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Induced Impulsiveness? Eating Behaviour and the Modulation of Behavioural Sub-Types of Impulsivity.

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Declaration

I hereby declare that this thesis has not been, and will not be, submitted in whole or in part to another University for the award of any other degree.

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Portions of the thesis have been accepted in the journals listed below:

Reported in Chapter 3: 'The reinforcing value of palatable snack foods and its relationship to subtypes of behavioural and self-report impulsivity.' Brace, A, and Yeomans, M.R. (2016), Eating Behaviours, Elsevier.

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Aaron Brace

Brighton, September 2015.

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Doctorate of Philosophy in Psychology

Cued Impulsiveness: Eating Behaviour and the Modulation of Behavioural Sub-Types of Impulsivity.

Summary

Previous research has implicated the role of food-associated cues or pre-task reward exposure with eating behaviour. Eating behaviour (specifically overeating) has itself been associated with subtypes of impulsivity. To date, no research has examined the direct relationship between (food) reward-associated cues, or rewarding food exposure on behavioural impulsivity – a possible underlying mechanism. This thesis aimed to examine how behavioural impulsivity may be modulated by external cues, or by hedonic reward consumption, and how this interacts with eating attitudes (TFEQ).

Experiment 1 examined the aims explicitly, giving participants a hedonic preload (or nothing) before they completed impulsivity tasks. Those who received a preload were more impulsive in terms of their impulsive choice, and inhibitory control than those who had not received a preload. This effect did not replicate in experiment 3, where 2 further conditions were added, a non-hedonic preload, and an anticipation condition, but no differences were found between the groups. Experiment 4 conditioned rewarding cues to novel stimuli, and presented them before the behavioural tasks, but again, no difference was found between the groups. This thesis discusses the theoretical and methodological concepts, which may explain some of these null findings.

Experiment 2 aimed to examine how the reinforcing value of food (RRV) may be associated with types of impulsivity. However, no relationship was found between RRV and impulsivity, but RRV was consistent in predicting ad libitum food intake, as shown in previous studies.

Chapter 6 of this thesis is a meta-analysis of our laboratory's research linking delayed discounting (DDT), the TFEQ, and cue exposure paradigms. The analysis showed that those in high in dietary disinhibition (TFEQ-D) who were shown food cues, or consumed a hedonic preload were more impulsive on the DDT than those high in TFEQ-D that did not consume anything. The key limitations of this thesis are discussed, most notably the lack of statistical power in the experimental studies conducted. The general discussion of this thesis discusses the important implications of this finding in understanding modulation of behavioural impulsivity.

Abbreviations:

DDT: Delayed Discounting Task

PDT: Probability Discounting Task

IC: Inhibitory Control

RI: Reflection Impulsivity

TFEQ: Three-Factor Eating Questionnaire

TFEQ-D: Three-Factor Eating Questionnaire Disinhibition Subscale

TFEQ-R: Three-Factor Eating Questionnaire Restraint Subscale

HDHR: High TFEQ-D and High TFEQ-R

LDHR: Low TFEQ D and High TFEQ-R etc.

BIS/BAS: Behavioural Inhibition/Behavioural Activation System Scale

RRV: Relative Reinforcing Value

RRVfood: Relative Reinforcing Value of Food

Kcal: Calories

BMI: Body-mass index

CA: Contingency Awareness

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1.0 Introduction

There is now broad agreement that the worldwide increase in obesity over the last 2-3 decades, and consequent health problems, are caused by an imbalance between energy intake and expenditure (e.g. McCrory et al, 1999). There is consequently great interest in what it is in our modern lifestyles, often referred to as the “obesogenic environment” that underlies this dramatic change in population body size. However, despite the general increase, there are considerable individual differences in propensity to become obese (Lawson et al, 1995), and genetic makeup is a strong predictor of individual differences in body-size (Hofker & Wijmenga, 2009). However, the genetics of obesity is highly complex, with as many as 100 genes implicated in obesity (e.g. Bell et al., 2005). Thus an alternative approach is to examine phenotypic differences that appear to be related to individual differences in propensity to have an imbalance in energy intake to expenditure. This thesis concentrates on one such cluster of phenotypic characteristics, impulsivity, and its relationship to overeating since there is now considerable evidence that impulsivity may be a risk factor for obesity (Dawe & Loxton, 2004). However before the examination of impulsivity, and further aspects that may be useful in understanding individual differences in eating behaviour, theories of adiposity and motivation are explored to understand the grounding of motivational concepts and how these are more broadly related to the thesis’ themes.

1.1 Theories of Motivation: Homeostasis, Set points, Settling-Points, and Incentive Salience.

Although overeating and obesity have been discussed as problematic in social, health and economic terms, without understanding theories for eating motivation, these problems are less easy to discuss. The fundamental nature of motivation in psychology and neuroscience has endured over 100 years of experimentation and theoretical

discussion, and has attempted to explain, as Berridge (2004) suggests, 'why people choose to do different things at different times'. Motivation in this sense is the integration of psychological and neurophysiological processes that shape decisions and the variability of human behaviours. Although external variables play a role in modulating these processes, this discussion will be explored in due course.

1.2 Homeostasis

The concept of homeostasis, originally coined by Cannon (1925) has been historically the primary motivational concept discussed in understanding human behavioural regulation. Homeostasis suggests that for any internal state that needs to be regulated (hunger, thirst, body weight regulation, temperature etc.) there is a set-point – a goal level or optimum point which must remain stable. Cannon suggested that this stability in physiological states was essential, and deviation from the goal-state could be dangerous for survival. Homeostatic regulation must consist of 3 mechanisms – the set-point, the online error-detector (should levels from the set-point deviate), and a motivational mechanism to rectify the error (Berridge, 2004). This idea was illustrated by the work of Kennedy (1953) whose early ideas consisted of a 'lipostatic model', the possibility that fat storage and signals could be regulated by the brain to rectify any discrepancy from an individual's set-point adiposity. Classically, Kennedy and Cannon's homeostatic regulation ideas have been discussed using the metaphor of a thermostat controlling temperature (and human body temperature regulation could be discussed with the same metaphor). For example, the set-point is the internal temperature required to remain stable, should there be an error detected (excessive deviation from the setpoint), the error rectifying mechanisms will adjust the temperature up or down depending on which direction the error was in away from the desired setpoint. The idea of homeostatic regulation of physiological mechanisms is one that is intuitively simple and attractive and which has gained substantial empirical support in relation to temperature control, fluid balances etc. For instance, traditional theorists seem to have converged on at least some homeostatic control of weight through homeostasis and set-points and concepts of hunger and satiety (hunger and satiety as error correcting mechanisms in the set-point level of energy and bodyweight, e.g. Herman & Polivy, 1984; Kaplan & Kaplan, 1957; Schachter, 1971). The basic homeostatic concept also

describes well how bodily fluid depletion and blood pressure activate the physiological mechanisms that account for the onset of drinking behavior in response to fluid depletion (Epstein, 1982; Fitzsimmons 1990), although homeostatic regulation of fluid intake has been criticised for poor control in over consumption e.g. diuresis (Mack et al, 1994). In relation to eating, some researchers have attempted to use the broad homeostatic concept to explain why in periods of overfeeding or chronic dieting, following the cessation of these periods, the individual often returns to their original weight or specifically, adiposity (Bouchard et al, 1996, Anderson, 2001).

The concept of homeostasis in weight and appetite regulation has gathered further some support from neuropsychological and physiological evidence. For example, classic evidence comes from hormonal studies examining hormonal changes and the commencement and termination of feeding. Gibbs (1979) famously in *Nature* demonstrated that the gut peptide cholecystikinin (CCK), if administered to rats prior to food availability would induce increased food consumption (relative to the CCK dosage) compared with control rats. Other work has highlighted potential peptides involved in the satiety mechanism, including glucagon, neuromedin and bombesin (Geary, 1990), antagonistic blocking can have potent effects on consumption. Smith and Gibbs (1992) for example demonstrated in rats that blocking the effects of these peptides involved in satiety produces a marked increase in meal size, and subsequent weight gain. Although a review of the history of physiological and hormonal homeostatic control of weight and food consumption is beyond the scope of this thesis (see Scott et al. 2014 for a detailed discussion), the evidence presented supports the notion of at least some homeostatic regulation in body weight.

1.3 Homeostasis without deficit?

Although homeostasis theory posits an attractive theory for weight regulation, its ability to account for levels of obesity is less strong. What also remains somewhat unclear from the homeostatic explanations of motivation and the traditional physiological set-point concepts is the issue of anticipatory motivation or homeostatic responding without physiological deficits. For example it is possible to activate homeostatic responses without any physiological deficit or fluctuation around the proposed set points.

Berridge (2004) discusses this concept in terms of drinking behaviour. Berridge posits that when sitting down for a meal, humans often commence drinking behaviour. Berridge suggests that the meal itself may elicit anticipatory drinking, or drinking behaviour when there is currently no physiological deficit. This is not to say that there is no predictive mechanism for drinking when we soon may need to drink, or that this thirst is subjectively different from the motivation obtained in situations of physiological depletion, but that in the current state often no fluid deficit exists. This is somewhat critical of traditional ideas of homeostasis and reactions resulting from depletions and deficits, and others have suggested that homeostatic responding and the commencing of physiological mechanisms are in fact commonly the result of this anticipatory or predictive system as opposed to deficit regulation (Epstein, 1962 food intake, Fitzsimons, 1990 thirst and salt intake).

1.4 Beyond Homeostasis...

As discussed above, if homeostatic mechanisms can be activated without the need for depletion or deficit, once considered a critical factor in the error detection system of homeostasis, where does this leave the notion of set points in motivation and weight regulation? Nisbett (1972) for example posited the idea that overweight was not per se due to individual differences in set points, but rather that homeostatic regulation within certain individuals was awry, resulting in increased weight. What about environmental contribution to weight gain – can weight be determined by biological means alone? Some argue that this is too simplistic (Symonds et al., 2011). Might this then suggest that other factors aside from a pre-determined internal-set point may play a role in the deregulation of homeostatic mechanisms? In this respect the possibility of external factors influencing homeostatic mechanisms, or even challenging the existence of homeostatic regulation have been presented. It was the attractiveness and plausibility of the idea of homeostasis moderating hunger that Bolles (1980) believed was simply kept due its nature as a 'comfortable idea', rather than an accurate reflection of the physiological systems on command in weight regulation. This criticism in light of the common lack of need for depletion in the instigation of homeostatic responding that in part drove theories not towards a set-point regulation of weight, but a settling point theory (Bolles 1980).

Bolles and previously Wirtshafter & Davis (1977) proposed that weight regulation might not be through a pre-determined setpoint, but through a settling point – equilibrium between opposing pressures (Berthoud, 2004). As Berridge (2001) discusses, it is possible for external environmental pressures and changes to alter one's settling point. For example, should an environmental change occur, this may shift the (neural) pressures in favour of one of the forces thus increasing or decreasing the settling point, leading to weight gain or loss. Many researchers have discussed this type of environmental change, commonly referred to as the 'obesogenic environment', or an environment full of highly palatable foods (Stanton, 2006), which due to their rewarding value may shift the neural pressures to consume them to levels of increased weight and settling points. Speakman (2011) and colleagues review the factors that may play a role in the balanced dynamic of set points, or what factors may unsettle these settle points. For example, factors such as variety (Rolls & Heatherington, 2011), increased portion sizes (Rolls et al, 2007), distraction whilst eating (e.g. television, Epstein, et al, 1992) all have been demonstrated as forces driving settling points and adiposity in the positive direction. Berridge (2001) also forwards the possibility that 'diet drugs' or lateral hypothalamic lesions which seem to cause stable body weight reductions shift the settling point pressures in the opposing direction, lowering settling points. However the settling point regulation model has also been criticized experimentally for its approach to weight maintenance. The Minnesota experiment (Keys, 1950) in which female participants were placed on a low-calorie diet lost around 25 % of their total bodyweight. Following the experiment, in non-accordance with both set-point and settling-point theories, individuals actually gained even more weight than their starting weight – which Dulloo et al, (1997) suggested was due to psychological compensation (e.g. dietary restraint) rather than dynamic settling point forces. This suggested that control over body mass composition seemed to be in some way psychologically controlled – however in the eating attitudes section of this chapter we present problematic evidence to the concepts of dietary restraint also. It has been discussed here that physiological deficit (or not), and neural settling point pressures generate behavioural outcomes, but what processes influence or modify the incentive value of the positive stimuli to seek and consume them in the first place? Settling point or set point, what factors contribute to the stimuli adopting any

motivational value? Although some reference has been made here to the role of hedonics as a force in driving settling points, the relationship between hedonic and motivational value in food types is of importance in understanding the wider themes of this thesis.

1.5 Incentive Motivation

Although I have discussed theories of weight regulation and adiposity, what is yet to be discussed is what could increase the motivation to consume or obtain rewarding stimuli to begin with. For example, what processes are involved (both cognitively and behaviourally) in determining the incentive value of a reward (food, drugs, sex etc), and how does that motivate the organism to obtain them? Once we understand these processes, the foundations are laid to understand the 'state' nature of impulsivity, and how motivational shifts can be posited to modulate behaviours. Before attention is turned to incentive motivation however, it is vital at this point to be transparent with some of the terminology used. In the instance of this next section, the term 'hedonics' refers to the actual subjective pleasure experienced, and 'incentive' refers to the learned motivational value belonging to a stimulus. Therefore, 'motivation' refers to the process of the 'drive to obtain the stimulus of interest'. It is only through the clarity of these terms can we explore incentive salience and motivation in reward.

Firstly however, to understand the process of incentive motivation, we must revisit the work of Bolles (1972) who proposed that an organism's motivation towards a rewarding stimulus was primarily due to predictive cognitive expectancies, arising from the previous experience with that reward. Bolles (1972) suggested that Pavlovian learning through pairing an unconditioned or neutral stimulus (UCS, which Bolles, 1972 termed as S) with a conditioned stimulus (CS+, or for Bolles, S*) such as a hedonic reward would result in 'incentive expectancy' from the S*. Bindra (1974) took Bolles (1972) ideas of incentive expectancies even further suggesting that the S* (CS+) doesn't simply invoke a prediction of hedonic reward, but actually causes the same motivational state and subjective feeling as the original reward itself, what Berridge (2001) discusses as the process of taking on the 'specific motivational properties that normally belong to the S*' (CS+). This type of motivation attribution can also be demonstrated

experimentally, a good example being flavour preference learning, specifically flavour-nutrient learning. Flavour nutrient learning is the process of pairing a novel (UCS) flavour with additional energy (CS+) repeatedly, resulting in later increased incentive value for the previously neutral flavour on its own - a developed liking for the neutral flavour without the presence of the calories. For a review of human flavour-nutrient learning in human subjects, I direct you to the work of Yeomans (2012, review), and the work of Sclafani (1994, 1999, 2005) using rodents.

As supported as the idea of incentive expectancies is, this theory assumes that motivation would remain constant independent of physiological state (Gallistel, 1973). For example, should this learning process give a previously neutral stimulus an incentive value, then the motivation to consume or seek to consume that stimulus should be present at all times,, and should not be modulated by physiological state e.g. hedonic food paired CS+'s incentive value should be as motivating when an organism is hungry as when it is sated (or has little physiological deficit). However, it is clear that physiological states and deficits do play a role in the moderation of incentive motivational value (Berridge, 2004). The work of Toates (1986) and Cabanac (1992) aimed to explain how physiological states might modulate motivation, suggesting an interaction between the UCS, the hedonic reward and the moderating impact of physiological state on motivational outcome. This modulation of motivation through physiological deficit was formally termed alliesthesis, and was experimentally examined by Cabanac (1992) who demonstrated with human subjects that the subjective pleasantness of sucrose was more pronounced in participants in a hungry state, than those in a sated state, demonstrating a state-dependent modulation of hedonic experience. This modulation of motivation presented from alliesthesis work lead to the suggestion from Toates (1971) that stimuli could be both wanted and liked, but as Berridge (2001) points out, these two words were used interchangeably in Cabanac's traditional model (Cabanac, 1971).

1.6 Liking vs. Wanting

The groundbreaking work of Berridge & Robinson (1993) aimed to unpick the incentive processes in wanting and liking. The researchers suggested that as opposed to the ideas

of Bindra and Toates (discussed above), the processes of 'wanting' and 'liking' are not the same mechanisms, but actually perform different reward functions, and are differently represented in the brain. Berridge and Robinson (1993) suggested that 'liking' represented the hedonic impact of the reward itself, the subjective pleasure associated with it, whereas 'wanting' could be considered as the motivational value of the reward, the drive to obtain it, not the pleasure associated with it (Berridge & Robinson, 1998). A full history of the wealth of work examining this dissociation is beyond this thesis (see Berridge, 2007 for a review), but some key experiments have demonstrated this. For example, it is possible in rodents to induce a 'liking' response without 'wanting' (the drive to obtain rewards) by lesioning dopamine pathways or through dopamine receptor blocking using pharmacological interventions (Pecina et al, 1997, Berridge & Robinson, 1993). By using these techniques, motivation to consume or obtain rewards is drastically reduced, but affective facial expressions are still in line with a 'liking response'. This link between dopamine and motivation has been experimentally demonstrated across a host of incentive stimuli, showing that dopamine dysregulation (specifically suppression) leads to a severe reduction in reward motivation (Kenny, 2011). Conversely, Berridge & Valenstein (1991) demonstrated that it is possible to experience 'wanting' without the subjective hedonics associated with 'liking'. Using hypothalamic stimulation in rodents, they were able to encourage sustained intake (ie the rats 'want' to continue eating), despite showing facial responses suggesting a dislike for the sweet solution they were ingesting. These studies and other evidence clearly demonstrate a functional dissociation of the neural systems underlying hedonic (liking) and incentive (wanting). The dissociation of concepts and neural mechanisms of wanting and liking has prompted research into the mechanisms that may be shared between food and drug rewards. But if 'wanting' and thus motivation can be dissociated from subjective hedonics, and if the motivational value of a stimulus can be modulated through physiological state (as suggested by Cabanac), how is motivation expressed behaviourally in human participants? And how is this relevant to eating behaviour and impulsivity, the core of this thesis?

1.7 The Relative Reinforcing Value of Food (RRV)

The relative reinforcing value of food (RRV), or the extent to which an individual finds a food reward reinforcing relative to a non-food alternative, has been considered a possible behavioural expression of wanting (Rollins et al. 2014) in relation to Berridge and Robinson's wanting vs. liking dichotomy of reward. Epstein et al (2003), whose laboratory and colleagues have been the primary research group examining RRV, has suggested that in understanding food choice, understanding the willingness to work for a food reward (interpreted as a behavioural index of wanting) may be more useful than is the subjective evaluation of liking for food alone. In terms of measuring this possible expression of wanting, behavioural measures attempt to assess how hard participants are willing to work to obtain food rewards compared with a non-food alternative, which can be conducted through a progressive ratio schedule based on a simple action (e.g. mouse clicks, palm squeezes, etc.). Understanding this reflection of motivation seems to stem from a galvanizing of behavioural economics and human choice, and behaviorism and ideas of reward reinforcement. Typically (e.g. Epstein et al, 2013), participants are required during the behavioural task to choose between working (through progressive mouse clicking) for either a food reward or a non-food (commonly reading in adults, or occasionally video-game time in child populations (Temple et al. 2008): for further methodological details, see Experiment 2 of this thesis, page 55) After assessing motivation for both food and non-food rewards using this type of behavioural methodology, it is possible to gauge an individual's level of food reinforcement relative to non-food.

Although a simple behavioural task, there does seem to be at least some relationship between RRV and the variables and concepts of interest to this thesis (Experiment 2, again provides further background on this). Significant evidence seems to link RRV with increased BMI (Rollins et al. 2014), short term snack intake (Hill et al. 2009), long term weight loss success (Best et al. 2012), impulsive choice (Rollins et al. 2010) and macronutrient and food choice (Epstein, Carr & Lin, 2011). RRV has also been shown to be modified by physiological and motivational state (e.g. acute food deprivation, Epstein et al. 2003). RRV is a primary focus of investigation of Experiment 2 of this thesis, and also helps to formulate and frame the rationale behind the wider concepts of this thesis, that motivation and more broadly 'state' (as opposed to stable 'trait') changes have the potential to impact widely on behavioural

responding, whether it is justified or otherwise to consider RRV a direct correlate to motivational 'wanting'.

In this section we have explored the origins or motivation for eating and rewarding behaviours, and the historical theories of homeostasis, set-points and settling points. We have also examined the process of incentive motivation and how it is possible to behaviourally examine human behaviour as a response to this. However, some would argue that these concepts are somewhat reductionist, using the role of biological pre-determinism or neural and environmental forces to drive ('settling point forces') eating behaviour and subsequent weight management and adiposity, with little emphasis on an individual's control over their own food consumption. Although motivational concepts are vital to consider, they give us little insight into the understanding of individual differences in eating behaviour. What factors might help explain some of the individual differences in food consumption, adiposity or reward motivation? The core factors posited to play a role in the individual variation in these behaviours are that of eating attitudes, primarily restraint and disinhibition, but first discussed is the original conceptualization of restrained eating.

1.8 Eating Attitudes: Restraint Scale and Three-Factor Eating Questionnaire

Understanding the possible individual differences in susceptibility for weight-gain or overeating behaviour has been a critical issue in appetite research for nearly half a century. Schachter and Rodin (1974) laid the foundations for this body of research in a survey attempting to characterize behavioural differences between those considered obese and those considered normal weight. Although itself an important study capturing behavioural differences in eating behaviour, it was the later focus on this survey by Herman & Mack (1975) who devised the Restraint Scale as a focus of differences in restraining from or restricting dietary consumption. Although counter-intuitive, Herman and Mack suggested that individuals high in 'restrained eating', the concept of purposefully restricting or inhibiting one's own dietary consumption, may be more susceptible to long term overeating and weight-gain, despite short term restriction. Although supported in Schachter and Rodin's findings, those obese and overweight individuals reported more conscious inhibition of food intake than non-

overweight individuals; the explicit interpretation of why this could be so counter-intuitive was discussed by Nisbett (1974) several years earlier. Nisbett conceptualized appetite motivation and weight maintenance through the set-point model (previously discussed), that individuals have a predispositional set-point, or level of body-weight at which they are predisposed to be. Given this, Nisbett suggested that chronic dieters, or what Herman and Mack (1975) would later discuss as 'restrained eaters' would chronically reduce their food intake below the level required to maintain their 'set-point' and would subsequently have significantly increased hunger and overeat when their self-imposed restraint was relaxed or broken.

It is this overeating behaviour in restrained eaters that Herman and Mack (1975) considered 'counter-regulatory eating', consumption greater than if no restrained eating behaviour was imposed. This process was demonstrated using a classic preload design, in which restrained and unrestrained participants were allocated to a milkshake consumption condition, a two-milkshake consumption condition, or no consumption condition, before being asked to complete a bogus ice cream 'taste-test'. Restrained eaters showed greater ice-cream consumption in both milkshake conditions than the no consumption condition, whereas unrestrained participants compensated for their preload appropriately relative to the milkshake consumed in the intake test.

Herman and Mack (1975) suggested that because restricting intake (as indexed using the Restraint Scale, RS, Herman & Polivy, 2008) was a key characteristic of restrained eating; those who are considered restrained must control intake not through homeostatic satiety mechanisms and internal cues, but through cognitive control. Therefore, at the point at which counter-regulatory eating takes place, restrained eaters are now relatively insensitive to their internal satiety cues and subsequently overeat beyond satiety (relative to unrestrained eaters). Herman and Polivy (1985) reconceptualised the idea of restrained eating, and suggested that although insensitivity to internal satiety cues and mechanisms may be useful in understanding excessive eating, it may be more useful to consider this process as a result of breaking a self-imposed cognitive dieting boundary. For example, the 'boundary-model' of eating regulation suggested that restrained eaters impose a cognitive boundary on their food intake, and if broken (by, for example the consumption of what are termed as 'forbidden 23

foods') would lead to excessive eating (what is termed as 'disinhibited eating', eating with a loss of inhibition) with disregard to the dieting boundary, and due to the insensitivity to satiety cues. There has been at least some evidence implicating restrained eating as a predictive risk factor in overeating behaviour, particularly as a function of insensitivity to internal hunger cues (Herman & Polivy, 1980; Heatherton, Herman & Polivy, 1989). However, assessing restrained eating as a unified predictive factor in weight gain or overeating has had little experimental support or replication (e.g. Jansen et al, 2008), with some research suggesting restraint linked to increased body-fat in normal weight individuals, but not overweight individuals (de Lauzon-Guillain et al, 2006), or restraint is linked to successful weight maintenance or weight loss (Koningsbruggen, Stroebe & Aarts, 2012). Leitch, Morgan & Yeomans (2013) further contributed to the mixed findings in the value of restraint as a predictive risk factor in overeating, demonstrating that restrained eating might be associated with increased behavioural inhibitory control.

It was posited by Westenhofer et al, (1994) that the counter regulatory consumption or risk factors for overeating might not be encapsulated totally by the construct of restraint, but rather a combination of restraint and disinhibition – a trait characterised by reactivity to external cues and a propensity to binge or display uncontrolled eating behaviours. Disinhibition was introduced as a subscale of the Three-Factor Eating Questionnaire (TFEQ; Stunkard & Messick, 1985) constructed as a factorially derived scale, not empirically derived, as was the RS. The TFEQ also posited that although some evidence provided that restrained individuals may counter regulate as a result of a contextual influence (forced preloads or stress manipulations), it might be the role of TFEQ's disinhibition that moderates the relationship between restraint, situational ('disinhibiting') manipulations, and overeating behaviour. What must also be noted is that Herman & Mack's (1975) Restraint Scale included measures of weight fluctuation, which may have been later underpinned by some of the behaviours indexed by Stunkard & Messick's (1985) trait 'disinhibition' subscale of the Three Factor Eating Questionnaire. More specifically, there is a key conceptual difference between trait disinhibition and the behaviour of disinhibited eating. Trait disinhibition here is scoring high on TFEQ-D, and likely to exhibit characteristics as indexed by this definition, whereas disinhibited eating is a behavioural response (overconsumption of foods)

following the breaking of a cognitive boundary. Therefore, Westenhoefer (1981; 1984) has suggested that it is the interaction between restraint and varying levels of dietary disinhibition that is critical in our understanding of disinhibited eating. Westenhoefer (1994) conceptualized this interaction as either having 'rigid' or 'flexible' control over one's dietary restraint, and the extent to which one is susceptible to a loss of control over consumption following the breaking of this self-imposed restraint. Westenhoefer (1994) discusses those highest in their expression of rigid dietary control – control that is dichotomous, completely controlled or broken, and those expressing high disinhibition - a greater tendency to lose control following broken dietary restraint, to be most at risk of problematic eating or 'susceptible to eating problems' (p.29). Conversely, those who are higher in restrained eating but who were low in disinhibition could be reflecting what Westenhoefer et al. (1994) described as 'successful dieting', due to the tendency to display restrained eating without the accompanying loss of control (period of disinhibition) following a breaking of the restraint. Westenhoefer (1994) then posited then that this interaction between restraint and disinhibition into high and low interactive groupings would allow for the examination of counter regulatory eating following preload consumption with the hypothesis that high disinhibition (TFEQ-D) and high restraint (TFEQ-R – HDHR group) would exhibit more counter regulatory eating than low disinhibition, high restraint (LDHR) participants. As hypothesised, those in the HDHR group ate significantly more than the LDHR group, demonstrating the role of disinhibition in restrained eating and counter regulatory behaviour. Although Westenhoefer et al. (1994) have posited the interactive TFEQ factors as risk for overeating following preload consumption, others have attempted to achieve a 'disinhibition effect' through other means (e.g. stress (Haynes et al, 2003), or alcohol consumption (Ouwens et al, 2001) with mixed success in replication.

Although the 2x2 classification of TFEQ-R and TFEQ-D has been useful in understanding the characterization of eating attitudes, pertinent to this thesis is the specific role of TFEQ-Disinhibition. Although restrained eating and the disinhibition interaction is of interest, given the subject matter of the thesis and impulsivity (subsequently discussed), the nature of TFEQ-D's 'loss of control' over overeating behaviours is particularly central. For example, TFEQ-D's increased responsiveness to food cues (Bryant, 2000) is vital to understand the role of external influences on motivation and behaviour. One of 25

the core external characteristics examined with regard to the relationship between disinhibition and overeating is the palatability of food. Typically this type of disinhibition-behaviour dynamic is investigated through an ad libitum 'taste test', with the food palatability being manipulated between test meals. This type of methodology is demonstrated in the work of Yeomans et al. (2004) who manipulated the palatability of a pasta meal using a bland (non-seasoned) and an added seasoning version on separate testing days. In alignment with what we know about the responsiveness of individuals with high TFEQ-D to external cues, in this case palatability, Yeomans et al. (2004) demonstrated increased intake in the palatable meal in HDLR individuals, but no difference in bland test meal intake. Although palatable meal intake increased also across HDHR and LDLR groups, the LDHR group did respond to the manipulation of palatability. These data contribute to the nature of disinhibition as a proxy for responsiveness to external factors. This type of responsiveness was also shown in the work of Haynes et al (2003). This design manipulated participant stress rather than palatability, and the same pattern of TFEQ groupings and intake was demonstrated; those groups with heightened levels of disinhibition (specifically HDHR) consumed more food ad libitum when exposed to external cues, in this case a stress manipulation. This pattern of results was also demonstrated by Loxton, Dawe and Cahill (2011), showing that in periods of negative affect and when exposed to cues of preferred foods, those high in disinhibition demonstrate greater urges to eat the preferred cued food. However, using pizza food-cues (exposing participants to 'close proximity' to the pizza), Brunstrom, Yates and Witcomb (2004) failed to find an association between responsiveness to the due to pizza exposure and disinhibition. However this may be due to the use of a salivary measure as the outcome variable compared to subjective preferences, desire or intake measured in the previous studies discussed above.

The relationship between external cues and TFEQ-D has been extrapolated slightly further through discussion of food choice, which may in itself reflect responsiveness to palatability. For instance, there is some evidence suggesting that higher scores on TFEQ-D are associated with preferences for high-fat, high-salt food and to some extent this data extends to sweetened beverages. Lahteenmaki and Tuorilla (1995) showed that high scores on disinhibition were an indicator of preference for foods in sweet, pastries, butter-based and margarine based food groups. Blundell et al (2005) also supported

this link, demonstrating that higher levels of disinhibition were associated with preference specifically for high-fat foods. This preference for high-fat foods was experimentally demonstrated in the work of Bryant et al (2006), showing that, using the Leeds Food Preference Questionnaire (LFPQ, Finlayson, King & Blundell, 2008), high scores on the TFEQ-D subscale were strongly associated with preference for high fat foods. Given the evidence for the relationship between food choice (or potentially more broadly – external cues and palatability) and scores on TFEQ-D, it is unsurprising that TFEQ-D has been associated generally with increased energy intake (Contento et al, 2005, Lindroos et al, 1997, Lawson & Williamson et al, 1995, Hays & Roberts, 2008, Chambers & Yeomans 2011), and this is even more so given the stability in heredity of disinhibition characteristics, especially in mothers to daughters (Cutting et al, 1999). It is this responsiveness to external cues and the potential for behavioural modification as a result (previously discussed as overeating following cue exposure or a loss of control) that is of most interest to this thesis, and further in this introduction the possible interrelationships between TFEQ variables and external cues linking to other vital aspects of this thesis (e.g. impulsivity) will be discussed. But what may underlie TFEQ-D and how might it be behaviourally manifested or associated with the key processes investigated in this thesis?

1.9 Impulsivity

What is impulsivity?

The multi-factorial make-up of impulsivity is not clearly and universally defined. However, there are some factors that seem to be consistently associated with impulsive or risky behaviours. Before we begin to discuss the role of impulsivity in eating behaviours and overeating, it is important to examine what these factors of impulsivity are proposed to be, and how they may relate to the current thesis. Before this, it is important to address the links between self-report and behavioural measure of impulsivity, and the issues distinguishing between state and trait impulsivity given the ‘state’ or ‘cues’ focus of this thesis.

