fact that it may violate individual freedom of choice, suggest it is appropriate to be considered a disorder of motivation. Addiction typically involves initial exposure to a stimulus followed by behaviours seeking to repeat the experience. After a number of repetitions of the behaviour–stimulus sequence, addiction becomes established. The character and severity of the addiction may change over time with attempts by the sufferer to abstain or regain control. In some cases, sufferers will achieve recovery for a sustained or even permanent period. (West, 2001).

West (2001) suggests classifying addiction models and theories in terms of mechanisms and processes. Theories of addiction may be construed in terms of biological, social or psychological processes or combinations of these (e.g. Drummond, 2001; Heather, 1998; Jones, Corbin & Fromme, 2001; Littleton, 2001; McCusker & Gettings, 1997; Orford, 1992, 2001; Volkow & Fowler, 2000). A second

set of theories focuses on the effects of addictive stimuli. A dominant theme is the positive and negative reinforcing properties of addictive drugs (and other stimuli) (e.g. Bozarth, 1994). Other authors argue the positive reinforcing effects of addictive drugs are enhanced rather than diminished by repeated exposure (e.g. Robinson & Berridge, 1993, 2001).

A third set of theories focuses on individuals' susceptibility to addiction. Individuals most at risk are those susceptible to biological, psychological and social effects of a given stimulus. Genetic susceptibility is a dominant theme (Cheng, Swan, & Carmelli, 2000; Cunningham, Niehus, Malott, & Prather, 1992; Buck & Finn, 2001). A fourth set of theories explores environmental factors affecting addiction such as stressors (Breslin, Hayward & Baum, 1995), social roles (Hajema & Knibbe, 1998), social influence and opportunities and economic factors in initiation and progression of drug use (Kenkel, Mathios, & Pacula, 2001).

The fifth set of theories involves theories of recovery and relapse including conditioning (Bradizza, Stasiewicz, & Maisto, 1994) and psychosocial factors (Annis, 1991). A dominant view in this area is the transtheoretical model (Prochaska, DiClemente, & Norcross, 1992). This theory states that modification of addictive behaviours involves progression through five stages: pre-contemplation, contemplation, preparation, action, and maintenance. Individuals typically recycle through these stages several times before terrnination of the addiction. However Sutton (2001) is somewhat critical of the application of this account to addictive behaviours, highlighting problems with existing methods of measuring discrete stages

of change; and evidence for its application to substance misuse being meagre and inconsistent.

West's (2001) classifications are a useful way of summarising the literature on addiction theories and models. West did not attempt to evaluate any one theory and suggested it was unlikely addiction theories would be truly testable because of a lack of clarity in concepts and problems in ruling out competing explanations in empirical studies. There was sufficient uncertainty in what addiction theories are claiming or in the strength of the methodology of the empirical tests for theorists to escape contradiction. West (2001) suggested successful theories should generate novel hypotheses and predict circumstances in which addiction was more likely to occur, giving insights into prevention and treatment. Theories might seek to predict whether a new drug will be addictive, who among a group of children will be at risk of developing addiction if exposed to particular stimuli, or whether changes in social factors will lead to an increase in the prevalence of particular forms of dependence (West, 2001).

Bearing in mind West's criteria for a successful theory, how could theories of addiction help us to understand overeating? If we could understand why people overeat would we be able to predict who would overeat? Could we predict which type of stimuli might enhance the risk of someone becoming an overeater? Should overeating be characterised as an addiction? In trying to answer some of these questions it was important to focus on neuroanatomy and reward circuitry of addiction.

Influence of Natural Rewards and Addictive Substances on Reward Circuitry

Volkow and Wise (2005) contend that feeding and drug use involves learned habits and preferences, affected by the reinforcing properties of powerful and repetitive rewards. Erlanson-Albertsson (2005) states when food is ingested, signals are transmitted from the gastrointestinal tract to the brain stem, where energy content and taste of the food are registered. In the absence of strongly attractive taste, the hypothalamus recognises and integrates various appetite signals and their receptors in the hypothalamic nuclei. When regulation of energy balance (homeostasis) is achieved, termination of food intake occurs. With palatable food (high in sugar and/or fats) the attractive taste of the food is registered, leading to an activation of brain reward circuitry (discussed below). The reward centre has close connections to the hypothalamus, thereby influencing the hypothalamic energy homeostasis mechanisms with the end result being prolongation of food intake.

It has been argued that addictive substances activate these pathways through direct pharmacological effects on the reward circuitry. Repeated physiological stimulation of reward pathways by addictive substances may not only create response habits and stimulus preferences, but also trigger neurobiological adaptations making behaviour increasingly compulsive and leading to further loss of control over intake (e.g. Volkow & Wise, 2005). Behavioural outcome of a reward is to make subjects 'come back for more' by conditioning an association between the approach and consumption activity and the hedonic (pleasurable) feelings derived from the anticipation and attainment of the desired object (Schultz, 2001).

Various reviews have examined the nature of the reward circuitry involved with addictions (Baxter & Murray, 2002; Rolls, 2000; Schultz, 2000, 2002; Tzschentke, 2001). Reviews concluded that there were two main circuits for 'reward' behaviour: (1) the reciprocal connection between the prefrontal regions of the brain and the amygdala with the nucleus accumbens and the ventral tegmental area (VTA) being two important sites on this circuit and (2) the limbic circuit integrating the amygdala with the hypothalamus and septal nuclei. The limbic system circuits are mainly focused on regulating the basic needs of life: food, sex, and water (Augustine, 1996; Denton et al., 1999). The discussion of reward in relation to addiction is relevant since individuals who are addicted continue to pursue the focus of their addiction in spite of punishing factors inherent in addictive substances i.e., insanitary environments, negative health effects, and disapproval of society, family and peers, as well as external factors such as arrest and legal implications (Joranby, Frost-Pineda, & Gold, 2005).

Long-term abuse of addictive substances results in physiological changes in the responsiveness of reward circuitry (Goldstein & Volkow, 2002). Investigators monitored neural activity of cocaine abusers exposed to cocaine-related cues and neutral cues (Bonson et al., 2002). Cocaine abusers demonstrated increased activation of the prefrontal cortex, orbitofrontal cortex, and amygdala in response to cocaine-related cues over neutral cues, but activation of these regions positively correlated to the self reported degree of cocaine craving experienced by the subjects. This increased activity was not observed in brain areas unrelated to reward, such as the paracentral cortex, posterior thalamus, and caudate nucleus. Activation was

specific to reward and not a global change in activity due to increased arousal (Joranby, Frost-Pineda, & Gold, 2005).

These patterns of reward circuitry activation generalise across addictions (Due, Huettel, Hal, & Rubin, 2002). Due et al. (2002) found nicotine-deprived smokers demonstrated increased activation of both limbic circuits (the amygdala, hippocampus, ventral tegmental area, and thalamus) in response to smoking over non-smoking cues. Activation of this reward circuitry could be triggered by substance-related cues or images, indicating possible long-term change in this circuitry once it is exposed to a substance.

Reward and Influence of the Opioid System

Opiate drugs such as heroin, opium, and morphine have long been recognised to have high potential for addiction. Naturally occurring opioid peptides in the mammalian brain are derived from ingested protein precursors. Opiate receptors are found in various networks in the brain such as the hypothalamic regions involved in the control of food intake and in the nucleus accumbens (reward centre). One important property of opiates is to reinforce behaviour best described as 'coming back for more' (Van Ree et al., 2000; Schultz et al., 2001). Systemic injection of morphine causes rats to overeat. Various opiate antagonists can prevent overeating.

Palatable food activates the opioid reward system in the nucleus accumbens (Helm, Rada, & Hoebel, 2003; Zhang, Balmadrid, & Kelley, 2003). In humans, the opiate

receptor antagonist naloxone was found to reduce preference for palatable food, without affecting subjective ratings of hunger and satiety (Drewnowski, Krahn, Demitrack, Nairn, & Gosnell, 1992). Opiate antagonists decrease attractiveness of food without affecting its taste, i.e. subjects are still able to sense sweet taste, but it does not provide the expected reward (Yeomans & Gray, 1996; Kelley et al., 2002). It has been hypothesised that opioids may stimulate food intake because the sensation of hunger is more intense. This conclusion was drawn from experiments where rats had to press a bar to receive food (Glass, O'Hare, Cleary, Billington, & Levine, 1999). Upon intravenous infusion of opioid agonists the number of bar presses (i.e. the urgency) increased before the rats gave up (Glass et al., 1999). In contrast naloxone reduced the urgency of the food-seeking behaviour. Together these observations suggest that endogenous opioids are important to induce food-seeking behaviour.

Berridge (1996) proposed that the opioid system is involved in 'liking' for food rewards. Studies have shown that stimulation of the opioid circuits in the nucleus accumbens induces facial expressions of pleasure, while blockade of the opioid system decreases the pleasure response to sweet tastes. When eating palatable food, the hypothalamic expression of opioid peptides and receptors is increased, supporting the involvement of the opioid system in palatable food consumption.

Reward and Influence of the Serotonin System

Observations suggest that serotonin may act as a satiety signal in the control of food intake (Lawton, Wales, Hill, & Blundell, 1995). Brain serotonin levels are affected by

circulating levels of tryptophan and certain macronutrients (Halford & Blundell, 2000). Whether serotonin specifically regulates carbohydrate intake (Wurtman & Wurtman, 1995) and/or fat intake (Blundell & Lawton, 1995) is debated. Dietary carbohydrates have been shown to raise brain serotonin turnover. Depressed patients have been observed overeating carbohydrates to increase feelings of well-being (Wurtman & Wurtman, 1995). Conversely, treatment with the anorexic agent fenfluramine (Bray 2001), which may act by releasing serotonin and inhibiting serotonin reuptake, has been shown to reduce fat intake (Lawton et al., 1995). In humans, a combination of fenfluramine and phentermine (fen-phen) has been used effectively as a diet drug by increasing serotonin levels. Weintraub, Hasday, Mushlin and Lockwood (1984) published a well-controlled four-year study demonstrating the efficacy of combining diet, exercise, and behaviour modification with phentermine and fenfluramine in patients with mild obesity to effect significant weight loss with little morbidity (Weintraub M, et al., 1992a; Weintraub M., 1992b). The drug combination, Phen-Fen, gained widespread acceptance and use, often being prescribed without behaviour-modification therapy. Observations of significant cardiac and pulmonary artery damage later led to the withdrawal of fenfluramine from the market (Fisher & Schauer, 2002).

Reward and Influence of the Dopamine System

Dopamine regulates food consumption and feeding behaviour operating within the hypothalamus and nucleus accumbens mesolimbic pathway. In the hypothalamus, dopamine is associated with the initiation and length of feeding (Wang, Volkow,

Thanos, & Fowler, 2004). It has been argued that dopamine release, in the nucleus accumbens, is associated with reinforcement aspects of food and drugs of abuse. Although the mesolimbic dopamine projections from the ventral tegmental area to the nucleus accumbens have been most frequently implicated in reward function, other forebrain dopamine projections are almost certainly involved (Wise, 2004). Various addictive substances lead to a chronic alteration of dopamine levels in the brain, specifically in the nucleus accumbens, which suggests there is a reinforcement mechanism that may be activated by both food and addictive substances. Similar neural substrates may underlie both drug addiction and overeating.

Ungerstedt (1971) demonstrated that selective destruction of dopaminergic neurons with a neurotoxin caused adipsia (absence of thirst) and aphagia (loss of ability to swallow) in rats, symptoms classically related to lesions of the lateral hypothalamic areas (Stricker & Zigmond, 1984). Szczypka et al. (2001) provided further evidence of dopamine's importance showing that mice unable to synthesise dopamine die of starvation by four weeks old. Restoration of dopamine production in the dorsal striatum can reinstate normal feeding behaviour (Szczypka et al., 2001). In animal models of obesity dopamine is reduced in the tuberofundibular pathway that projects into the hypothalamus (Pijl, 2003). Treatment with a dopamine agonist activates dopamine D2 and D1 receptors and reverses the obesity.

Dopamine agonists will increase size of meals consumed and length of feeding time while long-term administration of dopamine will increase body mass and feeding behaviour in rodents (Clifton, Rusk, & Cooper, 1991; Schwartz et al., 2000). Evidence

from animal studies suggests dopamine is involved in the modulation of behavioural arousal (Grigson, 2002). In monkeys, it has been shown that just touching hidden food elicits phasic activation of dopaminergic neurons (Romo & Schultz, 1990).

Wang et al. (2004) showed two mechanisms in the brain, which regulate food intake. They examined genetically engineered, dopamine deficient mice and found they died quickly because of decreased feeding behaviours. Mice given dopamine in the striatum, but not the nucleus accumbens, were able to restart feeding. Mice given dopamine in the nucleus accumbens were able to choose between pleasant and non-pleasant foods, but did not have enough motivation to prevent them from dying from low caloric intake.

Berridge (1996) proposed that activation of the nucleus accumbens mesolimbic dopamine systems was associated with a state of incentive salience or 'wanting' of a food or drug stimulus. This was a motivational state, involving activation of the animal and approach and attentional biases toward a desired stimulus. They argued this system could be dissociated from the 'liking' system associated with opioid and GABA circuitry (an inhibitory neurotransmitter acting on the central nervous system), leading to strong irrational desires. In pathological states such as addiction, this can lead to the organism experiencing strong desires for substances such as drugs that do not actually give much pleasure, even in the absence of withdrawal syndromes. It is possible that a similar phenomenon may cause excessive desire for food in obesity.

Human studies have examined effects of dopamine in drug addiction. Cocaine blocks dopamine reuptake in the brain, increasing synaptic dopamine levels which may act

to reinforce drug use. There are at least five different types of dopamine receptors. Those found in the striatum (D2 receptors) are critical for reinforcement of drug abuse. Higher levels of dopamine D2 receptors have been found to be protective against drug abuse in previously learned drug behaviours (Stein et al., 2001; Thanos et al., 2001). The neurophysiology of reward in humans has been extensively studied in the context of drug abuse (e.g. Volkow & Fowler, 2000); less is known about how the human brain specifically processes food reward.

Studies of reward processing in humans have only recently considered eating-related stimuli. Using positron emission tomography (PET) a type of brain scan that can be used to monitor the brain's activity and detect abnormalities, and a dopamine D2 receptor ligand that selectively binds to this receptor in the striatum, Wang et al. (2001) showed the availability of dopamine D2 receptors was decreased in the dorsal striatum of obese individuals studied at rest, a neurochemical feature previously described in drug and alcohol abusers (Volkow & Fowler, 2000). The same group demonstrated dopamine is released in the dorsal striatum of healthy subjects in response to the sight and smell of food. This effect is amplified by administration of the dopamine reabsorption blocker methylphenidate, linking the dopaminergic system to the anticipation and craving of a forthcoming meal (Volkow et al.2002). In obese subjects, but not in controls, dopamine D2 receptor abundance is inversely related to body mass index (BMI), suggesting that the dopamine system is involved in excessive food intake (Wang et al., 2001; Wang et al., 2004). In addition, clinical studies showing chronic treatment with antipsychotic drugs that block dopamine D2

receptors is associated with higher risk of obesity (American Diabetes Association et al., 2004).

Wang et al. (2004) demonstrated further correlation between eating disorders and addiction. Using neurofunctional imaging they examined the brains of methamphetamine users and obese subjects, finding lower levels of striatal dopamine D2 receptors in methamphetamine users and obese subjects compared with control subjects. These findings could indicate dopamine and the level of dopamine receptors are critical to reinforcement of drug behaviour and a tendency to overeat.

Limitations of Human Studies of the Role of Dopamine in Obesity

The first study (Wang et al., 2001) did not resolve whether low levels of dopamine D2 receptors in obese individuals were related to the predisposition to compulsive eating and other addictive behaviours. Low levels of dopamine D2 receptors could be downregulation in response to chronic hyperstimulation from overeating. The former explanation was not overwhelmingly supported by epidemiological data, because the association between functional mutations of dopamine D2 and obesity was weak at best (Comings et al., 1993; Jenkinson et al., 2000; Noble et al., 1994). It rests on the belief that food intake is associated with dopamine release in humans. The second study (Volkow et al., 2002) raised the question of whether dopamine mediated anticipatory responses to the sight and smell of food would have been greater in obese individuals.

Other neuroimaging studies suggested striatal responses to food stimuli changed from the anticipatory to the consummatory phase of feeding. O'Doherty, Deichmann, Critchley and Dolan (2002) found an increase of activity in the ventral striatum (nucleus accumbens) in response to anticipation of the pleasant taste of glucose. Small, Zatorre, Dagher, Evans and Jones-Gotman (2001) observed decreased neuronal activity in the dorsal striatum in parallel with decreases in the reward value of eating chocolate. Using PET, Gautier et al. (2000) and Tataranni et al. (1999) showed no significant change after tasting and a decrease in striatal neuronal activity after ingesting a satiating amount of a liquid meal in individuals who had fasted for 36 hours. No differences between lean and obese individuals were observed, suggesting the brain's response to food reward is not likely to be confined to the striatum.

Neuroimaging studies in humans have provided evidence that the central processing of food reward consistently overlaps with the sensory and visceral responses to ingestion of food in the insula, orbitofrontal cortex, and anteromedial temporal lobe (Small, 2002). These areas receive dopaminergic projections through the mesolimbic and mesocortical pathways. This is consistent with lesion studies in animals (Saper et al., 2002). Gautier et al. (2000) observed larger decreases in brain activity responses to satiation of obese men in comparison to lean men. This evidence suggests cortical processing of food sensory cues participates in motivational aspects of eating behaviour. Cortical processing may be affected by cognitive stimuli such as food advertisements in the media. How cortical processing reinforces eating habits remains to be established (Del Parigi et al., 2003).

Other imaging studies document abnormalities in the prefrontal cortex. When food-related stimuli are shown to obese subjects, the orbitofrontal cortex is activated and cravings are reported (Wang et al., 2004). The orbitofrontal cortex and cingulate gyrus in the prefrontal cortex are implicated in motivation to feed (Rolls, 2004). These prefrontal regions could reflect a neurobiological substrate common to the drive to eat or to take addictive substances. Abnormalities in this region could enhance either drug-orientated or food-orientated behaviour, depending on individuals' habits. Preclinical studies show adaptations in the opioid system after administration of palatable foods (Levine, Kotz, & Gosnell, 2003). Neuroadaptations resulting from chronic excessive food intake are likely to be more complicated than those observed with addictive substances. Changes may also occur in neuronal circuits that modify motivation to eat, energy efficiency and metabolic thresholds (Levine et al., 2003).

Why do some individuals abuse drugs and/or lose control over food intake and others do not? Volkow et al. (1999) found drug naïve normal participants, given methamphetamine (MP) and reporting it as pleasant, had lower levels of dopamine D2 receptors than participants who reported MP as aversive. The participants had no history of drug addiction. Some participants, reporting pleasant effects of MP, had dopamine D2 levels equivalent to those participants with a history of addiction previously reported by Volkow. This means dopamine D2 receptors may indicate vulnerability to addiction but this evidence is insufficient to explain the complexities. Studies assessing whether low levels of D2 receptors affect responses to food in non-obese participants are required to determine if low D2 levels may also affect the 'liking' responses for food stimulation (Wang, Volkow & Fowler, 2002).

Empirical evidence has demonstrated possible relevance of the differences between liking and desire (or incentive salience). Saelens and Epstein (1996) compared the reinforcement value of food and non-food rewards for obese and non-obese women. Both groups gave these rewards equivalent hedonic ratings with food reward having a greater reinforcing value for the obese women (in terms of willingness to work harder for it using a computer task). This confirmed earlier studies linking obesity to increased willingness to work for food rewards in the presence of food cues (Johnson, 1974). Although most behavioural literature has assumed a positive relationship between 'liking'/reward and eating, studies have reported low striatal dopamine D2 receptor availability in association with high BMI in a group of extremely obese subjects (Wang et al., 2001; Wang et al., 2004). These authors suggest obese subjects may have blunted psychological-reward responses to food and propose that overeating in these individuals might be a way of compensating for this deficit (Wang et al., 2001; Wang et al., 2004).

Reduction in dopamine receptor activity could be a response to repeated stimulation of dopamine release, due to chronic overeating, rather than a primary cause. Other researchers have reported a positive relationship between BMI and amygdala dopamine D2 receptor binding in non-obese subjects (Yasuno et al., 2001). The distinction between liking and motivational or incentive value may be valuable in both theory and practice. The behavioural and physiological measures cited above may be useful research tools to identify when individuals with weight control problems might better be aided by behavioural (Jansen, Broekmate & Heymans, 1992) or possibly pharmacological interventions (Mela, 2001).

In summary, addiction theories emphasise compulsion, loss of control, discomfort of withdrawal, positive psychoactive effects and harm to health and personal, social and economic functioning. Testing the theories empirically is complicated due to lack of clarity of concepts and ruling out competing explanations. Behavioural and biological theories of addiction are implicated in feeding behaviour, with palatable food activating the reward centres in the brain and hypothalamus that leads to prolonged feeding. Evidence suggests common neural circuits with opioid (Kelley et al., 2002; Pelchat, 2002; Saper et al., 2002), serotoninergic (Pelchat, 2002; Saper et al., 2002) and dopaminergic (Pelchat, 2002; Saper et al., 2002, Schultz, 2002) pathways triggering food and drug rewards.

Berridge (1996) suggests that reward contains separate psychological components, corresponding roughly to 'wanting' (incentive salience attribution) and 'liking' (hedonic/aversive evaluation). These wanting and liking processes are mediated by separate neural substrates. Opioid and GABA neurotransmitter systems and substantia innominata/ventral pallidal circuits that mediate feeding appear to be most directly related to liking. Mesotelencephalic dopamine neurotransmitter systems and the central nucleus of the amygdala appear to participate more directly in 'wanting' than 'liking'.

It appears that neurobiological and behavioural theories of addiction are best placed to further our understanding of overeating, with evidence pointing to lower levels of dopamine receptors in those who are obese, suggesting dopamine is central to understanding mechanisms maintaining overeating and obesity. Using Robinson and

Berridge's incentive-sensitisation theory predictions could be made regarding the neural and psychological reward systems involved in the incentive motivation for food.

Psychological Consequences of Addiction and Overeating

Evidence suggests overeating and addiction may have similar neural substrates and overeating could be characterised as an addiction, with similar reward circuits being activated for addictive substances and natural rewards such as food. Having examined evidence for similar underlying brain mechanisms, this section explores the psychological consequences of addiction and overeating and whether these consequences are functionally similar or the same concept.

Constructing a model characterising overeating as an addiction is complicated by ambiguous psychological and psychiatric definitions of addiction and a lack of understanding about the relationship between overeating, binge eating and obesity. There has been disagreement concerning strict definitions of drug use terminology (Altman et al., 1996). Previously the 'disease' model of addiction has been seen as one of physical dependence. However this premise has been questioned because of frequency of relapse among detoxified opiate addicts suggesting tolerance and withdrawal are not the only components of addiction. The World Health Organization (WHO; 1969) initially proposed abolishing the term, but instead introduced 'drug dependence' to encompass both *physiological dependence* (characterized by tolerance and withdrawal syndrome upon removal of the drug) and *psychological dependence* (characterized by intense craving, loss of control, compulsive drug-

seeking and drug-taking) to a specific substance. ICD-10 and DSM-IV continue emphasising these concepts, although both physiological and psychological symptoms are not necessary for a diagnosis of substance dependence (WHO, 1992; APA, 1994). This is important, as it is possible for an unconscious patient to become physically dependent on opiates, if given repeatedly, without becoming psychologically dependent (O'Brien, 1996).

James, Gold and Liu (2004) suggest overeating is like a substance dependence disorder, with food as the substance and obesity as a by-product of substance dependence. An analogous condition may be lung cancer as a consequence of nicotine dependence. Obese individuals and drug users experience similar issues when attempting to reduce substance intake. They consume more of the substance than planned; demonstrate failure to cut back on consumption; lose control; continue despite adverse consequences; and are often in denial of their difficulties. Vandereycken (1990) states obesity should be viewed as an addiction. The individual is dependent on a self-damaging pleasure-giving habit with immediate gratification and shows an absent or weak resistance (no self-imposed delay between stimulus and response). Vandereycken (1990) suggests if obese people are considered food addicts their dependence on food is psychological (without the features of tolerance and physiological withdrawal symptoms).

Orford (2001a, b) advocates a broader view placing some behavioural addictions on the same level as drug addictions. An advantage of a behavioural approach to abuse of addictive substances is that it takes into account excessive behaviour that does not

involve addictive substances and situations in which repeated exposure to addictive substances is not followed by addiction. He suggests 'addiction' is a commonly understood word when referring to appetites that have become excessive and argues that 'addiction' has become overly identified with addictive substances affecting the central nervous system. Orford maintains addiction is better defined as 'excessive appetites' than 'drug dependencies' (Orford, 2001a; b).

Psychological Theories of Overeating

The idea that overeating (an excessive appetite) might be linked to emotional regulation is not new. Kaplan and Kaplan (1957) suggested emotional tensions such as fear might become conditioned with feelings of hunger for food. Feeding might be associated with relaxation or tension relief, with food acquiring extra reinforcing properties in addition to its value as a hunger-reducing reinforcer (Kaplan & Kaplan, 1957). Van Strien (2002) proposed that individuals who overeat in response to emotional states are unable to recognise hunger, satiation or other discomforts and might overeat in response to any arousal state. Individuals who frequently resort to emotional eating are considered to be less well adjusted and to exhibit distinctive personality traits (Van Strien, 2002).

Schacter (1968) suggested obese and normal weight people respond differently to internal and external food cues. The 'externality theory', a personality theory of overeating, proposed that obesity was a consequence of being more reactive to external cues such as food palatability and less responsive to internal food cues

related to satiety. Empirical evidence supported Schacter's theory showing eating was triggered by psychic states such as anxiety, fear and loneliness or external food related cues in people who were obese (Schacter, Goldman & Gordon, 1968). Normal weight subjects with a history of obesity exhibited a similar pattern of externally controlled stimulus bound behaviour (Nisbett, 1968). Nisbett, Hanson, Harris and Stair (1973) challenged Schacter's theory by suggesting externality was a consequence of episodic food restriction practised by people who were trying to lose weight. External responsiveness was also found in healthy weight individuals (Ley, 1980).

The relationship between individuals and their excessive behaviour (alcohol for excessive drinkers, food for excessive eaters) and/or a general external responsiveness (a personality characteristic) conferring some 'addiction proneness' has caused confusion. Tucker et al. (1979) found evidence for externality among alcoholics but suggested externality to alcohol cues could have preceded the alcoholism or followed it. Recent personality studies show impulsivity is a common factor in individuals who are significantly overweight, addicted to drugs or alcohol or who suffer from bulimia (Björvell, Edman, Rössner, & Schalling, 1985; Fassino et al., 2002; Ryden et al., 2003). This raises the possibility of similar personality traits being associated with excessive eating and drinking (Palme & Palme, 1999). The psychosomatic and externality theories highlight an individual's inability to recognise internal cues of hunger or satiety and possible personality types associated with overeating and addiction.

Herman and Polivy (1975) developed the 'restraint theory' based on the concept of natural weight (a 'set point' or range of body weight an individual will return to through homoeostasis). Attempts to control food intake (restrained eating) were argued to disrupt 'natural' control mechanisms such as lowering metabolic rate and arousing hunger. When self-control is undermined by disinhibitors such as alcohol, anxiety or depression, the cognitive resolve to diet may easily be abandoned. Counter regulation may occur, resulting in excessive food intake. Continuous denial of hunger may result in loss of contact with feelings of hunger and satiety. Herman and Polivy (1975) used their Restrained Eating Questionnaire and showed low restrained eaters responded to internal cues of eating, supporting Schacter's characterisation of normal weight individuals. Highly restrained normal weight individuals responded to external cues; however when the restraint was gone they overate. This suggested normal weight people could overcome the biological set point (a direct function of the number of fat cells in the body) and reduce their weight through restrained eating.

The Relationship between Overeating, Binge Eating, Eating Disorders and Obesity

Stunkard (1959) described a number of eating patterns of individuals who were obese such as '*night eating syndrome*' characterised by excessive eating in the evening, insomnia and complete avoidance of eating the following morning; '*binge eating*' where large quantities of food were consumed in a short period of time, interspersed with longer periods of normal or restrained eating; and, '*eating without satiation*' where an individual found it difficult to stop eating once intake had started. Stunkard thought eating without satiation might be related to central nervous system damage.

'Binge eating' then became the focus of research and other authors have referred to binge eating as 'compulsive eating', 'bulimia nervosa' and 'bulimarexia', 'the dietary chaos syndrome' or the 'stuffing syndrome' (Wardle & Beinart, 1981).

A binge can be simple overindulgence with little effect on someone's life or loss of control of eating as having a significant negative impact. Failure to understand the distinction between these types of binges has caused confusion, e.g. when does an episode of overeating become a binge rather than everyday overeating? Researchers investigated those who binge eat and suggest 'true' binges feature eating a large amount of food and a subjective loss of control (Fairburn, 1995). Fairburn (1986) posited that it was the individual's perceived loss of control and perceived excessive consumption rather than actual amount of food consumed that should be considered when defining the eating episode. In the overeating section of Fairburn's Eating Disorder Examination, questions are asked about 'objective' and 'subjective' binge episodes, which both involve loss of control but differ solely on whether or not an objectively large amount of food was consumed (Cooper & Fairburn, 1987).The majority of people who binge do not have an eating problem or an eating disorder. Their bingeing is occasional and does not involve huge quantities of food. A smaller number who binge frequently causing distress affecting physical health may be regarded as having an 'eating problem' (Fairburn, 1995).

Many binge eating problems fit the criteria for bulimia nervosa, binge eating disorder or anorexia. Research suggests approximately one fifth of people who seek professional treatment for obesity meet the criteria for binge eating disorder.

However, DSM-IV (APA, 1994) does not officially recognise binge eating disorder but includes it in Eating Disorder Not Otherwise Specified (EDNOS). With so much overlap the classification and description of binge eating is an ongoing process (Fairburn, 1995). The relationship between binge eating and obesity is complex and not fully understood. Does binge eating cause obesity, or does obesity cause binge eating? Is it some other mechanism? Little research has been conducted but Fairburn (1995) suggests several routes that could lead to binge eating problems.