1.10 Self-report impulsivity – state vs. trait

Within impulsivity research, there are innumerable self-report 'trait'-like measures that seem to examine underlying 'trait' personality factors beneath 'impulsive' behaviours. Beneath these measures, there are a great number of factors, each attempting to encapsulate some component of impulsive behaviours. The breadth of this research is vast, from Buss & Plomin's (1975) original four-factor model (inhibitory control - decision time - lack of persistence - sensation seeking) to Cloninger's (1987) tri-factor model based around novelty-seeking, harm avoidance and reward dependence. However, despite the contributions of these measures, the models that seem to be the most prevalent in self-report impulsivity research are the Barrett Impulsiveness Scale (BIS-11, Barrett, 1994), Dickman's (1993) three-factor approach (Attentional - disinhibition - reflection impulsivity), and Carver & White's (1994) Behavioural Inhibition/Activation System (BIS/BAS, previously based on animal models posited by Gray, 1987). Although some of these measures contribute to the understanding of personality impulsiveness, what seems to be problematic is that often these measures fail to significantly load on one another, potentially suggesting that the concepts of which they claim to encapsulate are measuring subtly different constructs (Dawe & Loxton, 2004, Smith et al, 2007). Furthermore, there have also been mixed findings when examining the relationship between self-report measures of impulsivity, and behavioural measures of impulsivity (below). It is therefore possible that concepts that seem to overlap between self-report and behavioural measures of impulsive behaviour (e.g. 'inhibitory control') may potentially be examining different underlying constructs. For example, it seems possible that the self-report measures may be referring to underlying personality variables, whereas behavioural measures potentially tap-in to 'state' (situational or contextual) impulsivity. It is this possible distinction which should be considered when discussing the discrepancies between self-report, and the below discussed behavioural measures of impulsiveness. Although some discussion of self-report measures are included here, and a self-report measure is administered in Experiment 2 of this thesis, further discussion of impulsivity will be confined to behavioural measures, as the 'state' (behavioural) rather than trait ('self-report') outcomes of the potentially malleable impulsivity constructs are the primary focus of this research thesis.

1.11 Inhibitory Control

Inhibitory control is considered in this context as an ‘ability to prevent prepotent courses of action’ (Logan et al, 1997). Poor inhibitory control therefore is considered as an inability, or poor ability to inhibit prepotent motor responsiveness. In terms of the role of inhibitory control in impulsive behaviours, the origins of this research stem from the treatment of ADHD symptomology in adolescents. For example, sufferers with ADHD are often characterised as having poorer inhibitory control in comparison to individuals with no apparent psychological issues (e.g. Barkley, 1997; Lijffijt et al 2005), and specifically the inability to inhibit prepotent actions or processes. Inhibitory control has been associated not just in an ADHD suffering population, who seem to have issues with inhibition in a social context, but also populations who engage in ‘risky’ behaviour. The key population conceptualized as engaging in risky behaviour in the inhibitory control literature is those who engage in frequent drug use, or who suffer from substance addiction. Drug addiction has been relatively robustly associated with inhibitory control impairments in frequent cocaine users (Filmore, Rush & Hays, 2002; Filmore & Rush, 2002; Colzato et al, 2007), those suffering with alcohol addiction (Filmore & Vogel-Sprott, 1999; Field, Wiers & Christiansen, 2010; Weafer & Filmore, 2008) and heroin dependent individuals (Ful et al, 2008). Given these research findings, it does appear that there is evidence suggesting that engaging in risky behaviours, or a loss of control is associated with impairments in inhibitory control. The evidence linking these impairments in inhibitory control and eating behaviour are presented following the discussion of impulsivity subtypes.

In order to examine inhibitory control behaviourally in a lab context, Logan & Cowan (1984) originally posited what they coined as the ‘horse-race model’ of inhibition, an experimental expression of the investigation of motor inhibition in (rather romantic) real world examples (‘stopping ourselves from swinging at a baseball pitched outside of the strike zone’). The horse-race model itself assumes that inhibition of prepotent responses is a race between competing processes: the process of responding as quickly as possible, and the process of inhibiting a response. Logan and Cowan suggest that the winning process determines whether the successful inhibition occurs. For example, when attempting to respond, the response (‘go’) process is initiated, however at the

appearance of a cue (the 'stop' signal) the competing stop process is initiated, which if finished before the go process, leads to a successful inhibition of response.

Experimentally, these stop and go processes and signals are measured using stop-signal tasks, which ask individuals to respond to a go signal (which vary between tasks as arrows, abstract signals or letters), but to attempt to inhibit their response to a 'stop' signal (again, these vary between tasks, varying between visual to auditory stop stimuli). The trial types are split in these tasks, but in the original paradigm, go and stop stimuli were implemented as 66% go's/33% stop contingencies. The stop-signal reaction time (SSRT) is the difference between the onset and finish of the stop process in this original paradigm and is the latency that defines Logan and Cowan's inhibitory process speed. However, SSRT is not directly measurable ('covert', Verbruggen & Logan, 2008) and is estimated from a stochastic model integrating the distribution of stop trials, and from this distribution it is possible to make approximations of the average stop RT speed using the 'trimmed' distribution of stop RT's (trimmed as inhibition trims one side of the reaction time distribution) and the integration of the no-stop distribution (a detailed review is provided by Logan, 1994).

Other researchers have developed variations on the original stop-signal paradigm that doesn't require integration or calculus to derive values to understand the speed or success of the inhibitory process. For example, Dougherty et al (2003) developed the GoStop paradigm, a stop-signal task that allows for the adjusting onset of the stop-signal dependent on successful or unsuccessful inhibition (Experiment 1 Materials) for explanation of this task). To understand the inhibitory process, the researchers suggest that using this adjusting signal delay paradigm, the dependent variable is not necessarily the probability of inhibiting a response, but the incorrect responding RT to trials at which successful inhibition occurs at approximately 50% of the time, which does seem to lend itself to comparisons with the horse race model. It is this RT which the researchers suggest is the speed of the inhibitory process, which allows the investigation of inhibitory speed variation, not just commission or omission errors. As can be seen from the figure below (Figure 1) a behavioural measure consists of this type of initiation of pre-potent responding (as indexed by the left-right arrow responding) followed by the presentation of the stop signal. In Figure 1, the stop-signal presented is an auditory signal, but other tasks (and indeed the tasks used in the experimental

studies presented in this thesis) use a variety of stop signals other than an auditory signal, including the visual changing of the go stimuli (visual change, Verbruggen 2008) colour change as stop-signal, Dougherty et al, 2003), etc. Although behavioural tasks differ in their presentations of both the go signals and the stop-signals, there doesn't appear to be any task-dependent abnormalities as a result of these variations, and between task reliability seems to be robust.

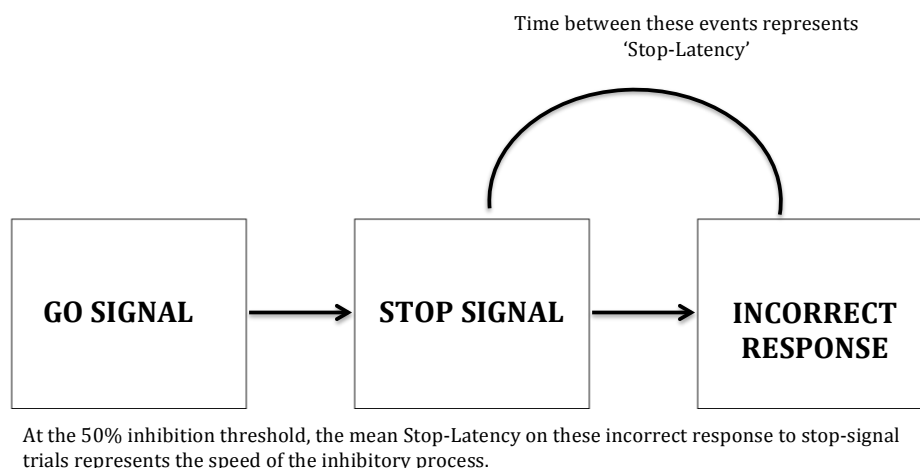


Figure 1 Representation of Stop-Latencies in relation to the Go and Stop Signal and the participant's response.

The work of Logan & Verbruggen, and subsequently Dougherty and colleagues has been tireless in developing behavioural methods to examine inhibitory control, but other researchers have attempted to identify the neural correlates of task performance surrounding inhibition. Typically at this point in the discussion of the neural basis of inhibitory control, one would highlight the case of Phineas Gage, a man who following an accident involving an iron bar damaging his frontal brain regions became disinhibited in his social attitudes, and made poor or risky business decisions – a far cry from the pre-accident man he was. Although a little anecdotal, the case of Phineas Gage appears to be some of the earliest evidence for the role of frontal regions in inhibitory behaviour. Since this case study, and minus the need for an iron bar, other researchers have attempted to examine the relationship between frontal regions and inhibitory control. Classic experimental work (Malmo, 1942) suggested (using frontal lesioned animals) that prefrontal deficits and inhibitory problems were a result of an inability to filter or suppress extraneous environmental or cognitive stimuli during the inhibitory

process – aptly discussed as the ‘distractibility hypothesis’. Although half a century prior to the formulation of stop-signal behavioural tasks, it is not difficult to draw parallels between the distractibility hypothesis and what is examined during behavioural measures of inhibitory responding. Although later work has suggested that the dorsolateral prefrontal cortex may modulate pathways leading to auditory and visual associative systems (Miller & Cohen, 2001), which seems to support the idea of multisensory requirements in successful experimental inhibition (Cohen, Braver & O’Reilly, 1996), a body of research has emerged robustly implicating prefrontal regions in inhibitory control. Strong evidence from animal experimentation supports this finding. For example, Walls et al (2001) demonstrated that primates who have been taught how to reach beyond a Perspex container to obtain food once lesioned in the PFC (prefrontal cortex) were unable to inhibit their response to reaching directly into the Perspex. These results were also supported experimentally some years prior by Iversen and Mishkin (1970) who demonstrated impaired performance on inhibitory control measures in primates with selective PFC regions.

We have explored here that the PFC does seem to have some association with impairments in inhibitory control, and as discussed these are robust associations. More recently, neuroimaging studies implicate the right lateralised inferior PFC (IFC) in inhibitory behaviours. These data have been shown predominantly in human adults and children (Garavan et al, 1999; Bunge et al, 2002; Konishi et al, 1999). A full review of the relationship between the IFC and inhibitory is explored in depth in the work of Aron, Robbins and Poldrack (2004). Interestingly, Aron (2007) discusses a novel criticism of the role of the PFC as a complete region for inhibitory control in – the nature of computational capacity. For example, Aron (2007) suggests that having a region that is engaged in ‘waking hours’ suppression of stimuli would be potentially computationally expensive (an idea reviewed by Miller & D’Esposito, 2005). Although an interesting philosophical limitation, as previously discussed in this segment, there does seem to be substantial evidence for the PFC as a neural correlate of the behavioural aspects of inhibitory behaviour.

1.12 Impulsivity and Impulsive Choice

What is being termed ‘Impulsive choice’ in this thesis for consistency is a subtype of impulsivity often referred to in the context of ‘reward sensitivity’ (e.g. Eppinger et al, 2012). This subtype is said to represent a trade-off between competing goals or rewards, often characterised as immediate vs. delayed rewards (Bickel et al, 2007). These ideas stem from, and seem to encapsulate the proposed underlying mechanisms of, the work on delay of gratification by Walter Mischel (1989). The famous marshmallow study conducted by Mischel seems to demonstrate this idea beautifully (Mischel, 1973) and has since produced some romantic images of the trade-off between competing goals in action in children. Children in that study were simply given the choice between eating a marshmallow at the time, or waiting a few minutes to receive two marshmallows, prompting visible scenes of the conflict between immediate and delayed gratification in action. Experimentally speaking, the concept of impulsive choice represents the extent to which an individual prefers immediate gratification as opposed to waiting for a longer-term goal. This assessment of this type of reward strategy is often measured using delayed discounting tasks (DDT). DDT’s present the participant with questions regarding their preference for a smaller immediate reward (often hypothetical and monetary), or a larger delayed reward, although a large variety exists (See Experiment 1 materials page 42, DDT task details for greater explanation). From these questions, and from varying time-periods of delays, it is possible to build a subjective discounting rate of delayed rewards for each participant, using either curve modeling (Mazur, 1987), or area-under-curve analysis (e.g. Green & Myerson, 1996, Yeomans et al, 2008).

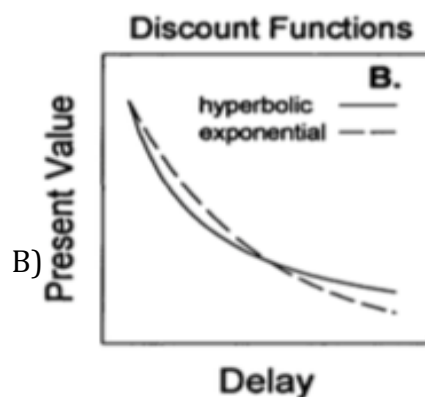
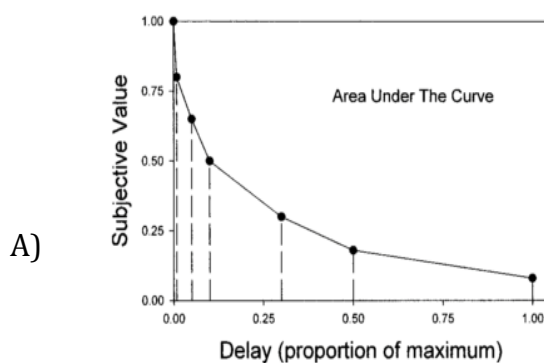


Figure 1.2: Graphical representation of A) AUC and B) hyperbolic and exponential methods of analysis DDT data.

From participants' data indicating their choice preferences, researchers are able to make comparisons of subjective discounting rates between subjects (Johnson & Bickel, 2008), for example with steeper curves representing 'higher' impulsive choice, e.g. that the person is willing to accept a smaller reward vs. a larger delayed reward sooner than someone considered to have 'lower' impulsive choice. Traditionally, discounting preferences are calculated by empirically deriving an individual's 'k' value (an overall measure of discounting) using either a hyperbolic formula ($V=A/(1+kD)$) or an exponential equation ($V=Ae^{-kD}$), where D represents the delay, A represents the amount of reward available at time D, and V as the subjective present value of the reward at time D (Figure 2, taken from Green & Myerson, 1996). As can be derived from the formulas, the k-value (Mazur, 1987) is the parameter that determines the value of the reward relative to the delay. Economists have classically preferred the exponential model to mathematically describe human behaviour, but research in the social sciences which attempts to unpick differences in choice preference has suggested that hyperbolic models account for human preferences more appropriately (Kirby & Marakovic, 1995). However, despite a vast number of studies using the hyperbolic and exponential equations of human preference, the models of choice rely on strict statistical assumptions. For example, the hyperbolic model relies on the data actually being hyperbolic itself, which in some cases does not happen. As Myerson et al (2001) discuss, a hyperbolic model, although useful in describing some individual's preferences, can often fit poorly to some data, with large variability and the potential for discounting parameter estimates to become skewed. In order to remedy this, Myerson et al propose the most useful and assumption-free method for examining discounting and choice data is through area-under-the-curve (AUC). The AUC is calculated by summing the plotted trapezoids, with each trapezoid calculated as $(x_2 - x_1)[(y_1 + y_2)/2]$, with x_1 and x_2 representing concurrent delay points, and y_1 and y_2 representing the subjective value of the reward at each delay point. The sum of the trapezoids, the AUC, gives an overall measure of discounting for each participant. This method does not require strict hyperbolic or exponential distributions of preference (figure 2), and can be calculated

without the use of complex integration or calculus. Work from our laboratory (e.g. Leitch, Morgan & Yeomans, 2013) has been useful in using discounting area-under-the-curve in between groups comparisons, more discussion of which comes later in this chapter.

Although we have discussed here the potential processes, types of analyses, and behavioural outcomes of discounting measures, there appears to be some evidence to suggest that there are possibly separate neurobehavioural mechanisms underlying both immediate and delayed gratification. This type of neural separation has been posited in the work of McClure et al (2004), demonstrating increased activation in the limbic system, associated with dopamine systems and the paralimbic cortex, whereas 'intertemporal choices' seem to increase activation in the 'lateral prefrontal cortex and posterior parietal cortex'. These ideas of independent neural activation between immediate and delayed gratification was also echoed in the work of Jimura et al (2013). The research suggests that steeper discounting of delayed rewards was positively associated with increased ventral striatum (VS) activation during both the period of choice, and also throughout a delaying period. Conversely, participants that were deemed as 'patient' (willing to wait) by the researchers displayed greater anterior prefrontal cortex (aPFC) activation throughout choice and delay periods. The researchers suggest that the results may represent 'dynamically evolving neural representations' of the subjective value of rewards.

This type of trade-off between reward systems has yielded some interesting behavioural findings concerning what are typically termed as impulsive populations e.g. substance abusers (Kirby et al., 1999) and alcohol abusers (Vuchinich & Simpson, 1998), but also now in populations with disordered eating. For example, studies in the last decade have implicated faster discounting of rewards in obesity (Weller et al, 2008), overeating behaviour (Nederkoorn et al, 2006), and increased BMI (Rasmussen et al, 2010). This link between impulsive choice and eating behaviour is examined in more detail later in this chapter.

1.13 Reflection Impulsivity

Until now, few experiments have examined the evidence for the role of what is termed 'reflection impulsivity' in impulsive behaviours. The concept of reflection impulsivity, developed by Kagan (1966), is the idea that when making decisions, certain individuals may not allow themselves enough time, or give themselves enough of an opportunity to reflect on or assess the relevant information to make an informed decision. Reflection Impulsivity arose from Kagan's similar ideas of cognitive tempo – the concept of the speed of perceptual or cognitive processing. This concept has been supported in the following years using Kagan's Matching Familiar Figures Test (MFFT), in which the participants are asked to choose an identical image to an example image from a number of similar images (e.g. similar looking Cowboys, Figure 1.3). Research has suggested that individuals who are more 'impulsive' often make more frequent, quicker errors, than those who make slower, correct reflective decisions (Drake, 1976; Devito et al, 2009).

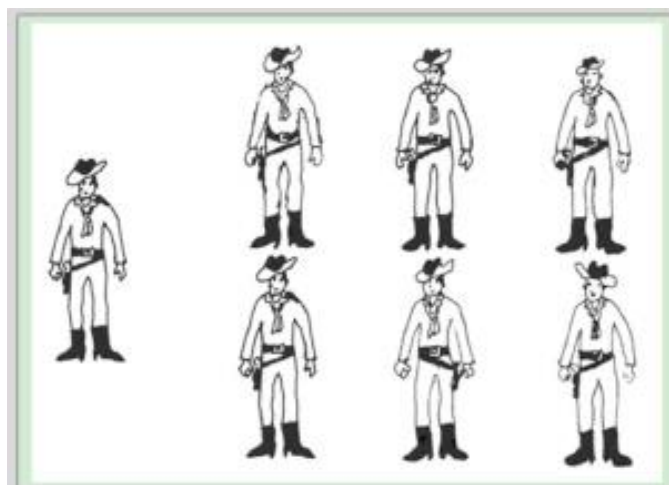


Figure 1.3: MFFT example (Cairns & Cammock, 1986).

Support for the role of reflection impulsivity, and specifically the use of the MFFT to assess this subtype of impulsivity, has come from research on ADHD sufferers (discussed as being more 'impulsive', Brown & Sleator 1979) and also, significantly, work with substance abusers. For example, Morgan et al (2002), using an extended version of the MFFT found that recreational MDMA users made significantly more errors than did non-MDMA users. However, despite the support for the MFFT, there have been more recent criticisms not only of its methodology, but also of its analysis.

For example, Clarke (2006) has criticised the idea of limiting the influence of the data of participants who respond quickly and correctly, and those who respond slowly and poorly on the task, because they 'do not meet the original definition of 'impulsive' or 'reflective' (Devito et al, 2004,p.2, Block et al, 1974). However, other researchers have highlighted possible limitations in the theoretical motivation underlying the MFFT. For example, Fox et al (2002) have suggested that the MFFT may not simply be a measure of reflective impulsivity, but may actually also put considerable constraints on visual working memory, and such working memory deficits have been found in populations studied who have a history of substance abuse (e.g. Ornstein et al, 2000).

It is due to these identified criticisms that another apparent measure of reflection impulsivity has been developed, known as the Information Sampling Task (IST, Clarke et al, 2006). The concept of information sampling is driven by researchers' rationale of developing a task that avoids over-emphasis on 'speed-latency' (Clarke et al, 2006, p.3) that has been seen in the administration of MFFT, but instead examines information sampling while reducing the demand on visual working memory. The IST presents participants with a 5x5 matrix of grey squares, which are revealed to be one of two presented colours when touched. The participant must then decide when they have revealed enough of the squares to make a decision as to which colour the majority of matrix is. Participants take part in two conditions, the fixed win (FW) condition, in which points are awarded for the correct decision of the majority colour, and the decreasing win (DW) condition in which points are deducted slowly each time another coloured square is revealed, therefore lowering the points offered following a successful decision on the matrix's majority colour. This task has received significant support in recent work, with research suggesting that it may be an efficient measure of reflection impulsivity (e.g. Delazer et al, 2011). In this thesis reflection impulsivity is considered as the third and final established behavioural subtype of impulsivity, which will be explored with regards to eating behaviour.

In experiment 1 of this thesis, in order to examine reflection impulsivity, the Matching Familiar Figures Task (MFFT, Cairns & Cammock, 1978) was used as the measure of this subtype. However, in the subsequent two experimental studies, the Information Sampling Task (Clark, 2005) replaces the MFFT as the measure examining reflection

impulsivity. This change occurred in the experimental design due not just the non-association between our experimental conditions in MFFT outcomes in experiment 1, but also due to criticism of the task itself. For example, Clark (2005) criticised the MFFT for potentially not measuring reflection impulsivity; but due to the visual search and working memory constraints that the tasks require (to examine minor differences in the stimuli presented). Fox et al (2002) then discussed the possibility that unmeasured deficits in these areas of visual working memory might therefore confound MFFT findings, and present findings that might not be simply differences in reflection impulsivity. The IST however according to Clark et al (2005) does not suffer from the same visual working memory shortcomings, as the stimuli on screen (the 'open boxes' that participants must decide the majority colour) remain on-screen throughout each trial, meaning that visual working memory is not relied upon heavily to examine key differences. Other criticism has also been leveled at the MFFT for participants' ability to speed through the task if they desire to leave the experiment as quickly as possible, as opposed to providing reliable estimates of reliability. The IST reduces this effect as much as possible by installing a minimum intertrial interval of 30 seconds so participants are aware that completing the task would not be achieved by speeding through trials. In terms of the measurable variables in administering the IST, traditionally research has used either the number of boxes opened in total, or $p(\text{correct})$ – the probability of being correct at any given trial at the point of decision as the key dependent variables. In this thesis, boxes opened in total were used as the dependent variable for two primary reasons – the first that the prototype version did not allow the practical calculation of $p(\text{correct})$ scores, but also because other work using the IST (Clark et al, 2005) have demonstrated that $p(\text{correct})$ and boxes opened provide 'statistically similar results'. It is for these reasons that the IST replaces the MFFT in experiments 2 and 3, and the boxes opened was chosen as the primary dependent variable in the IST.

1.14 Impulsivity and eating behaviour

Up to this point in this chapter, I have discussed the fundamental motivation for eating behaviour, and how shifts in this motivation can make important differences in behavioural responding. I have also discussed the role of dietary restraint and

disinhibition that may account for individual differences in eating behaviour. However now the focus of this chapter hones into the fundamental theme of the thesis as a whole, and another vital factor in understanding individual differences - the relationship between impulsivity and eating behaviour.

Inhibitory control & eating behaviour

In the last two decades, a body of research has emerged implicating subtypes of impulsivity with different aspects of eating behaviour. Response inhibition (inhibitory control as discussed earlier) is one subtype with a particularly strong association with eating. In the Netherlands, Nederkoorn & Jansen's research group has been successful in demonstrating the link between poor inhibitory control and increased ad libitum intake (Hofmann et al, 2008, 2009)

Research into inhibitory control and disordered eating has also been useful in understanding inhibition's role in eating behaviour. For example, research suggests a strong association between poor inhibitory control and greater frequency of disordered thoughts about eating measured using both the Restraint Scale (Herman & Polivy, 1980) and Fairburn & Beglin's EDE-Q; Eating Disorders Examination Questionnaire (Guerrieri, Nederkoorn & Jansen, 2007). Svaldi et al (2014) also provided evidence for a relationship between disordered eating and inhibitory control, demonstrating dissociation between control participants and participants with binge eating disorder through an inhibitory control (stop-signal) measure, and subsequently, Wu et al (2013) in their meta-analyses suggested that although small in effect size, individuals with bulimia-like disorders also show significant inhibitory control impairments. Ames (2014) supported this link between disordered eating, eating behavior and inhibitory control, demonstrating poor inhibitory performance on a go/no-go task and associations with binge-eating disorder in females, and increased sweet food and drink consumption in males. Lock et al (2011) also provided neuropsychological evidence of this association between inhibitory control and disordered eating, demonstrating in a youth sample that those who reported suffering with binge-eating disorder vs. anorexia

showed greater activation of hypothalamic and prefrontal brain regions, regions particularly pertinent to inhibition.

Research in non-clinical/eating disorder populations has also been extremely useful in attempting to understand how inhibitory control relates to eating behaviours, BMI, and adiposity. For example, some experimental research has reported an association between childhood obesity and response inhibition through increased stop-signal reaction times (Nederkoorn et al, 2006a, Nederkoorn et al, 2012), and with adult obesity through a higher number of failed inhibitions to stop-signals (Nederkoorn et al, 2006b). Also, Nederkoorn et al (2009a, 2010) also showed that a preference for snack foods in a laboratory test-session and poor inhibitory control in experimental conditions were significantly predictive of weight gain after a year follow-up to baseline. A rather novel study by the same research group (Nederkoorn et al, 2009b,) using a virtual supermarket and a subsequent ad libitum intake test, suggested that those with poor inhibitory control purchased the most food overall, the most snack food, and consumed the most ad libitum, but only when they were hungry. Batterink et al (2010) supported this association between inhibition and food intake in a population of adolescent girls, demonstrating a correlation (neurally and behaviourally) between response inhibition success throughout the task and BMI, however this finding was not contingent on levels of hunger (a behavioural finding also demonstrated by Jasinska et al, 2012). Further research has also been successful in demonstrating a trajectory of poor inhibitory control in early life (from age 7) leading to increased weight and higher BMI at age 15, which the authors equate as a risk of nearly 1.95x to gaining weight from having early life inhibitory problems (Anzman & Birch, 2009). Further research linking inhibitory control to BMI/bodyweight were supported subsequently in work by Koeber, Nederkoorn & Jansen (2014) who found interestingly that poor inhibitory control to food images but not neutral images was associated with increased BMI. Australian research groups including Kakoschke, Kemps & Tiggeman (2015) have also subsequently supported the relationship between poor inhibitory control to food-cues and overeating, demonstrating that poor response inhibition to a food-stimuli go/no-go test predicted greater snack intake in participants with attentional bias to food cues, which was previously mirrored using alcohol cues and predicted alcohol use in an adolescent sample (Peeters et al, 2012). Reinart et al (2013) attempted to draw

together the extent of the work linking inhibitory control and eating behaviours, or more specifically, body weight and brain region activation. Reinart et al's (2013) systematic review demonstrated in a body of child/adolescent sampled studies that body weight was associated with impairments in the orbitofrontal cortices, an area synonymous with inhibition of responses. It has to be noted however that the methodologies used here do not allow us to draw causal link between these deficits and eating behaviour.

Although these findings have drawn together useful evidence in assessment of inhibitory control and eating behaviour these findings have not always been replicated successfully, for example, Claes et al (2006) failed to dissociate controls from participants with different eating disorders, a useful proxy for disordered eating, through measures of inhibitory control. Houben (2014) and previously Meule et al (to food cues, 2012) also failed to find an association between general inhibitory control and BMI, which was surprising given the link demonstrated by Nederkoorn and Jansen's research group, and although work by Guerrieri et al (2007) showed an association between disordered thoughts about food and overeating, the researchers failed to find a significant association between response inhibition and either outcome measure. Loeber et al (2012) also failed to dissociate obese patients to normal weight patients with their responses on a food-cue specific inhibitory control task, further adding to the mixed findings between inhibitory control and eating behaviours.

Although there have been myriad mixed findings between eating behaviour and inhibitory control (Fay et al, 2014, good inhibitory control actually predicted snacking initiation, not inhibition), there is at least some evidence demonstrating an association. Further in this chapter, fitting with the rationale of this thesis, the role of food-specific inhibitory control is explored, e.g. inhibitory responding (or not) to food-related stimuli, a further focus of the 'state' nature of this type of impulsivity.

1.15 Impulsive Choice and Eating Behaviour

The second major subtype of impulsivity that we have discussed is impulsive choice. Impulsive choice, as indexed largely through delayed discounting task performance has been long associated with drug use in both humans (Bickel et al, 1999) and rodents

(Perry et al, 2006). However in recent years, there is an emerging body of work linking impulsive choice also to eating behaviour.

Firstly, the clearest link between discounting and eating behaviour is through the relationship between discounting BMI and body size. For example, consistent preference for small-sooner gratification vs. later-larger gratification either using curve modeling (k-values) or AUC has been shown in a number of studies to predict increased BMI (Epstein et al, 2003; Nederkoorn et al, 2006). Seeyaye et al (2009) in a remarkable longitudinal study were able to predict the trajectory successfully of children's weight status from age 4 in the first session to age 11, demonstrating delayed discounting as a key risk factor of childhood weight gain. Appelhans et al (2011) also demonstrated the ability to dissociate between obese and normal-weight individuals through discounting preferences, supported in the work of Kulendran et al (2013a), and Weller et al (2008). The same research group also demonstrated that as rates of obesity decreased in a youth weight management program so did preference for smaller sooner rewards (Kulendran et al, 2013b). For example, successful weight loss in a residential treatment was predicted by reduced discounting for immediate vs. delayed monetary rewards. Appelhans et al (2013) explored these relationships further, and their results suggested that not only did overweight status predict greater delayed discounting, but also that delayed discounting predicted preference for away-from-home foods with greater energy density.

Lee, Price & Higgs (2013) furthered these findings, and demonstrated that those with obesity weight status were more likely to discount hypothetical rewards more steeply than non-obese participants – independent of age and gender of their sample. More recent evidence supporting the relationship between discounting and weight status is provided by Buono, Whiting & Spong (2015) who demonstrated in a college student population that obese college students discount hypothetical monetary rewards more steeply than non-obese college students. Lawyer, Boomhower & Rasmussen (2015) in a sample of community volunteers continued the support for the relationship between discounting and weight status – those who meet obesity status showed significantly greater (steeper) discounting of hypothetical monetary rewards. Thomas (2015) using more novel, risk-estimation methods for examining weight gain risk showed evidence

that 'higher sensitivity to short term reward' e.g. steeper discounting was a key risk factor for higher BMI, independent of long-term goal perseverance, e.g. long term diet goals did not effect this relationship. Although little neuropsychological work has been conducted with regards to discounting research, Weller et al (2011) have demonstrated that impulsive discounting may be associated with hypoactivation of brain regions associated with or mediating executive function. The authors suggest that these differences in executive function may play a role in understanding impulsive decision making processes in those susceptible to weight gain.

However, as a recent review reveals (Story et al, 2014), there are mixed findings in the discounting data in relation to weight status. For example, both Borghans and Goldsteyn (2006) and Ikeda et al (2010) found that extraneous variables were related to BMI including under-saving in retirement and procrastination at work, but discounting did not. Nederkoorn et al (2006) also failed to find a consistent association with discounting and weight status. As can be seen from these data, there are a great number of mixed or confusing findings with regards to discounting and eating behaviour. For example, Davis et al (2010) demonstrated that binge-eating obese women were more impulsive than normal weight women, but not non-binge-eating obese women. Dodd (2011) added to this by demonstrating that the link between discounting and weight status was moderated by smoking status – which may be underpinned by general risk-taking moderating discounting preferences. Jarmolowicz et al (2011) support this idea, making the critique of their own and other experimental work in the field by claiming that too few studies control for smoker status in examining discounting and eating behaviour. Further limitations of the discounting literature are often aimed at the fact that discounting relies heavily on preferences for hypothetical, not real monetary rewards, which may not be a reliable way to capture participants true impulsive choice behaviour (Appelhans, 2013). A table review of the studies can be seen in Table 1.

Although this criticism has been leveled at the discounting literature, Reynolds (2006) has demonstrated that the relationship between discounting hypothetical vs. real monetary rewards seems to be correlated and consistent, however this is one of the few occasions that this point has been examined.

There are a number of possible reasons (possibly task-dependent differences) why some of these tasks may display mixed findings, and the meta-analysis conducted later in this thesis expands on this point. There is a body of work linking body size and discounting, albeit a little limited and sometimes mixed, but further in this chapter, the link between discounting and TFEQ variables are explored.

Table 1: Delay Discounting and Eating Behaviour Studies and Findings

Authors(s) and Year	Population	Discounting Method	Finding(s)
Nederkoorn et al (2006)	Lean vs. Overweight females	'seven delayed times are used, ranging from 1 week to 25 years', AUC	No difference in discounting between lean and overweight participants.
Richards & Hamilton (2008)	Obese vs. normal-weight women		Obesity linked to hyperbolic discount,
Weller et al (2008)	Obese vs. normal-weight women	2 weeks, 1 month, 6 months, 1 year, 3 years, 5 years and 10 years	Obese show steeper discounting than normal weight women.
Yeomans, Leitch & Mobini (2008)	Normal weight women	0,7,30,90,180, and 365 days, AUC	Steeper discounting associated with TFEQ-D
Harris et al (2010)	Binge eating vs. obese vs. normal weight women	(Not currently available)	Binge eating and obese more impulsive than normal weight women, but not different from each other.
Rollins, Dearing & Epstein (2010)	Normal weight women	1 day, 2 days, 1 week, 2 weeks, 1 month, 6 months, and 2 years, k-values	Discounting moderated relationship between RRV and food intake.
Appelhans et al (2011)	Obese & overweight women	1 day, 7 days, 30 days, 90 days, 180 days, 1 years, or 5 years, k-values	High food reward & steep discounting predicted food intake.
Dodd (2011)	Adults, ranging weights	Not reported	Suggested that smoking status may bias estimates of the link between discounting and bodyweight.
Manwaring et al (2011)	Binge eating vs. obese vs. normal weight women	1 week, 1 month, 6 months, 1 year, and 3 years, AUC.	Binge eating women discounted more steeply than obese and normal weight women.

Table 1 cont.

Authors(s) and Year	Population	Discounting Method	Finding(s)
Appelhans et al (2012)	Overweight and obese	1 day, 7 days, 30 days, 90 days, 180 days, 1 years, or 5 years, AUC	Steeper discounting predicted greater energy consumption in away-from-home food eaters.
Kishinevsky et al (2012)	Obese women	Not reported, k-value	Difficult vs. easy DD trials resulted in activation in exec. function areas - inferior frontal gyri, and medial PFC. Less activation in exec. function areas on difficult vs. easy DD trials predicted greater rate of weight gain over subsequent years
Fernandez (2013)	Female students	Not reported, k-value.	BMI associated with steeper discounting
Hendrickson & Rasmussen (2013)	Obese and healthy women	1, 2, 30, 180, and 365 days, k-value.	No changes in discounting before/after mindful eating training
Leitch, Morgan & Yeomans (2013)	Normal weight women	0,7,30,90,180, and 365 days, AUC	High TFEQ-D scores associated with steeper discounting.
Price, Lee & Higgs (2013)	Mixed weight males and females	(Not currently available)	Discounting associated with increased body weight.

Table 1 cont.

Authors(s) and Year	Population	Discounting Method	Finding(s)
Bickel et al (2014)	Crowd-sourced sample.	'small, medium, large' reward magnitudes and delays, k values.	BMI associated with more impulsive discounting
Dodd (2014)	Adults, ranging weights	Not reported	Impulsive discounting associated with higher BMI
Jarmolowicz et al (2014)	Range of body mass females	'small, medium, large delays'	Increased body mass associated with more impulsive discounting
Lu et al (2014)	12-13 year old female	1day 1 week, 1 month, 6 months, 1 year, 5 years, 10 years, and 20 years, AUC	Discounting moderated the link between cortisol reactivity and body fat %.
Aly, Howard & Lowe (2015)	Female, normal weight population	(Not currently available in full)	High discounting interacted with high power-of-food scores to predict food consumption.
Yeomans & Brace (2015)	Normal weight females	0,7,30,90,180, and 365 days, AUC	Food-cue exposure interacted with TFEQ-D to predict impulsive discounting.
Kulendran et al (2016)	Adults and adolescents on spectrum of weight management issues	Not reported. K-value.	Discounting associated with higher BMI, but discounting not associated with weight loss or weight loss surgery.

1.16 Impulsivity and TFEQ

The relationship between impulsivity and body size and disordered eating has been described and explored here in this chapter, but what about the relationship between impulsivity and TFEQ variables disinhibition and restraint, subscales indexing differences in eating behaviours? Research has documented some evidence suggesting that TFEQ variables may be associated with overeating, but it may be possible that the relationship between impulsivity and individual differences in TFEQ may aid the understanding of eating behaviours or to some extent the susceptibility to overeating.

Firstly, in terms of dietary restraint (as indexed by either the RS, TFEQ-R, or the DEBQ, Dutch Eating Behaviour Questionnaire), there is evidence to suggest that restraint is associated with subtypes of impulsivity. Nederkoorn, Van Eijs & Jansen (2004), using a split sample of highly restrained (DEBQ) participants and low restraint control participants, found that those highly restrained were poorer at inhibiting their motor responses on a stop-signal task, but this poor inhibitory control was not modulated by exposure to food cues. Jansen et al (2009) examined how the relationship between restraint and impulsivity impacted on laboratory eating behaviour, and found that inhibitory control modulated the pathway between dietary restraint and increased lab food consumption. This interaction between impulsivity and restraint was explored further in the work of Koningsbruggen et al (2013) who examined these variables with regard to short-term dieting success. As predicted, those who displayed low impulsivity (as indexed using the BIS-11) and high restraint were able to successfully lose weight. Ebner et al (2012) using a questionnaire methodology attempted to look more broadly at the relationships between these self-report measures (DEBQ, BIS-11), and showed specifically that attentional and motor subscales of the BIS-11 (subscales seen to be a self-report parallel of behavioural inhibitory control) were significantly correlated with dietary restraint; higher restraint associated with higher impulsivity.

As is discussed here, restraint clearly does have a role to play in understanding impulsivity and its associated behaviours, but other work has also demonstrated a key association between dietary disinhibition and some measures of impulsivity. Yeomans, Leitch and Mobini (2008) found no relationship between dietary restraint and any

impulsivity measures, but demonstrated a link between disinhibition (TFEQ-D) and more impulsive discounting on a DDT, and higher scores again on the motor and non-planning subscales of the BIS-11. The BIS-11 relationships are consistent with Ebner et al's (2012) work, and the non-planning subscale's association with disinhibition is particularly interesting given that the sample discounted more impulsively, and it could be suggested that these measures are associated, and work has often failed to associate behavioural and self-report measures of impulsivity. Work from the same laboratory (Leitch, Morgan & Yeomans, 2013) interestingly failed to find an association between TFEQ-D and discounting, but found that TFEQ-D was rather associated with reflection impulsivity using the MFFT. What was also unexpected about this study was that higher dietary restraint was associated with better inhibitory control (stop-signal). Although an unexpected finding, the link between better inhibitory control and dietary restraint has been demonstrated elsewhere, including the work of Meule et al (2011). The authors here make the suggestion that dietary restraint and inhibitory control may be 'situation specific', which to some extent taps into the ideas of this thesis, that there although there are some mixed findings between impulsivity and dietary disinhibition/restraint, and also eating measures, it may be possible that by its definition as 'behavioural' impulsivity is a 'state' concept, and that compromised subtypes of impulsivity could be modulated through a state motivational, environmental or physiological context, which to some extents seems to share at least some ideas with Cabanac's alliesthesis hypothesis of the state-modulation of motivation.

1.17 Impulsivity – State modulated, cue induced?

The concepts discussed in the last paragraph lead this chapter into the final defining theme of this thesis – the idea that behavioural impulsivity may be state-specific, or cue-induced. For example, some studies have failed to find a relationship between eating behaviour and some types of impulsivity (Nederkoorn et al. 2006), and Meule et al (2012) have demonstrated cue-specific (particularly food) responding to impulsivity measures. This leads to the fundamental crux of this thesis: is impulsivity state-dependent, and can it be modulated through experimental manipulation (e.g. cue exposures, anticipation for rewards, preload consumption)? The role of cue-exposure and subsequent behaviour is covered in depth in both the food and

drug/alcohol/smoking literature. For example, Olmstead et al (2005) demonstrated in rodents a consistent preference for immediate gratification following exposure to alcohol cues. Papachristou and colleagues (2012) have demonstrated similarly that cue-elicited craving for alcohol may interact with individual differences in impulsivity.

This type of pre-exposure (consumption or visual/olfactory associated cues) to the target substance is discussed alternatively in the alcohol, drugs and smoking literature as the concept of 'priming', rather than what some literature discussed here calls being 'disinhibited' (Westenhoefer, 1994). De Wit (1996) discussed this priming effect with habitual cigarette smokers who have remained abstinent from smoking for a prolonged period of time, and subsequently either relapse or report vastly increased desires to smoke following smoking a single cigarette. Much early work in understanding the role of pre-exposure to a substance and subsequent desire to obtain the substance was conducted on animal subjects. De Wit & Steward (1981) demonstrated that following periods of self-administration of cocaine or heroine, rats would reinstate responding behaviour after a period of extinction followed by a drug injection administered by the experimenter – a priming effect. Stewart (1984) continued to support these findings in rats, demonstrating that a direct morphine injection would instigate previous drug responding in previously abstinent rodents.

Of greater focus to this thesis, priming effects have also been examining with human subjects. Early work by Ludwig & Wikler (1974) showed that individuals meeting the criteria as alcoholic would work harder (increased button presses) and reported greater craving for ethanol if they had subsequently consumed an ethanol (vs. a placebo) preload. This ethanol vs. placebo preload design was particularly successful in demonstrating priming effects with human subjects. Bigelow et al (1977) took the idea of working for alcohol further in their design, showing that an ethanol preload would increase time riding a stationary bike for alcoholic participants vs. a placebo preload. Stockwell (1979) demonstrated not simply increased working for ethanol, but subjective feelings of craving for ethanol. Although the drug and alcohol literature provide a useful parallel for food investigated in this thesis, some work has look directly at the role of priming on actual food consumption – in both human and animal subjects. Beyond the early work of Pavlov (1919) who demonstrated appetitive behaviours in

canines following the presentation of a food-associated prime, Eiserer (1974) demonstrated with rats that the presentation of a food reward (in this case, pellets) would reinstate food responding, even after a period of extinction after a prior food reinforcement responding task, which was replicated successfully by Terry (1980). Cornell et al (1989, 1992) demonstrated parallel behaviours with human subjects, that consumption of a small amount of hedonic foods increased later consumption of the same foods, even in participants that reported being sated. Harris, Bargh & Brownell (2009) also demonstrated that both children and adults consumed more (healthy and unhealthy) foods in the guise of a taste test following exposure to snack food advert priming vs. a non-food advertisement, with the children showing a 45% increase in food consumption in the food prime group. Further food research demonstrated that prior to exposure reward cues (e.g. cues that predict tasty foods) have been shown to consistently elicit overeating behaviour in overweight children populations (Jansen et al, 2003), in restrained eaters (RS), in response to pizza food cues (Federoff, Polivy & Jansen, 1997), and that exposure to these food cues was not necessarily specific to the cued food (Federoff, Polivy & Herman, 2003). Epstein, Rocco & Coleman (1996) took this idea a little further, demonstrating a heightened salivatory response to hedonic food cues (a lemon yoghurt) in an obese vs. nonobese sample, a sample that has previously demonstrated heightened impulsive responding (Weller et al, 2009). These studies demonstrate the key role of 'priming' in modulating subsequent eating and food related behaviour.

Admittedly, although this work demonstrates a modulation of eating behaviour, or subjective feelings of craving or desire to eat, it tells us little about a modulation of impulsivity. However, the studies in this thesis aim to investigate whether this modulation of behaviour through a cue-induced experimental manipulation may actually be associated or possibly even underpinned by a modulation of some subtypes of impulsivity.

Early work stemming from this idea is becoming available from our laboratory. Yeomans & Brace (2015) demonstrated in a sample of undergraduate students that pre-exposure to food-cues lead to increased behavioural responding on a delayed

discounting. This early data seems promising in understanding the relationship between cues/food reward and impulsivity. Earlier work by Meule and colleagues (2014), although not attempting to induce impulsivity necessarily, found that if food images were included as part of an inhibitory control measure, those who failed to inhibit on food image trials would subsequently report greater food cravings. Taken together, the evidence for the state-specific nature of impulsivity is beginning to accumulate. Alternative paradigms have attempted to 'prime' impulsivity in a slightly different way to examine subsequent behaviours rather than impulsivity itself. For example, Guerrieri et al (2006) using a priming task that hinted at impulsive behaviours vs. a neutral story prime failed to increase intake following a prime. A follow up several years later (Guerrieri et al, 2009) attempted to use the same priming method by also splitting the sample into non-dieters and dieters. Specifically in non-dieters, priming of impulsivity lead to increased intake in the laboratory. This success lead to the same research group to turn the paradigm on its head, and attempt to induce inhibition rather than impulsivity (Guerrieri, Nederkoorn & Jansen (2012) using a stop-signal task where they participants were required to practice inhibition (low impulsivity condition) or were told to respond as quickly as possible. The data suggested that the low impulsivity condition managed to reduced ad libitum intake relative to the impulsivity condition, but not to a control group that were given no instructions. This suggests that inducing impulsivity may be more experimentally possible than strengthening inhibitory systems. Although not directly 'cued', Caswell, Morgan & Duka (2014) demonstrated that inhibitory control could also be modified through a depletion procedure, further providing evidence for the modifiable nature of impulsivity.

Taken together, the research discussed shapes the rationale for this thesis, the exploration of what may underpin impulsive responding, whether there is some contextual stability to impulsivity, or whether cued or motivational state differences may modulate impulsivity, and if so, what types? This may aid our understanding of the processes of overeating behaviours and how executive and decision-making processes contribute to this.

1.18 This thesis - Aims

The current thesis aims to tackle the questions outlined in the paragraph above: can impulsivity be induced through experimental manipulation, whether that may be through preload consumption or cue exposure, and which subtypes are particularly susceptible to these effects? In response to the work of Guerrieri et al. (2009) and the work of Meule et al (2012) who implicated dietary restraint as of interest as another variable which may interact with a cue-induced effect in predicting behavioural outcomes, this thesis also aims to examine to what extent dietary restraint and disinhibition may interact with our experimental manipulations to modulate subsequent behaviours.

Experiment 1 of this thesis is primarily concerned with revisiting the classic Herman & Mack (1975) milkshake preload paradigm of counter-regulatory eating. We propose a reconceptualization of the ideas proposed by Herman and Mack that overeating following cue exposure is as a result of a breaking of a cognitive boundary, but rather that preload consumption may heighten reward sensitivity and impulsivity which may account for overeating. Experiment 2 attempts to examine to what extent RRV as detailed by Epstein previously in this chapter is associated with other facets of impulsivity, and the extent to which RRV may be a vitally under researched component in impulsivity research. Experiment 3 attempts to unpick which preload characteristics may be pertinent in inducing subsequent impulsivity, including the hedonic value, the perceived energy content, or the anticipation of hedonic reward. Experiment 3 indicated that a replication of experiment 1 was not achieved, and the data presented that a measurement of post-preload satiety in preload designs is never measured which has been shown to dampen reward sensitivity (Nijs et al. 2010), therefore we attempted to condition a previously neutral cue as a CS+ in experiment 4 to negate any confounding variables with satiety or hunger, and to examine the role of a conditioned reward stimulus on impulsivity in a pseudo-applied context, which we liken to a branding mechanism, which has been shown to increase intake in our laboratory (Ridley-Siebert et al, 2015). Experiment 5 in this thesis is a meta-analysis of the data in the past decade from our laboratory linking TFEQ-D and DDT. It is felt that the relationship between TFEQ-D and discounting has been particularly mixed in our past research, so a meta-analysis was needed to integrate these findings. Interestingly, no correlation was found between the measures, but an overall interaction between exposure condition and

TFEQ-D across the studies included was significant, demonstrating that those high in TFEQ-D who have been exposed to an experimental manipulation act more impulsively on discounting measures.

Summary of Theoretical Framework

To understand the theoretical framework and paradigm under investigation, figure 1.4 is discussed below. Research demonstrates to some degree that impulsivity (as indexed by behavioural measures of different subtypes of impulsivity) is related to overeating behaviour (Hou et al, 2011). Other research has subsequently demonstrated that priming with either food or reward-cue exposure or consumption of a preload can lead to subsequently increased food or snack consumption (Westenhoefer, 1994), and that to some extent reward cue reactivity is associated with heightened impulsivity (Appelhans et al, 2011). This thesis therefore examines the following question: can exposure to a food cue or consumption of a preload not simply increase food consumption but actually modulate behavioural impulsivity performance, by which may act as a pathway to the increase consumption shown in previous work?

Neuropsychological research has demonstrated that exposure to reward cues may act to activate brain reward regions, and also regions associated with inhibition of responses, (Wang et al, 2014) which we could be posited to support the hypothesised modulation of inhibitory or impulsive task performance. Recent research from our laboratory has demonstrated that individuals high in TFEQ-D might be particularly susceptible to increased impulsive choice (delayed discounting) following food cue exposure, thus the possibly modulating role of TFEQ-D in cue exposure and modulated behavioural impulsivity. This thesis therefore aims to examine the extent to which impulsivity can be modulated by reward cue exposure, and the extent to which this modulation of impulsivity might interact with TFEQ-Disinhibition and TFEQ-Restraint (originally shown to interact with reward consumption leading to increased subsequent food intake). The figure below details a diagram of the theoretical framework under Examination throughout this thesis. Pathway A links overeating and impulsivity, a relationship that has been discussed at length in this introduction, B links cue exposure and overeating, an established pathway within the appetite literature (e.g. Federoff,

1997), and C represents the focus of this these – an attempt at understanding the relationship between cue exposure/preload consumption and behavioural impulsivity, and the exploration as to whether this relationship is moderated by TFEQ-Disinhibition.

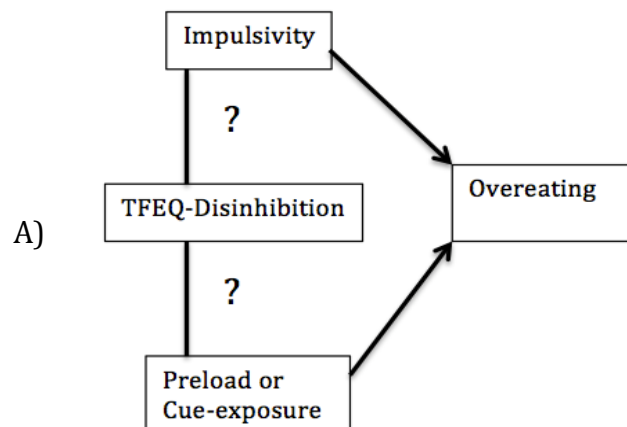


Figure 1.4 Theoretical model under examination,
Understanding the relationship between cue/preload exposure
And impulsivity, and the possible moderation through TFEQ-D
(Point A)

Taken together, the experiments in this thesis aid the current understanding of the possible state nature of impulsivity and its subtypes, and it is felt that there are extremely valuable methodological implications and suggestions for the implemented paradigms discussed. Although this thesis highlighted some mixed findings between measures, it also lays the foundations for experimental research into the area of inducing-impulsivity, and how this is associated with aspects of eating behaviours.

Experiment 1: Cued impulsivity: testing an alternative cognitive model of disinhibited eating

2.0 Introduction

The growth of obesity in recent decades is one that places an immeasurable burden not only in terms of the individual, but also in economic and financial terms through the use of resources and health support (Yang & Hall, 2007). What is of more concern is that this growth is not showing signs of a halt, in what is being called the 'Obesity epidemic' (James et al, 2001). For example, in the United States alone in 2010, there was an estimated 40 million individuals classed as obese, and recent research suggests that without effective intervention, that figure could double by 2050 (Fakhouri et al, 2012).

One of the theories central to the understanding of this 'obesity epidemic' is the concept of the 'obesogenic environment' (Egger & Swinburg, 2007). This idea suggests that in the most part, there is ready and cheap access to high-fat, high-sugar foods in contrast to past times of scarcity (Egger & Swinburn, 1997). Despite the compelling nature of the obesogenic environment theory of obesity, it is an environment in which all of us are contained, but yet not everyone within it is overeating or becoming obese. It is due to this shortcoming that other research has attempted to identify possible genetic factors that may reveal susceptibilities to this energy-dense environment. However, such genetic research has proven unclear, with a vast number of genes implicated in their possible role in obesity (Bell et al., 2005; Chung & Leibel et al., 2012). Due to the complexity in the genetic basis of obesity, we cannot yet be reasonably certain as to the genetic underpinning of the condition. What has seemed to be more fruitful than exploring the genetics of obesity has been the examination of possible behavioural expressions of such traits that may underpin individual differences in the susceptibility to obesity.

One particular focus of investigation that has seemed to prove useful in understanding overeating is the role of impulsivity. There is considerable evidence supporting the association between impulsive behaviours and overeating (Nederkoorn et al. 2006). This evidence comes from not only from work with obese populations and a tendency towards impulsive behaviour (e.g. Epstein et al, 1996; Nederkoorn et al, 2006), but

critically also with healthy populations which seems to link dysfunctional eating to impulsive behaviour (e.g. Guerrieri et al, 2007). The link between overeating and impulsive behaviours is also supported via the understanding of ADHD, a condition of which the population are traditionally considered 'impulsive'. There seems to be consistent evidence that those from the ADHD population have a greater rate of overeating and subsequent obesity (Holtkamp et al, 2004; Davis et al, 2006), potentially implicating impulsivity in these eating behaviours.

2.1 Impulsivity and cue-interactions

Although it seems that impulsivity and its subtypes do have a role in understanding eating behaviour, this study also attempts to examine the interaction between impulsivity and reward (specifically food) cues. Much work has assessed the potentially 'state' nature of impulsive nature in terms of exposure of substance abusers with associated cues (Doran et al, 2007) and also specifically the role of impulsivity and food-cue exposure in food intake (Larsen et al, 2012). Herman & Polivy (1985) classically demonstrated this food-consumption and overeating interaction effect. The researchers suggested that the consumption of a milkshake preload subsequently lead to overeating in restrained eaters, forcibly breaking their dietary restraint. This type of effect has been expanded with the use of both restrained and disinhibited eaters, in what Westenhoefer (1994) called 'the disinhibition effect', e.g. dietary disinhibition induced by the consumption of a dietary-boundary breaking preload.

Although research has suggested that 'impulsive' individuals are prone to over-eating during an intake task (Guerrieri et al, 2009), or that high-calorie preloads can induce later food consumption in high restraint, high disinhibition participants (Westenhoefer, 1994), this study posits an alternative approach. This study suggests that exposure to food cues (in this case, consuming a preload) may interact with eating attitudes (restraint and disinhibition, TFEQ) in inducing behavioural state impulsivity, not in terms of food-intake, but in terms of general tasks assessing subtypes of impulsivity. This position is supported by the previous work by Yeomans and Brace (2015) who found that individuals scoring highly on TFEQ-Disinhibition were significantly more impulsive on a delayed discounting task only when previously exposed to food cues. In terms of our theoretical model, this experiment aims to examine the extent to which a

milkshake preload may modulate behavioural impulsivity (inhibition, discounting, and reflection impulsivity) rather than overeating behaviour as shown through counter-regulatory eating (Herman & Mack, 1975), and the extent to which this modulation is specific to those high in both disinhibition and restraint scores (failed dieters) of the TFEQ - which Westenhoefer discussed as 'disinhibition effect'.

2.2 Hypotheses:

H: Those scoring high on TFEQ-Disinhibition (HD) will be significantly more impulsive (greater GoStop stop-latency (H1), steeper DDT/PDT area under-the-curve (H2), and greater MFFT i-score (H3)) than those in the Low Disinhibition groups, and this effect will be enhanced with the consumption of the preload in the experimental group (H4). As an exploratory analysis, we also explore the 3-way interaction between TFEQ-D, TFEQ-R and condition on outcome impulsivity variables.

2.3 Method

2.3.1 Design

A between-participants design was used to examine the interactive role of restraint and disinhibition on behavioural impulsive scoring in participants with or without the consumption of a preload. Participants were categorised into either high or low TFEQ-Restraint and TFEQ-Disinhibition according to the criteria suggested in Westenhoefer et al (1994) using median split (6 for R and D). A 2 (High/Low Restraint/Disinhibition) x 2 (experimental vs. control condition) ANOVA was used to examine this interaction in terms of the 3 types of behavioural impulsivity measured, and planned comparisons were conducted. Pearson's R correlational analysis was also used to examine the intercorrelation between variables of interest.

2.3.2 Participants

100 female participants took part in the study ranging from ages 18-46 [$M=21.29$, $SD=3.71$], with BMI ranging from 18-30 [$M=23.25$, $SD=3.29$], one participant was excluded due to their BMI not meeting our criteria ($BMI > 30$). Participants were recruited through the University of Sussex internal experimental advertising system (SONA). In order to participate in the study, participants must have previously completed an appetite recruitment questionnaire, containing the TFEQ and any allergy or aversion to food details. This was done in order to exclude any participants within the experimental group if they met the exclusion criteria, and to also analyse the TFEQ restraint and disinhibition scores in the control group without providing them with direct food cues. When comparing experimental condition, TFEQ-D and TFEQ-R, analysis is conducted on a cell size of 12/13 participants.

2.3.3 Materials

Go/Stop Paradigm (Dougherty et al. 2003)

The computerised Go/Stop task was used as a measure of inhibitory control, or the ability to inhibit a response on a visual 'Go' cue immediately followed by a 'Stop' cue. The task presents participants with a five-digit number sequence followed by either the same number sequence ('Go' cue), the same number sequence which turns red ('Stop') or a novel number sequence. The rate at which the go cue changes to red is adjusted faster or slower by 25 milliseconds by the task, from the starting rate of 200 milliseconds, depending on the inhibitory performance by the participant. Participants were exposed to two blocks of 64 trials with a 30 second rest between blocks, a figure which was deemed appropriate due to the reliability in GoStop data and impulsivity measures in Leitch (2009). With regard to the frequency of different trial types, 50% were novel trials, with 25% of the trials being Stop trials, and 25% being Go trials.

Within this task, there are several variables of interest. Due to the adjusting nature of the task, Dougherty et al (2003) have suggested that the most reliable data is the point at which participants' have the ability to inhibit their responses 50% of the time, as it represents a 'tie' between inhibition and response. Within this set of 50% inhibition

trials, some research has assessed what is termed as 'stop –latency' as the pertinent variable. Stop-latency represents the point at which the participant responds after a stop-signal has been presented, therefore being a measure of 'the speed of the inhibitory response' (Dougherty et al). However, other studies have cited their variable of interest as simply the ratio of Go's on Stop trials to Go's on Go trials (Dougherty et al. 2010). This task was utilised due to its unique ability to self-adjust stop-signal onset times by 25 milliseconds, as opposed to previous tasks which have used 50 millisecond adjustments (e.g. Jansen & Nederkoorn et al. 2009). It is thought that the smaller adjustments may have the ability to detect more sensitive differences in participants' inhibitory control responses. Due to the use of the adjusting version of this task, the dependent variable in use is stop-latency.

To remain transparent, it is important to establish set rules for when patterns of performance require an element of judgement. For example, there are occasions in this task when participants are performing at approximately 50% on more than on stop-interval. If this is the case, the stop latency that shall be adopted will be at the stop-interval which had the most trials, or adjacent to the stop-interval with the most trials if it is closer to a 50% inhibition rate. This is because at this point, it is said that the task is adjusting between stop-intervals on a close margin to the participants' 50% inhibition rate. However, should the number there need to be judgement on two similar stop-intervals, the stop-latency adopted will be the one in which the participant reached approximately a 50% inhibition rate with more responses in the time-period required (not late).

2.3.3.1 Delay Discounting and Probability Discounting Task (Baumann & Odum, 2012)

This task was used as a measure of participants' impulsive decision making, often referred to as 'Reward reactivity'. It was used to examine to what extent participants subjectively value a hypothetical larger monetary reward at the expense of either a delay in receiving it, or only a percentage chance of receiving it, versus a smaller but immediate or guaranteed reward.

2.3.3.1.1 Delay Discounting (DDT)

Programmed and launched via MATLAB (v. R2012b), this is a computerised version of Baumann & Odum's (2012) discounting task. This part of the task required participants to state their preference for either the hypothetical immediate short term reward or a hypothetical delayed larger reward. The immediate amount offered always starts at £50, versus £100. The task self-adjusts depending on the participants' choices. For example, the next immediate amount offered is half of the difference between the previous immediate and delayed amount. So for the next trial, depending on their previous choice, participants will be offered hypothetical £50 (+/- £25) versus hypothetical £100 delayed, and the next trial, either £25 or £75 (+/- £12.50 – half of the difference between the previous immediate and delayed amounts) and so on. The 'indifference point', the point at which participants become indifferent towards the immediate and delayed reward is deemed to be the tenth smaller, immediate amount offered. This process is repeated for 7 randomised delays of 1 day, 2 days, 1 week, 2 weeks, 1 month, 2 months and 6 years. Green & Myerson's (1996) Area-under the curve analysis, which has been successful in distinguishing between high and low impulsive choice participants was used as an outcome measure. This AUC ranges from 0 to 1, with 1 indicating the least possible impulsivity, and 0 signifying the most impulsive. $((x_2/x_1)[(y_1+y_2)/2] - x_1 \& x_2 \text{ represent parallel delays, and } y_1 \& y_2 \text{ represent the delays' subject})$.

2.3.3.1.2 Probability Discounting (PDT)

The probability-discounting task (Baumann & Odum, 2012) is based on the same paradigm and algorithm as the delay-discounting task. Participants are asked whether they'd prefer a smaller, guaranteed reward or risk it for a percentage chance of receiving a larger reward. The percentage chance corresponds to the 'delay' in the delay-discounting task. There are 7 randomised percentages of a guaranteed smaller reward versus 95%, 90%, 70%, 50%, 30%, 10% or 5% chance of a larger reward. As with the DDT version, the indifference point is the tenth offered smaller reward. Green & Myerson's AUC was again used as an outcome variable (as above).

2.3.3.2 Matching Familiar Figures Test (Cairns & Cammock, 1978)

This is a modified, 20-item computerised version of Kagan's original measure. The task consists of asking participants to identify the identical sample image with one of 6 similar images (see figure below). Participants are asked to complete two simple practice trials, and 30 recorded trials. This is considered an assessment of reflection impulsivity – the concept that highly impulsive individuals do not give themselves adequate opportunity to consider all of the available information before making a decision. The variables of interest in this study are reaction time before making a decision, number of errors on each trial, and the trade-off between these variables (Braet, 2007). To interpret the relationship between errors and latency, MFFT scores were converted into an i-score, the standardised errors minus the standardised mean time to first response/latency (Ze-ZI).

2.3.3.3 Mood Questionnaire

Participants were told in both the experimental and control group that the experiment was concerning 'mood and cognitive performance'. Therefore participants completed a mood questionnaire, which utilised a question in order to control for hunger in both groups. The questionnaire was conducted using the Sussex Ingestive Pattern Monitor (SIPM) and asked participants a series of questions about their current mood (calmness, clear-headedness, hunger, happiness, liveliness, fullness, nervousness and nausea) to which the participants had to rate their feelings on a visual analogue scale (VAS) ranging from 1-100 between two polarised statements on a fixed line. For example 'I am feeling (target word)' was presented, and participants are asked to move the cursor to the point on the scale that the feel is appropriate polarised between 'not at all (target word)' and 'extremely (target word)'.

2.3.3.4 Milkshake – Experimental Group.

Participants in the experimental group received a chocolate milkshake (466kcal total). This consists of 200g of Sainsbury's brand triple chocolate ice cream (338kcal), and 200g whole milk (128kcal), blended and refrigerated for one hour to ensure consistency

between milkshakes in viscosity. The milkshake was presented in a beige milkshake tumbler with a straw. The milkshake is based on disinhibiting preload milkshakes as used in Herman & Mack (1975), Polivy, Heatherton & Herman (1988), and similar to that used in Jansen & Nederkoorn et al. (2008).

2.3.5 Procedure

The experiment was approved by the University of Sussex Ethical Review Board. The study was advertised via SONA as a study assessing 'mood and cognitive performance'. This was used in order not to expose the control group to food cues. Therefore, once participants requested to take part in the study, they were randomly allocated to either the control or experimental condition. Participants in the control group continued to be told that the study examined mood and cognitive performance, whereas the experimental group were told that it was thought that hunger may play a role in mood, so a milkshake needed to be consumed to 'normalise hunger' across participants. Participants completed the TFEQ online at the point to which they signed up for future consideration for appetite studies in our laboratory. The difference in time between TFEQ completion and study participation varied greatly depending on their selection, eligibility, and point at which they decided to take part in the study.