Pathway 1:

Dieting ⟶ Anorexia Nervosa ⟶ Binge Eating ⟶ Bulimia Nervosa

Pathway 2:

Obesity ⟶ Dieting ⟶ Binge Eating

Pathway 3:

Overeating in childhood ⟶ Dieting ⟶ Binge Eating

A review of the literature (Howard & Porzelius, 1999; Guertin, 1999) concluded there was support for 'restraint theory' with evidence of severe dieting preceding binge eating and vice versa.

Another less common pathway is that individuals with binge eating problems also have problems with addictive substances such as drugs and alcohol and more generally with impulse control. The use of impulsive behaviour to release tension seems to be a key factor, with dieting playing a minor role (Fairburn, 1995). Evidence for this pathway comes from personality studies showing impulsivity is a common factor in individuals who are significantly overweight, addicted to drugs or alcohol or

who suffer from bulimia (Björvell et al., 1985; Fassino et al., 2002; Palme & Palme, 1999; Ryden et al., 2003).

Classical Conditioning and Theories of Addiction and Overeating

The 'externality theory' (Schacter, 1968) highlights internal and external responsiveness to cues, which is not dissimilar to cue-reactivity. Research has focused on reactivity to a variety of alcohol and drug related cues (Drummond, 2000). The cue reactivity paradigm has been derived primarily from the framework of classical conditioning (Tiffany, 1995a). Addiction related stimuli (the use of drug paraphernalia) are reliably associated with the administration of drugs. It is assumed these stimuli are paired with the drug unconditioned stimulus and become conditioned stimuli (CSs), which elicit conditioned responses (CRs). Addicts' responses to addiction related stimuli are considered CRs. Withdrawal models predict addicts' responses should resemble withdrawal-like states. Wikler (1948) proposed stimuli repeatedly paired with withdrawal states in addicted individuals could become CSs that elicit CRs which are physiologically withdrawal-like. Similarly, Siegel (1975) proposed environmental stimuli reliably paired with drug administration could become CSs that elicit CRs which are physiologically opposite to direct effects of the drug. Although the Wikler (1948) and Siegel (1975) models vary with regard to the nature of critical CSs, they essentially predict similar response, i.e. responses to drug-related stimuli should be withdrawal-like in nature (Carter & Tiffany, 1999).

In contrast, Stewart, de Wit and Eikelboom (1984) use a conditioned appetitive-motivational model to explain the relationship between drug relevant cues and responses. The positive-reinforcing value of drugs, in contrast to negative reinforcement of the withdrawal-based models, plays a prominent role in maintaining drug-seeking behaviour. Drug-relevant stimuli become CSs that elicit central motivational states producing physiological responses consistent with direct, positively reinforcing properties of the drug. This model predicts responses to drug-related stimuli should be consistent with a positive incentive motivational state.

There have been other accounts of conditioning effects in drug addiction including incentive salience (Robinson & Berridge, 1993) which provides an account of continued compulsion to use drugs long after withdrawal or cessation of physical dependence (O'Brien, Childress, Ehrman, & Robbins, 1998). In a meta-analysis Carter and Tiffany (1999) concluded that drug cues (regardless of drug class) induce a physiological state corresponding to arousal (increased heart rate and sweat gland activity and decreases in skin temperature). They reported that effect sizes for physiological changes were much smaller than effect size for psychological changes or reported craving.

Robinson and Berridge's theory (1993, 2001) proposed two different neural and psychological brain reward systems: a system mediating the pleasurable effects of drugs (drug 'liking') and a dopaminergic system involved in the incentive salience attribution (drug 'wanting' or craving). They proposed addiction related stimuli act as reinforcers becoming conditioned to drug taking and are associated with the

activation of dopamine transmission. Repeated administration of drugs results in the neural system becoming sensitised and addiction related stimuli are perceived as highly attractive, become especially 'wanted' (craved), grab attention, cannot be ignored and elicit approach behaviours. It is suggested these processes occur automatically and are outside awareness. Mesolimbic dopaminergic circuits are thought to have evolved because they signalled desire ('wanting') for natural rewards such as food and sex. Addictive drugs are often described as having 'hijacked' the brain's natural reward (dopamine and opiate) systems. Dopamine release triggers attention towards conditioned incentives such as the sight of cues associated with drug use.

Research using the Stroop and visual probe tasks has shown attentional bias is an important factor in the development and maintenance of drug taking behaviour (Bauer & Cox, 1998; Franken, Kroon, Wiers, & Jansen, 2000; Lubman, Peters, Mogg, Bradley, & Deakin, 2000; Mogg, Bradley, Field, & De Houwer, 2003; Rosse, Miller, Hess, Alim, & Deutsch, 1997; Ryan, 2002). Drug-related environmental cues seem to capture the attention of drug-dependent individuals. Studies suggest that drug-relevant cues interfere with ongoing cognitive processing in drug-dependent populations, but do not reveal the precise nature of processes involved in this interference effect (Mogg et al., 2003).

The sight of food promotes dopamine release or conditioned orienting to the edible stimulus (Schroeder, Binzak, & Kelley, 2001). Similar processes to those occurring in attentional biases to drug dependent cues may apply to overeating. An over-

responsive reward system may imply over-activity of the dopaminergic system to the sight of food, with individuals who are obese being perceived as food dependent and having excessive attentional bias to food cues. Franken (2003) suggests an integrated approach to craving and addiction with cognitive processes mediating between drug stimulus, the subject's response to this stimulus and subsequent behavioural response (e.g., drug use, relapse). This would bias behaviour towards eating through the cognitive processes (Franken, 2003). Several studies have investigated disruption of attention to food and body stimuli in eating disorders, such as anorexia and bulimia (Dobson & Dozois, 2004; Lee and Shafran, 2004), but further research using non-clinical samples is needed.

Classically conditioned mechanisms might play a role in binge eating (Jansen, 1998). The conditioning model of binge eating states that when cues (sight, smell and taste of binge food) are systematically paired with actual binge eating, an association is learned between the cues and the intake of binge food. It is argued that the learning of this association will be stronger when the cues are more reliable predictors of intake, and when the amount of food eaten is larger in terms of consumed kilocalories. During confrontation with predicting cues (sight, smell and taste of binge food), the binger's body will expect a binge and will prepare physiologically for excessive intake. These preparatory responses are subjectively experienced as an irresistible urge to binge or craving (Jansen, 1998).

Research into validity of the conditioning model of binge eating is scarce (Nederkoorn, Smulders, Havermans & Jansen, 2004). Empirical support is evident for

the hypothesis that self-reported craving during exposure to food cues differentiates eating disorder patients from normal controls (Bulik, Lawson, & Carter, 1996; Karhunen, Lappalainen, Tammela, Turpeinen, & Uusitupa, 1997; Staiger, Daw, & McCarthy, 2000). Exposure to food or food cues evokes physiological pre-absorptive responses in normal subjects, which prepares the body to make better use of nutrients (Mattes, 1997; Nederkoorn, Smulders, & Jansen, 2000). Some studies show increased pre-absorptive responding in binge eaters (Carter, Bulik, McIntosh, & Joyce, 2001; Karhunen et al., 1997; Karhunen, Lappalainen, Vanninen, Kuikka, & Uusitupa, 1997; Teff & Engelman, 1996; Tepper, 1992; Tuomisto et al., 1999; Vögele, & Florin, 1997). It has not yet been convincingly demonstrated that increased physiological responding during exposure is positively related to an increased self-reported urge to eat and increased food intake after the exposure, a prediction that is made by the conditioning model (Nederkoorn et al., 2004).

Addiction as an Excessive Appetite

Orford (2001b) sees the idea of excessive appetites as a 'model' rather than a 'theory'. He proposes that an addiction is a combination of operant conditioning (Skinner, 1974), with powerful emotions acting as a reinforcer with elicitation of conditioned responses to cues. This occurs within diverse social contexts leading to amplification of an initially unremarkable liking for a behaviour/substance to a strong potentially troublesome attachment (Orford, 2001b). Secondary processes (acquired emotional regulation cycles e.g. the abstinence violation effect (AVE) and consequences of conflict and dissonance) amplify the individual's state of attachment

to the excessive appetite. The stronger an attachment becomes, the more likely it is that these new processes will 'kick in', providing further incentive for consumption by serving new emotional regulating functions. The AVE sets up a new cycle of emotions and furthers appetitive drive towards excess. First described in the context of excessive alcohol use this now represents one of a small but growing number of points where literatures on excessive drinking and excessive eating overlap (Cummings, Gordon & Marlatt, 1980).

Ambivalence (a consequence of dissonance) is often present when appetitive behaviour starts. The distinction between a strong and troublesome appetite and a relatively trouble free, restrained, moderate, or normal appetitive behaviour, is the upgrading of a state of balance (between inclination and restraint) into one of conflict resulting from the harms or 'costs' associated with growing attachment to an appetitive activity. It is the *consequences* of the conflict brought about by strong attachment to appetitive behaviour that are of concern.

Consequences of Conflict

Astin (1962) and Heilizer (1964) used Miller's (1944) model of approach-avoidance competition to demonstrate appetitive conflict in animals trained to approach food and to avoid shock in the same place. Results showed two gradients: an approach tendency (inclination towards appetitive indulgence) and an avoidance tendency (restraint) with the strength of the tendencies increasing with the nearness to the goal (act of 'consumption'). Astin (1962) suggested if rewarding consequences of the

appetitive act followed soon after consumption, while punishing consequences were delayed, the inclination to approach might be stronger than the inclination to avoid the appetitive goal as the goal became closer (temporal contiguity and reinforcement). Heilizer (1964) proposed that the relative steepness of both gradients depended on the relative importance of *internal and external* cues. Appetite may be more strongly cued by external stimuli (odours or sights relating to food) which become more prominent as the act of eating food approaches, while restraining cues may be largely internal cognitive representations of past and likely future events (e.g. what an individual will look like next week), the force of which is likely to remain relatively constant as the act of eating gets nearer.

Orford (2001) suggested behavioural self-control was relatively easy when an individual was far from the goal (act of eating) but this could be lost if they found themselves in situations where they could not avoid the goal. People who overeat often describe the struggle to avoid temptation, only to find that when they give in to temptation, they 'lose control' and consume far more than they wanted to. Astin and Heilizer's idea of avoidance-approach conflict suggests that 'loss of control' is an expected consequence because balance between inclination and restraint no longer exists.

Wardle and Beinart (1981) suggested some overweight individuals who were under social and medical pressure to reduce weight and were restrained eaters, were in an approach-avoidance conflict in relation to food. They argued this lead to 'externality' (Schacter, 1968) - eating being largely determined by external cues. This supported Heilizer's model of approach-avoidance conflict that stressed the importance of

external cues being associated with the appetitive object. Wardle and Beinart (1981) proposed that an eating binge was an individual's 'capitulation' in light of the belief that the decision to diet had already been broken. They argued this was analogous to the AVE in people who drink excessively. Cummings et al. (1980) described the AVE as: cognitive dissonance about the 'relapse', affected by length of the preceding period of abstinence; degree of private or public commitment to abstinence; and self-attributions of blame, personal weakness and failure, which might predict and justify continuing the excessive behaviour. Empirical evidence from Grilo and Shiffman (1994) showed the time between binges became progressively shorter: women made more global and internal attributions following the first binge, with less consistent results for uncontrollability and guilt feelings. This suggests people who experienced stronger AVE following a lapse from self-control would be more likely to experience escalation in binge episodes.

In drawing parallels between theories of overeating and addiction, it is clear that the excessive appetites model should lead to more comparative research. Greater priority should be given to research including two or more forms of addiction, and particularly to studies combining substance and non-substance addictions (Orford, 2001b).

Is the Development of Dependence Linked to Palatable Food?

Another factor which may cause overeating to escalate and explain the increasing problem of obesity worldwide is increased availability of palatable food. Erlanson-Albertsson (2005) suggests palatable food, rich in fat and sugar, offsets normal

appetite regulation. Being attracted to palatable food has provided an evolutionary advantage, because such food can be rapidly converted into energy (Nesse & Berridge, 1997). Evidence suggests palatable food will increase food intake, i.e. the activity and expression of signals controlling appetite is balanced in favour of prolonged eating (Erlanson-Albertsson, 2005).

Erlanson-Albertsson (2005) asserts palatable food activates the opioid and dopamine reward systems described above. Opiates and dopamine, when injected into the nucleus accumbens, stimulate food intake, particularly sucrose and fat, creating a vicious circle. This behaviour, induced by stimulating the reward system, has been described as 'coming back for more' (Kelley et al., 2002). The reinforcement mechanism is similar to that induced by many drugs of abuse. Long-term over-consumption of palatable food has been compared to drug addiction (Berridge, 1996; Gosnell, 2000). Unlimited/unrestrained access to palatable food may lead to over-eating, characterised by prolongation of meals because the normally induced sensation of satiety is overridden. This is consistent with the view that palatable food may cause dependence (Gosnell & Krahn, 1992; Nestler & Aghajanian, 1997).

The attractiveness of food depends on taste, carbohydrate and fat content and on whether individuals are fasted or well fed (Cabanac & Lafrance, 1990; Berridge, 1991). The development of dependence is facilitated by factors enhancing the attractiveness of palatable food (Erlanson-Albertsson, 2005). Intermittent feeding has been shown to increase rewarding effects of food (Colantuoni et al., 2002). They demonstrated a model of sugar dependence in rats by using an intermittent feeding

protocol and concentrated sugar solutions. After a week sugar intake had increased three times. Withdrawal of sugar precipitated symptoms such as anxiety, autonomic nervous system abnormalities, changes in body temperature, teeth chatter, forepaw tremor and head shakes. Indirect evidence that the opioid system had been activated during the escalated sugar intake was provided by precipitation of withdrawal symptoms in response to naloxone (Colantuoni et al., 2002). During the development of sugar dependence there was an increase of dopamine and a decrease of acetylcholine in the nucleus accumbens; withdrawal of sugar reversed the effects (Colantuoni et al., 2002). This is in contrast to previous researchers who hypothesised there would be no tolerance and withdrawal to food. Further research is needed to establish whether humans develop dependence to palatable food with associated withdrawal when intake of palatable food ceases (Erlanson-Albertsson, 2005).

Researchers have studied the possibility of addiction to palatable food substances in humans. Chocolate is a highly palatable, preferred food (Hill & Heaton-Brown, 1994; Rozin, Levine, & Stoess, 1991). Hetherington and Macdiarmid (1993) found 'chocoholics' reported once they opened a box of chocolates, they were compelled to consume it entirely. Tuomisto et al. (1999) found chocolate 'addicts' were more aroused, reported greater cravings, experienced more negative affect and ate more chocolate, than control subjects in the presence of chocolate cues. Self-report measures of eating attitudes and behaviour, body image, and depression also confirmed a relationship between 'chocolate addiction' and problem eating. Chocolate 'addicts' showed more aberrant eating behaviours and attitudes than controls, and

were also significantly more depressed. Chocolate 'addicts' may be considered to be a parallel group with addicts generally, because they differ from controls in craving for chocolate, eating behaviour, and psychopathology (in respect of eating and affect), although this has been disputed and is discussed below.