Participants were excluded if they met any of the exclusion criteria, including: the use of regular medication (other than the contraceptive pill), smoking more than 5 cigarettes per week, allergies or aversions to foods used, currently pregnant or breastfeeding, diabetic or diagnosed with an eating disorder. If participants met the appropriate criteria, they were asked to come to the University of Sussex Psychopharmacology Laboratory at a timeslot between 3-5pm on a convenient day. Participants were then asked to complete a mood rating (SIPM) before consuming the milkshake in the experimental condition, or waited for the set of tasks to begin. All participants completed the DDT/PDT, GoStop and MFFT in a random order, which was recorded. Following completion, which took approximately 20-25 minutes, all participants were debriefed about the nature of the experiment and thanked for their time. Participants were given a choice of either 2 course credits, or £2 cash for their participation.

2.3.6 Statistical Analyses

The results of this study are analysed using a 2 (milkshake preload vs. no preload) x 2 (high vs. low TFEQ-D) x 2 (high vs. low TFEQ-R) ANOVA. Pre-preload hunger is used as a covariate, but removed where the covariate is not a significant one.

2.4 Results

2.4.1 Preliminary Analyses

Independent samples t-tests were conducted to check pre-existing differences between conditions in BMI, hunger, TFEQ-D and TFEQ-R.

There was no significant difference between the experimental (M=22.8, SD=2.8) and control condition [M=23.8, SD= 3.6] in BMI, [t(90)=-1.55, p = .13], nor was there any significant difference between the experimental (M=40.6, SD=21.1) and control condition [M=39.0, SD= 27.6] in hunger, [t(88)=-31, p = .76].

There was no significant difference between the experimental (M=8.7, SD=5.7) and control condition [M=8.9, SD= 5.4] in TFEQ-R, [t(93)=-23, p = .82.] There was also no significant difference between the experimental [[M=7.0, SD=3.4] and control condition [M=7.7, SD= 3.4] in TFEQ-D, [t(93)=-.92, p = .36].

2.4.2 Condition and Impulsivity Measure ANOVAs and correlational analysis

Women responded more impulsively on the DDT after consuming the preload [DDT AUC 0.47 ± 0.04] than without the preload [DDT AUC 0.60 ± 0.04 : $F(1,84) = 5.19$, $p=0.03$, $\eta^2 = .05$]. However, the effect of condition did not depend on either classification of women in terms of scores on the TFEQ-D [$F(1,84) < 1$, $p=.95$) or TFEQ-R ($F(1,84) < 1$, $p=.66$], and likewise performance on the DDT did not differ significantly overall depending on TFEQ R*TFEQ-D [$F(1,84) < 1$, $p=.81$, or TFEQ-D*Condition ($F(1,84)=1.21$, $p=.27$], or TFEQ-D*TFEQ-R*condition interaction [$F(1,84) < 1$, $p=.97$]. Neither BMI nor hunger at the start of testing were significant covariates in these analyses (Table 2 A and Figure 2).

There was no significant difference between the experimental ($M = .32$, $SD = .03$) and control conditions [$M = .39$, $SD = .030$] on their respective PDT AUC scores [$F(1,85) = 2.54$, $p = .11$, $\eta^2 = .03$] when including TFEQ groups in the model. There was no significant effect of TFEQ group interaction [$F(1,85) < 1$, $p = .46$], TFEQ-D [$F(1,85) < 1$, $p = .48$], TFEQ-R [$F(1,85) = 1.21$, $p = .28$], or TFEQ-D*Condition interaction [$F(1,85) < 1$, $p = .77$], or TFEQ-D*TFEQ-R*condition interaction [$F(1,85) < 1$, $p = .98$]. Hunger and BMI were non-significant covariates ($p > .05$) so were removed from the analysis.

GoStop data were non-normally distributed, so a Log10 transformation was used to rectify the distribution. There was a significant difference between the experimental [$M = 188.98\text{ms}$, $SD = 18.88$] and control condition [$M = 137.97\text{ms}$, $SD = 16.82$] on the GoStop task, with those consuming the milkshake preload displaying poorer inhibitory control [$F(1,81) = 5.65$, $p = .020$, $\eta^2 = .07$]. There was no significant effect of TFEQ-groups interaction [$F(3,83) < 1$, $p = .48$], TFEQ-D [$F(1,81) < 1$, $p = .84$], TFEQ-R [$F(1,81) = 1.32$, $p = .25$], TFEQ-D*condition [$F(1,81) < 1$, $p = .77$] and no significant TFEQ-group*condition interaction [$F(3,83) = 1.49$, $p = .23$]. Hunger and BMI were non-significant covariates at $p < .05$ so were removed from the analysis (Table 2 B and Figure 2).

There was no effect of condition on i-score [$F(1,88) = 1.07$, $p = .30$], and no effect of TFEQ-R and TFEQ-D groups interaction [$F(3,88) = 1.3$, $p = .26$] or TFEQ-group*condition interaction [$F(3,88) = 1.20$, $p = .28$]. Hunger and BMI were non-significant covariates at $p < .05$ so were removed from the analysis (Table 2C and Figure 2).

Tables 2: Means and SEM for conditions between high and low TFEQ-D and TFEQ-D and TFEQ-R interaction groups for A) DDT AUC, B) GoStop Stop Latency and C) MFFT i-score.

A)	Milkshake	No Milkshake
Condition Means	0.47±.04	0.60±.04
Low TFEQ-D	0.50±.05	0.57±.06
High TFEQ-D	0.44±.44	0.63±.57
LDLR	0.51±.06	0.57±.09
LDHR	0.50±.07	0.58±.07
HDLR	0.43±.11	0.60±.08
HRHR	0.45±.07	0.66±.08

B)	Milkshake	No Milkshake
Condition Means	195.0±18.25	136.6±16.42
Low TFEQ-D	196.1±20.27	130.6±23.24
High TFEQ-D	193.9±30.36	142.7±23.20
LDLR	162.1±26.54	119.0±37.54
LDHR	230.1±30.65	142.2±27.42
HDLR	207.3±53.09	118.5±33.58
HRHR	180.5±29.45	166.9±32.01

C)	Milkshake	No Milkshake
Condition Means	-.21±.27	.19±.26
Low TFEQ-D	0.02±.32	0.12±.38
High TFEQ-D	-0.43±.43	0.25±.37
LDLR	-0.03±.41	-0.13±.59
LDHR	0.07±.49	0.37±.49
HDLR	-0.47±.72	0.83±.53
HRHR	-0.39±.47	-0.33±.51

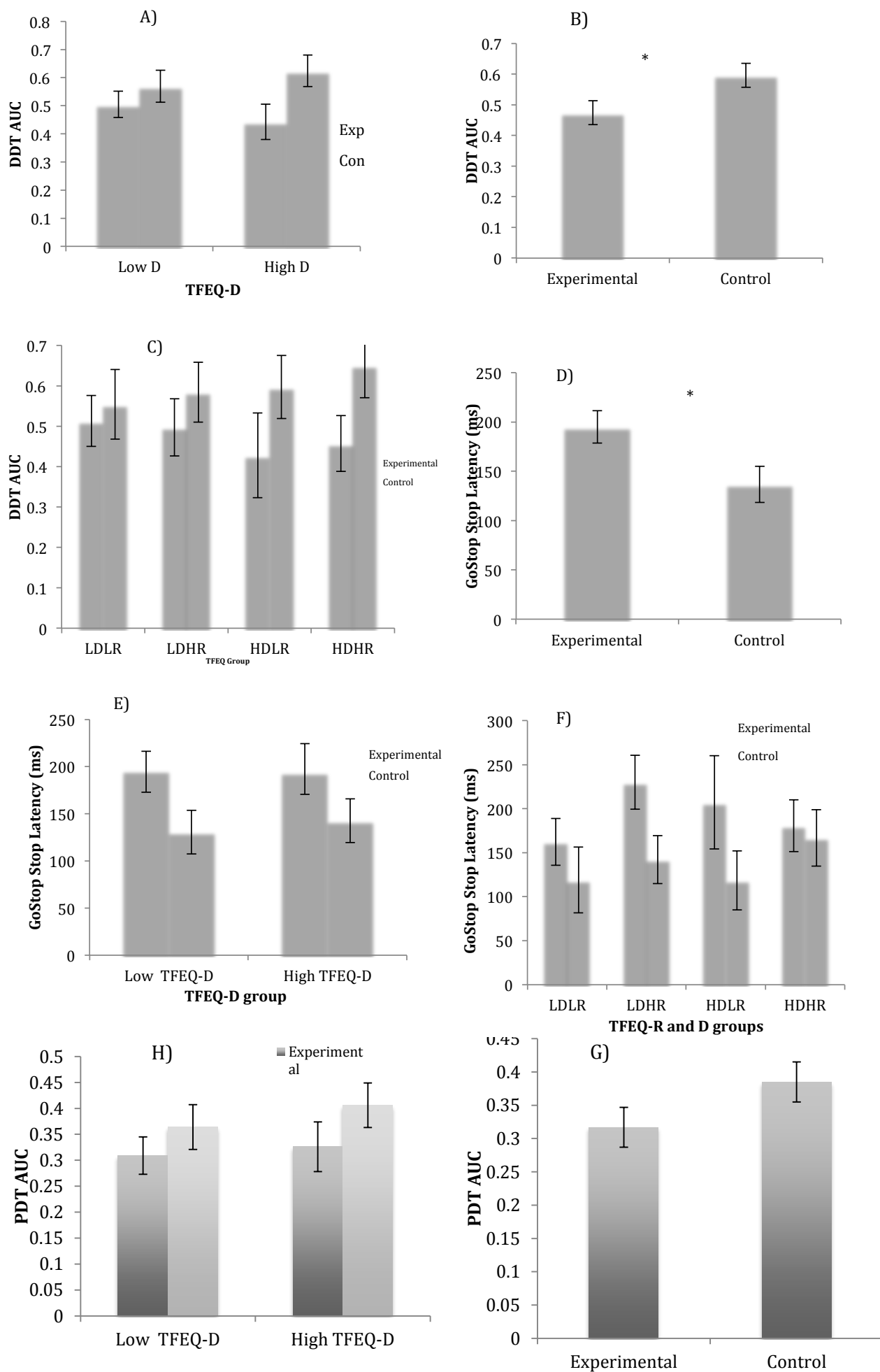


Figure 2: Error bars indicate 1 +/- SEM A) DDT scores of TFEQ-D groups between conditions, B) DDT scores between condition, C) DDT scores of TFEQ-D/R groups, D) GoStop Latency (ms) scores between conditions, E) GoStop SL scores (ms) of TFEQ-D groups between conditions, F), GoStop SL (ms) scores of TFEQ-D/R groups between conditions, G) PDT AUC scores between conditions, H) PDT AUC scores of TFEQ-groups between conditions. * $p < 0.05$.

2.5 Discussion

This study found that participants who consumed a preload were significantly more impulsive on delayed discounting and inhibitory control tasks, but not reflection impulsivity tasks. This effect was found independent of TFEQ scores and their interactive effects. This suggests that following the consumption of the preload, participants are poorer at controlling their inhibitory responses, and consistently prefer smaller immediate rewards versus delaying their gratification for larger rewards. Although what is termed as ‘the disinhibition effect’ (Westenhoefer, 1994) has been demonstrated in ad libitum eating tasks following preload consumption (Herman & Mack, 1975), little research has demonstrated this disinhibition effect on general behavioural impulsivity. However, we did not observe this effect on reflection impulsivity, which will be explored further later. Nonetheless, these results of this experiment support the previous work in this laboratory (e.g. Yeomans et al, 2013) and the concept of behavioural impulsivity subtypes being manipulated across states.

So although research has suggested that ‘impulsive’ individuals over-consume (e.g. Guerrieri et al, 1999), and that preload consumption increases food intake, this study integrates the two ideas – preload consumption can induce general impulsiveness, specifically the inability to delay gratification and poorer ability to control inhibitory processes. It is thought that this finding may reflect an activation of dopamine reward pathways. For example, a great deal of work has assessed the potential of hedonic food rewards to activate dopamine reward pathways, in this case the ‘olfactory and gustatory stimuli of food’ (Wise, 2006). From this, it is possible to discuss dopaminergic pathway activation as the initiation of motivational processes, with vast supporting evidence from animal evidence of motivational differences induced through dopamine antagonists (e.g. de Wit & Wise, 1977). Given this possible food consumption-dopamine pathway activation explanation, it is of no surprise that delayed discounting and inhibitory control measures reflect these changes in motivation. For example, multiple studies have demonstrated the role of dopamine in both inhibitory control and delay of gratification (Dalley & Roiser, 2012). From this, it is feasible to suggest that

consumption of food reward may activate dopamine reward circuitry, thus compromising inhibitory control and also the ability to delay gratification.

It was interesting here, and indeed unexpected, that reflection impulsivity was not modified by preload consumption, given both the previous literature implicating overeating behaviour and reflection impulsivity (Braet et al. 2007), and the link here between preload consumption and inhibitory control and delayed discounting measures. It is possible that reflection impulsivity may not be underpinned by this motivational reward-circuit mechanism, and may represent a more cognitive, decision-making process. However it is also possible that this result is task specific, and it may be appropriate to examine reflection impulsivity in this paradigm with both the MFFT and the information-sampling task (Clarke, 2009), which may examine reflection impulsivity without the critiques often applied to the MFFT. It would also be of great interest to examine how these task differences are unpicked in a multiple preload design, which would allow a fuller understanding of the underlying mechanisms of reflection impulsivity without questioning task-specificity.

This research also seems to suggest a lack of specificity to the reward cue. For example, although the preload increased some aspects of impulsive behavior, with regard to later consumption of other food rewards, e.g. in participants with disinhibited and restrained eating behaviours (Westenhoefer, 1994), the results of this experiment suggest that consumption of a food reward potentially acts as a general behavioural disinhibitor. Although we demonstrated this preload effect, contrary to our hypotheses, there was no effect or interactive effect of TFEQ disinhibition or restraint. These hypotheses were generated in response to the significant role TFEQ variables have had in previous impulsivity research (Yeomans et al, 2008), but also the work of Yeomans & Brace (2015) demonstrating the manipulation of behavioural impulsivity through the exposure of food-cues versus a non-food-cue exposed control group. However, in this study, it is possible that the consumption rather than food-cues alone could produce a stronger effect in general, and therefore the role of TFEQ scores becomes less detectable. This finding is one that seems unusual given past research linking impulsivity and TFEQ scores (Yeomans et al. 2008), so this role is something that should be explored further in future.

One of the theoretical questions arising from this study is the nature of the relationship between delay and probability discounting, and the way in which this is interpreted. This study highlighted a positive correlation between probability and delay discounting AUC's. Traditionally, one would expect this relationship to be negative, with lower DDT AUC indicated lower impulsivity (preference for immediate rewards), and higher PDT AUC indicating greater impulsivity (preference to risk for larger rewards). These results, which are not experimentally unique (Baumann & Odum, 2010), possibly suggest that those who have preference for immediate rewards display this preference for 'immediacy' in their probability discounting choices – preferences for immediate reward despite the smaller nature against a riskier larger preference. Although theoretically this is logical, this explanation depends on the way in which probability discounting is interpreted, for example some have suggested that delayed discounting may itself represent probability discounting, in the sense that a longer delay signals a reduced probability of actually receiving the reward (Reynolds et al. 2004). Given these differences in possible interpretation, it would be useful to explore the dynamics between delay and probability discounting, and the nature of the possible murkiness between the concepts.

Although the milkshake-induced impulsivity provides an interesting insight into the malleability of inhibitory control and impulsive choice, there are limitations as to which conclusions can be drawn because of the nature of the preload. For example, as the control group selected did not consume anything prior to the behavioural tasks, it is not known at this stage whether the pertinence of the milkshake is due to its hedonic nature (thus supporting a reward-activation paradigm), the belief of calorie content possibly due to its sensory characteristics (which research has shown important in ad libitum designs, Mills & Palandra, 2006), or simply the consumption vs. no consumption design. It is felt that targeting this discrepancy in future work would aid our understanding of the specificity of reward-impulsivity cues, and may give an opportunity to dissociate the existing explanations.

Despite the unknowns arising from the preload-control design, the concept itself of state differences in behavioural impulsivity seems to be a positive result in terms of the 70

existing and on-going research examining impulsivity interventions or training. For example, several studies have attempted to either directly 'train' (Houben & Jansen, 2011) or manipulate (Guerrieri et al, 2009) inhibitory control towards 'food-related responses' (Houben & Jansen, 2011, p. 346). Given the results in this experiment that inhibitory control has the potential to be compromised by consumption, and previous work (Yeomans et al, 2013) linking inhibitory control reductions from cues alone, it would suggest that state-dependent interventions or training concepts could be supported, however the longitudinal aspects of these are far from being known.

Experiment 2: ‘The reinforcing value of palatable snack foods and its relationship to subtypes of behavioural and self-report impulsivity.’

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3.0 Introduction

In eating behaviour, the pleasurable (hedonic) and nutritional consequences of eating a particular food shape the extent to which we find these foods reinforcing, thus influencing our motivation to obtain and consume them. In some cases, the nature of the reinforcement and subsequent motivation is elastically ‘biologically pre-determined’ (Epstein, 2010), in the sense that bodily changes modify the strength of the reinforcer depending on physiological need (e.g. hunger, Cabanac, 1971). For example, when acutely deprived of access to food, the reinforcing value of food will be increased in a state of hunger, thus leading to greater motivation to obtain food.

The reinforcing value of food (RRV_{food}) refers to the extent to which someone is willing to work or allocate resources, in terms of time or effort, for food, and it has been suggested (e.g. Epstein et al, 2007) that RRV_{food} may be a useful behavioural measure of ‘wanting’ as defined in the ‘wanting vs. liking’ distinction from the incentive salience model of motivation (Berridge, 1997). The reinforcing value of food, as related to Berridge’s neurobiological ‘wanting’, is considered as a behavioural and motivational willingness to attain reward, as opposed to the subjective pleasure of experiencing it or ‘liking’. As discussed at length by Berridge & Robinson (2010), dopaminergic activity is a core neurobiological mechanism in the motivational acquisition of reward-seeking activities. There is a wealth of literature building on these core foundations of dopamine as an active agent in motivational behaviour, despite some remaining uncertainty about the underlying behavioural mechanisms.

The reinforcing value of a reward, in this case food, is conceptualised behaviourally as the extent to which our motivation drives us to obtain that reward. Existing reinforcing-value tasks using progressive variable (Epstein et al, 2008) or more commonly progressive-ratio scheduling tasks aim to examine the extent to which an individual is willing to allocate time or resources to obtain rewards: in the case of the present study palatable snack foods. The way that progressive-ratio tasks work is to ask the participant to work progressively harder to obtain reward, usually using a simple response such as pressing the keyboard spacebar or clicking a computer mouse. For example, participants might at first be required to make 20 clicks to obtain the food reward, then 40 clicks. Critically, the amount of clicks doubles following each receipt of a reward. The critical measure is the point at which the participant is no longer willing to work for the reward, the break-point. This value has been shown to have predictive value in eating research: Epstein's group and others have shown higher break-points using RRVfood tasks predict aspects of eating implicated in poor control: higher measures on RRVfood were related to higher ad libitum intake (Epstein et al., 2004a, Epstein et al., 2004b and Epstein et al., 2007b), and has been associated with obesity (e.g. Temple et al., 2008; Giesen et al, 2010).

One key question is how individual differences in RRVfood relate other factors that also may pre-dispose people to react to the opportunity for reinforcement, such as impulsivity. Although some studies have discussed this relationship, no study to date has systematically examined the relationship between RRVfood and the three main subtypes of behavioural impulsivity: inhibitory control, impulsive choice and reflection impulsivity. Epstein et al. (2010) suggest that delayed discounting (impulsive choice) and RRVfood, although fundamentally different behavioural models, could be integrated and developed into a model that encapsulates critical risk factors for understanding weight gain. There is an emerging body of evidence linking RRV to delayed discounting preferences. The work of Rollins et al (2010) suggests that there is a moderating relationship between delayed discounting and RRV on increased weight gain in nonobese individuals.

Taking a behavioural economic approach to human food choice and acquisition, and with much focus on impulsive choice through delayed discounting tasks; Carr & Epstein 73

(2012) describe a model of 'reinforcement pathology'. This concept refers to the interaction between motivational and executive systems, or top down and bottom up, with RRVfood indexing the motivational system, and executive referring to constructs of impulsivity, specifically inhibitory control. Extrapolating the link between preferences on delayed discounting tasks and RRV; this model proposes an interaction between RRV and impulse or inhibitory control. The authors suggest that dopamine reward pathway activation is associated with RRV (as discussed previously), and suggest that this activation may also in part be linked to reduced impulse control (Volkow, Wang and Fowler, 2008).

Although this provides a relatively interesting mechanism for understanding a possible relationship between RRV and inhibitory control as mediators of short-term overeating, that dopamine dependent incentive mechanisms may drive impulsive behaviour, there has been little behavioural work carried out directly examining this idea. The primary aim of the present study is therefore to investigate the inter-relationship between RRV and behavioural impulsivity as predictors of increased snack food consumption. Notably, most prior work using the RRV methodology has been conducted in a paediatric setting, often with obese children (Temple et al, 2008). The present study therefore also examines for the first time how RRV (as measured by progressive-ratio procedures), impulsivity and uncontrolled eating were inter-related in a normal weight, healthy adult population.

Additionally, although the association between RRV, inhibitory control and delayed discounting has been discussed and tested, the relationship between RRV and a third subtype of impulsivity, reflection impulsivity – the ability to reflect adequately on the available evidence before making a decision – has yet to be explored to our knowledge. Thus the present experiment was the first attempt to examine the relationship between behavioural motivation (as measured by RRV) and the three main behavioural constructs of impulsivity, in relation to increased snack consumption and scores on the disinhibition scale of the Three Factor Eating Questionnaire (TFEQ-D). TFEQ-D was included since it was previously found to moderate the relationship between RRVfood and 12-month weight gain (Carr et al, 2013). Given the literature suggesting that those high in both disinhibition and restraint (TFEQ-R) are characterised as most likely to

overeate or are ‘unsuccessful dieters’ (van Strien, 1999), this investigation aims to assess the interaction between these two components TFEQ-R and TFEQ-D, and their interactive product on RRV and other subtypes of impulsivity, not just disinhibition alone.

We hypothesized that higher scores on the RRV will be associated with faster delayed discounting and weaker inhibitory control, and given the association between TFEQ-D and reflection impulsivity (Leitch et al., 2013), we also predicted RRV to relate to this third component of impulsivity. We also hypothesize that dietary attitudes, RRV, delayed discounting and subtypes of impulsivity will be related to ad libitum intake.

3.1 Method:

3.1.1 Participants

Participants were 80 women between the ages of 18-35 who were students or staff at the University of Sussex. All approached participants had previously completed a recruitment questionnaire, which contained the TFEQ and dietary requirements and allergies. Participants were told that the study was about ‘snacking behaviour and cognitive performance’. Participants were ineligible to take part if they smoked (> 5 cigarettes per week), did not meet the BMI requirements (between 18-30), were taking regular medication, or were allergic to any ingredients in the snack foods used. All participants gave their written informed consent and were paid either £6 or 6 course credits for their participation. The University of Sussex ethical review committee approved the study. Participants were excluded based on either not responding throughout the GoStop task, or for non-systematic erroneous responding on the DDT in accordance with Bickel et al (2007).

3.1.2 Materials:

RRV Slot-Machine Task:

The relative reinforcement task (RRV) was in the form of a slot machine style game with 3 shapes that rotate on the screen. A point was earned each time the three shapes match in shape and colour. For every five points earned, the subject received a portion of his or her preferred snack food selected in the ad libitum task or 2 minutes of reading time, depending on which reward they chose to work for (participants could choose to work for food or reading time, or could alternate according to choice on separate windows of the software on the same monitor). The programmed reinforcement schedules for food and reading were progressive fixed-ratio schedules with response requirements of 4, 8, 16, 32, 64, 128, 256, 512 and so forth for each point, and 5 points were required to obtain a reward. This meant that in the FR 4 schedule, participants earned a point every 4 responses, meaning that they needed 20 responses to progress to the next schedule, and therefore earn a reward. Whenever the software informed participants when a reward was won, they were required to alert the experimenter using an external light activation switch from inside the cubicle, who provided the participant with the allocated reward portion (14-20g, 100kcal). Participants could end the task when they no longer wanted to earn either reward by contacting the researcher. Water was provided ad libitum.

The last reinforcement schedule (P_{max} , the dependent variable) was the last schedule at which subjects met requirements for 1 point towards either reward, and the proportion of responses for food compared with the alternative (RRV_{prop}) was calculated $(P_{max \text{ food}})/(P_{max \text{ food}} + P_{max \text{ reading}})$ as the dependent variable to have a metric for understanding the reinforcing nature of food versus and non-food alternative, the RRV_{food} vs. $RRV_{reading}$.

Snack foods used in taste test and in RRV_{food} task:

The snack foods used in the taste test and RRV_{food} task were Walker's ready salted crisps (Walkers, UK), cheese corn tortilla chips (Sainsbury's, UK), M&M's (Mars, USA) and chocolate buttons (Sainsbury's, UK), See table 1 for nutritional information.

Table 3.0: Nutritional Information of the snack foods used (per 100g)

	Crisps	Chocolate Buttons	Tortilla Chips	M&Ms
Calories (Kcal)	526	542	492	485
Fat (g)	31.9	31.3	23.6	20.5
Carbohydrates (g)	51.5	56.0	60.7	68.8
Protein (g)	6.1	7.5	6.4	4.8
Salt (g)	1.4	0.3	1.27	0.1

Impulsivity Measures:

Go/Stop Paradigm (Dougherty et al. 2003)

The computerised Go/Stop task was used as a measure of inhibitory control, or the ability to inhibit a response on a visual 'Go' cue immediately followed by a 'Stop' cue. The task presents participants with a five-digit number sequence (500ms) followed by the same number sequence ('Go' cue), the same number sequence that turns red ('Stop') or a novel number sequence. The rate at which the go cue changes to red is adjusted faster or slower by 25 milliseconds by the task, from the starting rate of 200 milliseconds, depending on the inhibitory performance by the participant. Participants were exposed to two blocks of 64 trials with a 30 second rest between blocks, with an interval of 1500ms between trials, a figure which was deemed appropriate due to the reliability in GoStop data and impulsivity measures in Leitch (2009). With regard to the frequency of different trial types, 50% were novel trials, with 25% of the trials being Stop trials, and 25% being Go trials. Due to the adjusting nature of the task, Dougherty et al (2003) have suggested that the most reliable data is the point at which participants' have the ability to inhibit their responses 50% of the time, as it represents a 'tie' between inhibition and response. Within this set of 50% inhibition trials, some research has assessed what is termed as 'stop -latency' as the pertinent variable.

Delay Discounting Task (Baumann & Odum, 2012)

Programmed and launched via MATLAB (v. R2012b, Windows XP OS, Dell Computer), the DDT task was computerized version of Baumann & Odum's (2012) discounting task. This task required participants to choose their preference for either a immediate reward or a delayed larger hypothetical reward. The immediate amount offered starts at £50, versus £100. The task adjusts depending on the participants' choices. The next immediate amount offered is half of the difference between the previous immediate and delayed amount. So for the next trial, participants will offered £50 (+/- £25) versus £100 delayed, and the next trial, either £25 or £75 (+/- £12.50 – half of the difference between the previous immediate and delayed amounts) and so on. The reward offered were always hypothetical. The 'indifference point', the points at which participants become indifferent towards the immediate and delayed reward is deemed to be the tenth immediate amount offered. This process is repeated for 7 randomised delays of 1 day, 7 days, 30 days, 90 days, 180 days and 365 days. Green, Fry & Myerson's (1994) Area-under the curve analysis was used as an outcome measure. This AUC ranges from 0 to 1, with 1 indicating the least possible impulsivity, and 0 signifying the most impulsive. $((x_2/x_1)[(y_1+y_2)/2] - x_1 \& x_2$ represent parallel delays, and $y_1 \& y_2$ represent the delays' subject).

Information Sampling Task (Clark et al, 2003)

Participants are presented with a 5x5 grid matrix and are told that beneath each square on the grid is one of two colours (image in Appendices). Participants are told to open as many boxes on the grid by clicking the mouse on the selected box until they feel confident to make a decision as to the overall majority colour on the grid. Participants were awarded 100 points for a correct majority decision and lost 100 points for an incorrect decision. Participants were made aware of the outcome of each trials with the relevant message displayed to them: "Correct! You have won [x] points" or "Wrong! You have lost 100 points" which was presented for 2 seconds. The task consisted of 10 trials, with a variable delay of 1 second between trials. The dependent variable in this task is the average number of boxes opened per trial (Clark et al, 2003).

Behavioural Activation System/Behavioural Inhibition System (BIS/BAS, Carver and White, 1994)

20-item questionnaire, with 4 subscales. The BIS subscale measures participant expectancies and aversions to impending punishment, and the BAS and its three subscales (drive, reward responsiveness and fun-seeking) measures reward approach, motivation and intention behaviour. Each question is rated on a four-point likert scale and all items are reverse-scored except items 2 and 22. The total BAS score is calculated by summing its relevant subscales, comprising of 13 items.

Mood Questionnaire

Participants were told in both the experimental and control group that the experiment was concerning 'mood and cognitive performance'. Therefore participants completed a mood questionnaire, which utilised a question in order to control for hunger. The questionnaire was conducted using the Sussex Ingestive Pattern Monitor (SIPM, Window XP OS, Dell Computer) and asked participants are series of questions about their current mood (calmness, clear-headedness, hunger, happiness, liveliness, fullness, nervousness and nausea) to which the participants had to rate their feelings on a 100-point visual analogue scale (VAS) between two polarised statements on a fixed line. For example 'I am feeling (target word)' was presented, and participants were asked to move the cursor to the point on the scale that the feel is appropriate polarised between 'not at all (target word)' and 'extremely (target word)'.

3.1.3 Procedure:

Participants were recruited using the University of Sussex subject online participation pool advertisements. 80 non-smoking female participants were scheduled in for a taste-test of snack foods on the first occasion, with their highest liking rating used as the food reward in the RRV task. This taste test also acted as an ad libitum snack intake measure. Participants were then scheduled into testing times for a second session between 2pm-5pm approximately 1 week later, in accordance with Epstein et al (2012). Participants then completed the randomized battery of 3 impulsivity tasks in a randomized order and shortly after, completed

the RRV slot machine task. Participants chose to click on a progressive fixed interval ratio schedule for snacks or reading time alternatively in two separate forms of the slot machine task on the same CPU monitor, to give a measure of the relative reinforcing value of food versus a non-food alternative (see RRV task details). When participants completed each reinforcement schedule, they would activate a light external to the cubicle so the researcher could provide them with the snacks or reading material depending on which task they chose to complete. Participants ended the task when they no longer wanted to participate in the task for snacks or reading time. Participants then completed the BIS/BAS questionnaire on study and a measure of their height, weight and age was taken. Participants were debriefed and thanked for their time. Participants were compensated with £6 or 6 course credits for participation.

3.1.4 Statistical Analyses:

Multiple regression models were constructed using RRVprop, and TFEQ-D and R and subtypes of impulsivity and ad libitum intake in the taste test. Subsequently, regression analysis was run to examine the role of absolute responding rate across FR schedules, and continuous TFEQ measures on behavioural and self-report impulsivity, and the continuous nature of BIS/BAS subscales.

3.2 Results

3.2.1 Descriptive Statistics

Descriptive statistics shown below for participant demographic data, and variables used in model selection and analyses.

Table 3.1: Variables of Model Interest Means and SDs.

Variable	Mean (+/- SD)
TFEQ-Disinhibition	7.81 ± 4.31
TFEQ-Restraint	6.96 ± 3.05
BMI	22.92 ± 2.67
DDT	.50 ± .20
GoStop Stop Latency	170.24 ± 94.15
IST Mean Boxes Opened	13.91 ± 5.31
RRVprop	.68 ± .24
Grams Eaten In Taste Test	32.76 ± 23.01
Hunger	54.37± 17.67
Calories Eaten in Taste Test (kcal)	166.22 ± 166.70

3.2.2 Model selection

Models were constructed to predict each dependent variable of interest (RRV, and total calories consumed ad libitum) in two stages. Hierarchical models were constructed with eating attitudes and BMI, followed by the behavioural impulsivity measures and RRV scores, with the third step of the mode then also including BIS and BAS total scores from the BIS/BAS self-report measure. Variables with which we have a theoretical reason to believe to have a critical impact on other model variables were always present in the final model. These variables consist of the two TFEQ measures and their interaction term, and BMI, all of which have been shown to be associated in some capacity to the other predictors in the model and the dependent variables. Hunger was uncorrelated with any other variables ($P > .45$) so was not included in the regression analysis. GoStop scores violated normality assumptions and were log10 transformed to rectify this. The intercorrelation between TFEQ-D and TFEQ-R, and TFEQ-D and RRV was high ($R = .234$, $P < .05$) so the TFEQ-D/RRV interaction term was removed from the model. Model tables included as Tables in appendices (Tables A.1 and A.2).

3.2.3 Models Predicting RRV food

In predicting RRVfood, the first model including TFEQ-R, TFEQ-D, their product, and BMI (Table 3), no variables significantly predicted the outcome variable (all $p < .05$) and the model did not account for a significant amount of variance ($F(4, 61) = .291, P > .05$). The second model, adding impulsivity variables DDT, Go-Stop Stop latency, and IST mean boxes opened did not significantly predict RRV ($F(7, 61) = .702, P > .05$, all predictors $P > .05$). The final model, adding BAS and BIS total scores, did not significantly predict RRV ($F(9, 61) = 1.120, P > .05$), however BIS total trended as a significant predictor of RRV (Beta = -1.98, $P = .053$).

3.2.4 Models Predicting Total Calories Consumed

In predicting total calories consumed [Table 4], the first model including TFEQ-R, TFEQ-D, their product, BMI, and RRV food, the model did not account for a significant amount of variance [$F(4, 61) = .693, P > .05$], and no variables significantly predicted calories consumed [$P > .05$]. The second model, adding impulsivity variables DDT, Go-Stop Stop latency, and IST mean boxes opened did not significantly predict RRV [$F(8, 61) = 1.190, P > .05$] and no variables significantly predicted calories consumed [$P > .05$] other than RRV food [Figure 1, Beta = .280 $p = .04$]. The final model, adding BAS

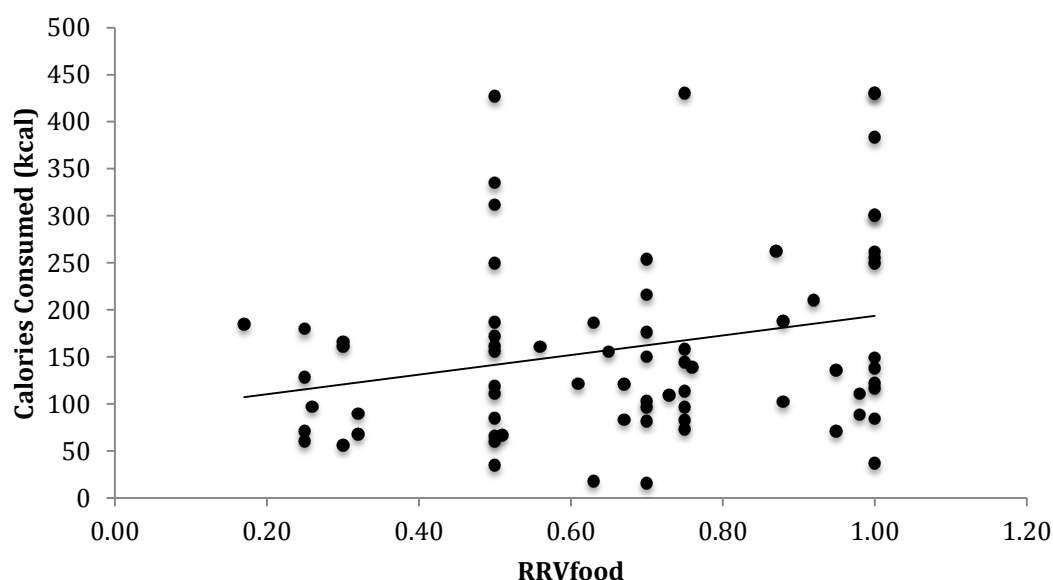


Figure 3.1: Relationship between RRVfood and Calories consumed in taste test (kcal), analysis shown in Appendix Tables A1 and A2 coefficients

and BIS total scores, did not significantly predict RRV [$F(10,61) = 1.143, P > .05$], and no variables predicted calories consumed other than the trending RRV food [$Beta = .241, p = .09$].

3.2.5 Models Predicting Total Grams Consumed

In predicting total grams consumed [Table 5], the first model including TFEQ-R, TFEQ-D, their product, BMI, and RRV food, the model did not account for a significant amount of variance [$F(4, 61) = .693, P > .05$], and no variables significantly predicted grams consumed [$P > .05$]. The second model, adding impulsivity variables DDT, Go-Stop Stop latency, and IST mean boxes opened did not significantly predict RRV [$F(8,61) = 1.158, P > .05$] and no variables significantly predicted grams consumed [$P > .05$] other than RRV food [$Beta = .239, p = .042$]. The final model, adding BAS and BIS total scores, did not significantly predict RRV [$F(10,61) = 1.104, P > .05$], and no variables predicted grams consumed other than the trending RRV food [$Beta = .241, p = .095$].

3.3 Discussion

As discussed, little work has examined the relationship between RRV and facets of impulsivity except for delayed discounting. However, the model assessing this relationship here failed to replicate those findings, since DDT did not account for a significant amount of variance in RRV scores. The only factor reaching significance in the model predicting RRV scores was the BIS of BIS/BAS. Carver and White (1994) suggest that BIS subscales underpin an individual's motivational avoidance of negative stimuli and situations. This seems to be an unexpected association with RRV. However this relationship was negative, suggesting that those low on the BIS (low avoidance) were willing to work harder for snack foods. Could it be that the trade-off between working for food and exiting the task created a 'conflict' interacting with BIS scores (Berkman et al, 2009)? This would suggest that those high in BIS would have found the food reinforcement task as conflicting, which seems an unlikely interpretation, but further work needs to potentially assess BIS in food reinforcement. However if

this were to be considered an explanation, it would make sense to further examine the levels of arousal and/or stress throughout studies of this nature as this may be a moderating factor in the relationship between BIS and RRV, particularly due to previous work on the disinhibiting nature of stress manipulations (Wallis & Heatherington, 2009). It may however as Berkman et al (2009) suggest, be a product of situational or decision-related conflict, but the mechanism for which is currently unknown. It has to be noted that rates of responding for both rewards were considerably lower than that of work that has also implemented these types of designs (Epstein et al, 2010). This may suggest that it is not necessarily that food or reading time is not a reinforcing reward, but maybe that leaving the experiment is a greater reinforcer, where participants can indulge in rewards of their choice without the necessity to work for such a reward. This may be a product of using a design commonly used with children with adult participants. Therefore in these types of design, it is recommended here to evaluate the reinforcing value of leaving the experiment when implementing this methodology. The limitations of this methodology and the implications of this are discussed further in the study.

The second factor assessed was the total grams and calories consumed in the pre-experiment snack foods, which has been shown to be an applied correlate to the reinforcing value of food (see Epstein et al, 2008 for a review of this evidence). Unexpectedly, no models significantly predicted snack food intake, but RRV_{food} trended as predictive of snack food intake. This therefore suggests that the extent to which an individual finds a snack food reinforcing (higher RRV), the more they will consume when given free access to it ad libitum. This supports the work of Epstein et al, and this not only supports the task as a possible reflection of short term snacking behaviour, but also reveals an interesting insight into the behavioural correlates of the reinforcing value of snack food rewards.

However it is possible that there are limitations with the task methodology. For example, the overall mean intake in the snack session were low (32.7g), which may not have reflected snacking behaviour accurately thus effecting variable

relationships. This suppression of intake may have been caused by the calling of the session a 'snack tasting session', which potentially alludes to consumption of smaller amounts, despite participants being told that they could eat as much as they wanted. Future designs of this kind should bear in mind these possible semantic effects, and it would be fruitful to explore these relationships without this type of constraint.

3.3.1 Limitations

Although this experiment provided some interesting insights into the role of RRV and behavioural/self-report impulsivity, there are some methodological limitations to discuss particularly with regard to the implementation of the RRV procedure. Although the RRV proportional values are not dissimilar to those seen in previous work (Epstein et al 2008), this may be misleading given that participant responding was low between both food and reading RRV tasks. It has to be kept in mind that a large amount of RRV literature has been conducted on child samples, and the reduction in responding might be a reflection of our adult sample. For example, participants were told that they could continue the RRV task until they wanted to stop, so the reduction in responding might be an illustration not necessarily of the reinforcing value of food or a non-food alternative, but possibly a greater reinforcing value for the participant of leaving the experiment, which might be what is reflected in the high variances for calories/grams consumed. This could be negated in future research by implementing a set-time for the duration of the study, which may prevent participants prematurely ending the task. Also with regard to the lack of responding on this task, the rate of responding for reading was particularly low. Work with children has previously adopted a non-boredom alternative such as playing video-games (Temple et al, 2008). It may be here that a reading alternative did not provide a stimulating enough alternative, possibly meaning that the food reinforcing value isn't 'relative' *per se*. Future work may look to assess an alternative non-food task whilst providing a set-time in the experiment in an attempt to prevent premature decisions to abort RRV tasks.

3.4 Conclusions

Through the statistical models, it seems that few measures are consistently predictive of the variables of interest. However it seems although tentative and with limitations discussed, that RRVfood may represent a risk factor for increased snack consumption ad libitum. It is felt that despite a lack of association with food intake, this study provides a springboard for exploring impulsivity in light of reinforcement of reward, whilst also highlighting some methodological limitations surrounding food reinforcement tasks.

Experiment 3: Exploring differences in the perceived hedonic value of a milkshake preload, the anticipation of a milkshake preload, and the relationship with subsequent behavioural impulsivity.

4.0 Introduction

Previous work in this thesis (Brace & Yeomans, this thesis Experiment 1) demonstrated that the consumption of a chocolate milkshake preload induced subsequent behavioural impulsivity specifically inhibitory control and the ability to delay gratification, independent of TFEQ subscales restraint and disinhibition. This supports previous work conducted in this laboratory examining the malleable 'state' paradigm of impulsive behaviour (food cue exposure, Yeomans & Brace 2015), and other work assessing the ability to prime or 'train' subtypes of impulsivity, specifically inhibitory control (Houben & Jansen, 2011). However, in the previous study we only contrasted the effects of consumption of a preload versus no consumption, and this does not allow identification of what effect the preload had which enhanced subsequent impulsivity. For example, we cannot determine whether the effect was a result of the hedonic or palatable nature of the preload, the perceived high calorie nature of the preload, the anticipation of reward, or effects of general consumption relative to no consumption. Further investigation of the nature of the impact of the preload is needed to dissociate these potential explanations.

Some ideas about the possible explanations for the effects of preload consumption on behaviour can be inferred from studies of the effects of preloads on actual ingestion in acute snack tests immediately following the preload, as used classically in studies of disinhibited eating in the context of dietary restraint (Herman and Mack 1975). Some previous work (e.g. Polivy, 1976; Mills and Palandra, 2008, Knight and Boland, 1989) has suggested that overconsumption of food following preload consumption was not due to the actual energy content, but due to the perceived energy content. However Mills & Palandra (2008) suggested that this perceived energy overconsumption effect was dependent on dietary restraint scores. In contrast, neither impulsivity induced by exposure to

food cues (Yeomans and Brace, 2015) or in Experiment 1 in this thesis were affected by dietary restraint. This would suggest that the effect of consuming the preload was less likely to be due to the perceived energy content (although this is a factor which must be controlled), but elsewhere. Given that reward-reactivity has been related to desire for pleasure (Berridge, 1996), the present experiment examined an alternative explanation for the outcome of Experiment 1, the hedonic nature of the preload. The present experiment also examined further how the effects of the preload on impulsivity depended on restraint and disinhibition scores, with particular focus on impulsivity differences in those characterised as most likely to gain weight (HDLR, Experiment 1).

To attempt to understand the pertinent characteristics of the milkshake that may have induced the impulsive effects, the first potential explanation is the role of hedonic reward. There is a wealth of research documenting the association between palatable food reward and reward pathway activation. The seminal work of Berridge (1996, as reviewed in chapter 1) who previously posited a neural dissociation between wanting (incentive motivation) and liking (subjective pleasure) associated with drug-taking behaviour, suggested that a similar dissociation is demonstrated with regard to food reward. Berridge (1996) suggested that as with drug use, the dopaminergic system plays a pivotal role in mediating the incentive and motivational value of the rewarding behaviour, without altering the subjective pleasure associated with it. That the anhedonia hypothesis forwarded by Wise (1982) which suggested that as a result of dopamine function suppression, there would be a reduction in the subjective 'liking' of the reward, was ineffective in exploring the role of dopamine in reward. This criticism from Berridge (1996) was due to the nature of the experiments also demonstrating a marked reduction in 'wanting' behaviours, and the evidence from taste-reactivity measures resulting in unchanged liking 'affective' measures. Other work then continued to demonstrate a role of dopamine pathways in the motivational aspects of food reward but not the subjective and affective aspects. For example, work has continued to emphasise that dopamine antagonists are ineffective at altering liking measures or affective ratings of food reward (Treit and Berridge), and this

is further highlighted in the animal literature of dopamine depleted rats with 6-OHDA lesions showing little to no difference in affective responses to sweet solutions, but marked differences in the motivational behaviour to obtain the sweet rewards. This argument is enhanced by work providing evidence that suggests that in animals extinguished of food-reward (e.g. show no reward-seeking behaviour towards a stimulus due to repeated lack of reward), dopamine agonists effectively reinstate behaviours concurrent with reward seeking (e.g. de Wit & Steward, 1981). Further experimental work has provided more evidence for the role of dopamine in both food reward motivation (Salamone & Correa, 2002) and motivational reinforcement - the learned food-prediction-incentive cues (McFarland & Ettenberg, 1998).

Although it is currently unclear as to the complete relationship between reward pathways and subtypes of impulsivity, findings suggest that there is at least an association between these constructs. Specifically it seems that dopaminergic reward pathway activation, as discussed functionally as having a role in motivational incentive behaviour, seems to be linked to impulsive behaviours (van Gaalen et al, 2006a). The involvement of dopamine reward pathways in impulsivity seems to be associated with several impulsivity subtypes, including impulsive choice (Winstanley, Theobald & Dalley, 2005), inhibitory control (van Gaalen et al, 2006b; Pattij et al, 2007, sample with children) and self-report measures of impulsiveness (Cools, Sheridan & Jacobs, 2007). This explanation seems to link food consumption/exposure and the activation of reward pathways thus subsequently reducing the ability to delay reward or inhibit responses behaviourally through enhanced reward sensitivity. It is this direction that will be the focal point of this experiment. We also consider the limited significant association of TFEQ restraint and disinhibition in the original experiment, which therefore again will be further explored. As discussed earlier in this chapter (Mills & Palandra, 2004) with regards to the link between disinhibited overeating and TFEQ-variables contingent on perceived energy content, this factor will also be controlled in order to isolate differing hedonic values alone.

Although we have discussed the relationships between impulsivity and hedonic reward, using our previous experimental design (preload vs. no consumption) it is not possible to understand the role that anticipation to the reward may play in impulsivity, or how to control for it. For example, the cued impulsivity effect may not be driven by direct consumption of the preload, but actually just the anticipatory response to the reward, consistent with the finding that viewing pictures of food can increase impulsive responding (Yeomans and Brace, 2015). Other evidence from the literature also suggests that this may be the case, that anticipation to reward from cue-exposure can increase behavioural and motivational responding to the reward. For example, Meule et al (2014a), using a stop-signal inhibitory task demonstrated participants' reduced inhibitory control when exposed to hedonic food images, compared to matched control images. However it is important to note that in that study, participants had not eaten for over 5 hours, so the results may be confounded by acute food deprivation. These findings were replicated in the same laboratory (Meule et al, 2014b), however this time the researchers further explored the nature of the cue exposure, including a high-calorie, and low calorie set of food stimuli. Again the researchers found a reduction in inhibitory control (greater stop latencies and omission errors) in trials using high-calorie cue exposure (vs. low calorie), which was predicted by the interaction of trait food craving, and trait impulsiveness. However, in this variation of the experiment, the researchers, used an affective shift paradigm, using food stimuli, however they did not use a neutral-cue control condition, which would have been useful in understanding responding to low-calorie stimuli, as currently we cannot be sure if this poor inhibitory control demonstrated is relative to low calorie stimuli, or whether there is a linear effect (e.g. perceived as twice as hedonic leading to twice as impulsive) of 'hedonic responding' based on the perceived hedonics associated with each reward. We cannot yet also be sure whether the milkshake-preload-impulsivity effect previously discussed was a result of anticipation of food reward, or exposure to food cues, in this case prior exposure before consumption. With these data in mind, it is vital to the current investigation that an anticipation condition is included – a condition in which participants are told that they are going to

receive a chocolate milkshake to 'standardise hunger', but never actually receive it.

Therefore this experiment aims to firstly replicate the findings of Experiment 1 by repeating the contrast of effects of consumption of a liked preload (milkshake) relative to nothing (control) on measures of impulsivity, but included two further conditions to try and start to tease apart alternative explanations for any effects of the preload: an anticipation condition where participants expected to consume the hedonic preload but did not do so, and a milkshake that's flavour was adjusted to make it less palatable to test explicitly the role of hedonic impacts of consumption. If it is the actual ingestion of food that leads to enhanced impulsivity, the same effects should be seen for the liked and disliked preloads, but no effects of anticipation without consumption, whereas if it is actual or anticipated consumption of a liked food that alters impulsivity, no effects should be seen in the disliked preload condition. In terms of our theoretical model, this experiment aims to examine the extent to which a hedonic milkshake preload vs. a non-hedonic may modulate behavioural impulsivity (inhibition, discounting, and reflection impulsivity) rather than overeating behaviour as shown through counter-regulatory eating (Herman & Mack, 1975), and the extent to which this modulation is specific to those high in both disinhibition and restraint scores (failed dieters) of the TFEQ - which Westenhofer discussed as 'disinhibition effect'. Two further conditions (no consumption, and an anticipation condition) are also added in order to be able to examine the extent to which any experimental modulation of impulsivity is due to differences in the hedonic value of the milkshake preload (theoretically driven by hedonic reward activation leading to a potential compromise of inhibitory systems, Volkow et al. 2009) rather than simply a priming of reward through anticipation.

Hypotheses

It is hypothesised that those in the hedonic experimental condition will be more impulsive on the behavioural measures than the other conditions, and the anticipation condition will be more impulsive than the control (no consumption) and non-hedonic experimental conditions. In addition, although no effects of TFEQ-D or TFEQ-R were seen in Experiment 1, effects of cue exposure on impulsivity have been found to depend on TFEQ-D scores. It was also therefore hypothesised that the effects of the hedonic preload and anticipation of that preload on impulsivity would also vary as a function of TFEQ-D scores.

4.1 Method

4.1.1 Participants

When comparing experimental condition, TFEQ-D and TFEQ-R, analysis is conducted on a cell size of 10 participants. 80 female participants took part in the study ranging from ages 18-46 ($M=21.29$, $SD=3.71$), with BMI ranging from 18-30 ($M=23.25$, $SD=3.29$). Four participants were excluded non-responding on either behavioural tasks, or non-systematic trends in their discounting decisions (see Johnson & Bickel, 2008). Participants were recruited through the University of Sussex internal experimental advertising system (SONA). In order to participate in the study, participants must have previously completed an appetite recruitment questionnaire, containing the TFEQ and any allergy or aversion to food details. The University of Sussex Ethical Review Board approved the experiment. Potential participants were excluded if they met any of the exclusion criteria, including: the use of regular medication (other than the contraceptive pill), smoking more than 5 cigarettes per week, allergies or aversions to foods used, currently pregnant or breastfeeding, diabetic or diagnosed with an eating disorder. When comparing experimental condition, TFEQ-D and TFEQ-R, analysis is conducted on a cell size of 6 participants.

4.1.2 Materials

Participants in the hedonic experimental group received a chocolate milkshake (466kcal total). This consists of 200g of Sainsbury's brand chocolate ice cream (338kcal), and 200g Sainsbury's whole milk (128kcal), blended and refrigerated for one hour to ensure consistency between milkshakes in viscosity. The milkshake was presented in a beige milkshake tumbler with a straw. The milkshake is based on disinhibiting preload milkshakes as used in Herman & Mack (1975), Polivy, Heatherton & Herman (1988), and similar to that used in Jansen & Nederkoorn et al. (2008). The non-hedonic milkshake was identical to the hedonic milkshake, but it was adulterated with 1.5g of Schwartz garam masala seasoning, piloted to ensure that it was perceived as significantly less hedonic, but was not perceived as being significantly less healthy.

4.1.3 Pilot

To formulate an experimental preload for the non-hedonic condition, a pilot study was conducted. In accordance with previous research (Bobroff & Kissileff) which used adulterated formulas to reduce the hedonic appeal of a standard food, incongruent flavour pairings were attempted. The base preload was identical to the hedonic condition (200g Sainsbury's whole milk, 200g Sainsbury's chocolate ice-cream) but with the addition of one of the following: monosodium glutamate (MSG, 1.5g), salt (2g), Schwartz garam masala spices (2g), or Sainsbury's cumin (2g), generating five variations of the milkshake. 8 participants evaluated each of these samples on two occasions, and asked to sample and rate them on characteristics indulgence, pleasantness, familiarity, healthiness and novelty using a Visual Analogue Scale using SIPM and were also asked to estimate the number of calories they perceived to be in a full glass (330ml) of each sample, with the glass presented as a reference point. Participants were asked 'How (target word characteristic) is the sample' and had to select from 1-100 on a visual analogue scale using their mouse on a computer using the Sussex Ingestive Pattern Monitor Software (SIPM). The non-hedonic preload selected in from the pilot study (n=8) for the main experiment from salt adulterated, cumin adulterated, MSG adulterated and

garam masala adulterated was the garam masala milkshake. Participants in the pilot rated the garam masala milkshake as significantly less pleasant ($t(7) = -4.08, p < .05$), but not significantly less subjectively healthy ($t(7) = -1.413, p = .200$) than the control (hedonic) milkshake.

Table 4.1 Means (\pm SD) of pilot milkshakes rated on healthiness and pleasantness

	Healthy	Pleasant
Control (chocolate)	14.3 \pm 26.6	40.3 \pm 28.5
MSG	39.5 \pm 36.8	43.4 \pm 25.0
Garam Masala	17.0 \pm 24.5	30.6 \pm 29.7
Cumin	49.4 \pm 20.9	10.3 \pm 14.0
Salt	23.6 \pm 26.6	44.8 \pm 20.1

SIPM Mood Scale (see Methods, Experiment 1)

Go/Stop Paradigm (Dougherty et al. 2003, see Methods, Experiment 1)

Delay Discounting and Probability Discounting Task (Baumann & Odum, 2012)

This study employed the same algorithm used in the previous experiment in this thesis (Experiments 1 and 2), however some details have been modified.

Whereas Baumann & Odum 7 used randomised delays of 1 day, 2 days, 1 week, 2 weeks, 1 month, 2 months and 6 years, it is felt that the incongruence in the difference between the last delay and the penultimate delay has the potential to create a framing or anchoring effect (Tversky & Kahneman, 1974). For example, it may lead participants into anchoring their choice decisions based around the incongruent anchor, leading to a deflation in overall AUC scores on this measure. Other behavioural economic researchers discuss the possibility of framing and anchoring at length including (Tversky & Kahneman, 1974), demonstrating how a disparity in delay estimates can bias decisions on alternative delays. Therefore, we have altered the delay points in this experiment to 1 day, 2 days, 7 days, 14 days, 30 days, and 180 days, in the hope that this limits any possibility of

anchoring around an incongruent delay. The probability discounting measure was identical to the one used in the original study, and the algorithms for calculating indifference points were also the same.

Information Sampling Task (Clarke et al, see Methods, Experiment 2)

4.1.3 Design

A between-participants design was used to examine the interactive role of restraint and disinhibition on behavioural impulsive scoring in participants between experimental and control conditions. Participants were categorised into either high or low TFEQ-Restraint and TFEQ-Disinhibition according to the criteria suggested in Westenhoefer et al (1994) using median split (6 for R and D). A 2 (High/Low Restraint) x 2 (High/Low Disinhibition) x 4 (experimental hedonic vs. experimental non-hedonic vs. anticipation vs. control condition) ANOVA was used to examine this interaction in terms of the 3 types of behavioural impulsivity measured, and planned comparisons were conducted.

4.1.4 Procedure

The study was advertised via a university-wide online participant recruitment pool as a study assessing 'mood and cognitive performance'. This was used in order not to expose the control group to food cues. Initially, participants were randomly allocated to either the control, hedonic, experimental non-hedonic or anticipation conditions. Participants in the control group continued to be told that the study examined mood and cognitive performance, whereas the hedonic and non-hedonic groups were told that it was thought that hunger may play a role in mood, so a milkshake needed to be consumed to 'normalise hunger' across participants: this also allowed relevant screening for potential adverse reactions to the milkshake. Participants in the anticipation condition were told that they would receive a chocolate milkshake, but never did. Participants in the control (no consumption) condition were not subject to the exclusion requirements based on allergies or regular medication use. Participants

completed the TFEQ online at the point to which they signed up for future consideration for appetite studies in our laboratory. The difference in time between TFEQ completion and study participation varied greatly depending on their selection, eligibility, and point at which they decided to take part in the study.

If participants met the appropriate criteria, they were asked to come to the University of Sussex Psychopharmacology Laboratory at a timeslot between 2-5pm on a convenient day. Participants initially completed a mood-rating questionnaire (SIPM, see methods, Experiment 1) before consuming the milkshake in the hedonic and non-hedonic experimental conditions, or waited for the set of tasks to begin. All participants completed the DDT/PDT, GoStop and IST in a random order. Following completion, which took approximately 35-45 minutes, all participants were debriefed about the nature of the experiment and thanked for their time, and a measurement of their height, weight and age was taken. Participants were given a choice of either 4 course credits, or £4 cash for their participation.

4.2 Results

4.2.1 Preliminary analysis of existing group differences

There was an unexpected significant difference in BMI between control (23.65 +/- .66), anticipation (24.96 +/- .66), hedonic (26.52 +/- .67) and non-hedonic (24.34 +/- .74) conditions ($F(3,71) = 3.32, p = .025$), therefore BMI was used as a covariate in subsequent analyses. There was no significant difference in TFEQ-D ($p < .05$) or TFEQ-R ($p < .05$) between conditions.

4.2.2. Drink perceptions

Participants in the hedonic and non-hedonic condition completed ratings of pleasantness, perceived healthiness (using a visual analogue scale (1-100)

between 'not at all' and 'extremely' and estimation of caloric content (relative to a reference point – e.g. a glass of orange juice the same size as the milkshake). As expected, participants in the hedonic condition (81.1 ± 21.4) rated the milkshake as significantly more pleasant [$t(33) = 2.574, p = .015$] than the non-hedonic condition [56.9 ± 29.5]. However participants in the hedonic condition [28.0 ± 16.4] also rated the milkshake as significantly less healthy [$t(33) = -2.273, p = .030$] than the non-hedonic condition (43.1 ± 22.8). There was no significant difference in estimate caloric content of the two drinks: hedonic $500 \pm 178\text{kcal}$, non-hedonic $466 \pm 198\text{kcal}$ [$t(33) = .545, p = .589$].

4.2.3. Effects of preload conditions on impulsivity subtypes

Unexpectedly, there was no significant difference between control, anticipation, hedonic, or non-hedonic conditions in DDT area-under-the-curve [$F [3,59] = .<1, p = .92 \eta^2 = .01$], and there was no significant effect of TFEQ-D [$F [3,59] = .<1, p = .64 \eta^2 = .01$], , TFEQ-R [$F [3,59] = 2.32, p = .13 \eta^2 = .03$], , the interaction of TFEQ-D/R [$F [3,59] = .<1, p = .57 \eta^2 = .01$], the interaction between condition and TFEQ-D [$F [3,59] = .107, p = .37 \eta^2 = .05$], , and condition, TFEQ-D and TFEQ-R [$F [3,59] = .<1, p = .71 \eta^2 = .02$], and no significant covariates of pre-test hunger or BMI [all effects at $p > .05$], See table 4.2.A and Figure 4].

There was no significant difference between control, anticipation, hedonic, or non-hedonic conditions in PDT area-under-the-curve [$F [3,59] = .137, p = .94$], and there was no significant effect of TFEQ-D [$F [3,59] = <1, p = .49 \eta^2 = .01$], , TFEQ-R [$F [3,59] = 1.39, p = .24 \eta^2 = .02$], , the interactive effects of TFEQ-D/R [$F [3,59] = 1.00, p = .32 \eta^2 = .02$], the interaction between condition and TFEQ-D [$F [3,59] = .<1, p = .95 \eta^2 = .01$], or condition and TFEQ-D and TFEQ-R [$F [3,59] = .<1, p = .90 \eta^2 = .01$], , and no significant covariates of hunger or BMI [all effects at $p > .05$].

The GoStop stop-latencies were log10 transformed in the analyses to correct for normality violations, but for sake of interpretability, the means are reported here in their original metric. There was no significant difference between control, anticipation, hedonic, or non-hedonic, conditions in DDT area-under-the-curve

[F [3,58] = 1.11 p = .35 η^2 = .05], and there was no significant effect of TFEQ-D [F [3,59] = .<1, p = .90 η^2 = .01], TFEQ-R [F [3,59] = .<1, p = .92 η^2 = .01], the interactive effects of TFEQ-D/R [F [3,59] = .<1, p = .68 η^2 = .01], the interaction between condition and TFEQ-D [F [3,59] = 1.29, p = .29 η^2 = .06], or condition and TFEQ-D and TFEQ-R [F [3,59] = .<1, p = .65 η^2 = .03], and no significant covariates of pre-test hunger or BMI [all effects at p< .05] See table 4.2.B and Figure 4..

There was no significant difference between control, anticipation, hedonic [155.90 +/- 12.45], or non-hedonic, conditions in IST amount of boxes opened [F [3,54] = 1.738, p = .170 η^2 = .07], and there was no significant effect of TFEQ-D [F [3,54] = <1 p = .35 η^2 = .01], TFEQ-R [F [3,54] = <1, p = .84 η^2 = .01], the interaction between condition and TFEQ-D [F [3,59] = <1, p = .54 η^2 = .03], or condition and TFEQ-D and TFEQ-R [F [3,59] = .<1, p = .98 η^2 = .002], the interactive effects of TFEQ-D/R [F [3,59] = .<1, p = .78 η^2 = .01], , and no significant covariates of pre-test hunger or BMI (all effects at p>.05) See table 4.2.C and Figure 4..