Craving

Several reviews have been published outlining the concept of craving (Altman et al., 1996; Kozlowski & Wilkinson, 1987; Pickens & Johanson, 1992; Rankin, Hodgson, & Stockwell, 1979; Verheul, Van den Brink, & Geerlings, 1999) with little consensus on the concept of craving. It has been argued that craving should be reserved for states of extreme desire (Kozlowski and Wilkinson, 1987). However, this restriction is somewhat artificial and results in a dichotomous representation of the concept (craving is present or absent). It may be more appropriate to interpret craving as a continuous measurable state, which, in addition to pathological states, can also be present in non-addicted subjects (Franken, 2003).

The desire for addictive substances is regarded as an abnormal subjective motivational state, the result of substance dependency. The homeostatic dysregulation theory states that craving was needed to establish a new homeostasis after drug withdrawal with motivational aspects of internal states being 'simple' biological needs that could be excluded from 'the emotions' (Franken, 2003). Frijda (1986) noticed that in emotion theories, desire is not regarded as an emotion. It could be argued that desire is the emotion that accompanies approach behaviour—in the

same way fear is the emotion that accompanies avoidance behaviour (Franken, 2003). Craving could be the accompanied emotional state produced by conditioned stimuli that are associated with the reward effects of substances or behaviour.

Studies have showed unconditioned and conditioned stimuli play a central role in motivational learning and accompanying emotional components (incentive motivation). Incentives act as triggers to reinstate drug use behaviour and elicit craving with craving being a conditioned appetitive motivational state (Franken, 2003). Drug and food craving are all affective states resulting from appetitive processes (Orford, 2001). Addictive behaviour results when the brain's approach mechanism is hypersensitised (Franken et al., 2000). Craving could be the accompanied emotional state, produced by conditioned stimuli, that is associated with reward effects of substances or behaviour. Tiffany and Carter (1998) proposed that craving requires cognitive capacity, and therefore reduces capacity available to 'block' automatised behaviours such as drug use.

If chocolate 'addiction' is based on the classical conditioning paradigm, some authors have assumed that eating chocolate acts as an unconditioned stimulus for physiological and subjective responses, like hunger and craving (Lappalainen & Sjödén, 1992; Wardle, 1990). These responses can be conditioned to external cues such as the smell or sight of chocolate (Rodin, 1985). Powell, Gray and Bradley (1993) maintain drug addicts may be distinguished from non-addicts by their 'tendency to respond to the presentation of drug related cues with changes in craving,

and in other affective and physiological responses.' The same may be true of those who identify themselves as chocolate 'addicts'.

Many factors influence appearance or satisfaction of food cravings, but it seems clear cravings can occur in the absence of a homeostatic disturbance in similar ways to drugs of abuse. Food cravings are not necessarily produced by a nutritional deficit (Pelchat & Schaeffer, 2000). Flavour, rather than a pharmacological outcome, is an important factor in the (short term) satisfaction of food cravings (Michener & Rozin, 1994). Like drug cravings, food cravings are readily triggered by exposure to sight, smell, or imagery of craved foods (Fedoroff, Polivy, & Herman, 1997; Tuomisto et al., 1999). Gibson and Desmond (1999) suggest chocolate craving can be learned. They found craving for chocolate is strengthened when subjects repeatedly eat chocolate when hungry but not when repeatedly eating chocolate candy when satiated. Pelchat & Schaeffer (2000) found subjects who spent five days on a monotonous diet consisting of vanilla-flavoured nutritional supplement beverages showed no change in liking for the flavour of the beverage over the course of the study. Once they returned to their regular diets, several of them reported craving the vanilla beverage. Pelchat (2002) argues that all of the above is consistent with an acquired incentive salience view of food cravings.

Long-term food restriction has been shown to augment the rewarding effect not only of food, but also various drugs of abuse (Cabeza de Vaca & Carr, 1998; Carr, 2002). It can also provoke binge eating (Hagan, Chandler, Wauford, Rybak, & Oswald, 2003). A similar sensitising effect on the reward system by food intake restriction is

observed for alcohol (Soderpalm & Hansen, 1999). Thus food restriction may sensitise the reward system, which triggers craving not only for food (palatable food in particular), but also for addictive drugs.

Compulsion, overeating, addiction and Obsessive Compulsive Disorder (OCD)

There is considerable overlap within the OCD, overeating, and addiction literature of terms such as urge, drive, habit, impulse and compulsion, which refer to a certain 'force' through which man's cognitive self-mastery, is put to the test. In the context of obsessional compulsion the term compulsion can be defined as when a person cannot resist (except with great strain and anxiety) the urge to do or think something although he/she is aware of the absurdity and is distressed by its existence, but experiences it as enforced upon him/her against his/her will (Vandereycken, 1990).

The compulsive nature of addiction has been compared to OCD (Modell, Glaser, Cyr, & Mountz, 1992) as the severity of compulsivity and obsessionality in drug addiction is at a comparable level to that seen in OCD (Friedman, Dar, & Shilony, 2000). The two disorders are clearly clinically distinct with commonalities in clinical features (i.e. difficulty inhibiting specific thoughts and inappropriate behaviours). This raises the possibility of a shared neurobiological circuitry underpinning the compulsive behaviour seen in both disorders. Neuropsychological studies in OCD and addiction have shown dysfunction in the anterior cingulate cortex and orbitofrontal cortex, especially during symptom provocation (e.g. presentation of drug/OCD-related cues) (Barnett et al., 1999; Bechara & Damasio 2002; Childress et al., 1999; Maruff, Purcell,

& Pantelis, 2002; Volkow & Fowler, 2000). In non-pathological states, these two highly interconnected cortical regions are critically involved in assessing the future consequences of one's own actions (response selection) and placing inhibitory control over inappropriate behaviours (response inhibition) (Bechara & Damasio, 2002; Elliott, Friston, & Dolan, 2000; Fan, Flombaum, McCandliss, Thomas, & Posner, 2003; Kiehl, Liddle, & Hopfinger, 2000).

Lubman, Yücel and Pantelis (2004) propose that in chronically addicted individuals, maladaptive behaviours and high relapse rates can be conceptualised as being 'compulsive' as a result of dysfunction within inhibitory brain circuitry, particularly during symptomatic states. Affected individuals cannot make adaptive behavioural changes despite explicit knowledge of the consequences of their actions. This model may help to explain why some addicts lose control over their drug use, and engage in repetitive self-destructive patterns of drug seeking and drug-taking that takes place at the expense of other important activities (Lubman et al., 2004).

Davis, Levitan, Muglia, Bewell and Kennedy (2004) demonstrated overeating is not simply a passive response to salient environmental triggers and powerful physiological drives; it is also about making choices. Decision-making deficits were found in patients with ventromedial prefrontal cortex lesions and in those with substance dependence. These impairments reflect an inability to advantageously assess future consequences and choosing immediate rewards in the face of future long-term negative consequences. The research was extended to overeating and suggests cortical and subcortical processes, which regulate one's ability to inhibit

short-term rewards when the long-term consequences are negative, might also influence eating behaviours.

Obsessive thoughts and feelings of loss-of-control or compulsion-to-consume are salient characteristics of food and drug cravings (O'Brien, et al., 1998; Tuomisto et al., 1999; Volkow & Fowler, 2000). Activity in the orbitofrontal cortex is implicated in the pathology of OCD (Insel, 1992), receives projections from reward circuits (e.g., nucleus accumbens and ventral tegmental area) and is associated with cocaine and alcohol craving (Volkow & Fowler, 2000). While there is no direct evidence linking orbitofrontal activity with food cravings, there are anatomical and behavioural data consistent with the premise. The orbitofrontal cortex is activated by two major sensory components of flavour, gustatory (Baylis, Rolls, & Baylis, 1995) and olfactory stimuli (Wiesman et al., 2001).

There is also higher-than-expected co-occurrence of obsessive–compulsive behaviour and the major eating disorders, anorexia and bulimia (Bellodi et al., 2001; Pasquale, Sciuto, Cocchi, Ronchi, & Bellodi, 1994). Individuals with a diagnosis of OCD report higher-than-normal levels of carbohydrate craving (O'Rourke et al., 1994). It has been noted that OCD and eating disorders can be treated with serotonergic drugs (O'Rourke et al., 1994).

How is Overeating Different from an Addiction?

Vandereycken (1990) suggests it may be more problematic to demonstrate tolerance to eating than to addictive substances because of the variety of food. One might interpret increasing food intake as an indication of tolerance but many people do not experience the act of overeating as pleasurable or satisfying. DSM-IV (APA, 1994) does not recognise food as a substance of abuse. Humans are physiologically dependent on food for survival so one could argue that everyone suffers from tolerance to, and withdrawal from, food (two of three conditions necessary for a diagnosis of substance dependence). Few studies have been able to demonstrate tolerance and withdrawal from food in humans. It is likely dependence is psychological rather than physical. Individuals vary in their vulnerability to addiction due to factors such as socio-economic circumstances and inherited traits [e.g., genetic predisposition to alcoholism (Cloninger, 1987). Substances vary in their addictive potential according to their capacity to produce positive psychoactive effects and neuroadaptive changes that occur with continued substance use (including, tolerance and withdrawal effects). Wilson (2002) argues that starvation, or withdrawal of food produces physiological and psychological symptoms including irritability, distractibility, preoccupation with food and weight and binge eating but argues these symptoms are not equivalent to drug withdrawal effects.

There is lack of agreement as to what substances (and activities) are 'addictive'. Claims that caffeine, nicotine, and cocaine are addictive have all been disputed (Akers, 1991; Hughes, 1993; James, 1997; Robinson & Pritchard, 1992; Robinson &

Berridge, 1993; Stolerman & Jarvis, 1995; Warburton, 1989). There is a tendency to treat addiction and addictiveness as 'all-or-nothing' phenomena (Rogers & Smit, 2000). Implicit in the concept of the alcohol dependence syndrome (Edwards & Gross, 1976) is the view that alcohol-related problems constitute a dimension which is conceptually separate from dependence. Alcohol dependence and alcohol-related problems are seen as lying on continua of severity rather than 'all-or-nothing' phenomena (Edwards, Gross, Keller, Moser, & Room, 1977).

The pharmacological hypothesis of chocolate addiction suggests there are psychoactive or mood-altering compounds (phenylethylamine, theobromine) in cocoa containing products which are used to argue that chocolate is addictive (Rogers & Smit, 2000). This speculation has continued in the light of what little is known about the concentrations of potentially psychoactive compounds in products that are most widely eaten, and their likely effects on the brain when administered orally. The evidence showed such constituents play little or no role in chocolate 'addiction' and craving, or indeed in influencing appetite or liking for chocolate (Gibson & Desmond, 1999; Max, 1989; Michener & Rozin, 1994; Rogers & Smit, 2000; Rozin et al., 1991; Tarka, 1982). Chocolate can contain relatively high concentrations of theobrornine but this is a relatively weak central nervous system stimulant and does not have strong subjective effects (Mumford et al., 1994).

Results of Weingarten and Elston (1991) and Hetherington and Macdiarmid (1993) cast doubt on the pharmacological hypothesis of chocolate addiction. The majority of subjects reported that when they were craving chocolate, there was no substitute to

be found; even foods which had the same psychoactive properties appeared not to satisfy the cravings. In accordance with this, Rozin et al. (1991) found little evidence to suggest a pharmacological link to chocolate liking or addiction.

Eating does not appear to produce the powerful neuroadaptive effects, including associated withdrawal effects, which are central to drug addiction. It has been argued that labelling the *perceived* over consumption of chocolate as 'chocolate addiction' (Hetherington & Macdairmid, 1993; Tuomisto et al., 1999), even if this is associated with high levels of comfort (emotional) eating and somewhat unstable eating patterns, risks trivialising serious addictions. However the author would dispute this, as health complications associated with overeating and obesity are no less serious than those associated with prolonged drug use. The balance of evidence is against the hypothesis of food craving and food 'addiction' being linked specifically to the effects of eating on the activity of brain serotonergic or endogenous opioid systems (Rogers & Smit, 2000).

Rogers and Smit (2000) investigated self-reported food craving and 'addiction' (and 'moreishness'). They gave a prominent role to psychological processes of restraint, ambivalence and attribution (Gold et al., 1997; Levison et al., 1983; Orford, 2001a, b; Wise, 1997; Woods, 1991), operating together with normal mechanisms of appetite control, hedonic effects of certain foods, and socially and culturally determined perceptions of the appropriate intakes and uses of those foods. They suggest that ambivalence about chocolate (e.g., 'nice but naughty') arises from the attitude that

while this is a highly palatable food, it is not a staple component of the diet but instead a 'treat' that should be eaten with restraint.

Attempts to restrict intake may cause desire for chocolate to become more salient, and this experience is then labelled as a craving (Rogers & Smit, 2000). This, together with the need to provide a reason for why resisting eating chocolate is difficult and sometimes fails, can, in turn, lead the individual to an explanation in terms of addiction (e.g., 'chocoholism'). According to this view, chocolate is the most frequently craved food because it is the food that people most often try to resist eating. In contrast to craving, 'moreishness' (causing a desire for more) occurs during rather than preceding an eating episode. Restraint is again an essential feature, because 'moreishness' is experienced when the eater attempts to limit consumption before appetite for the food has been satisfied (Rogers & Smit, 2000).

Implications for Treatment and Future Research

Manipulation of dopamine function might be beneficial in the treatment of obesity. Historically appetite suppressants contained amphetamines but this is now contraindicated because of the addictive and psychoactive nature of the drug concerned. Strategies to enhance dopamine function could include behaviour interventions such as exercise. Studies of physical activity in obesity have found that physical activity decreases as percentage of excess body weight increases (Tryon, Goldberg, & Morrison, 1992). Dopamine is an important neurotransmitter in the mediation of reinforcement, directly involved in motor control in the striatum and the

key to the mechanisms underlying increases and decreases in physical activity. Animal studies have shown that release of dopamine is influenced by exercise (Wang et al., 2002). In general, a better understanding of brain regions and mechanisms associated with addictive behaviour may help to identify groups who are vulnerable or have difficulties controlling use, aid in the development of strategies that reduce the addictive power of drugs, and suggest new avenues for treatment and prevention.

There may be merit in studying individuals with Prader-Willi Syndrome (PWS), a Neurogenetic multisystem disorders characterised by infantile hypotonia, mental retardation, short stature, hypogonadism, dysmorphic features, and hyperphagia with a high risk of obesity (Clarke et al., 2002). This may help us to understand the aetiology of overeating leading to being overweight and obesity

Conclusions

Obesity is a multi-factorial disorder with genetic, psychological, environmental, biological and neuropsychological influences. There has been a significant increase in the numbers of people who are overweight and it is likely scientists will be under increasing pressure to try and explain why. One cause is likely to be maladaptive overeating behaviour.

Common features of overeating and drug use include psychoactive (mood) effects, the external cue (environmental) control of appetites, and the cognitive factors of restraint, ambivalence, and attribution (Gold et al., 1997; Levison et al., 1983; Orford,

2001a, b; Wise, 1997; Woods, 1991). There is also a wide overlap of the brain mechanisms underlying the rewarding effects of foods and drugs (Berridge, 1996; Robinson & Berridge, 1993) and foods are, like drugs of abuse, strong reinforcers.