Tables 4.2. Means and SEM for condition, TFEQ-D low and high, and TFEQ interaction groups for A) DDT AUC, B) GoStop SL (ms) and C) IST boxes opened

A)	No Milkshake	Anticipation	Hedonic Milkshake	Non-Hedonic Milkshake
Condition	0.54±.07	0.58±.07	0.54±.06	0.60±.07
Low TFEQ-D	0.57±.12	0.50±.11	0.63±.10	0.62±.10
High TFEQ-D	0.52±.08	0.66±.07	0.46±.08	0.57±.10
LDLR	0.52±.15	0.51±.13	0.66±.15	0.50±.15
LDHR	0.61±.18	0.50±.18	0.60±.13	0.74±.13
HDLR	0.42±.15	0.59±.12	0.36±.13	0.55±.18
HDHR	0.62±.08	0.73±.09	0.55±.09	0.59±.10

B)	No Milkshake	Anticipation	Hedonic Milkshake	Non-Hedonic Milkshake
Condition	144.0±29.61	151.0±25.79	113.5±25.60	134.1±30.22
Low TFEQ-D	160.04±45.41	149.75±43.08	93.30±40.62	139.37±45.41
High TFEQ-D	127.96 ±38.00	152.23±28.36	133.78± 31.18	128.85±39.89
LDLR	194.83 ±57.45	175.00±49.75	83.27±57.45	146.40±70.36
LDHR	125.25±70.36	124.50±70.36	103.33± 57.45	132.33±57.45
HDLR	86.00±70.36	143.14±44.50	124.88± 49.75	127.50±70.36
HDHR	169.93±28.72	161.31±35.18	142.69±37.61	130.20± 37.61

C)	No Milkshake	Anticipation	Hedonic Milkshake	Non-Hedonic Milkshake
Condition	123.5±16.67	154.1±13.07	155.9±12.45	121.4±15.00
Low TFEQ-D	115.3 ±29.11	162.8± 21.83	138.4±19.25	111.3±21.83
High TFEQ-D	131.7±16.27	145.4±14.37	173.4±15.80	131.6 ±20.58
LDLR	95.67±29.11	132.00± 25.21	158.00±29.11	110.50±35.77
LDHR	135.00±50.42	193.50±35.65	118.75±25.21	112.00±25.21
HDLR	131.33±29.11	130.80± 22.55	204.00± 25.25	135.50±35.65
HDHR	132.00±14.55	160.00± 17.82	142.86±19.06	127.67±20.58

Table 4.3: Means (+/-SD) of impulsivity measures, TFEQ-subscale scores, and hunger/BMI across conditions

	No Milkshake	Anticipation	Hedonic Milkshake	Non-Hedonic Milkshake
DDT AUC	.573 ±.27	.619 ±.28	.546 ±.21	.629 ±.22
PDT AUC	.354 ±.12	.332 ±.19	.346 ±.12	.419 ±. 42
GoStop SL (ms)	152.98 ±143.91	150.22 ±86.45	113.59 ±64.83	114.25 ±76.22
IST M Boxes Opened	126.32 ±40.68	149.96 ±-50.94	156.59 ±58.31	118.15 ±49.44
TFEQ-D	9.11 ±3.46	7.84 ±3.35	8.41 ±3.65	7.31 ±4.36
TFEQ-R	9.89 ±5.84	6.37 ±4.49	8.24 ±4.89	7.77 ±3.68
Hunger	69.94 ±17.00	45.31 ±28.81	52.00 ±23.61	52.38 ±23.69
BMI	23.91 ±2.40	25.05 ±3.07	26.77 ±2.66	23.95 ±3.58

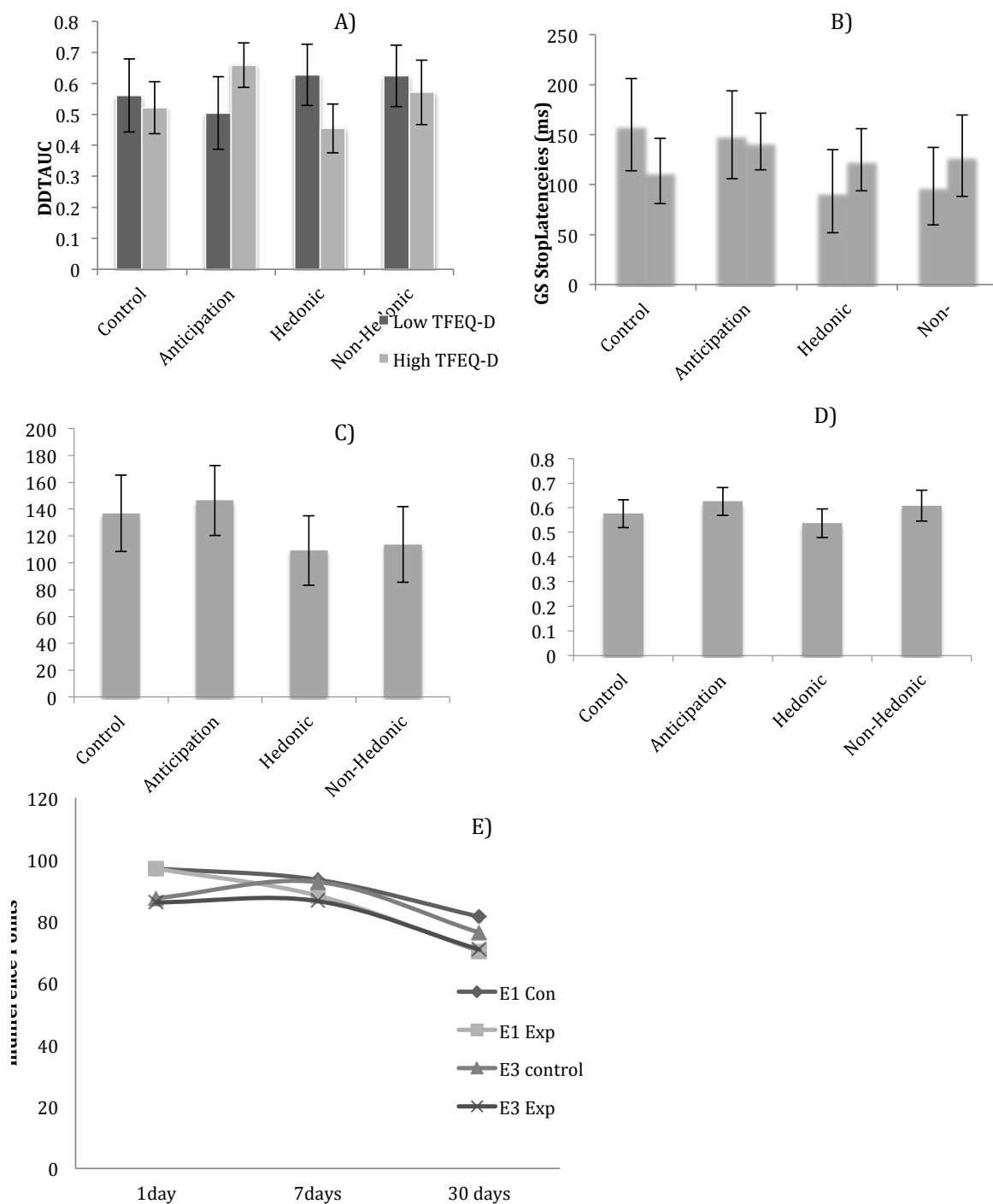


Figure 4: A) means (\pm SEM) of DDT AUC between conditions in high/low TFEQ-D groups, B) GS Stop latencies (ms) between conditions in high/low TFEQ-D groups, C) GS Stop latencies between conditions, D) DDT AUCs between conditions, E) common timepoints of indifference points between E1 and E3 experimental and control conditions.

4.3 Discussion

Traditional preload counter-regulation studies (Herman & Mack etc.) originally posited that consumption of a milkshake preload leads to overeating compared to no preload consumption, specifically in restrained eaters. This was later discussed as a 'what the hell' effect (Herman & Mack, 1975), referring to restrained eaters' all-or-nothing attitude towards overconsumption; they have broken their self-imposed cognitive dieting boundary, so continue to over-consume in that episode. However, although some studies have replicated the original findings of preload consumption leading to excess calorie intake, several studies have failed to replicate this effect (e.g. Van Strien, 2007, Ouwens, Van Strien & Van der Staak. 2003). From our laboratory, we have since posited an alternative paradigm to interpret the seemingly counter-intuitive counter-regulatory effect. We have posited that preload consumption may not lead to a break of cognitive dietary boundaries, but may actually serve to enhance reward-reactivity as a result of exposure to hedonic food rewards. Therefore, it may not be that 'counter-regulation' and food consumption is as a result of broken cognitive boundaries, but may be a food-associated reflection of the behavioural impulsivity linked to activation of reward pathways through exposure of hedonic reward, which also has been strongly implicated with inhibitory pathways. In the first study in our laboratory, preload consumers were more impulsive (inhibitory control and delayed discounting) than a non-consuming control group. However, in our follow-up study, we failed to replicate this finding. This again, despite not measuring direct caloric intake, adds to the inconsistency in preload counter-regulation research.

Although we are not the first research group to show mixed findings with regards to delayed discounting data (Fernandez, 2013), it is equally possible that these results may be as a result of reduced sample size to the original study (due to the inclusion of additional experimental groups), therefore further examination of the nuances of DDT mechanisms are required within the literature. This is further example of the inability to detect the relationship

between DDT scores and TFEQ variables as hypothesised and demonstrated in Yeomans & Brace (2015, Yeomans et al. 2008), and to some extent in the contrasting of interacting TFEQ-D and TFEQ-R conditions in study 1 provides adequate rationale for the meta-analytic review presented later in this thesis.

It is possible that preload and reward activation results are a statistical artifact, but it is also possible that the history of research in this area has failed to account for a potentially confounding variable underpinning these effects. For example, although original preload-consumption studies and our recent preload-impulsivity have controlled participant hunger prior to participating (with an enforced food abstinence period), very few of the studies in the area account for hunger, or more specifically, satiety, post-preload. For example, due to the capacity for variability in physiological and psychological satiety, there is potential for some in the research population who reach satiety through the consumption of the preload to drastically reduce or weaken any reward-reactivity-induced behavioural responding. Fundamentally this is the discussion of the difference between being 'disinhibited' by the preload, and satiated by the preload, two different but entirely possible outcomes from preload consumption. Those who are not sated, or are disinhibited, may respond in a reward reactive manner (as shown in experiment 1), however those who have reached satiation in the same experimental condition may reduce the ability for researchers to detect reward-reactive responding especially if using statistical methods aimed at detecting between-groups mean differences as commonly used in these experimental designs. This may therefore be why in some studies, there is a clear demonstration of a disinhibition effect, and why in others there is not – a shift or discrepancy in the studied population who are either disinhibited or satiated.

Although this proposal of satiety vs. disinhibition has not been directly examined experimentally, there is a great deal of evidence investigating the role of hunger and satiety in reward and behavioural responding. Several research groups (e.g. Kringlebach et al, 2003, Padoa-Schioppa & Assad, 2006) have explored this idea with primates using neuroimaging methodologies. The findings suggests that in states of satiety orbitofrontal cortex regions (also a region associated with

behavioural inhibition) show a reduced response to food reward, both behaviourally to consume the reward, and in the cued presence of the reward. Other work has suggested that in states of satiety, primate OFC neurons stop responding in the presence of food reward (Rolls et al, 1986). Again, OFC activation has also been associated with mechanisms implicated in inhibitory control (Volkow et al, 2012). The work of Del Parigi et al (2002) using PET scanning, also demonstrated the relationship between satiety mechanism and inhibitory control, specifically a blunted response to satiety signals, and reduced inhibitory control in those prone to overeating behaviour following enforced satiety. Tetley & Brunstrom (2010) discusses the possibly disruptive role of satiety in the relationship between portion-size selection and impulsivity. As is demonstrated by these studies, we are yet to directly examine the relationship between satiety and impulsivity, but it would seem that there is evidence to at least suggest an association between satiety and blunted response to reward cues, specifically food reward, which may provide adequate rationale for the implication of satiety in impulsivity research through a reduction in reward reactivity.

It is not known from this study or any preload-counter-regulation studies where or why the differences in satiety may have arisen. However, work from our laboratory (McKrickard et al, 2014) has suggested that contextual information of a product can influence its satiety value. For example, in a high-sensory preload, participants reported feeling fuller following consumption if they were told that the preload was a 'snack' rather than a 'thirst-quenching' drink. The concept here of priming expectations about consumption leading to changes in satiety and expected satiety have been explored further in the work of Brunstrom et al (2011), who demonstrated that participants feel fuller, and have less hunger immediately and over a 3 hour period after consuming a fruit smoothie preload with an image shown to them to contain a large portion of fruit, versus the same fruit smoothie with an image shown to contain a small portion of fruit. The concept of expectations of food effecting later experiences and consumption has since been explored behaviourally and physiologically (e.g. Jesudason et al, 2007). Although in these two discussed studies participants were randomly allocated to

experimental conditions priming expectations of a product, it is also possible that these expectations can occur organically. That is to say that individuals form expectations of products (and our milkshake) based on myriad factors which can influence subsequent satiety, many of which very few studies in the literature have measured or controlled for. It is currently not known how participants in counterregulation studies perceive the milkshake, or more pertinently in terms of this research, how they contextually frame the milkshake based on their own expectations, e.g. drink vs. snack and it's subsequent effect on satiety.

An informal questionnaire was conducted online through our subject pool to gauge general expectations and beliefs of our milkshake beverage. Participants (n=23) were shown a picture of the milkshake, and were asked for a number of questions on what type of beverage they consider it (e.g. a snack vs. a drink) and on their expectations of fullness and hunger of the beverage (VAS 0-100). As expected, there was a great deal of variation in the data. In terms of how filling the milkshake was perceived, participants rated it on average 65.2 (25.8), and how hungry they would be following consumption 35.6 (27.2), demonstrating how much variation there is in terms of expectations of the satiety value of the milkshake. Of particular interest concerning the work of McKrickerd et al. (2014) was the question regarding beliefs about what type of beverage the milkshake was perceived to be. Sixty eight percent of respondents perceived the milkshake to a snack on it's own, 13% as a drink, 9% as a drink with a meal, and 4% as a snack with a meal. Given the contextual expectations effect of satiety demonstrated by the work discussed, it is possible that this belief (68%) of a milkshake as a snack vs. a drink may have enhanced the satiety value of the product.

As is demonstrated here, there is a great deal of variation in the expected satiety value of a milkshake preload. And as shown in other work discussed, expectations or prior beliefs (primed or organic) can alter behavioural and physiological responses. Given this variation and expectation, which are not controlled for traditionally in preload-counterregulation studies, it is possible that they have a confounding effect on the sensitivity to detect a disinhibition

effect, and this may suggest why results have been inconsistent in methodologies of this type, due to the possible ‘dampening’ effects of differences in satiety due to one or several of these mechanisms involved. As of yet it is unknown the direct statistical effects of satiety on impulsivity, and although samples drawn from a normally distributed population should ensure systematic variation in the differences in post-preload satiety, the relationship may not be a linear one. For example, we cannot be certain, if satiety is a factor, how much tolerance for satiety in detecting reward reactivity is possible, particularly so with reduced sample-sized groups.

Using our current paradigm, we also intended to control for reward anticipation, or the anticipation of a milkshake reward. We hypothesised that participants in the anticipation condition would act more impulsively in the behavioural measures (e.g. Beck et al, 2009; Hahn et al, 2009), but this was not statistically supported. An explanation for this may be through the differential techniques used in inducing reward anticipation. For example, other work has used olfactory exposure (Larsen et al. 2012) or visual cue exposure (Sobik et al, 2005). However in our paradigm, participants were not exposed to direct reward cues but were informed that they would later consume a ‘delicious chocolate milkshake’. This method may have been problematic as we are currently unaware exactly what is being anticipated for the participants. There may have been different representation between participants, which may be problematic in understanding anticipatory inducement in research. It may be useful in future work to expose participants to the same stimuli, or attempt to form similar reward representations in order to control (as best as can be controlled) for differing reward representations.

One of the critical limitations with this experiment is that although the hedonic and non-hedonic milkshakes were rated as significantly different in pleasantness in the pilot, but not significantly different in healthiness (as was the aim in the formulation of the preloads), this did not replicate in the experiment itself. Participants in the experiment itself did indeed rate the hedonic milkshake as more pleasant, but also healthier than the non-hedonic milkshake. As discussed

in this thesis, the role of perceived healthiness has been shown to impact subsequent behaviour independent of actual caloric content (Mills & Palandra, 2008). This may be a product of a within-subjects pilot vs. a between subjects experiment (e.g. those in the pilot may anchor their beliefs about the preload relative to the other preloads, whereas those in the experiment may be driven by the preload alone, and as we know, hedonic value may drive beliefs around healthiness, therefore this could be an explanation as to why differences in perceived preload healthiness may have been detected. Although we found no impulsivity differences between groups, this may have been in some part due to individual differences in satiety as previously discusses, but differences in perceived preload healthiness may have also in some part played a role in this.

To conclude, we failed to find evidence that differences in types of preload or preload consumption would modulate behavioural impulsivity. However, we failed to replicate our earlier findings of GoStop stop-latency associations to preload consumption. We have proposed a number of possible reasons as to why this could be the case, and have made further suggestions as to how these problems could be negated in future reward-impulsivity paradigm research.

Chapter 5: Experiment 4 – The role of a reward-conditioned stimulus on subsequent behavioural impulsivity

5.0 Introduction

Classically, as discussed at length in this thesis (Experiment 1), consumption of a pre-task milkshake preload has often been shown to lead to an increase in ad libitum food consumption (Herman & Polivy, 1975), explained as a breakdown of self-imposed cognitive dieting boundaries. We originally posited (experiment 1) that this supposed counter-regulation hypothesis may not be as a result of a ‘disinhibition effect’ (Westenhoefer, 1994), but could be due to enhanced reward sensitivity due to the preload, which has been linked to some subtypes of impulsivity (Tetley et al. 2010), which posits an alternate pathway to counter-regulatory eating.

However, this type of effect has been explored not just in terms of intake following food consumption, but also following explicit food cue exposure. That is to say that participants who have been exposed to food cues vs. non-food matched cues subsequently consume more food ad libitum (Jansen et al, 2003). This effect has been robustly displayed in restrained eaters in the work of Federoff et al (1997), although due to the use of the Revised Restraint Scale (RRS), we are unable to ascertain the role of dietary disinhibition, which has also been posited to play a role in understanding individual differences in the susceptibility and sensitivity to external food cues (Stunkard & Messick, 1985). These are not isolated examples of the role of food-cue exposure on behavioural modification (e.g. Tetley et al, 2010; Lawrence & Hinton et al, 2012).

This effect has also been demonstrated by manipulating physiological differences, e.g. acute food deprivation (Epstein et al, 2003), further enhancing the effect of food cues and their interactions with dietary restraint individual differences and physiological manipulations. Through work in our laboratory (Yeomans & Brace, 2015), we have conceptualised this cue-to-consumption effect as a heightening of reward mechanisms leading to increased impulsivity. Direct research from the drugs and addiction

literature reinforces this possible mechanism conceptualisation between cue exposure and impulsivity. In that work there is some evidence for a direct association between explicit drug or alcohol cue reactivity and impulsivity. For example the work of Noel et al (2006) demonstrated that in a clinical sample of alcohol abusers, those exposed to alcohol cues were significantly poorer at inhibiting initiated responses (reduced inhibitory control). Doran et al (2007) explored this issue further in a population of smokers using smoking-related cues. The work demonstrated that reactivity to ‘environmental smoking cues’ (expressed functionally as subjective smoking-related craving) vs. non-smoking cues was related to heightened impulsivity in the form of consistent preference for immediate vs. delayed rewards. The authors suggest that this is strong evidence for the link between impulsivity, cue reactivity and outcome response – in this work, smoking.

However, thus far we have discussed the role of what I term here as ‘explicit’ cues. In this context explicit cues refer to cues that are explicit to the rewards themselves, e.g. images of the food directly, exposure to the food sources, or olfactory and sensory exposure to the food rewards. What has yet to be explored in detail is the role of ‘implicit’ food reward cues in over-consumption, or in this case, behavioural impulsivity. ‘Implicit’ here are cues associated with food rewards but which have no direct representation of the real food (as apposed to pictures of the actual foods). The most common example in the literature for this type of cue is that of exposure to food branding. It could be argued that a food-brand logo is an example of a food reward cue, a conditioned stimulus associated with the food reward, but not the reward itself. Several laboratories have explored the effects of exposure to food branding on subsequent food intake and consumer behaviour. For example, Forman et al (2007) employed the typical experimental design used to examine the role of exposure to food branding. Overweight and non-overweight children were exposed to both branded and non-branded food items. The overweight children consumed significantly more branded food items, and appeared to show greater preference when the branding cue was present on those items. These findings have been followed up and replicated a number of times. Halford et al (2007a; 2007b) explore not the role of branding vs. non-branding on food choice and intake, but the role of exposure to branded foods vs. non-food advertisements. The researchers suggest that this exposure can lead to consumption that is beyond just ‘brand choice’, and can lead to

increase food intake via exposure to cue-branded foods, specifically energy dense food in overweight children. This design was employed and presented a successful replication in the work of Harris, Bargh and Brownell (2009), except using cartoons interspersed with food branding vs. non-food content. Although vital in terms of the rationale for the current investigation, the critical limitation from this research, which we hope to address, is the lack of a food-cue control condition. For example, although participants were exposed to branded foods, research also suggests that food-cues (e.g. sight of foods alone) can increase subsequent intake (Harris et al, 2009). Therefore we cannot be sure as to whether it is simply exposure to food cues alone, and not implicit (branding) that are behind this increase in preference and intake in designs using only branded foods vs. non-food exposures.

Although the inter-relationship between branding, intake and preference has been examined, we investigate here an alternative paradigm integrating the relationship between cue exposure and food-associated stimulus exposure. For example, if explicit cue exposure (smoking, Doran et al, 2007., alcohol Papachristou, 2012, food, Lawrence et al, 2012) can stimulate intake, or craving, and on select occasions impulsivity, and if food-branded/associated stimuli can increase intake or craving, what is the relationship between conditioned-cue exposure and impulsive behaviour? For example, it is possible that the data suggesting that brand exposure leads to heightened intake and food preference may be a behavioural and applied expression of heightened impulsivity – either reduction in inhibitory control, or impulsive decision making as a function of that cue exposure?

In order to explore this paradigm, a previously neutral cue (an abstract symbol) without prior learning opportunities or expectations as a result of past associations must be used as the conditioned stimulus. As previously discussed as a limitation (Experiment 3, this thesis), the potential for participants to reach satiety following consumption, and therefore lower food motivational value also needs to be controlled. This study therefore intends to condition through Pavlovian conditioning a previously neutral stimulus with a natural reward (sweet taste, 10% sucrose, CS+). This stimulus will then be exposed to participants during two behavioural impulsivity measures (Stop-signal, and a delayed discounting task), with the aim of examining the role of reward conditioned stimulus in the modification of behavioural impulsivity – a

possible alternative explanation to the food branding exposure and increased intake data discussed by some of the authors in the previous paragraph (e.g. Harris et al 2010). The rationale for this experiment is driven by the failure to replicate our previous milkshake preload-impulsivity findings, possibly due to the role of satiety (which is negated in this design), and allows for the examination of an alternative paradigm: a mechanism of environmental cues and subsequent behavioural processes. As well as negating the physiological confounds of the previous studies (hunger state), this study also attempts to negate the potential confound of exposure to explicit food cues. For example, as discussed exposure food cue exposure (visual or olfactory) can modify behaviour, and it is difficult to determine the role of either branding or conditioned food-cues alone without the explicit food cue exposure potentially underpinning these effects. Therefore this study examines the role of a previously neutral reward-conditioned cue whilst negating direct food cue exposure effects.

We also acknowledge that in tasks such as stop-signal or go/no-go tasks the display of any stimulus outside of the focus area of the task may act as a distractor potentially reducing inhibitory scores or increasing errors of commission (which would appear in the data misleadingly as a reduction in inhibitory control), therefore we employed a condition using the display of a novel cue (a previously unseen symbol) and a control condition (no-cue) to be able to make comparisons between exposure to a reward cue and a control condition whilst negating the confound of attention distraction, with the inclusion of the novel cue. We also employed the use of a neutrally conditioned cue (CS-); a cue associated with a neutral substance ('artificial saliva'), chose not to be water as water has been shown to have a 'taste' and is not necessarily deemed as neutral, a methodology shown to be successful in conditioning such cues in previous research (Ridley-Siegert, Crombag & Yeomans, 2015). This condition allows for the further exploration of the role of reward-associated stimuli comparative to a range of adequate controls. In terms of our theoretical model, this experiment aims to examine the extent to which a reward cue vs. a neutral may modulate behavioural impulsivity (inhibition, discounting) if presented during behavioural tasks, a priming effect of reward cues leading to behavioural modulation as shown in work by Yeomans & Brace, 2015). This experiment also aims to examine the extent to which this modulation is specific to those high in both

disinhibition and restraint scores (failed dieters) of the TFEQ - which Westenhoefer discussed as 'disinhibition effect'. Two further conditions (no cue, and a novel cue condition) are also added in order to be able to examine the extent to which any experimental modulation of impulsivity is due to differences in the cue value (theoretically driven by hedonic reward activation leading to a potential compromise of inhibitory systems, Volkow et al, 2008) rather than simply a distraction effect of cues on screen detracting from performance.

It is hypothesised that those in the CS+ condition would show greater behavioural impulsivity than those in all other conditions, and that those in the CS- condition would show reduced impulsivity in both tasks compared to the other conditions. It is also hypothesised that there will be an interactive effect between TFEQ-D and CS+ exposure leading to greater impulsivity, building on a possible mechanism discussed by Yeomans & Brace (2015), and given other work exploring the role of restraint, but not as of yet disinhibition in behaviours following food-cue exposure (Federoff et al, 2002). We also aim to explore the interactive effects of TFEQ-D and TFEQ-R on behavioural impulsivity independent of condition.

5.1 Method

5.1.1 Participants

To achieve 90% power at an effect size of $d = .47$ (the effect size of the difference in HDHR groups in the between conditions contrast in experiment 1), a sample size of 120 was calculated. This was the smallest effect detected as was chosen to represent sample size calculations. Participants were 120 female members of staff or students at the University of Sussex, who had previously completed a recruitment questionnaire that also included the Three-Factor Eating Questionnaire, as well as smoking status, food preferences and allergy information. Participants were excluded from participation if they had any known allergies, smoked regularly (>5 per week) or were taking regular prescribed medication (other than the contraceptive pill) or if self-reported BMI was greater than 30 (actual BMI ranged from 18.5-31.4, $M = 25.2$, $SD = 2.9$). Participants were ineligible to participate if they had previously taken part

in a previous experiment conducted in this thesis or other similar studies in our laboratory. Seven participants were excluded for erratic responding on the DDT task or inhibitory control measures (as detailed in previous studies in this thesis). Groups were characterised as high and low TFEQ-Disinhibition and TFEQ-Restraint using a median split, and further characterised into four interaction groups of high and low TFEQ-D and TFEQ-R (HDHR, HDLR, LDHR, LDHR) in accordance with the successfully employed methodology of past studies from our laboratory (Yeomans, Leitch & Mobini, 2008). Experimental conditions did not significantly differ in TFEQ-D ($F(3,109) = 1.243, p = .30$), TFEQ-R ($F(3,109) = .406, p = .75$), BMI ($F(3,108) = .770, p = .51$) or hunger ($F(3,107) = 1.167, p = .18$).

Table 5 BMI, TFEQ-R and TFEQ-D (+/-SEM) between TFEQ-D and TFEQ-R interaction groups

	HDHR	HDLR	LDHR	LDLR
BMI	25.09 +/- .42	25.14 +/- .54	25.16 +/- .69	25.67 +/- .65
TFEQ-R	11.52 +/- .39	3.69 +/- .53	11.56 +/- .64	3.48 +/- .59
TFEQ-D	9.83 +/- .31	9.23 +/- .42	4.11 +/- .50	4.48 +/- .46

5.1.2 Materials:

Cues: The cues used were abstract symbols (Figure 7) previously used in conditioning procedures in our laboratory (e.g. Ridley-Siebert, Crombag & Yeomans, 2015). The symbols were randomised per condition, other than the novel cue (the right hand cue in Figure 7), which was not used in the conditioning procedure to maintain its novel nature to the participants.



Figure 5.1: Cues/symbols used in the conditioning procedure and later in the impulsivity measures.

DDT (Delayed Discounting Task): This is the same DDT task with adjusting algorithm as used in Studies 2 and 3 (see methods of these studies for algorithm and analysis details) of this thesis. However, between blocks (each delay point) participants were displayed their condition relevant symbol in the centre of a black screen for 2 seconds before returning to the task. The programme was launched on a Windows 7 computer using MATLAB v.2013 software and the Psychtoolbox add-in.

Stop-Signal Task: The stop signal task is a modified version of the program created by Verbruggen et al (2008). The programme was launched on a Windows 7 computer using MATLAB v.2013 software and the Psychtoolbox add-in. Participants are required to attend to either a white left or right facing arrow on a black screen (1 trial) by pressing the corresponding left or right key on the keyboard (go-trials). On stop trials, the arrow turns blue after a variable delay and participants are instructed to try and withhold their response to these trials. Depending on the successful or unsuccessful inhibition of response to stop-trials, the time at which the stop-signal (arrow turning blue) occurs on stop trials either increases or decreases – stop-signal onset. The onset time is increased, making the task more difficult if inhibition is successful. This procedure is the same as discussed by use in the GoStop task and methodology by Dougherty et al (2003) in Studies 1, 2 and 3 of this thesis. On each trial, the condition-appropriate cue is presented above the task arrows for the length of each trial, or no cue in the no-cue control condition. Participants completed a practice block of 32 trials, followed by 3 blocks of 64 trials. Two outcome variables are determined from this task – commission errors (incorrect responses to stop-trials – 25% of trials) and stop-latency (as used in previous studies in this thesis, the average incorrect response time to stop-trials in ms).

Hunger ratings: VAS mood ratings using SIPM (Sussex Ingestive Pattern Monitor) software (see previous chapters' experimental methods materials).

Conditioning Procedure: The conditioning procedure of the experiment used was a triangle test (e.g. Yeomans et al, 2009) disguised as an odd-one-out test. There were

five sessions in total, with five trials in each session (one presentation of set A, B, C, D and E). During each trial, participants were presented with three solutions. Using E-Prime (version 1.2) participants were instructed the following:

“For this task, you will be presented with an odd-one-out task. You will be asked to take one of the Sets of drinks and place it in front of you. Your task will be to taste each solution and determine which solution is the odd-one-out based on its taste. Some trials will be hard and some will be easy. You must select the solution that you think is the odd-one-out by its location on the tray. For example, if you think the solution on the left of the tray is the odd-one-out then click the picture on the left of the screen. When you try a solution put it back on the tray. Do not pick up the next solution until you have returned the first solution to the tray. Spit the solution into the bottle provided then swill your mouth with water. Please only take one sip of each solution per task.”

The would-be conditioned stimuli (figure 7) associated with each set were presented in two locations; first using 4 x 4 cm stickers directly placed on the cups and secondly on a 17" LED screen located on a desk in front of the participant (the three images presented equidistant along the screen with the question above them). The three cups in each set had the same CS sticker on the side of it that was specific to the solution in the cup (i.e. one CS for sweet solutions, one CS for neutral solutions, etc.). All three CS' on screen and the cups were the same image. Participants were choosing which CS to pick based on its location corresponding to the samples on the tray. Participants tasted the three samples and once they identified an odd sample had to click on one of the three CS images on the screen. The CS+ condition solution was a 10% sucrose solution. the CS- was 'artificial saliva' (1.865g/l of potassium chloride (KCl), and 0.210g/l sodium bicarbonate, NaHCO_3), and two filler/bogus sets of solutions consisted of either raisin or kiwi flavouring 35 drops/250g of water.

To ensure participants understood the task a practice trial (one solution orange cordial and two water) was conducted with the experimenter present. For sets A and B the three solutions were identical. This forced participants to closely

attend to the flavour of the solutions and also to the stimuli presented on the screen. Sets C, D and E were control stimuli, used to keep the guise of the study (an 'odd-one-out-task'). Participants were instructed to only take one sip of the solutions per session, as this would ensure a standard 25 CS-US pairings for all participants. After each US sampling, participants were instructed to spit the solution and rinse their mouth with water.

All images, tastes and order of tasting was counterbalanced and randomised across participants.

Contingency Awareness Test: Following the completion of the conditioning task, participants were shown each symbol again separately and were asked 'How likely do you think it would be to taste a liquid with (fruity/sweet/salty) flavour if you saw this symbol?' on a VAS scale anchored from 'very likely (scored 100) to 'very unlikely' (scored 0). Participants were deemed aware of the association between the stimulus symbol and the stimulus sample (US-CS contingency) if they said the paired US symbol was more likely to be paired with the paired CS than any of the unpaired USs. For example, to be aware of the sweet-paired CS a participant would have to give a higher rating for that CS on the sweet scale than the fruity or salty scales.

Sweet Liking Questionnaire: Participants were then asked to rate how much they liked the liquid with the (sweet/salty/fruity) flavour on a VAS scale on 1-100.

5.1.3.Procedure:

Participants were recruited using an online recruitment method (SONA) at the University of Sussex. Participants were scheduled into a timeslot between 13:00 and 16:00. Upon arrival participants completed their consent forms and filled in the mood (hunger) questionnaire. Participants were then asked to complete the conditioning procedure, followed by the contingency awareness test and the sweet-liking questionnaire. Participants then had a 30-minute break. Following the break, participants completed the Stop-Signal Task and the DDT measure in a randomised

order. Those in the CS+ condition were presented with the symbol associated with sweet solution before discounting trial blocks and during stop-signal trials, CS- were presented with the artificial saliva conditioned symbol, the novel condition were presented with a previously unseen symbol, (to control for the effects of symbol distraction rather than specific effects of each conditioned symbol) and the no-cue condition did not get shown a symbol throughout the impulsivity tasks. In this experiment, a reflection impulsivity subtype task is removed from the battery of impulsivity tasks. This is due to the lack of association with reflection impulsivity with our experimental manipulations thus far in this thesis, and also in the interests of time and the prevention of participant fatigue. For example, with an additional conditioning procedure, followed by a break and the two impulsivity tasks (Go/No-Go, and DDT) which participants are asked to retain concentration, it is thought that a further impulsivity task would increase the chances of fatigued performance on one of the randomised tasks, therefore reflection impulsivity was removed from the battery. Participants' height, weight and age was measured. Participants were debriefed, thanked for their time and were reimbursed with course credits or £6 for participation.

Table 5.2 Means (SEM) for TFEQ variables, BMI and impulsivity measures

	No Cue	Novel Cue	CS+	CS-
TFEQ-R	6.9±.5	7.4±.5	8.9±.6	7.2±.6
TFEQ-D	7.1±.4	6.6±.4	6.7±.5	6.2±.5
BMI	24.7±.5	25.6±.5	25.8±.7	24.5±.7
DDT auc	.53±.1	.54±.1	.53±.1	.57±.1
StopSignal Commission	40.5±1.6	41.2±1.6	37.3±1.9	40.6±1.9
Stop Signal Stop Latency (ms)	557.4±36.4	532.4 ±29.9	561.9±36.4	557.4±36.4

5.1.4 Statistical Analyses

Participants were median split into high and low restraint and disinhibition groups which each interacted with condition (4 x 2 ANOVA). The interaction groups were also examined with the dependent variable (TFEQ-D x TFEQ-R (2x2)). The three-way interaction was not analysed due to much reduced group sizes at that interaction level, and the rationale of TFEQ-D as the core moderator in the relationship between condition and outcome.

5.2 Results

The analyses were performed originally by separately covarying hunger, BMI, awareness of cues (contingency awareness), and in the CS+ condition whether the participant indicated sweet-liking preference. However, none of these variables affected any of the models in terms of variable significance and were thus removed from the final analyses. Condition and TFEQ interaction variables' means and SEMs can be seen in Tables 7 and 8. In terms of contingency awareness or awareness of what the cues in the study were associated with, 47/80 participants were deemed as aware of the cue association. However due to the statistical similarity between analyses with and without contingency aware participants, results here are reported without the covarying contingency awareness.

DDT: The prediction that the CS+ condition would increase impulsivity on the DDT [i.e. lower DDT AUC scores] was not supported: there was no significant difference in DDT AUC scores overall between the four conditions [$F(3,97) = .09, p = .97, \eta^2 = .01$; Figure 8.A]. TFEQ-Disinhibition group [$F(1,97) = .16, p = .69, \eta^2 = .01$], TFEQ-Restraint group [$F(1,97) = .001, p = .97, \eta^2 < .01$] and their interactive product [$F(1,97) = .13, p = .72, \eta^2 = .001$] also did not differ significantly on DDT AUC scores. The interaction between condition and TFEQ-D [$F(3,97) = .83, p = .48, \eta^2 = .026$] and condition and TFEQ-R [$F(3, 97) = .75, p = .524, \eta^2 = .02$] was also not significant with regards to DDT area-under-the-curve scores [See figures 5.2.A, B and C and Table 5.3].

Stop-signal Commission Errors: The prediction that the CS+ condition would increase impulsivity on the Stop-signal task [i.e. higher stop-signal commission errors] was not supported: Stop-signal commission errors did not differ significantly between conditions [$F(3,97) = .87, p = .46, \eta^2 = .03$], TFEQ-D [$F(1,97) = .72, p = .40, \eta^2 = .007$], TFEQ-R [$F(1,97) = .001, p = .97, \eta^2 < .01$] and their interactive product [$F(1,97) = .13, p = .72, \eta^2 = .001$].

=.01], TFEQ-R [$F(1,97) = 2.40, p=.13, \eta^2=.02$], the interaction between TFEQ-D and TFEQ-R [$F(1,97) = .44, p=.511, \eta^2=.01$] was also not significant. The interaction between condition and TFEQ-D [$F(3,97)=1.73, p=.17, \eta^2=.05$] and condition and TFEQ-R [$F(3,97)=.36, p=.78, \eta^2=.01$] were also not significant with regards to stop-signal commission errors. [e.g. disinhibition and restraint groups did not differ on commission errors depending on condition].

Stop-signal Stop-Latency: The prediction that the CS+ condition would increase impulsivity on the stop-signal task [i.e. increased stop-latency scores] was not supported: there Conditions [$F(3,97)=.17, p=.92, \eta^2=.01$], TFEQ-D [$F(1,97)=1.13, p=.290, \eta^2=.01$] and TFEQ-R [$F(1,97)=2.06, p=.16, \eta^2=.02$] were not significantly different on stop-latency performance. The interaction between TFEQ-D and TFEQ-R [$F(1,97)=2.41, p=.12, \eta^2=.03$], condition and TFEQ-D [$F(3,97)=.846, p=.47, \eta^2=.026$], and condition and TFEQ-R [$F(3,97)=.18, p=.91, \eta^2=.01$] was also not significant in terms of stop latency performance [See figures 5.2.D, E and F and table 5.4].

There was no significant difference between control, anticipation, hedonic, or non-hedonic conditions in PDT area-under-the-curve [$F(3,59) = .137, p = .94$], and there was no significant effect of TFEQ-D [$F(3,59) = <1, p = .49, \eta^2=.01$], , TFEQ-R [$F(3,59) = 1.39, p = .24, \eta^2=.02$], , the interactive effects of TFEQ-D/R [$F(3,59) = 1.00, p = .32, \eta^2=.02$], the interaction between condition and TFEQ-D [$F(3,59) = <1, p = .95, \eta^2=.01$], or condition and TFEQ-D and TFEQ-R [$F(3,59) = <1, p = .90, \eta^2=.01$], , and no significant covariates of hunger or BMI [all effects at $p>.05$].

The GoStop stop-latencies were log10 transformed in the analyses to correct for normality violations, but for sake of interpretability, the means are reported here in their original metric. There was no significant difference between control, anticipation, hedonic, or non-hedonic, conditions in DDT area-under-the-curve [$F(3,58) = 1.11, p = .35, \eta^2=.05$], and there was no significant effect of TFEQ-D [$F(3,59) = <1, p = .90, \eta^2=.01$], TFEQ-R [$F(3,59) = <1, p = .92, \eta^2=.01$], the interactive effects of TFEQ-D/R [$F(3,59) = <1, p = .68, \eta^2=.01$], the interaction

between condition and TFEQ-D [$F [3,59] = 1.29, p = .29 \eta^2 = .06$], or condition and TFEQ-D and TFEQ-R [$F [3,59] = .<1, p = .65 \eta^2 = .03$], and no significant covariates of pre-test hunger or BMI [all effects at $p < .05$] See table 4.2.B and Figure 4..

There was no significant difference between control, anticipation, hedonic [155.90 +/- 12.45], or non-hedonic, conditions in IST amount of boxes opened [$F [3,54] = 1.738, p = .170 \eta^2 = .07$], and there was no significant effect of TFEQ-D [$F [3,54] = <1, p = .35 \eta^2 = .01$], TFEQ-R [$F [3,54] = <1, p = .84 \eta^2 = .01$], the interaction between condition and TFEQ-D [$F [3,59] = <1, p = .54 \eta^2 = .03$], or condition and TFEQ-D and TFEQ-R [$F [3,59] = .<1, p = .98 \eta^2 = .002$], the interactive effects of TFEQ-D/R [$F [3,59] = .<1, p = .78 \eta^2 = .01$], , and no significant covariates of pre-test hunger or BMI [all effects at $p > .05$] See table 4.2.C and Figure 4..

Table 5.3: DDT Means (\pm SEM) of condition alone, high and low TFEQ-D groups, and high and low TFEQ-R and TFEQ-D interaction groups between conditions.

	No Cue	Novel	CS+	CS-
Condition	40.49 \pm 1.59	41.22 \pm 1.61	37.34 \pm 1.95	40.58 \pm 1.01
Low TFEQ-D	39.31 \pm 2.48	38.08 \pm 2.34	36.000 \pm 3.35	46.40 \pm 3.67
High TFEQ-D	41.66 \pm 1.98	44.35 \pm 2.22	38.68 \pm 2.00	37.07 \pm 2.19
LRLD	41.80 \pm 3.67	38.80 \pm 3.67	37.50 \pm 3.35	46.40 \pm 3.67
LRHD	44.14 \pm 3.10	44.83 \pm 3.35	38.00 \pm 3.35	38.86 \pm 3.10
HRLD	36.83 \pm 3.35	37.38 \pm 2.90	34.00 \pm 5.80	40.00 \pm 5.80
HRHD	39.18 \pm 2.47	43.88 \pm 2.90	39.38 \pm 2.19	37.07 \pm 2.19

Table 5.4: Stop-Signal Stop-Latency Means (\pm SEM) of condition alone, high and low TFEQ-D groups, and high and low TFEQ-R and TFEQ-D interaction groups between conditions.

	No Cue	Novel	CS+	CS-
Condition	543.392 \pm 29.55	532.416 \pm 29.10	561.969 \pm 36.24	557.459 \pm 36.43
Low TFEQ-D	576.42 \pm 46.14	518.07 \pm 43.44	614.25 \pm 62.22	557.17 \pm 63.75
High TFEQ-D	510.37 \pm 36.84	546.76 \pm 41.15	509.69 \pm 37.18	557.75 \pm 35.27
LRLD	516.82 \pm 68.15	482.67 \pm 68.15	533.08 \pm 62.22	534.99 \pm 68.14
LRHD	515.05 \pm 57.60	572.71 \pm 62.22	520.04 \pm 62.22	524.71 \pm 57.60
HRLD	636.01 \pm 62.26	553.46 \pm 53.88	695.43 \pm 107.76	579.35 \pm 107.77
HRHD	505.68 \pm 48.95	520.81 \pm 53.88	499.33 \pm 40.73	590.78 \pm 40.73

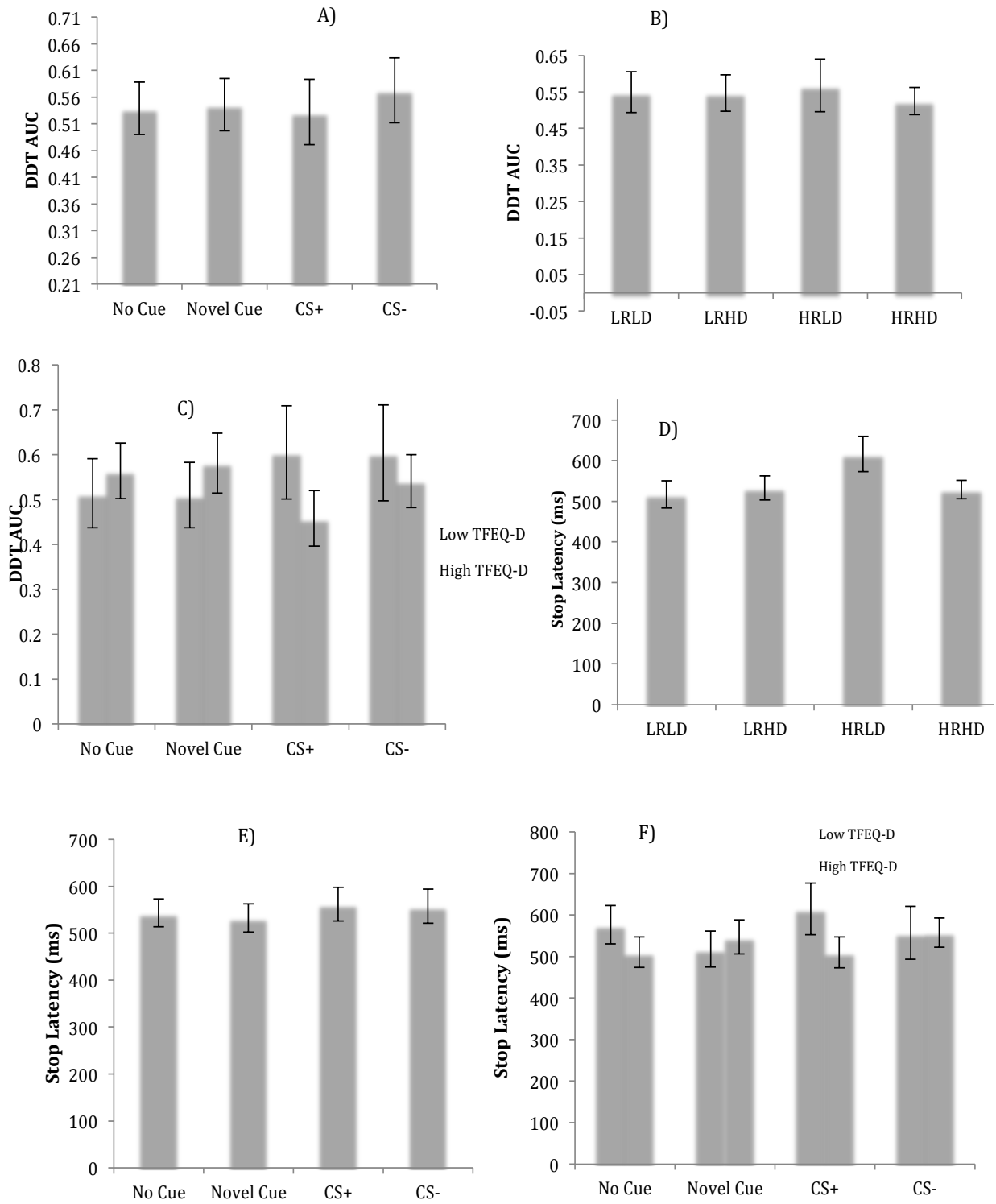


Figure 5.2: Error bars indicate +/- SEM A) DDT AUC between conditions, B) Between TFEQ interaction groups, C) between high and low TFEQ-D groups between conditions, C) Stop latency between TFEQ interaction groups, D) between conditions, E) between high and low TFEQ-D groups between conditions.

5.3 Discussion

This experiment attempted to use a conditioned reward (sucrose) cue to modulate impulsive responding on a DDT and Stop-Signal task. However, there was no evidence of a significant difference between our experimental and control conditions.

This study provided no evidence of a conditioned reward cue modulating behavioural impulsivity, which fails to support previous research from our laboratory demonstrating the modulation of monetary preferences in the DDT (Yeomans & Brace, 2015). However the previous study demonstrates this effect using the exposure of explicit food-cues immediately before the completion of a DDT, this may then suggest that explicit food cues may produce a more pronounced effect on subsequent responding. However this is not the first study to find limited differences in discounting following cue exposure. For instance, Field et al (2009) demonstrated in smokers, that independent of subjective craving, that smoking cues did not modulate decision-making. In terms of the relationship between TFEQ-D and TFEQ-R, the high/low interaction groups did not reach significance.

With regards to Stop-Signal results, our data produced quite unexpected patterns – we failed to find any significant differences with regards to condition, or the role of TFEQ-D and TFEQ-R. Mixed findings with inhibitory control are not unique to this thesis; The work of Caswell (unpublished thesis,) also failed to consistently modify impulsivity, suggesting that without manipulating cognitions, state impulsivity can be overridden. Van Holst et al (2012) also posited unexpected findings. In their study of problem gamblers and inhibitory modulation using gambling cues, they found that problem gamblers were more successful at inhibiting responses on gambling salient trials. This would be unexpected given the hypotheses we constructed, but Holst et al forward the idea that exposure to participant salient contexts (in their case gambling cues, in our a reward conditioned cue) may facilitate more successful inhibition, which may also support why we have the opposite to the expected findings between our high/low TFEQ- condition groups, maybe suggesting that in high TFEQ-D participants exposed to the reward cue, this reward cue may differentially facilitate

inhibitory performance. However of course these groups were not significantly different, and these explanations are discussed purely as potential mechanisms. On the other hand, given the small differences between groups, and the lack of statistical significance for these effects, this may demonstrate yet another example of the mixed findings in the impulsivity and food literature (Faye et al. 2015, inhibitory leading to initiated snacking behaviour), particularly the mixed findings of cue (conditioned or existing-food) and impulsivity mirroring the failure to detect participant differences in cue-exposed groups in inhibitory control in the work of Luijten, Little and Franklin (in smokers and smoking cues, 2011), Forzano et al (2010, food cues) and Nederkoorn, Van Eijs and Jansen (2003, food cues), although the latter demonstrated differences between restrained and unrestrained participants which we did not detect.

We must however consider why these cued impulsivity effects were not visible or potentially detectable from the paradigm employed. The rationale for this study stemmed from previous work in this thesis in cueing impulsivity through a milkshake preload, and other work in our laboratory linking food-cue exposure to impulsivity and reward-conditioned stimuli to overeating (e.g. Doran et al. 2007, Papachristou et al. 2012). However, one possible explanation for our findings not replicating the conditioned-stimuli to behavioural mechanism may be either the possibility of extinction or the lack of subsequent reinforcement. For example, in this paradigm (Ridley-Siebert et al, 2015), the Pavlovian conditioning phase was the same, but rather than impulsivity measures, the participants completed an ad libitum intake task, with their condition-relevant stimuli on the food containers. However it is possible that CS+ participants are again reinforced during the intake phase, as again the CS+ stimulus is being paired with rewarding outcomes. In our paradigm, although the impulsivity measures present the CS+ stimulus, the rewarding outcomes are not present. It is possible then that the presentation of the CS+ stimulus without the (possibly interactive) presentation or reinforcement may lead to extinction in some participants. It would be possible to examine this in a more complex design, using conditioned cues, food-cues and the combination of the two to understand the relationship between the food and would-be conditioned cues to determine if subsequent reward pairings are what limit the effects of our current paradigm.

Alternatively, others have posited the idea that motivational modulation through cue exposure may be highly specific. For example, Federoff et al (1997) suggested that in restrained eaters, following cue exposure the increased intake was associated only with the reward in which they had subsequently been exposed to. This may then suggest the possibility that if the conditioned stimulus is no longer paired with a food-reward, or if a reward is not available for participant receipt, then behavioural motivation or responding may remain unaltered. This specificity to the cued reward is also mirrored in the work of Tetley, Brunstrom & Griffiths (2009) and that changes in subjective motivation to consume rewards only appeared to be modified to by the target cue to the target food-reward.

Limitations

As mixed findings in the literature and in the current thesis suggest, the role of cues or consumption in subsequent behaviour, specifically behavioural impulsivity, are difficult to detect and seem to be particularly sensitive. In this design (not to mention the impulsivity literature generally), it is acknowledged that there are a great number of variables. For example, there is a possibility to manipulate a wide number of variables to attempt to detect these effects in a way that may make the relationship a little clearer. For example, other researchers have attempted to focus on the experimental manipulation of hunger state as way of enhancing the ability to detect the relationship between cue exposure and outcome. For instance, hunger state may increase reward motivation thus leading to a clearer pathway between cue exposure and subsequent behaviour, and some researchers have been successful in demonstrating this (Sobik, Hutchison & Craighead, 2003; Loeber et al, 2013) and the previous experiment discusses at length possible link between hunger/satiety and impulsive behaviours. Although we controlled for hunger as a covariate in the statistical design, this did not allow us to examine the controlled manipulation or enforcement of a state of acute deprivation, which appears to have been the most successful in dissociating cue-exposure conditions. Another variable which seems to be a contributing factor in cue-reactivity/exposure paradigms is that of weight status. Although we had a range of BMI scores, our sample was distinctly healthy-weight. Other researchers have found it fruitful to interact weight status ('lean' vs. overweight) in cue-exposure-outcome paradigms (Ferriday & Brunstrom, 2010),

which was not possible to investigate with our current normal-weight student population. It is possible that the variables associated with task specificity may also provide difficulties in detecting group differences. For example, there may be subtle differences in the processes involved in Stop-Signal vs. Go-No/Go paradigms (which are often clustered together as measuring similar phenomena, not always with justification, Caswell et al, 2014; thesis). In terms of the DDT, there are innumerable manipulations possible in terms of the delay periods used, to attempt to elicit cue-induced group differences. For example, our longest delay period, one year may not require a sufficient amount of ‘future planning’ to be able to detect the greatest differences between groups, which may become more pronounced at delay points of greater lengths of time (e.g. 5 years as demonstrated in Experiment 1 of this thesis), but this is simply one of many manipulable factors possible that may enhance the ability to detect these currently sensitive effects following cue exposure.

One additional limitation to the methodology of the study is the time difference between the conditioning procedure and the impulsivity measures (30 minutes). It is possible for example, that any cued effects may have faded at the point at which the impulsivity tasks were completed, another possible explanation as to why there was no significant difference between our experimental conditions. Although participants’ awareness was tested, this was before the 30-minute break before the impulsivity tasks. Although this timeframe has been used successfully in previous work in our laboratory, we can be sure as to the lasting effects of conditioning, or the potential for the effects to fade in the experimental session. Future conditioning work in this type of study would be fruitful in considering implementing a second contingency awareness measure following the 30-minute break, to evaluate the length to which the conditioning effect may last.

5.3.1 Implications and Future Directions

As discussed, through the implemented cue-exposure paradigm, we failed to detect a significant difference between our CS+(experimental) condition and our control conditions. However future work may allow the detection of such potentially sensitive effects. For example, it is suggested here that future work aim to isolate polarised

TFEQ differences that given our current population was not possible. For example, although the range of TFEQ scores in our sample was large, without a large sample with identified participants at the extreme scoring points of the TFEQ scales, it is difficult to split groupings in this way. It may therefore be that TFEQ-interaction effects with cue exposure are more pronounced in the extreme scoring conditions, something that is yet to be examined. Also, future work may be fruitful in employing an enforced period of acute food deprivation in order to create a controlled hunger vs. non-hunger condition paradigm. As previously discussed, cue-exposure and to some extent disinhibition effects seem to be more pronounced or detectable between hunger state groups, and may allow for the investigation of the role of enhanced reward motivation through hunger (Epstein et al. 2010) and its interaction with conditioned cue exposure which seems to have been an important moderator of these effects in past research (Loeber et al. 2013). It is felt that it would be interesting to explore these either polarised or experimentally manipulated group differences with regard to possible task-specific differences. For example, it is possible that these group differences may interact with between-task differences in DDT delays, and it may be possible that under certain conditions (e.g. hunger, cued, extreme scoring TFEQ groups, weight status) it is possible to detect not only DDT differences but also reward and delay magnitude differences between DDT tasks.

5.3.2 Conclusion

As previously discussed, this study failed to find significant differences between our cue conditions. We have posited possible mechanisms behind this instance of inability to detect sensitive cued effects on impulsive behaviours. We also forward possible future directions for this type of cued paradigm, paying particular attention to the need to experimentally control and split participant hunger/acute food deprivation states, and to be vigilant in the recruitment of participants who fulfill the quota of extreme TFEQ scores required to investigate the nature of the effects of polarised TFEQ scorers, potentially by way of the outermost groups in a TFEQ tripartite split. A further examination of some of the paradigm and task's variables was also forwarded, specifically in terms of DDT reward and delayed magnitude.

Experiment 5: A Meta-Analysis examining Dietary Disinhibition (TFEQ-D) and Delayed Discounting (DDT) and the moderating role of cue exposure or preload consumption

6.0 Introduction

The role of impulsivity in eating behaviour has received much attention in the past decade. Research has attempted to understand the association between subtypes of impulsivity, and eating disorders, dietary attitudes, eating behaviours and weight gain. However, although some work has yielded interesting findings between these concepts (Yeomans et al. 2008), what has become clear is that the relationship between measures of delay of gratification (impulsive choice) through delayed discounting tasks and measures of dietary attitudes are inconsistent. Specifically studies examining the relationship between the disinhibition (TFEQ-D) subscale of the Three-Factor Eating Questionnaire (TFEQ; Stunkard & Messick, 1985) and discounting measures have revealed mixed findings. In some studies, it appears that TFEQ-D is strongly correlated with discounting behavior (e.g. Yeomans et al, 2008), or at least provides a moderating role (e.g. Rollins et al, 2010), whereas some studies failed to reveal a significant relationship between these variables. In particular, 3 of the experiments conducted in this thesis (Experiment 1, Experiment 3, Experiment 4), failed to replicate earlier findings of both a relationship between TFEQ-D and discounting (Leitch, Morgan & Yeomans, 2013), or TFEQ-D as a moderator in the relationship between cue-exposure or preload consumption and delayed discounting (Yeomans & Brace, 2015).

As discussed at length in the introduction to this thesis, there is a body of evidence linking delayed discounting and adiposity (Weller, 2010), and at least some evidence to suggest TFEQ variables are also implicated in weight gain. Recent data from our laboratory also seems to demonstrate an association between discounting and TFEQ (Leitch et al. 2013). Why then has there been such inconsistency in replicating these findings? This may be an example of

publication bias, with a failure to publish non-significant findings, but alternatively it is possible that there is a relationship between discounting and TFEQ, yet this is difficult to detect statistically, even when studies are designed using power calculations based on published successful studies, perhaps because the real effect size of the reported relationship is smaller than that in published studies. Much work in the literature also seems to address the link between weight status and discounting in samples of lean vs. obese participants; it may be equally possible that the difference between these groups has a greater probability for statistical detection, or is more difficult to detect in between normal-weight samples. Delayed discounting or impulsive choice seems to have the most support in its relationship to TFEQ-Disinhibition (Leitch et al, 2013), particularly with regard to cue interactions (Yeomans & Brace, 2015). Therefore, discounting is the main focus of this meta-analysis. It would be possible to examine the relationship between cue-exposure, TFEQ-Disinhibition and other measures (particularly inhibitory control), but as of yet there is little literature other than that conducted in this thesis that examines inhibitory control or reflection impulsivity and TFEQ-Disinhibition in a cue exposure paradigm. Therefore the body of work in our laboratory and in this thesis allows us to specifically explore discounting, disinhibition and cue exposure.

The meta-analysis reported here intends to re-evaluate the relationship between discounting and TFEQ-D, and to examine the combined relationships between the variables of interest. It is possible that the inconsistencies in results may stem from fundamental differences in the tasks and methodologies used. For example, there are no established standardized delays between studies using delay-discounting tasks, and the longest delay point between tasks can range from 6 months, up to 6 years. It is not clear what effect this variation in delays might have in modifying the sensitivity of the DDT task to detect individual differences in responses. For example, it is possible that there is great variation in the way an individual values shorter delays, which are clustered together, but we cannot be sure that these responses are not subsequently anchored by the presence of an extremely large delay, and we cannot be sure which individuals or

populations may be sensitive to an anchoring effect. (Explain) Having said this, this study intends to examine the relationship between discounting measures and TFEQ-D scores, whilst attempting to negate the issue of differences in delay methodologies. This study takes the past decade's research from the Sussex Ingestive Behaviour Unit's (SIBU) research using delayed discounting measures to examine whether there is an overall association between discounting and TFEQ-D, and whether there is consistent evidence to suggest a moderating role of TFEQ-D in exposure to experimental manipulation (preload, food-cues, food-associated cues) to subsequent discounting. The studies included were contingent on their completion at the Sussex Ingestive Behaviour Unit, and included both the data available for the DDT measure and TFEQ-D information. The studies included were conducted by myself, Leitch et al (a previous SIBU doctoral student; (Leitch, 2011)), and a number of undergraduate students collecting data for psychology research projects, all of which were supervised by Professor Martin Yeomans.

6.1 Methods:

6.1.1 Study Selection:

Traditionally when formulating a meta-analysis, one would search the online databases (e.g. PsychInfo, Google Scholar). However, when that search was conducted in the present context it was clear that there are comparatively few studies that have examined both variables of interest (TFEQ-D and DDT together), and the two main published examples were both studies conducted at SIBU. Moreover in the second planned component to the meta-analyses (the moderating relationship between experimental preload or food cue exposure and TFEQ-D on DDT), no other studies to date other than from our laboratory have examined these relationships, thus the only included datasets are from the previous work conducted at SIBU. Other laboratories have examined discounting in relation to scores on the Dutch Eating Behaviour Questionnaire (DEBQ, Van Strien, 1986), and the outcome of those studies are referred to in the discussion for comparison, but were not part of the reported meta-analysis.

6.1.2 Meta-Analysis studies included (Participant BMI and age means included where available):

Experiment 1 (thesis, 2013)

- Study aimed to contrast impulsivity responses between a preload consumption (experimental) and a no consumption (control) condition. 100 female participants. Aged 18-46 ($M=21.3$, $SD=3.7$), with BMI ranging from 18-30 ($M=23.3$, $SD=3.3$). Chocolate milkshake preload condition vs. no consumption condition prior to battery of impulsivity measures. TFEQ, DDT (1 day, 2 days, 1 week, 2 weeks, 1 month, 2 months and 6 years timepoints, AUC included), GoStop measure, MFFT.

Experiment 2 (thesis, 2014)

- Study aimed to examine the interrelations between the reinforcing value of food (RRV) with subscales of impulsivity, and a measure of self-report impulsivity. 80 female participants. 2-session experiment, 1st session a snack taste test, 2nd session the RRV task (see chapter X), a GoStop task, Information Sampling Task, BIS/BAS, and DDT measure (1, 7, 30, 90, 180, and 365 days timepoints).

Experiment 3 (thesis, 2014)

- Study aimed to contrast impulsive responding between a hedonic preload, a non-hedonic preload, a preload anticipation, or a no consumption condition. 100 female participants aged between 18-42 ($M=20.9$, $SD=3.1$), with BMI ranging from 18-30 ($M=24.9$, $SD=3.1$). 4-condition between-groups design; hedonic milkshake vs. non-hedonic milkshake vs. anticipation no consumption vs. no consumption conditions prior to impulsivity task battery. TFEQ, DDT measure (1, 7, 30, 90, 180, and 365 days timepoints), GoStop Measure, IST.

Experiment 4 (thesis, 2015)

- Experiment aimed to contrast impulsive responding between groups following different cue exposures; hedonically associated cue, neutrally associated cues, non-conditioned cue, or no cue. 120 female participants. BMI ranged from 18.5-31.4, $M=25.2$, $SD=2.9$). 4-condition between groups design; sweet conditioned cue (CS+) vs. neutral conditioned cue (CS-) vs. novel cue vs. no cue (presented during impulsivity battery blocks/trials). TFEQ, DDT (1, 7, 30, 90, 180, and 365 days timepoints), Go/No-Go.

Leitch, M. - Thesis (2008) Experiment 1.

- 100 female participants, general associations study between measures of dietary restraint and behavioural/self-report measures of impulsivity. TFEQ, DEBQ, DDT (0, 7, 30, 90, 180, and 365 days timepoints), BIS-11, BIS/BAS, SPSRQ, BART, BES.

Leitch, M. - Thesis (2008) Experiment 2

- 64 female participants. A preload vs. no preload condition study examining behavioural impulsivity following chocolate sundae consumption. Fixed ice-cream preload condition vs. no consumption condition. TFEQ, DDT (0, 7, 30, 90, 180, and 365 days timepoints), BART, MFFT.

Yeomans, Leitch & Mobini (2008)

- 147 female participants (mean BMI=23.03, SD=2.82, mean Age = 21.97, SD = 4.79). General association study. Measures included TFEQ, Dickman's, BIS-11, DDT measures (0, 7, 30, 90, 180, and 365 days), k-value included and standardized.

Leitch, Morgan & Yeomans (2013)

- 80 female participants (mean BMI=22.88, SD=2.77), controlled vs. unrestricted consumption conditions prior (overnight) to experimental session to examine heightened hunger on impulsivity. TFEQ, BIS-11, Go/No-Go, MFFT, DDT (0, 7, 30, 90, 180, and 365 days timepoints, AUC presented).

Yeomans & Brace (submitted, conducted in 2013)

- 98 female participants (mean BMI=22.88, SD=8.24, mean Age=21.39, SD=8.05). Food-cues vs. pair-matched neutral cues (exposure prior to battery of other measures to explore the relationship between food-cue exposure and impulsivity). Measures included DDT same timepoints as above, AUC included. Included TFEQ, BIS/BAS, BART, Go/No-Go.

Undergraduate Project (Conducted in 2008)

- Undergraduate project 1. (Conducted 2004-2005): 71 female participants (mean BMI = 23.1, SD = 3.1, mean age = 22.4, SD = 4.9). Project was part of a larger project examining the relationship between cognitive distortions and impulsivity (Published as Mobini et al., 2007), but only this subset completed the DDT. Relevant data were DDT and TFEQ scores for all 71 participants.

Undergraduate Project 2 (Conducted in 2015)

- 60 female participants (mean BMI = 24.32, SD = 4.81, mean age = 27.2, SD=10.63) Project examining the role of high-hedonic (food), low-hedonic (food) and neutral cue exposure on behavioural impulsivity (DDT, GoStop).

Table 6.1: Study Means (M), +/- Standard Deviation (SD) and N (number of participants) for experimental (exp) and control (con) groups.

Study Name	M exp	M con	Sd exp	Sd con	N exp	N con
Experiment1	0.44	0.62	0.24	0.24	20	21
Experiment3	0.48	0.58	0.28	0.28	12	15
Experiment4	0.46	0.56	0.25	0.25	20	33
UG project 2015	0.45	0.54	0.22	0.22	6	20
Leitch et al. (2013)	3248.66	3128.31	245.24	251.96	40	38
Yeomans & Brace (2015)	14200	20641	8885.42	8886.67	27	23

6.1.3 Statistical Analyses:

The meta-analysis takes the form of two separate analyses. The first is a random-effects correlation model examining the correlations and meta-correlation between DDT scores and TFEQ-D scores as continuous measures. The second analysis is a between groups standardized mean difference analysis examining differences between those high in TFEQ-D in treatment vs. control conditions.

6.2. Results

6.2.1 Random Effects Correlational Model (RE)

The studies included in the main model were all statistically homogenous ($Q(10) = 9.94, p = .44$). The random effects correlational model demonstrated no significant correlation between DDT measures and TFEQ-D scores, negative correlations are indicative of greater impulsivity (Figure 1), estimated meta-correlation -0.03 , SE 0.03 (CI lower -0.10 , CI upper $.003$).