Low levels of dopamine receptors are present in people who are obese and people who are addicted to drugs. It is unclear whether lower levels of receptors in obese individuals are related to the predisposition to compulsive eating and other addictive behaviours. Psychological consequences of this anomaly may be increased levels of desire for food or drugs (Berridge, 1996; Robinson & Berridge, 1993, 2001; Wang et al., 2001; Wang et al., 2004), and/or a deficit in inhibitory control (Lubman et al., 2004). Dopamine receptors may be relevant for vulnerability to addiction but are not sufficient to explain the complexities. Studies assessing whether low levels of D2 receptors affect responses to food in non-obese participants are required to determine if low D2 levels may also affect the liking responses for food stimulation (Wang et al., 2002).

Characterising overeating as an addiction is difficult because of ambiguity in psychological and psychiatric definitions of addiction and a lack of understanding about relationships between overeating, binge eating and obesity. The author agrees with Orford (2001a, b) that the word 'addiction' has become overly identified with drugs that have an effect on the central nervous system. A broader view is advocated placing some behavioural addictions on the same level as drug addictions, as the consequences of overeating are no less serious than the consequences of drug taking.

Triggers and maintaining factors in overeating are complex with overlaps in theories and terminology and a lack of clarity regarding definitions. Conditioning (classical/operant) underpins a number of theories of addiction (Robinson & Berridge, 1993, 2001; Siegel, 1975; Stewart et al., 1984; Wikler, 1948) and has been linked to theories of overeating with some evidence of classical conditioning in binge eating. Palatable food is also implicated in increased food intake, and long-term over-consumption of palatable food has been compared to drug addiction (Berridge, 1996; Gosnell, 2000). Free access to palatable food may lead to over-eating because normally induced sensations of satiety are overridden. This is consistent with the view that palatable food may cause dependence (Gosnell & Krahn, 1992; Nestler & Aghajanian, 1997).

Although it may be reasonable to label compulsive eating seen in bulimia and binge-eating disorder as food addiction, Rogers and Smit (2000) suggest that vast majority of cases of self-reported food 'addiction', 'chocoholism', and food craving should not be viewed this way. In drawing parallels between theories of overeating and addiction, it is clear more comparative research is needed. Greater priority should be given to research that includes two or more forms of addiction, and particularly to studies combining substance and non-substance addictions (Orford, 2001b).

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UNIVERSITY OF SOUTHAMPTON

Attentional and approach biases for food related stimuli in individuals who are overweight

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Tanya Griffiths

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School of Psychology

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Abstract

The study investigated if being overweight is related to enhanced attentional and approach biases for food related stimuli in overweight individuals. Relationships between being overweight, attentional bias and stimulus valence were also explored. Participants who were healthy weight (n=26) and overweight (n=36) took part in a single session in which their attentional and evaluative responses to food-related and matched control pictures were recorded. Attentional and approach biases for food-related cues were assessed on the visual probe and stimulus response compatibility tasks, respectively. Participants also rated the stimuli for pleasantness.

Participants of a healthy weight showed a greater attentional bias for food cues than overweight participants, not differing significantly in approach bias or subjective rating of the food cues compared to overweight participants. Explicit ratings of food were significantly correlated with measures of approach bias and attentional bias. The results did not support the hypothesis that participants who are overweight have an over-responsive reward system for processing food-related pictorial stimuli. Further research is needed to understand the cognitive mechanisms involved in processing of appetitive stimuli.

INTRODUCTION

Attentional Bias and Eating Disorders

Cognitive biases for eating-, shape- and weight-related stimuli have been demonstrated in eating disorders, such as anorexia and bulimia (Ainsworth, Waller, & Kennedy, 2002; Faunce, 2002; Huon, 1995; Williamson, Muller, Reas, & Thaw, 1999). These biases have been hypothesised to arise from 'maladaptive schemata' (Vitousek & Hollon, 1990) and may contribute to the maintenance of eating disorders. Lee and Shafran (2004) reviewed studies of attentional biases in eating disorders. Individuals with eating disorders take longer to name the colour of eating-, shape-, and weight-related words than other words (Channon, Helmsley, & De Silva, 1988; Cooper, Anastadiades, & Fairburn, 1992; Cooper & Fairburn, 1993; Fairburn, Cooper, Cooper, McKenna, & Anastasiades, 1991; Davidson & Wright, 2002; Long, Hinton, & Gillespie, 1994; McManus, Waller, & Chadwick, 1996). Attentional biases are not limited to clinical groups and have been noted in various samples including restrained eaters, dieters, and women with high drive for thinness (Green & McKenna, 1993; Green & Rogers, 1993; Perpina, Hemsley, Treasure, & De Silva, 1993). Debate has occurred about cognitive processes which contribute to interference in the Stroop task and the extent to which they reflect attentional rather than response biases.

The dot probe task is a widely used alternative but few studies have examined information processing biases using the visual probe in clinical eating disorders. The visual probe task is sensitive to clinical severity, with biases noted in individuals with

eating disorders (Reiger et al., 1998) but not in restrained eaters (Boon, Vogelzang & Jansen, 2000). Further work is needed with this alternative technique, placing greater emphasis on increasing ecological validity of stimuli used.

Attentional Bias and Addiction-related Disorders

Robinson and Berridge (1993; 2001) propose two separate neural and psychological brain reward systems are involved in the incentive motivation for drugs: a system mediating the pleasurable effects of drugs (drug 'liking') and a system involved in the incentive salience attribution (drug 'wanting' or craving). Through conditioning, repeated administration of drugs results in the dopaminergic 'reward' system becoming sensitised. Incentive-sensitization theory of addiction proposes a reward system that is over-responsive to drugs (Robinson & Berridge, 1993). The drug-related stimuli are perceived as highly attractive, become especially 'wanted' (craved), grab attention, cannot be ignored and elicit approach behaviours. These processes may occur automatically and outside an individual's awareness. The mesolimbic dopaminergic circuits mediating this system may have evolved because they signalled desire ('wanting') for natural rewards such as food. Addictive drugs are often described to have 'hijacked' the brain's natural reward (dopamine and opiate) systems. Dopamine release may trigger attention towards conditioned incentives, such as the sight of cues associated with drug use, as well as cues associated with natural rewards such as food.

Biases in selective attention and attractiveness of drug-related stimuli are important factors in development and maintenance of drug taking behaviour. Modified Stroop and visual probe tasks have shown selective attentional biases in addiction-related disorders including alcohol dependence (Bauer & Cox, 1998; Ryan, 2002), nicotine dependence (Mogg, Bradley, Field & De Houwer, 2003), cocaine dependence (Rosse, Miller, Hess, Alim & Deutsch, 1993) and opiate dependence (Franken, Kroon, Weirs & Jansen, 2000; Lubman, Peters, Mogg, Bradley, & Deakin, 2000). Drug-related environmental cues appear to capture the attention of drug-dependent individuals.

The sight of food promotes dopamine release or conditioned orienting to edible stimuli (Schroeder, Binzak, & Kelley, 2001). Wang, Volkow and Fowler (2002) found obese volunteers had similar brain dopaminergic abnormalities (lower levels of dopamine receptors) as cocaine dependent individuals. In response to the sight of food over-activity of the dopaminergic system may occur in some individuals, which may increase their desire for food making them prone to overeating and becoming overweight. This over-responsive reward system may result in excessive attentional bias to food cues. Franken (2003) suggests an integrated approach to craving and addiction in which cognitive processes, e.g. attentional biases mediate between the motivationally salient stimulus (e.g. drug cue) and subsequent behavioural response (e.g., drug use, relapse). An increased attentional bias to food might bias behaviour towards eating. It could also be a form of hypervigilance, akin to anxiety facilitating active avoidance of the food related stimuli.

Hunger may affect attentional bias for food- and eating-related cues as suggested by studies showing hunger-related Stroop interference effects for food-, body shape- and weight-words. Attentional bias for food stimuli was shown in healthy controls following relatively short periods of food deprivation (Mogg, Bradley, Hyare, & Lee, 1998; Stewart & Samoulek, 1997). Chronic dietary restriction produced longer Stroop latencies for food relative to control words (Stewart & Samoulek, 1997). The effect of chronic deprivation may be specific to the processing of food-words and not body size words (Channon & Hayward, 1990). Impairments in colour naming of food-related words have been shown to vary according to subjects' self-reported hunger levels (Green, Elliman, & Rogers, 1996). Placanica, Faunce, and Soames-Job (2000) found that fasting increased attentional bias towards high calorie food-words for all subjects. Further research is needed to clarify the role of hunger and eating disorder pathology in determining food, body shape and weight related attentional biases.

Is There an Attentional Bias Towards Food-Related Cues in Overweight Individuals?

Attentional biases for food cues are likely to be relevant to eating behaviour. Very few studies have investigated attentional biases to food stimuli in individuals who are healthy weight or overweight. In the present study participants who were healthy weight and overweight were recruited to investigate information-processing biases to food stimuli. A modified version of the visual probe task was used to assess attentional bias. In pictorial versions (Lubman et al., 2000; Bradley, Mogg, Wright, & Field, 2003) two pictures were presented briefly simultaneously side by side on each trial (e.g. an experimental stimulus and a control stimulus). In the present study the

experimental stimuli were food pictures while control stimuli were non-food pictures. Immediately after the pictures disappeared, a probe stimulus (e.g. a small arrow) appeared in the location of one of the pictures, and participants were required to press a key as quickly as possible in response to the probe. The rationale for the task is that people responded faster to stimuli that appeared in an attended, rather than unattended, region of a visual display (e.g. Posner, Snyder, & Davidson, 1980). People's attention to the pictures can be inferred from their response times (RTs) to the probes.

The visual probe task gives a snapshot view of attentional biases with Reaction Time (RT) measures being obtained after the offset of the display of the pictures (i.e. when the probe appears). When the picture pairs are shown briefly (e.g. 500 ms or less), the RT bias measure is more likely to reflect *initial shifts* in attention. When the picture pairs are presented for longer durations (e.g. 2000 ms), there is greater opportunity for attention to shift repeatedly between the pictures while they are displayed, so the bias measure is more likely to reflect *maintained* attention (Mogg et al., 2003).

Recent theories of selective attention highlight the distinction between different processes involved in the initial shift versus maintenance of attention (LaBerge, 1995). Bradley et al. (2003) found using the visual probe task that smokers showed greater vigilance for smoking-related pictures than non-smokers when the pictures were presented for 2000 ms. This suggests addiction-related biases may operate in the maintenance of attention. One aim of the present study was to investigate whether participants who were overweight showed biases in initial orienting to food

stimuli (will react faster to probes presented in the location of food stimuli after 500ms), and maintained attention to food cues (will react faster to probes presented in the location of food pictures at 2000ms).

It was predicted that participants who were overweight would show a greater bias in orienting attention to food related stimuli than control participants i.e. they should react more quickly to probes presented in the same location of food stimuli than control stimuli. Individuals who were overweight were predicted to have an ambivalent response to food cues. They might show an automatic initial orienting to food due to an overactive dopamine response (Robinson & Berridge, 2001) but then use effortful strategies to try and resist attending to the food cues. If so, overweight participants may show a pattern of initial hypervigilance followed by avoidance of food stimuli that would be reflected by biased initial orienting to food stimuli at 500ms (i.e. react faster to probes presented in the location of food stimuli), and later avoidance at 2000ms (i.e. react faster to probes presented in the location of control pictures).

Do Overweight Individuals Show a Positive Subjective Evaluation Bias and an Approach Bias for Food-related Cues?

To assess biases in explicit evaluation of food cues, a rating task was used. Participants evaluated subjective pleasantness of the food stimuli. To assess approach bias a modified version of a task used by De Houwer, Crombez, Baeyens and Hermans (2001) was used providing an implicit measure of the motivational valence of the stimuli. Previous research has shown people categorise positively

valenced stimuli faster if the appropriate categorisation response is an approach movement, rather than an avoidance movement, but the reverse is true for negatively valenced stimuli (Chen & Bargh, 1999; Neumann & Strack, 2000).

De Houwer et al. (2001) obtained similar findings using a task in which participants made symbolic approach and avoidance movements to positive and negative words by moving a manikin figure towards or away from the stimuli. The task used is termed the 'stimulus response compatibility' (SRC) task, because responses to positive stimuli are compatible with behavioural tendencies to approach that stimulus, whereas responses to negative stimuli are compatible with behavioural tendencies to avoid that stimulus (De Houwer, 2003). In the SRC task, participants were asked to decide whether or not each picture was related to food and to respond by moving a manikin figure either towards or away from each picture. Previous research has shown such tasks are sensitive to the motivational or affective valence of the stimuli. It was expected that if participants evaluated the food-related pictures as positive they should be faster to make approach than avoidance movements to those pictures relative to the control pictures. Conversely, if they evaluated the food-related pictures as negative the opposite pattern of results would be seen.

It was hypothesised that, in comparison with healthy weight individuals, participants who were overweight would be faster to make an approach than avoidance response to food-related stimuli than control stimuli as if the food cues were motivationally positive. An advantage of the SRC task is that it does not require participants to make explicit judgements of the affective valence of the stimuli, so it may be less

susceptible to demand effects that are associated commonly with direct measures of stimulus valence, such as pleasantness ratings.

Are Attentional Biases for Food-related Cues and their Perceived Pleasantness Linked?

Another aim of the research was to investigate the relationship between attentional bias and the motivational and affective valence of the food stimuli. According to incentive models of addiction, attentional biases for drug-related cues should be associated closely with the perceived attractiveness of those cues because a common mechanism underlies both, namely, a dopamine-based incentive-sensitisation system (Robinson & Berridge, 1993, 2001). Recent theories of emotion propose the valence of a stimulus is important in determining its capacity to capture attention. Lang, Davis and Ohman (2000) proposed that stimuli with high affective valence (either highly pleasant or unpleasant) are more likely to attract attentional processing than stimuli with mild affective valence.

Mogg et al. (2003) examined the relationship between the perceived valence of smoking-related pictures and their effects on attentional orienting. Smokers were faster to detect probes that replaced smoking-related than control pictures, consistent with an attentional bias for smoking-related cues. Smokers showed greater preferences for smoking-related than control pictures, compared with non-smokers, on both the subjective (explicit) and cognitive-experimental (implicit) indices of stimulus valence. These results demonstrated smokers show biased attentional

orientating to smoking cues, which was related to craving and the affective and motivational valence of the stimuli. The present study aimed to investigate whether individuals who were overweight showed biased attention to food cues and whether this would be linked to the affective and motivational valence of the stimuli.

In summary, the present study investigated attentional biases for food stimuli in individuals who were overweight. This might be reflected by biased initial orienting to food stimuli (reacting faster to probes presented in the location of food stimuli after 500ms), and maintained attention to food stimuli (reacting faster to probes presented in the location of food pictures at 2000ms). A further aim of the study was to examine whether they showed more positive subjective evaluation of food cues on a rating task and an approach bias for food cues on the SRC task. The relationship between the predicted attentional biases and direct and indirect measures of the affective and motivational valence of the pictorial stimuli (as measured by subjective rating and SRC tasks) was also examined.

METHOD

Design

The study employed a mixed design with one between-subjects variable (healthy weight group and an overweight group). The study aimed to match the two groups on variables such as age, gender and educational level.

In the visual probe task, the dependent variable was the mean reaction time with group (healthy weight, overweight) as the between-subjects variable, and probe position (probe in same versus different location to food picture) and picture duration (500ms, 2000ms) as the within-subject variables. In the picture-rating task, the dependent variable was the mean pleasantness ratings, with group (healthy weight, overweight) as the between-subjects, variable and picture type (food-related, control) as the within-subjects variable. In the SRC task, the dependent variable was the mean reaction time with group (healthy weight, overweight) as the between-subjects variable, and assignment type [(1) approach food-related and avoid non-food (control) pictures versus (2) avoid food-related pictures and approach non-food (control) pictures] as the within-subjects variable. Order of the explicit rating task and SRC task was counterbalanced, and the order of 'assignments' within the SRC task was counterbalanced.

Participants

Data were collected from 72 participants, recruited from the local community in Southampton and surrounding areas by poster advertisements in the local media and community, local radio and slimming groups. Staff and students from the University of Southampton were recruited through an online booking system and by poster advertisements. All participants were male or female and aged between 18 and 68 years. All participants spoke fluent English and had visual acuity within the normal range.

Following the World Health Organisation (1995) guidelines participants with a BMI of between 18.5 and 25 were allocated to the healthy weight group. Participants with a BMI of over 25 were allocated to the overweight group. Following the screening process, participants with a BMI of less than 18.5 and participants who were vegetarian/vegan were excluded (14% of the sample). In the healthy weight group (n=26), there were 7 males and 19 females, with a mean age of 28.9 years (SD = 14.5). In the overweight group (n=36), there were 7 males and 29 females, with a mean age of 45.7 years (SD = 15.4).

Two further participants were excluded from the visual probe task because their overall error rates were extreme outliers, i.e. percentage error > 20%.

Six participants were excluded from the SRC task due to having outlying error rates greater than 40%.

Materials

Experimental Tasks

The computer tasks were presented using Presentation 9.81software on a Toshiba SPA10 portable computer attached to a purpose built response box.

Pictorial Stimuli

The pictorial stimuli consisted of 20 colour photographs of food-related pictures. Each was paired with a photograph of a non-food item matched for colour and shape but lacking any food-related cues (See Appendix A). These photographs were piloted prior to the study (see Appendix B). An additional 20 pairs of pictures (non food-related) were used as fillers, and there were 10 food-control pairs used for practice and buffer trials. The pictures were 1600 x 1200 pixels stored in jpeg format.

Questionnaires

Dutch Eating Behaviours Questionnaire (DEBQ: Van Strien, Frijters, Bergers, & Defares, 1986)

This is a 33-item questionnaire used to assess the structure of an individual's eating behaviour. There are three scales, restrained eating (DEBQ-E), external eating (DEBQ-D) and emotional eating (DEBQ-A). The emotional eating scale is two-dimensional and gives scores for eating in response to diffuse emotions such as feeling lonely or bored (DEBQ-B) eating in response to clearly labelled emotions such as anger or irritation DEBQ-C). All the sub scales are reasonably reliable with Cronbach's Alpha coefficients greater than 0.80 (Van Strien, Frijters, Bergers & Defares, 1986b).

Eating Attitudes Test (EAT-26: Garner, Olmstead, Bohr, & Garfinkel, 1982)

The EAT-26 was used to assess current attitudes and behaviours in order to establish that the groups did not contain people with a clinical eating disorder. A cut-off of 20 is believed to reliably identify "maladaptive eating" consistent with anorexic or bulimic disorders. The EAT-26 has good validity (r = .79; Mintz & O'Halloran, 2000) and high internal reliability (ά =.90; Garner et al. 1982).

Items from the Dieting Scale include: 'I feel extremely guilty after eating; I like my stomach to be empty'. Items from the Bulimia and Food Preoccupation Scale include: 'I have gone on eating binges where I feel that I may not be able to stop; I vomit after I have eaten'. Items from the Oral Control Scale: 'I avoid eating when I am hungry. I feel that others pressure me to eat'.

Satiety Labelled Intensity Magnitude scale (SLIM: Cardello, Shutz, Lesher & Merrill, 2005)

The Satiety Labelled Intensity Magnitude (SLIM) scale was used for measuring perceived hunger and satiety and has an average reliability coefficient of 0.90 (Cardello et al, 2005). This scale was used to assess an individual's perceived satiety.

Hunger Scales (Grand, 1968)

The hunger scale was used to assess subjective level of hunger. The scale consisted of 4 hunger indices:

1. Time since last eating (number of hours, estimated to nearest 15 mins);
2. Subjective hunger (rated on 7 point scale: 1 = not hungry at all, 7 = extremely hungry);
3. Subject's estimate of the amount of their favourite food that they would be able to eat at the time of testing (rated on 6 point scale: 1 = none at all, 6 = as much as I could get);
4. Estimate of time until next expected meal (estimated to nearest 15 min).

Questions about recent food intake (See Appendix C)

This is an unpublished 4-item questionnaire used to obtain further information about eating habits and to see whether participants subjectively felt they had eaten enough before taking part.

Procedure

Ethics Committee approval was obtained from the School of Psychology Ethics Committee. Each participant was tested individually; the test session took approximately one hour and took place in a dimly-lit quiet room at the University or at the participant's home. Prior to testing, the participants were asked eat no more or less than they would usually eat. Verbal instructions for all tasks were given following a script.

Participants were given an opportunity to read the information sheet (see Appendix D), provide informed consent (see Appendix E) and then complete the SLIM scale (Satiety Labelled Intensity Magnitude scale; Cardello, Shutz, Lesher, & Merrill, 2005) and Hunger Scales (Grand, 1968). Participants were seated at a desk at a distance of 110cm from the screen. To check the readability of instructions participants were asked what they could see on the screen. Once the visual probe task was loaded participants were instructed to focus on a cross appearing on the screen followed by a picture pair and an arrow (pointing up or down) and to press the corresponding arrow key on the response box as quickly and as accurately as possible. They were reminded that they could ask questions at any time. They were advised to put the thumb of their left hand on the up button of the response box and the thumb of their right hand on the down button before starting.

The visual probe task was modeled on that used by Bradley et al. (2003, Experiment 2). Each trial started with a central fixation cross shown for 500ms which was replaced by a pair of pictures, side by side for a designated length of time (500 or 2000 ms). Immediately after the offset of the picture pair, a probe was presented in the position of one of the preceding pictures, until the participant gave a manual response. The probe was either an up or down arrow. The inter-trial interval varied between 500ms and 1500ms.

There were 12 practice trials followed by a short break and two buffer trials. There were 240 trials (160 critical trials and 80 filler trials). There was a short break after

120 trials. The picture pairs were presented for 500ms in half of the critical trials and for 2000ms in the remaining 80 critical trials. During the critical trials, each of the 20 food-related picture pairs were presented four times. Each food-related picture appeared twice on the left side of the screen and twice on the right. The probe appeared in the location of either the food picture or control picture with equal frequency. The 20 filler picture pairs were presented twice each. Critical and filler trials were presented in a new random order for each participant. Each picture was 90 mm high by 120 mm wide, the distance between their inner edges was 60 mm and the distance between the two probe positions was 17.8 mm (visual angle of 10.7°).

Immediately after the visual probe task, participants were asked to rate their subjective hunger using the SLIM scale. Participants then completed the picture rating task and the SRC task; the order of these tasks was counterbalanced.

The picture-rating task consisted of two practice trials, which used filler pictures, followed by 40 test trials in which each food-related picture and control picture from the visual probe task was presented, one at a time, in a new random order for each participant. Each picture (90mm by 120mm) was presented for 2000ms and after a pause of 500ms a seven-point anchored rating scale was displayed on the screen until the participant's response. The rating scale ranged from very unpleasant to very pleasant and participants were asked to press one of seven keys labelled –3 (very unpleasant) to +3 (very pleasant), to indicate how pleasant or unpleasant they found each picture. The intertrial interval was 500ms.

The SRC task was modelled on that used by Mogg et al. (2003). It consisted of two blocks, each of 100 trials. In each trial, a picture was displayed in the centre of the screen and a manikin figure was presented either above or below the picture. The picture was either a food picture or a control picture (i.e. those used in the critical trials in the visual probe task). Each block of trials had a different stimulus-response assignment: In assignment one, participants were instructed to move the manikin towards the picture if it was food related and away from the picture if it was not food related. In assignment two, these stimulus response relationships were reversed (i.e. participants were instructed to move the manikin away from food related pictures and towards non-food related pictures). The order of the assignments was counterbalanced across participants.

For each assignment there were 20 practice trials, in which 10 food-related and 10 non-food related were presented, followed by 80 test trials, with a short break after 40 trials. During the test trials, each of the 20 food-related and 20 control pictures was presented twice. Each picture was presented twice and was 90 mm high×120 mm wide. The manikin was 18 mm high by 10 mm wide; it appeared 25 mm above or below the picture with equal frequency. The manikin appeared above the picture in 50% of the trials and below on the other 50%. Participants responded by pressing the up or down arrow keys on the keyboard, which moved the manikin figure up or down the screen, respectively. The picture and the manikin disappeared as soon as the manikin reached the edge of the screen or the picture. There was a 500ms interval between trials. The latency between picture onset and the response was recorded.

Within each assignment block the trials were presented in a new random order for each participant so that picture type and manikin position varied over trials. The SRC task avoids one-to-one mapping between the required response on each trial and the approach/avoid instructions because, within each block, the manikin appears above the pictures on half the trials (when 'approach food') required a 'down' response to food-related pictures) and below the pictures on the other half of trials (when 'approach food' required an 'up' response food related pictures).

After the computer tasks, participants completed a SLIM scale and questions about recent food intake. They also completed the Dutch Eating Behaviours Questionnaire (DEBQ) (Van Strien, Frijters, Bergers & Defares (1986) and Eating Attitudes Test (EAT-26) (Garner, Olmstead, Bohr & Garfinkel (1982) which were given in a randomised order. Participants' height and weight were measured on a flat, even surface. At the end of the session, participants were debriefed about the aims of the study (see Appendix F), offered an information pack and reimbursed for travel expenses.

Data Analysis

The data was analysed using the statistics package SPSS Version 12.0 for Windows. The dependent variable for the visual probe and SRC tasks was reaction times (RT). For each participant a mean reaction time was calculated in each condition, after removing outliers and error trials. As there were no major departures from the

parametric assumptions, data for these tasks was analysed using mixed model Analysis of Variance (ANOVA).

Bias scores were calculated separately for each task and each participant, such that positive values reflected a bias favouring food related pictures relative to control pictures: (i) for the visual probe task, the mean RT to probes replacing food pictures was subtracted from the mean RT to probes replacing control pictures, so that positive values of the bias score reflected relative speeding of RTs to probes replacing food pictures, i.e. an attentional bias to food cues; (ii) for the rating task, the mean pleasantness rating of control pictures was subtracted from that of food-related pictures, so that positive scores reflected a more positive evaluation of food pictures; and (iii) for the SRC task, the mean RT for approaching the food pictures and avoiding the non-food pictures (assignment 1) was subtracted from the mean RT for avoiding the food pictures and approaching the control pictures (assignment 2). The overall bias score reflects relatively faster RTs when participants are instructed to approach, rather than avoid, food-related pictures, i.e. suggesting a bias to approach food-related cues. The data were also checked for normal patterns of distribution.

Results

Group Characteristics

Table 1 shows descriptive statistics and t-tests for age, BMI and measures of eating in the healthy weight and overweight groups. There was no significant difference in gender distribution, x^2 (1)=.29, p=.59. The healthy weight group appeared to have a higher level of education. However some of the cell values were less than five, indicating that calculating a chi-square was not possible (illustrated in Table 2). The groups differed significantly in age, $t(1,58) = -4.04$, $p < .01$; and the DEBQ-E restraint scale, $t(1,58) = -3.01$, $p < .01$. The overweight group were older and showed more restrained eating. They also tended to show more emotional eating (see Table 1). Three members of the healthy weight group and five members of the overweight group scored about the cut off point on the EAT-26. However it was not possible to calculate a chi-square because two of the cells had an expected count of less than 5.

Table 1

Group Characteristics

	Healthy weight group (n=26)		Overweight group (n=36)		*t*	*p*
	M	*SD*	*M*	*SD*		
Age	28.92	(14.50)	45.72	(15.42)	-4.33	.00**
BMI	22.01	(1.89)	31.91	(7.74)	[a]	[a]
[b] BMI $^{-2}$	.0021	(.0004)	.0011	(.0003)	10.73	.00**
DEBQA – Emotional Eating	2.40	(0.75)	2.86	(0.95)	-2.15	.04*
DEBQD – External Eating	3.29	(0.60)	3.18	(0.65)	0.81	ns
DEBQE – Restrained Eating	2.43	(0.85)	3.03	(0.72)	-3.02	.00**
EAT Total	7.46	(10.12)	11.00	(9.82)	-1.31	ns

* $p<.05$ **$p<.01$

Note. BMI = Body Mass Index; DEBQ = Dutch Eating Behaviours Questionnaire; EAT = Eating Attitudes Test

[a] Due to violation of homogeneity of variance assumption t-tests were not conducted on BMI data. [b] BMI data were transformed to meet homogeneity of variance assumptions for t-test.

Table 1

Group Characteristics (cont.)

	Healthy weight group (n=26)		Overweight group (n=36)		*t*	*p*
	M	*SD*	*M*	*SD*		
Minutes since last meal	115.96	(85.42)	147.03	(123.26)	-1.11	.273
Subjective Hunger Rating	2.96	(1.59)	2.69	(1.39)	.703	.485
Amount of favourite food the participant could eat at time of testing	3.5	(1.21)	2.89	(1.41)	1.786	.079
Minutes until next meal	168.46	(91.91)	190.28	(142.77)	-.683	.497

Visual probe task

Reaction Time (RT) data from filler trials, and from trials with errors, were discarded. RTs of less than 200ms or more than 2 SD above the mean were excluded as outliers. Mean reaction times and standard deviations are shown in Table 3.

To test the hypotheses, a 2 X 2 X 2 mixed ANOVA of the probe RT data was carried out with group (healthy weight, overweight) as the between-subject variable, and probe position (probe in same versus different location to food picture) and picture duration (500ms, 2000ms) as the within-subject variables. This showed no significant main effect of group, $F(1,58)=2.20$, $p = 0.14$. There was no significant main effect of probe position, $F < 1$ or interaction between probe position, duration of presentation and group, $F < 1$. There was a significant interaction between probe position and group, $F(1,58)= 4.74$, $p=.03$.

The healthy weight group was 9 ms faster to respond to probes replacing food than control pictures, $t(24)=1.63$, $p=.12$, while the overweight group was 4 ms slower on average to respond to probes replacing food than control pictures, $t(34)=1.30$, $p=.20$. This interaction is illustrated in Figure1. An ANOVA was calculated which excluded those who scored above 20 on the EAT but this did not alter the pattern of results significantly i.e. no main effect of group $F(1,50)=0.95$, $p = 0.34$ and there was a significant interaction between probe position and group, $F(1,50)= 3.57$, $p=.06$. An ANCOVA was calculated with hunger as the covariate and there was a significant result, $F(2,58)= 6.29$, $p=.02$, but the pattern of results already obtained remained unchanged.

Table 2

Contingency table showing the level of education of participants in each group

		Healthy weight group (n=26)	Overweight group (n=36)
Education	School	1	10
	College	2	11
	Degree	22	14
	Post Grad	1	1

Table 3

Mean RTs and standard deviations to probes (in ms) replacing smoking-related and control pictures in healthy weight and overweight participants

	Healthy weight participants (n=25)		Overweight participants (n=35)	
	Mean	Std. Dev.	Mean	Std. Dev.
Food picture and probe in same position at 500ms	649.10	(142.44)	702.73	(116.54)
Food picture and probe in opposite position at 500ms	656.12	(140.90)	699.55	(122.43)
Food picture and probe in the same position at 2000ms	646.60	(130.71)	703.60	(115.41)
Food picture and probe in opposite position at 2000ms	657.90	(134.08)	697.81	(120.54)

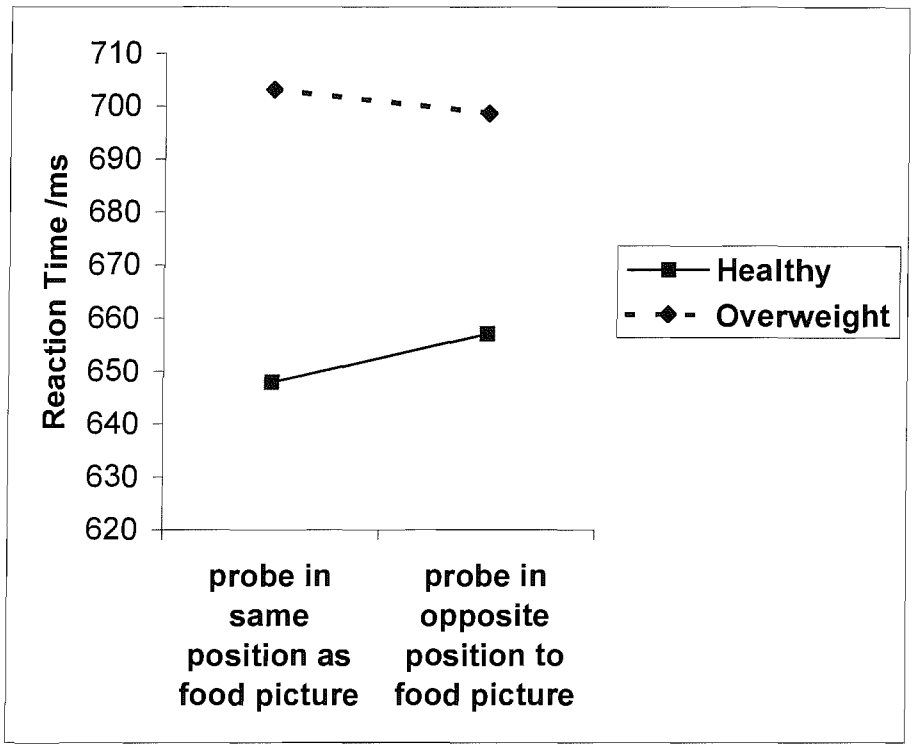

Figure 1. Mean RTs to probes replacing food-related and control pictures in healthy weight and overweight groups

It was not possible to calculate a chi-square using educational groups due to the cells being less than 5. The four educational groups were then collapsed in two groups (school/college and degree). An ANOVA was calculated to assess whether educational level was a factor with reaction times as the within subjects variable and group and educational level as between subjects variables. There was a main effect of educational level, $F(1,56)=30.02$, $p = <.01$. Participants in the school/college group were slower than the participants in the degree group (see Table 3 below). However this did not alter the pattern of results.

Table 4 *Mean RTs and standard deviations to probes (in ms) replacing smoking-related and control pictures in healthy weight and overweight participants who are school/college or degree educated*

	Healthy weight participants (n=25)				Overweight participants (n=35)			
	School/ College		Degree		School/ College		Degree	
	Mean	Std. Dev.	Mean	Std. Dev.	Mean	Std. Dev.	Mean	Std. Dev.
Food picture and probe in same position at 500ms	895.68	(316.31)	615.48	(61.54)	747.14	(123.32)	636.11	(64.49)
Food picture and probe in opposite position at 500ms	922.29	(295.10)	619.82	(53.80)	745.13	(132.53)	631.17	(61.56)
Food picture and probe in the same position at 2000ms	884.90	(234.93)	614.10	(71.07)	744.89	(129.31)	641.66	(47.13)
Food picture and probe in opposite position at 2000ms	913.77	(240.58)	623.01	(66.36)	742.21	(130.71)	631.22	(61.30)

Picture rating task

Mean pleasantness ratings were calculated for the food-related and control pictures for each participant. A 2 X 2 mixed design ANOVA of the ratings, with group (healthy weight, overweight) and picture type (food-related, control) as independent variables, showed no significant main effect of group, $F < 1$, or significant group X picture type interaction, F (1, 58)=2.13, p = 0.150. There was a significant effect of picture type, F(1,58)=46.94, $p < .01$, indicating that, in general, all participants rated food pictures as more pleasant than control pictures (See Figure 2). An ANCOVA was calculated with hunger as the covariate and there was no significant result, F (2,58)= .063, p=.80, but the pattern of results already obtained remained unchanged.

Stimulus–response compatibility task

Reaction Time (RT) data from filler trials, and from trials with errors, were discarded. RTs of less than 200ms or more than 3000ms were excluded as outliers. Means and standard deviations are shown in Table 4. A 2 X 2 mixed ANOVA was carried out with group (healthy weight versus overweight) as a between-subjects variable, and assignment type [(1) approach food-related and avoid food-unrelated (control) pictures versus (2) approach food-unrelated (control) pictures and avoid food-related pictures] as a within-subject variables.

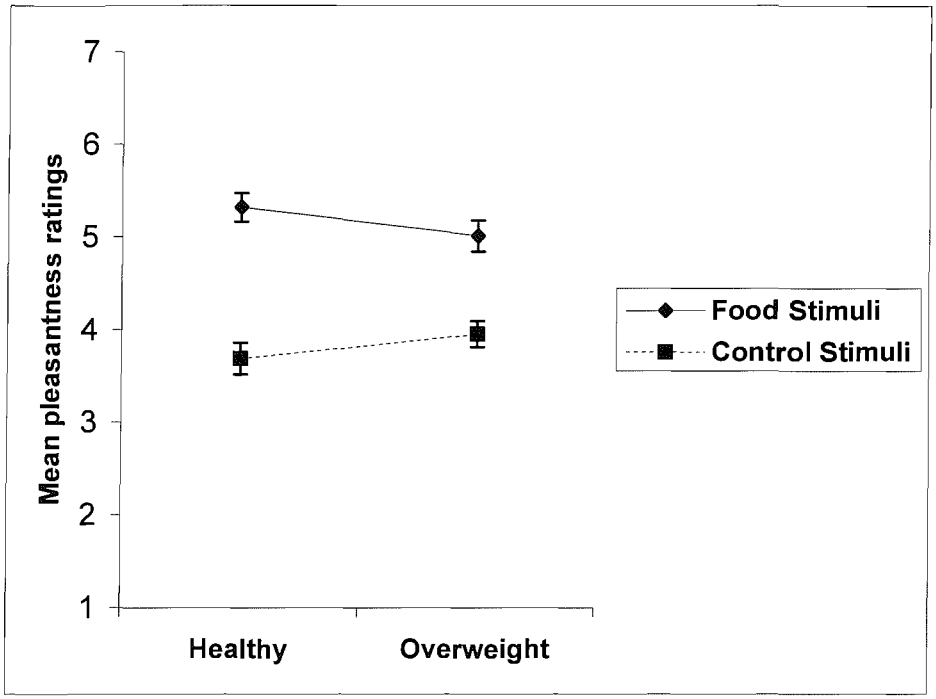

Figure 2. Mean pleasantness ratings (with standard error bars) for food-related and control pictures for healthy weight and overweight groups.

	Healthy weight participants (n=24)		Overweight participants (n=32)	
	Mean	Std. Dev.	Mean	Std. Dev.
Assignment 1	794.82	(129.80)	981.37	(218.81)
Assignment 2	898.31	(169.49)	1096.73	(284.77)

Table 5

Mean RTs (in ms) during assignment 1 ('approach food, avoid control') and assignment 2('avoid food, approach control') for healthy and overweight participants

This showed a significant main effect of group, $F(1,54) = 11.86$, $p < .01$ and a significant main effect of assignment, $F(1, 54)=42.12$; $p < .001$, but no significant interaction, $F < 1$. The healthy weight people were faster in both conditions than the overweight people. All participants were faster in assignment 1 than assignment 2. An ANCOVA was calculated with hunger as the covariate and there was no significant result, F (2,53)= .995, p=.323, but the pattern of results already obtained remained unchanged.

Correlations

Pearson correlations were calculated to examine relationships between the attentional bias, approach bias and pleasantness ratings. Overall, the approach bias to food was significantly correlated with more positive evaluations of the food pictures, r (58) = 0.330, p = 0.01. The attentional bias at 500ms was significantly correlated with more positive evaluations of the food pictures, r (58)=0.282, p=0.04.

Correlations were also calculated to investigate relationships between the questionnaire measures and experimental tasks. The external eating scale of the DEBQ was correlated with attentional bias at 2000ms, r (58) = 0.31, $p < .05$ and with the positive evaluations of food, r (58)= .42, $p < .01$. Participants' ratings of the amount of their favourite food they could eat right now (Grand, 1968) was correlated with attentional bias at 2000ms, r (58)=. 28, $p < 0.05$ and with the positive evaluations of food, r (58) = .32, $p < .05$ (illustrated in Table 5).

Corrections for multiple comparisons were not made to avoid the possibility of type II errors i.e. if statistical control is too strict, interesting results might be overlooked. Therefore the correlations highlighted as being significant could be due to chance.

Table 6

Pearson correlations between questionnaire measures, age and BMI and the experimental tasks

	VP bias (500)	VP bias (2000)	Explicit Ratings	SRC bias
Age	.069	-.046	-.149	.202
BMI	-.120	-.015	-.076	-.014
DEBQA [1]	-.286	.006	-.055	.261
DEBQD[2]	-.060	.309*	.423**	.268
DEBQE[3]	.071	.018	-.072	.152
SLIM scale A (pre)	.094	.097	-.094	.061
SLIM scale B (mid)	.153	.041	-.198	.034
SLIM scale C (post)	.217	.092	-.228	-.018
Mins since last eaten	-.056	-.175	.134	-.066
Subjective Hunger rating	-.242	-.079	.074	-.179
Favourite food eat now	.016	.285*	.322*	.272
Mins until next meal	.157	.038	-.129	.119

*$p < .05$ **$p < .01$ [1]emotional eating, [2] external eating, [3] restrained eating

DISCUSSION

The results of the visual probe task showed participants who were overweight had a reduced attentional bias for food cues, compared with participants of healthy weight. All participants showed an approach bias for food pictures but the two groups did not differ significantly in either the subjective evaluation (pleasantness ratings) of the food cues, or in approach bias to the food cues (SRC task). The results will be discussed in turn.

Visual Probe Task

This present study investigated whether participants who were overweight showed increased biased attention to food cues compared to healthy weight controls. Results showed that participants who were healthy weight and overweight differed in their attentional bias for food cues. The healthy weight group showed a greater attentional bias for food cues than the overweight group. Participants of a healthy weight showed a trend (p=.12) for an attentional bias to food cues (i.e. a tendency for responding slightly faster to visual probes appearing in the location of food). The results did not show participants who were overweight as having a greater attentional bias to food stimuli indicating there was no evidence that food cues were 'grabbing attention' in this group as predicted. Participants who were overweight showed no attentional bias towards or away from the food cues.

One explanation of the pattern of results could be participants of a healthy weight show a greater attentional bias for food cues than participants who are overweight. The bias may be 'reduced' in participants who are overweight and they perceive the food stimuli as being aversive. Individuals with eating disorders and anxiety disorders show hypervigilance and orientation towards threatening stimuli, (Ben-Tovim & Walker, 1991; Reiger et al., 1998). Ambivalence about food cues could account for participants who were overweight showing reduced bias to food compared to healthy weight controls. This 'ambivalence' might have resulted from the participants having a difficult relationship with food over a number of years. However given that the overweight participants showed no attentional bias for the 500ms presentation there may be other explanations related to the timing of conscious complex decision making. Further exploration of this idea was outside the scope of this study. The present study could be extended to include other types of stimuli in order to explore this further and could use a semi-structured interview to ascertain each participant's relationship with food.

Placanica, Faunce, and Job (2000) and Sackville et al. (1998) suggest that high and low calorie food words do not produce identical attentional biases and that biases may be more evident for high calorie foods. Future research considering these issues will be required in order to explore the specificity of attentional biases in individuals who are healthy weight and overweight.

The present results suggest attentional biases for appetitive cues previously found in addiction (Bradley et al., 2003) do not generalise to attentional processing in

participants who are overweight. Another aim of the study investigated whether participants who were overweight showed a pattern of hypervigilance and cognitive avoidance of food stimuli, which might be reflected by biased initial orienting to food stimuli (reacting faster to probes presented in the location of food stimuli after 500ms), and later avoidance (reacting faster to probes presented in the location of control pictures at 2000ms). Recent theories of selective attention highlight the distinction between different processes involved in the initial shift versus maintenance of attention (LaBerge, 1995). There was no effect of picture duration on attentional bias and the results did not show differences in selective attention. The visual probe task provides a limited snapshot view of attentional responses (i.e. at the time of offset of the pictures). It might be interesting to assess eye movements of the participants as these provide a more dynamic, ecologically valid index of visual orienting.

The external eating behaviour scale of the DEBQ and attentional bias to pictures shown for 2000 ms were correlated suggesting individuals who are high in responsiveness to external food cues maintain attention on salient food stimuli. Individuals high in responsiveness to external food stimuli have been characterised as prone to overeat when faced with palatable foods (Johansson, Ghaderi & Andersson, 2004) and this could be because their attention is drawn to food cues (Franken, 2003).

Attentional bias also correlated with the amount of their favourite food participants reported they could eat "right now". This can be considered an index of hunger

(Grand, 1968). This is consistent with findings of (Mogg et al., 1998) who reported that hunger is a predictor of attentional bias to food stimuli. However the ratings of subjective hunger did not correlate with the attentional bias measures. There was a trend towards group differences and this correlation could be a reflection of this result.

Picture Rating Task

A second aim was to examine the affective valence of food-related cues in healthy weight and overweight participants using both implicit and explicit measures, and to examine the relationship between the stimulus valence and attentional bias measures. In the picture-rating task, the explicit measure of participants' evaluation of stimulus valence, the healthy weight group and the overweight group both rated the food-related pictures more positively than the control pictures. This result is inconsistent with previous research into addiction. In relation to nicotine dependence Mucha, Geier and Pauli (1999) found smokers rated pictorial scenes of smoking as more pleasant than non-smokers.

The SRC Task

The SRC task provides an implicit measure of stimulus valence, which is inferred from the speed of behavioural responses in a symbolic approach/avoidance paradigm. This task does not require participants to make explicit judgements about the attractiveness of the stimuli; thus, it may reflect an individual's affective or motivational disposition towards food cues, while being less confounded by response

bias. It was predicted that participants who were overweight would respond faster to make an approach to food related stimuli than control stimuli as if they were motivationally positive. There was a significant main effect of group with participants of a healthy weight responding faster than participants who were overweight. Both groups showed faster reaction times when completing assignment 1 than assignment 2 and were faster in approaching food and avoiding control indicating an approach bias for food. This result is consistent with findings from Mogg et al. (2003), which shows evidence for approach bias for appetitive stimuli.

Affect is determined by the individual's motivational state (Lang, 1995; Lang, Bradley & Cuthbert, 1990; 1992; 1997). Two brain circuits are suggested, one determining appetitive responding (e.g. approach, attachment, consumption) and positive, pleasant affects and the other prompting defence (e.g. avoidance, fight-flight) and unpleasant affects. These systems may be co-active and the emotive significance of cues can be modified by experience. However, emotion and mood (pleasant and unpleasant) at any given time are determined by the dominant emotive system (appetitive or defensive).

Appetitive cravings under certain conditions can prompt an aversive state in substance abuse in humans (Baker et al., 1987; Drobes & Tiffany, 1997). While reward cues generally prompt positive affect, under conditions of deprivation or denial (frustration) such cues can lead to unpleasant affect, and perhaps, a different pattern of reflex modulation. According to frustration theory, an aversive state may be prompted by direct activation of the defence motivation system or through blocking of

appetitive drive. It is proposed that appetitive food cues presented to organisms in a high drive state – when actual consumption is not possible – promote a state of frustrative non-reward (Ansel, 1958, 1992). Food pictures appear to prompt a state of motivation ambivalence in food deprived individuals and binge eaters (Drobes et al., 2001), with self-report and psychophysiological responses being consistent with an appetitive reaction to these cues, but other responses (e.g. augmented startle response) being consistent with an aversive emotional state (e.g. frustrative non-reward). Drobes et al. (2001)'s study suggested opposing motivational circuits and evaluative systems were co-active (Cacioppo & Berntson, 1994; Lang, 1995; Miller, 1944). It is possible participants who were overweight were in state of motivational ambivalence and experiencing frustrative non-reward. This might explain why the predicted approach biases and positive explicit ratings for food were not found.

The present findings suggest the incentive salience model (Robinson & Berridge, 1993, 2001) does not apply to food cues in individuals who are overweight, as the overweight group showed a lack of attentional or approach biases to food cues. There is no evidence to support the hypothesis that individuals who are overweight have an over-responsive reward system implying over-activity of the dopaminergic system to the sight of food. Further research is needed to understand the mechanisms of natural rewards such as food and addictive drugs.

Relationship between attentional bias and stimulus valence

Another aim of the research investigated the relationship between attentional bias and motivational and affective valence of the food stimuli. More positive evaluations of food cues were significantly correlated with attentional and approach biases towards food cues. According to incentive models of addiction, attentional biases for drug-related cues should be associated closely with the perceived attractiveness of those cues because a common mechanism (dopamine-based incentive-sensitisation system) underlies both (Robinson & Berridge 1993, 2001). Recent theories of emotion also propose that the valence of a stimulus is important in determining its capacity to capture attention. Lang, Davis & Ohman (2000) proposed that stimuli with high affective valence (either highly pleasant or unpleasant) are more likely to attract attentional processing than stimuli with mild affective valence.

Although there was no evidence to support the hypotheses of greater attentional, approach and evaluative biases for food cues in participants who were overweight, the correlations between the bias measures and between them and some eating-related measures (hunger, external eating) suggest these measures may be useful tools in further research into cognitive mechanisms controlling eating behaviours.

Limitations

The food and control stimuli were piloted and matched where possible for colour, size and content. The pictures were not standardised, were generated in different settings

on two different cameras and were culturally biased. Although two of the filler pictures were indirectly related to the food stimuli in some way (e.g., cooking utensils) (Lee & Shafran, 2004), none of the control pictures were related directly or indirectly to food. Future studies could take photographs from a standardised system such as International Affective Picture System (IAPS; CSEA, 1995). However this may be problematic in terms of providing the required number of food pictures. Future studies could also choose control photographs picked from a particular category such as cars or items of furniture.

The study instructed participants to eat no more or less than they would usually eat before attending the study. Previous research has shown that hunger affects attentional biases (Mogg et al., 1998; Stewart & Samoulek, 1997). If the study were repeated it might helpful to manipulate hunger e.g. by asking half the participants to fast for 24 hours beforehand. Attentional biases for food cues may be stronger in participants who are overweight who are food-deprived.

While studies examining information-processing biases in clinical eating disorders have been somewhat sparse, there is growing evidence of the visual probe task being sensitive to clinical severity, with biases noted in clinical cases but not in restrained eaters (Boon et al., 2000). Further work needs to be carried out with this alternative technique, with greater emphasis being placed on increasing the ecological validity of stimuli used.

There was a significant difference in scores on the restraint scale of the DEBQ between the groups. The differences are to be expected because participants in the overweight group were mainly recruited through local slimming groups who were restraining their food intake. There were also a number of individuals in each group who scored above the cut-off point on the EAT-26 but excluding those individuals did not affect the pattern of results. This may suggest a lack of difference between the healthy weight and overweight groups which could account for the lack of difference in attentional bias. Future research could focus on comparing clinical and non-clinical samples to explore this.

Despite attempts to match the participants for age, there was a significant difference between the groups, which may have affected group differences in reactions times. In order to control for this age would usually be inputted as a covariate but age was not used because Miller & Chapman (2001) argued that it was inappropriate to use a variable that had a group difference as a covariate. Raw reaction times were not the variable of interest in this study and the correlations between age and the cognitive bias measures were not significant. Therefore the results for the cognitive bias measures were probably not confounded by the age difference.

Matching of the groups was problematic because the recruitment process attracted people to the overweight group who were older, retired and with more time available to take part in studies. The healthy weight group attracted a number of students because of targeted advertising at the university. It was more difficult to recruit participants of working age, a probable cause being work, occupational and family

commitments. Participants in the overweight group were mainly recruited through a slimming club and it could be argued this was not representative of the general population of people who are significantly overweight. In addition, the participants were grouped by BMI with some participants who were only slightly overweight being in the overweight group. Although the groups did differ significantly on BMI, future studies may benefit from stricter criteria. It may be helpful to compare individuals who are healthy weight with those who meet the criteria for obesity, who may have greater dysfunction than individuals who are overweight in mechanisms controlling cognitive and behavioural responses to food cues.

Individuals who are overweight may be a relatively heterogeneous group, and the extent to which their eating behaviour has characteristics of addictive behaviour (e.g. compulsive eating; irresistible urges) may vary considerably. This should be explored in future research.

Clinical Implications

Research shows attentional biases to relevant stimuli are elevated in people with eating disorders. Some research suggests biases may reduce as a function of treatment and reduced symptomatology (Lee & Shafran, 2004). However, these associations are not sufficient to support the causal role of biases in this disorder. Researchers in anxiety have experimentally manipulated attentional biases to emotional stimuli using the modified visual probe task to address causality (MacLeod, Rutherford, Campbell, Ebsworthy & Holker, 2002). These results support the

hypothesis that attentional biases play a causal role in anxiety (in the form of emotional reactions to a stress task). MacLeod et al. (2002) have developed a novel paradigm whereby attentional biases towards anxiety relevant stimuli are enhanced or lessened as a result of attention training. Such biases have causal effects on vulnerability to anxiety via their influence on how significant events are processed (Mathews & MacLeod, 2002). It is possible that this could be extended to individuals with eating disorders to examine the specificity of such a mechanism.

Lee and Shafran (2004) suggest research is needed using all individuals with eating disorders presenting for treatment. Inconsistencies between individual diagnoses (Cooper & Todd, 1997; Perpina et al., 1993) and the current trend for a 'transdiagnostic' approach to eating disorders (Fairburn & Harrison, 2003) suggest clinical utility in investigating cognitive biases in people with any clinical eating disorder or maladaptive patterns of eating behaviour rather being restrictive to specific diagnostic groups. Greater emphasis on increasing the ecological validity of stimuli used and research using pictorial (rather than individual word) stimuli is needed with the visual probe task. Research on cognitive biases is more likely to lead to clinical advances if they can be used in experimental paradigms that indicate the direction of causality. It is possible reduced attentional bias for food cues in individuals who are overweight is an effect (not a cause) of overeating and being overweight. It may be helpful to investigate what parallels might exist between overeating and addictive behaviour because if overeating can be shown to be psychologically similar to taking addictive substances this has implications for the treatment of both obesity and addictions.

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APPENDIX A

Pictorial Stimuli

APPENDIX B

Pilot Study

PILOT STUDY

Aim

To assess whether people could recognise what was depicted in a set of pictures and to rate how much each picture made them want to eat in order to decide which pictures to include in a stimulus set for an investigation of psychological reactions to food.

Design

Participants of healthy weight and people who were overweight took part in a single session in which their responses to food and matched control pictures were recorded.

Participants

Twelve people participated.

Measurements

The task was presented using Presentation 9.81software on a Toshiba SPA10 portable computer attached to a MEL version 2-response box. The photographs were presented twice. The first time the pictures were presented individually for 500ms each. The participant was asked to name each picture, and the experimenter used the response box to record whether the participant correctly identified the stimuli. On the second presentation the participant rated how much each picture 'made them want to eat' on a 9-point scale (1 being not at all and 9 being very much). On this presentation pictures were shown until the participant made a response.

Findings

The stimuli, which had the highest frequency of participants failing to recognise it correctly, were excluded from the study. A picture was considered suitable for inclusion in the task if more than 90% of participants could correctly name it after 500ms exposure. Pictures were considered suitable as controls if they received mean ratings of less than 1.75 (i.e. they did not make people want to eat). Pictures were considered suitable as 'food stimuli' if they received mean ratings of more than 4.5 (indicating that they did make people want to eat). Pictures where the standard deviation of ratings was high (greater than 3) were excluded from the task, as this indicated that there was variation in how appetising people found them.

Appendix C

Questions about recent food intake

Questions about recent food intake following the experiment

Which meal did you last eat (breakfast, lunch, dinner)?

What did you eat at that meal?

Did you eat enough?

Have you had any snacks, teas, coffees?

APPENDIX D

Participant Information Sheet

University of Southampton

School of Psychology
Doctoral Programme in Clinical Psychology

University of Southampton
Highfield
Southampton
SO17 1BJ United Kingdom

Tel +44 (0)23 8059 5321
Fax +44 (0)23 8059 2588
Mob: 07740776339
Email tg503@soton.ac.uk

A PSYCHOLOGICAL STUDY OF ATTENTION TO FOOD RELATED STIMULI

You are being asked to take part in a research study. Before you decide it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information carefully and discuss it with others if you wish. Thank you for reading this.

What is the purpose of the study?
To investigate how thought processes may affect overeating and contribute to weight gain. The study is looking at differences in peoples' attention and reaction to a variety of photographs to help us try and understand links between weight and psychological processes.

Why have I been chosen?
The study is looking for people of different weights in order to compare differences in peoples' attention to a variety of photographs.

Do I have to take part?
It is up to you whether or not you take part. You will be given a copy of this information sheet to keep and be asked to sign a consent form.

What does the study involve?
You can choose where to take part in the study (at home or at the university) at a time and date convenient to yourself. You will take part in three computer tasks, two questionnaires and complete some rating scales which will last about an hour. Your height and weight will be measured and you will be asked questions about your recent food intake. We hope to finish the study by May 2006.

Please eat no more or less than you would usually eat before attending the study.

Following your participation in the study, any reasonable expenses incurred will be reimbursed.

Will my taking part in the study be kept confidential?
All information, which is collected during the course of the research, will be kept strictly confidential. The results of this study will have all identifying information removed to protect anonymity.

What will happen to the results of the study?
A report of the study will be written. A summary of the results will be made available on request following submission of the dissertation in June 2006.

Who is organising and funding the research?
I am a third year clinical trainee at the University of Southampton, Doctoral Programme in Clinical Psychology. This research is being conducted as part of my training. The University of Southampton will act as a sponsor.

Who has reviewed the study?
The School of Psychology Ethics Committee, University of Southampton has reviewed this study. If you have any questions about your rights as a participant in this research or you feel that you have been placed at risk, you may contact the Chair of the Ethics Committee, School of Psychology, University of Southampton, Southampton, SO17 1BJ Tel: 023 80593995
Contact for further information

If you have any questions, wish to take part or wish to request a summary please contact:
Tanya Griffiths, Department of Clinical Psychology, Shackleton Building (44), University of Southampton, SO17 1BJ Tel: 07740776339 Email: tg503@soton.ac.uk

Appendix E

Consent Form

University of Southampton

School of Psychology

Doctoral Programme in Clinical Psychology

University of Southampton
Highfield
Southampton
SO17 1BJ United Kingdom

Tel +44 (0)23 8059 5321
Fax +44 (0)23 8059 2588

Email tg503@soton.ac.uk

Centre Number:

Study Number:

Participant Identification Number for the study:

CONSENT FORM

Title of Project:

A PSYCHOLOGICAL STUDY OF ATTENTION TO FOOD RELATED STIMULI.

Name of Researcher: Tanya Griffiths

	Please initial box
1. I confirm that I have read and understand the information sheet (Version 2) for the above study and have had the opportunity to ask questions.	☐
2. I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason and without my legal rights being affected.	☐
3. I consent to have my height and weight measured at the end of the experiment.	☐
4. I agree to take part in the above study.	☐

Name of participant	Date	Signature
Researcher	Date	Signature

1 for participant; 1 for researcher

APPENDIX F

DEBRIEFING STATEMENT

A PSYCHOLOGICAL STUDY OF ATTENTION TO FOOD RELATED STIMULI

DEBRIEFING STATEMENT

The aim of this research was to investigate the difference in attention to food related stimuli between healthy weight and obese participants. It is expected that:

- Participants with higher body weight will show more biased attention towards food pictures. Therefore they are expected to respond faster to the probes (crosses) that appeared on the same side of the computer screen as the food pictures.
- Heavier participants may also show a greater unconscious liking for food pictures. Therefore, they are expected to be quicker to move the manikin figure towards, than away from, the food pictures. They may show this effect even if they do not show greater conscious liking for food pictures.
- Conscious liking was measured in the picture rating task.

The results of the study will not include your name or any other identifying characteristics. A summary of the research findings will be available to you once the project is completed.

If you have any further questions, please contact Tanya Griffiths on 07740 776339 or tg503@soton.ac.uk

Thank you for your participation in this research.

Signature _____ Date _____

Name _____

If you have any questions about your rights as a participant in this research, or you feel that you have been placed at risk, you may contact the Chair of the Ethics Committee, Department of Psychology, University of Southampton, Southampton, SO17 1BJ. Phone: (023) 8059 3995