6.2.2 Standardized Mean Difference Model (SMD)

The studies included in this second analysis were all statistically homogenous [$Q(5) = 3.67, p = .50$]. Negative numbers are indicative of greater impulsivity towards the experimental group; positive numbers indicate greater impulsivity in the control condition. As Discounting differs in the units reported between studies, the outcome variable is standardised with 0 representing no difference between groups, and negative and positive representing positive or negative differences between groups. Experimental groups consist of either a preload consumption, or food-cue exposure condition together vs. no consumption or neutral cue exposure condition. The standardized mean difference between those high in TFEQ-D in the experimental conditions and those high in TFEQ-D in the control conditions was significant [SMD estimate = $-.44$, SE $=.14$, CI lower = $-.71$, CI upper = $-.17$, $p = .001$, Figure 2]. Those in the experimental condition were $-.44$ SMD points more impulsive on the DDT than those in the control condition (See table 1 for means and SD of groups).

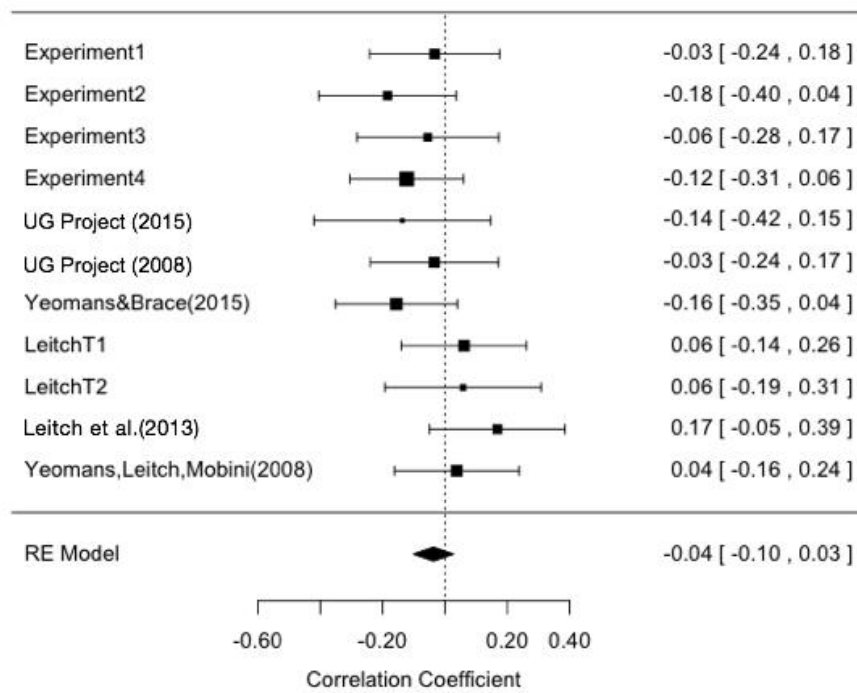


Figure 6.1: Correlation Meta-analysis between TFEQ-D scores and DDT AUCs. Points indicate correlation effect size (R) in favour of negative correlation (left) or positive correlation (right) +/- 95% confidence intervals.

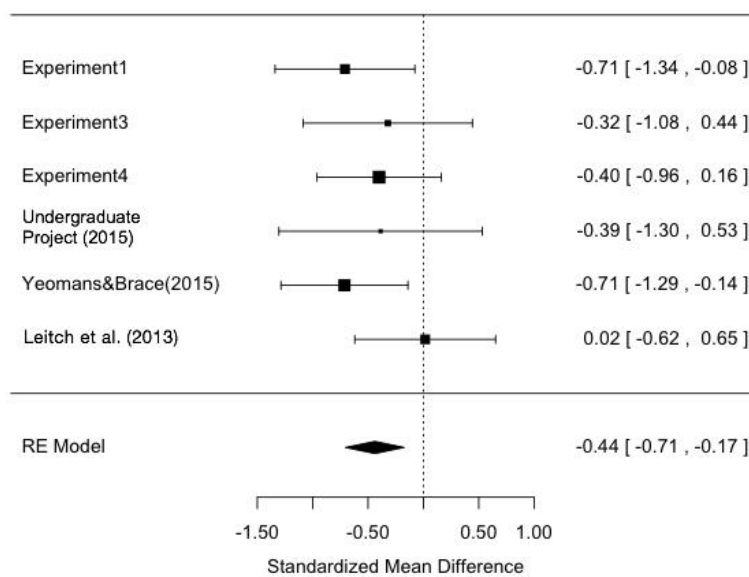


Figure 6.2: Standardized mean difference analysis between those high in TFEQ-D in experimental vs. control conditions. Points indicate mean differences points in favour of experimental groups as more impulsive (left) or control groups more impulsive (right), +/- 95% confidence intervals.

6.3 Discussion

The present meta-analysis has provided some useful insights into the relationships between TFEQ-D, Delayed Discounting and experimental moderators of this relationship. The analysis revealed no significant correlation between discounting scores and TFEQ-D overall, and some correlations were in the opposite direction to that predicted (e.g. Leitch 1, Leitch 2, Leitch 3, Yeomans, Leitch & Mobini, 2008). However, when looking at the moderating relationship of experimental condition (consumption of a preload, pre-test exposure to food cues), the meta-analysis provided evidence for a significant difference in DDT scores between those high in TFEQ-D who were in their respective experimental conditions, and those high in TFEQ-D who were in their respective control conditions. This statistically demonstrates in the same of studies conducted in this thesis (using a cue-exposure/preload design) and over the past decade at the Sussex Ingestive Behaviour Unit that those characterised as being high in TFEQ act more impulsively on discounting measure when exposed to their experimental condition's stimuli than those in the control (no exposure/consumption conditions).

Despite research from our laboratory previously demonstrating an association between discounting and TFEQ-D (Yeomans et al. 2008, Leitch et al. 2013), the collation of the subsequent data did not find a significant correlation ($R = -.04$, non-sig. CI $(-.10, .03)$). This finding also contrasts a large number of studies demonstrating the relationship between discounting and weight status, potentially characterised by disinhibited eating (Weller et al. 2010). However, as discussed mixed findings between these variables are not uncommon, and Fernandez' (2013) study also fails to uncover an association between discounting and TFEQ-D. What this may tell us then is the extremely sensitive nature of this relationship. For example, although some studies reveal significant or close to significant associations between variables (Yeomans et al. 2008), the overall relationship in our sample is potentially negligible, or at least extremely small and difficult to detect.

What now becomes of more interest is the potential moderating variables between these relationships – a theme central to this thesis as a whole. I have demonstrated

here that although in the studies of thesis there were not significant differences between high TFEQ-D experimental vs. control groups, if meta-analysed with studies with comparable designs we see a significant over standardized mean difference. Without this methodology, the file drawer conundrum would have been fully active – a disregarding of potentially useful findings due to a lack of statistical significance. However for the first time using a meta-analysis it is shown here that although a small effect, those high in TFEQ-D module their preference for delay vs. immediate gratification following exposure to food cues, hedonically associated cues or preload consumption. In fact the mean difference of all studies included except one (Leitch thesis, experiment 3, which was a satiety vs. control, not a preload study, so the lack of group differences are not particularly surprising) had their mean in the hypothesised direction. In the context of the overarching themes of the thesis, the proposed idea of cue-induced impulsivity, this is an extremely positive finding from the analysis. Therefore, there appears to be at least some support for the tentative hypothesis discussed in the discussion section of Experiment 1 of this thesis: that exposure to the sight/smell or consumption of hedonic food or hedonic food associated cues may activate reward mechanisms (Wise, 2006), which in turn has been linked to differences in subtypes of impulsivity (Wade, de Wit & Richards, 2000; Pine et al. 2010). This analysis for the first time provides at least some evidence that the nature of some subtypes of impulsivity (specifically delay discounting/delay of gratification) may be malleable. This suggests then that correlational analysis of median-split differences between levels of TFEQ-D without moderators may not be useful in understanding the relationships between these variables, and that is demonstrated in the correlational meta-analysis.

The malleable nature of impulsivity, sometimes discussed as ‘state’ vs. ‘trait’, therefore seems to also be very positive in light of studies not looking to induce impulsivity, but to strengthen it. For example, Guerrieri, Nederkoorn & Jansen (2012), Jones et al. (2011), and Houben & Jansen (2010) have attempted to either prime ‘restrained’ concepts, or to ‘train’ inhibitory control through practice trials. Although this is a different subtype of impulsivity, our findings here do suggest that in certain populations (those high in TFEQ-D), impulsivity can be induced. This may therefore then be supportive of findings where the opposite is possible or attempted, impulsivity to be reduced or strengthened through similar mechanisms.

Given the small study sample size available for this meta-analysis, upcoming work would be fruitful in examining other moderators of the relationship between discounting and dietary disinhibition. For example it would be extremely important to understand not just the how the interaction between dietary disinhibition is moderated by what is tentatively discussed here as enhanced reward-reactivity, but also how the relationship may be moderated by other factors influencing motivation. For instance, the work of Epstein et al (2003) explores hunger state as a motivational modulator, particularly with regards to the reinforcing value of food (RRV; see experiment 2 for history of this concept). In particular, Epstein explores comparisons of RRV with regard to states of satiety vs. acute food deprivation, and attempt to formulate a model of 'reinforcement pathology', linking RRV and top-down executive systems. Although this thesis (Experiment 2) found no association between discounting and TFEQ-D (limitations with regards to the methodology are discussed in the experiment 2 chapter), future studies may be fruitful in exploring different motivational and physiological hunger states to examine these as further moderators of discounting and disinhibition.

A point to note here is that all of the studies' samples were normal weight female individuals, and as with a great deal of other psychological investigations, conducted primarily on young undergraduate students. As can be seen from figure 6.2, the effects discussed here are significant in a meta-analysis but as stand-alone experiments are relatively small and difficult to detect. Therefore, future work should be designed to detect these differences as effectively and clearly as possible. This may be done using obese vs. normal weight individuals (as shown in discounting work by Weller et al, 2010), or as previously mentioned by manipulating physiological hunger state. It would also be possible to spend a large amount of time experimenting with discounting indifference timepoints. For example, could there be any differences in discounting rates between TFEQ-D groups in using discounting timepoints of greater range, for example may discounting differences be more expressed when participants consider monetary preference at longer times in the future? It is logical to consider this a useful manipulation to attempt to exacerbate the differences between groups of interest as much as possible.

6.3.1 Limitations

Although this meta-analysis provides useful insight into the interaction between experimental manipulation and dietary disinhibition on discounting data, it does have a number of limitations. Firstly relatively speaking as a meta-analysis it is somewhat limited due to its small study sample size. This is not something that can be directly addressed as only studies included have examined the variables and relationships required for inclusion. However, although insightful, in order to examine these effects further, a broader collection of studies from other laboratories and populations would be desirable. Secondly, in this analysis, I have equated the same effects to preload consumption, food-cue exposure, and food-associated cue exposure. Although if the mechanism behind the subsequent effects is attributable to a reward sensitivity enhancement effect (e.g. that reward sensitivity may be enhanced by consumption/exposure thus leading to increased impulsivity), it may be logical to equate these designs together. However as of yet, there is insufficient work to do anymore than hypothesise this as the possible mechanism. Therefore for the sake of logic and existing knowledge, these designs have been clustered together, further analyses with greater within-design experimental group type to unpick differences in cue exposure vs. preload consumption etc.

Summary

As can be seen from this meta-analysis, although a small number of studies are included, from what is a relatively small number of available studies investigating the concepts of interest to this study, there appears to be a significant difference in discounting between those high in TFEQ-D who have been exposed to food cues or who have consumed a milkshake in discounting than those high in TFEQ-D who have not been exposed to food cues or have consumed a milkshake. This was only conducted for those high in TFEQ-D however. This for the first time demonstrates a possible ‘reward-reactivity’ mechanism in those who have an inability to control their food consumption not as a blanket effect, but under specific circumstances, which

interestingly here is manifested through behavioural delayed discounting measures, not simply measurable ad libitum food consumption.

Chapter 6 - General Discussion

7.0 Introduction

Traditional models of overeating behaviours have typically focused on ad libitum food intake following an experimental preload. For example, Herman & Mack (1975) noted counter-intuitively that restrained eaters consumed more ad libitum ice cream after a milkshake preload than those restrained eaters who had not received a preload. This was typically considered ‘counter regulatory’, and as a result of breaking a self-imposed cognitive dieting boundary by those who restrain or control their own intake. Westenhoefer (1994) explored this idea further, and suggested rather than just those who restrained their eating behaviour, those who display traits characterised as being high in both dietary restraint and dietary disinhibition (high restraint, high disinhibition, HRHD) are most at risk of this overconsumption following the experimental preload – displaying what Westenhoefer discussed as ‘disinhibited eating’. This thesis attempted to conceptualize this counter-regulation for the first time not as a breaking of restraint or as a period of disinhibited eating, but as a general ‘disinhibition’ effect, manifest as eating behaviour, but better captured as a modulation of behavioural impulsivity. We postulated tentatively from this conceptualization that the consumption of hedonic food might lead to heightened reward sensitivity as a result of reward pathway activation (as shown by Wise, 2006), leading to a greater display of impulsivity. Having this in mind, this thesis attempted to examine the extent to which human behavioural impulsivity could be modulated through preload consumption or food cue exposure.

The subsequent discussion will discuss the key findings from the 4 experimental studies and the meta-analysis included in this thesis, before highlighting the

theoretical and methodological implications, and finally discussing the possible future directions for research of this type.

7.1 Summary of Thesis Chapters and Findings

Experiment 1 – ‘The Role of A Milkshake Preload On Subsequent Behavioural Impulsivity.’

Experiment 1 of this thesis aimed to examine the explicit hypothesis for the first time that consumption of a pre-task milkshake preload would lead to enhanced behavioural impulsivity (GoStop task, DDT, and MFFT) compared to a no-consumption control condition. It was also hypothesised that preload consumption would interact with TFEQ-D, with those scoring higher on the TFEQ-D and consuming the preload acting more behaviourally impulsive than other conditions. As predicted, those women who consumed the milkshake preload acted significantly more impulsively on the GoStop inhibitory control task, and the DDT than those in the control condition. However, there was no effect or significant interaction with dietary disinhibition, which was unexpected given the relationships previously highlighted implicating TFEQ-D in impulsivity (Leitch et al. 2013) and interactive with food cue exposure (Yeomans & Brace, 2015). Interestingly, when the data were explored further examining the median split of both TFEQ-D and TFEQ-R groups, a trending but non-significant contrast was revealed. Those high in disinhibition but low in restraint (HDLR), those characterised by Westenhoefer (1994) as having the greatest propensity to gain weight, were more impulsive on the DDT and GoStop following the preload in comparison to their TFEQ counterparts who had not consumed an experiment preload. This suggested for the first time that there may be an interactive link between dietary attitudes and preload exposure on inhibitory control and impulsive choice preferences. However, the grouping design employed in this experiment consisted of a milkshake preload vs. no consumption. From this design, it was impossible to determine the pertinent characteristics of the

milkshake condition that elicited this cued-impulsivity effect. For instance, it was not possible to suggest that the hedonic characteristics of the preload was the key in stimulating cued-impulsivity, or whether the perception of the energy content drove this effect (as demonstrated in an ad libitum eating experiment following manipulated beliefs of energy content by Mills & Palandra, 2008).

Experiment 2 – ‘The reinforcing value of palatable snack foods and its relationship to subtypes of behavioural and self-report impulsivity’ (Accepted for publication to ‘Eating Behaviours’, Jan, 2016).

The second experiment of this thesis attempted to examine the relationship between the reinforcing value of food (RRV) and impulsivity. Previous work (Carr et al. 2012) has proposed a model of weight gain or overeating using both RRV and delayed discounting, demonstrating an association between the concepts. This experiment was designed to examine any predictive relationships between TFEQ variables, impulsivity measures, and a self-report measure of impulsivity (BIS/BAS) and RRV.

The results of this study demonstrated although no impulsivity measures predicted intake ad libitum or RRV, ad libitum snack food intake itself was predicted by RRV. Some caution is needed in interpreting the lack of association between RRV and measures of impulsivity, since the RRV task used here was shorter than in other studies adopting similar methodologies (Epstein et al. 2008), which may have limited the ability to detect relationships with impulsivity variables. It is proposed in Chapter 3 that a minimum play timer should be installed in tasks of this type that use adult participants to ensure that there is a reduction in the possible floor effect displayed here. Nonetheless, the finding of intake predicted by RRV is a theoretically interesting development, and particularly useful in understanding how the laboratory task (RRV) may translate to eating behaviour.

Experiment 3: 'Exploring differences in the perceived hedonic value of a milkshake preload, the anticipation of a milkshake preload, and the relationship with subsequent behavioural impulsivity.'

The third experimental study in this thesis attempted to replicate our findings from Experiment 1, that preload consumption would lead to heightened behavioural impulsivity (specifically DDT and GoStop), whilst further exploring what the pertinent characteristics of the preload may be to elicit this effect. It was highlighted prior to the execution of this study that the characteristics which have the potential to elicit a modulation of behaviour and possibly impulsivity, were the hedonic value (as shown to elicit craving, and neural reward system activation, Kringlebach, 2004) and the participants' perception of the healthiness of the preload (shown by Mills & Palandra, 2008, to modify subsequent eating behaviour). In order to achieve this, a piloted preload was formulated which contained garam masala (see Experiment 3 for formulation procedure of preloads) which was rated as significantly less liked, but did not differ significantly on perceived healthiness from the original preload in Experiment 1. This piloted preload also did not differ on actual energy or perceived content which allowed the examination of the role of hedonic value whilst controlling for the confounds of perceived energy content and perceived healthiness. In order to control for the potential confound of the expectation or anticipation of a hedonic preload, an anticipation condition was also included in which the participant was told that they would receive a chocolate milkshake but never did. Participants then completed the battery of impulsivity tasks.

The results of this experiment showed a failure to replicate the findings of Experiment 1: preload consumption did not produce heightened impulsivity, and there was no significant difference between any of the conditions on any of the impulsivity measures. There were also no relationships between condition and TFEQ-variables. Unfortunately, although the piloted preloads demonstrated the differences required (no difference in healthiness perception, but difference in hedonic value), this was not replicated in the main experiment (preloads were rated as significantly different on healthiness), which may have also contributed

to the lack of difference between experimental conditions, and it may be that participants during the pilot were evaluating the preload samples relative to one another, not as a standalone sample, which may account for the significant difference on healthiness. This again is explored later in the implications section of this chapter.

In terms of the lack of difference between the conditions, I propose that the lack of a post-preload hunger measure in this thesis (and in fact in all preload experiments through the appetite literature historically) may be why there is such inconsistency, due to potential individual differences in satiety vs. 'disinhibition' between studies' samples. This theory and the theoretical implications and methodological shortcomings of this experiment are explored in the forthcoming latter sections in this chapter.

Experiment 4: 'The role of a reward-conditioned stimulus on subsequent behavioural impulsivity'

In our earlier work (Yeomans & Brace, 2015), exposure to pictures of food increased impulsive responding, particularly in women scoring higher on TFEQD. In Experiment 1, consumption of a chocolate milkshake preload had similar effects, and while Experiment 3 failed to replicate Experiment 1, there was some hint again of an effect in women scoring higher on TFEQD.

Interpretation of the effects of exposure to pictures of food, and consumption of a liked food widely seen as unhealthy, is complicated since in both instances it is impossible to determine what aspect of the food stimulus drove the increased impulsivity. One way around that is to use visual cues that have been specifically associated with one rewarding aspect of eating. Experiment 4 attempted this using hedonic value, here defined as liked sweet taste, as the reward. The study then pre-associated novel visual cues with experience of the sweet taste and then examined how this cue modified impulsive responding.

In order to complete these objectives, a between groups design was employed, with each group being tasked with conditioning a previously neutral cue with either a

hedonic taste (CS+, glucose), a neutral taste (CS-, artificial saliva), with no taste (a novel symbol) or no symbol, which was presented to them prior to and during a battery of impulsivity tasks. So to reiterate, in this study depending on experimental group, participants were shown either a cue associated with reward (CS+, sweet taste), a cue associated with a neutral taste (CS-, artificial saliva), a cue that they have not seen before (to control for visual distraction alone), and no cue before and during the trials of a DDT and a Stop-signal task. It was hoped also that this experiment could provide an insight into a potential ‘branding’ mechanism, and would help to build on the understanding of some of the mechanisms discussed in Yeomans & Brace (2015, revised) published outside of this thesis.

The results of this study failed to find significant differences in impulsivity scores on any of the measures between experimental groups. There were no significant effects of TFEQ-D or TFEQ-R on impulsivity, or any condition/TFEQ interaction. This is proposed to have been potentially due to a lack of power (expanded on further in the limitations sections, below).

Experiment 5: ‘A Meta-Analysis examining Dietary Disinhibition (TFEQ-D) and Delayed Discounting (DDT) and the moderating role of cue exposure or preload consumption’

One of the overriding outcomes of this thesis was how results for delayed discounting seems to often be in the predicted direction, that is more impulsive after exposure to food or food-related cues, but rarely reached significance which might imply that the effect size for this relationship was smaller than predicted, and consequently studies lacked the power to pick up these smaller effects. This to some extent seems also seems to be the case with the interaction between experimental conditions (preloads, cue exposures) and TFEQ-D (Figure 6.3). Given the work previously from our laboratory (Yeomans et al. 2008, Leitch et al. 2013) which seems to find a clear association between TFEQ and discounting, the final empirical chapter used meta-analysis to explore further the relationship between DDT and TFEQ-D, and subsequently how this relationship might be moderated by experimental conditions. The meta-analysis suggested that although no general correlation was found between TFEQ-D and DDT measures, there was a significant mean difference between those

high in TFEQ-D in the experimental groups and those high in TFEQ-D in the control groups. This may then demonstrate a state-dependent sensitivity to reward or modulation of gratification preferences in those high in TFEQ-D following preload consumption or hedonic cue exposure.

7.2.1 Theoretical Contributions

- Lack of consistency in cue or preload conditions in impulsivity

The key theoretical contribution of this thesis arguably arises from the analysis of the effects of food-cue exposure or preload consumption on subsequent impulsivity. Importantly, the Meta-Analysis (Chapter 5) in this thesis demonstrated a modest increase in impulsive choice (measured using the DDT) after exposure to food-related stimuli relative to controls when analysed across a variety of cue-exposure conditions. This type of impulsivity modulation draws particular parallels to work in the drug and alcohol literature. For example, a number of experiments have attempted to demonstrate the modulation of sub-types of behavioral impulsivity following cue exposure. Kambouropoulos & Staiger (2001) demonstrated in problem drinker participants that following an alcohol cue exposure participants were more sensitive to monetary incentive tasks (using the Card Rearranging Reward Responsivity Objective Test, CARROT, as opposed to a DDT). Van Gaalen et al (2006) drew parallels to this study using cocaine exposure in rodent subjects – demonstrating premature responding (discussed by these authors as ‘behavioural disinhibition’) following acute cocaine exposure. Vezina (2004) showed earlier that acute cocaine exposure did not simply demonstrate modulated behavioural impulsivity, but also increased risky behaviours following drug exposure, including increased subsequent drug taking and seeking. This type of modulatory effect also seems to support some of the findings found in the literature on priming as discussed at length during the introduction of this thesis (e.g. de Wit, 1996, Cornell et al, 1998, 2002), that reward cues (drug and alcohol cues in the case of the priming studies cited) has the potential in some instances to modulate impulsivity.

Although this drug and alcohol literature draws a useful parallel, it has to be kept in mind that these effects seem to be general, whereas the meta-analysis presented here from a somewhat limited sample of studies does seem to be modulated specifically by high TFEQ-D, rather than the unreplicated general effects of milkshake preload consumption shown in experiment 1 of this thesis. It has to be noted that the modulation of impulsivity may have been demonstrated in our meta-analysis and in experiment 1, individual studies 3 and 4 failed to produce significant differences between experimental and control condition in impulsivity.

A second theoretically relevant issue relates to individual differences in impulsivity, and especially the idea that a tendency to show uncontrolled eating (indexed by the TFEQD) is related to impulsivity. We failed to find significant differences between experimental and control conditions and between those in high and low TFEQ-D throughout our laboratory's data, and further only experiment 1 in this thesis significantly dissociated experimental and control conditions in subsequent impulsivity. Experiment 3 and experiment 4, although tending towards the hypothesised direction of DDT and GoStop, failed to reach significance between conditions. This is one of the most surprising outcomes of this thesis - the general lack of association between TFEQ restraint and disinhibition and subtypes of impulsivity. Previous work from our laboratory (Yeomans, Leitch & Mobini, 2008; Leitch, Morgan & Yeomans, 2013) suggested that disinhibition may be related to discounting data, but in the latter work failed to find this association, instead producing an association with reflection impulsivity. Interestingly the latter study, despite the hypothesis that dietary restraint would result in subsequently poor inhibition, participants showed greater ability to control their inhibitions if they were high in restraint. From these two studies alone, there is beginning to be a somewhat unclear picture developing of the role of TFEQ in impulsivity. There is not currently a large body of research published exploring the direct role of TFEQ variables on impulsivity, and much data has focused more on the relationship between restraint and disinhibition on ad libitum laboratory food consumption (Chambers & Yeomans, 2011) or weight status (Burton, Smith & Hightowler, 2007), which is sometimes tentatively linked to impulsivity (Nederkoorn et al, 2005). However, even the link between TFEQ and these alternative behavioural and biometric outcomes have been inconsistent. For example, in the introductory chapter of this thesis, I explored at

length the relationship between TFEQ and eating behaviour, and noted several studies that failed to find any significant relationship between these variables (Broadbent et al, 2014, Lowe et al, 2013). So taken together, what does this tell us about the nature of the relationship between TFEQ and impulsivity? In terms of this thesis, it is fair to suggest that these effects are small, and difficult to detect. The meta-analysis conducted in this thesis (Chapter 6) supports this idea, particularly in the case of disinhibition. However, other research has been fruitful in using TFEQ variables as a moderator between either eating behaviours (ad libitum intake etc) or impulsivity (e.g. Loeber et al. 2013 in non-hungry participants). The meta-analysis presented as the final experimental chapter in this thesis provides the most striking evidence for a moderating role of TFEQ variables, specifically disinhibition. This maybe then suggests that disinhibition as a moderator between cue exposure and impulsivity is, although sensitive, a statistically small but useful way of understanding TFEQ's relationship to impulsivity.

A third key theoretical issue arising from the designs employed in experiments 1 and 3 is that of post-preload satiety. It was posited that in experiment 1 and experiment 3 of this thesis (and indeed in the preload literature as a whole), there has since been no post-preload hunger assessment prior to the impulsivity measures, meaning that we unable to control for hunger. Therefore, it is possible that there is potential for individual differences in hunger or more specifically satiety (as opposed to 'disinhibition'), which may account for a number of differences (e.g. perception of the preload as a drink vs. a snack, McKrickerd et al, (2014) which would have been unforeseen. Past research has shown that satiety and hunger do separately influence reward reactivity (Epstein et al 2003), or food and eating related behaviours (Nederkoorn et al. 2009), which would potentially dampen the effects of the preload on subsequent impulsivity, which might be attributable for the failure of the data presented here to reach significance. In experiment 4 however, the issue of hunger was controlled more strictly, using cue exposure as opposed to preload consumption, but again no significant difference between groups was found. This was particularly surprising given the work of Yeomans & Brace (2015) and Ridley-Siegert, Crombag and Yeomans (2015) who demonstrated heightened impulsivity following cue exposure, and increased food consumption following the presentation of hedonically

associated cues respectively. The crucial difference in experiment 4 is that at no point in the impulsivity tasks was there any actual hedonically present reward. For example in Yeomans & Brace (2015), hedonic cues were available, and Ridley-Siegert et al (2015) the associated cue was further reinforced by the availability of ad libitum snack food consumption. However, in the experiment 4 of this thesis, following the conditioning procedure, there was no further reinforcement from the cues, which may not have been sufficient to produce any cue-induced impulsivity. This may then tentatively suggest that hedonically associated cues may enhance hedonic cue effects in combination, but without further reinforcing factors may not produce subsequent statistically detectable effects.

7.2.2 Methodological Implications

- DDT and anchoring reward magnitude?

Another factor, which was of particular interest, may have been the task-dependent differences in the DDT's used between experiment 1 and experiment 3. For example, the DDT task used in Experiment 1 utilized a wide range of delays (5 years, Baumann & Odum, 2010), contrasted with 1 year in Experiment 3.. I suggested in the meta-analysis of this thesis that this may have elicited an anchoring effect with some individuals, for those that may have received questions regarding the longest delay early in the randomized order may anchor their other responses relative to the amount selected for the longest delay (Tversky & Kahneman, 1974. On the other hand, this may suggest that delay magnitude may be a vital component in dissociating control and experimental groups, and large timescale magnitude preferences may be required to detect any differences.

Experiment 2 – the reinforcing value of leaving the experiment?

As briefly discussed in the discussion of Experiment 2 of this thesis, one of the methodological implications of this experiment lies with the low rate of participant responding on the RRV task relative to other experiments using this

methodology. I propose the reason for this rate of responding may have been due not to the low reinforcing value of snack foods used to participants, possibly that what may actually be being measured is the reinforcing value of leaving the experiment. For example, participants were told that when they wanted to stop playing the RRV task, they were free to leave the experiment. This therefore may have been measuring how reinforcing leaving the experiment and getting on with their day is relative to how reinforcing the snack foods were. Although there was some evidence that the RRV task reflects snacking behaviour to some degree (shown through ad libitum) snack intake, it is also possible that the low rate of responding may have been problematic in detecting the relationship between RRV and impulsivity variables rather than just RRV and snack intake. I would recommend that future experiments using the RRV methodology should implement a minimum play time during the RRV task, which would allow a fuller assessment of the relationship between relative reinforcing value of food (vs. a non-food alternative) and variables of interest.

7.2.3 Limitations

Although this thesis has revealed some novel methodological and theoretical insights into impulsivity and eating behaviour, there are a number of limitations to the studies conducted. The first limitation is one, which could be levied at all counter-regulation studies that have adopted preload designs – the nature of controlling post-preload satiety. I discussed at length in the discussion of experiment 3 of this thesis that there is no way of assessing the extent to which there may be individual differences in satiety following a preload may impact impulsivity (there is evidence linking satiety to a dampening of reward mechanisms, or a reduction in reward motivation, James et al. 2004) as there was no inclusion of a post-preload hunger measure. I would suggest that this type of design in future employ a post-preload measure of satiety (in the guise of a mood questionnaire), which although a simple addition to the design, would allow for the examination of preload effects whilst controlling wherever possible for the role of satiety.

One limitation that has not to this point been discussed or explored within this thesis surrounds the reliability of Stunkard & Messick's (1985) Three-Factor Eating Questionnaire. For example, within this thesis, there was no control over the timespan from which the participant completes the TFEQ to the experimental session, nor was there any assessment of the internal validity of the factor structure of the measure itself. For example, the reliability of Stunkard & Messick's (1985) original factor structure comprising of three core factors (disinhibition, restraint & hunger) has come under experimental scrutiny. Karlsson (2000) in a very large sample of obese Swedish participants, failed to replicate the original reliability of the Disinhibition subscale, explaining their findings as demonstrating that disinhibition has 'weak' reliability, and there was a significant correlation with the hunger subscale. Neale, Mazzeo & Bulik (2002) compounded some of the criticism of the disinhibition subscale, demonstrating that disinhibition significantly covaried with the hunger subscale of the TFEQ. It is not well understood why there appears to be some overlap in experimental validations of the TFEQ in the hunger and disinhibition subscale, but it is possible that this difference may be due to the studies examining the factor structure being conducted on obese populations. For example, Ogden & Wardle (1990) discuss the evidence that obese individuals may be insensitive to hunger or internal satiety cues. If this is the case, there is potential that a validation study would be beneficial in normal-weight individuals. Karlsson speculates that the relationship between TFEQ-D and hunger might represent 'episodic, compulsive overeating', a type of behaviour less likely to occur in normal weight individuals. In terms of the factor structure, it has to be acknowledged in hindsight that there is potential that specifically the disinhibition subscale that was a core factor in this thesis might be unreliable, and a reliability and validity study would be beneficial in a normal-weight sample.

Another limitation of the use of the TFEQ in this thesis may also lie in the long-term reliability of the measure itself. For example, participants who took part in the experimental studies of this thesis (and from our laboratory) complete the TFEQ some time prior to completing their experimental session. This can possibly range up to 1 year prior to their participation, as participants complete

the TFEQ upon registering their interest to taking part in our laboratory studies, and are recruited at a later date when required if eligible. It was felt important for the participant not to complete the TFEQ just prior to or during the experimental session in order not to prime any disinhibited thoughts, or to expose participants to hedonic imagery (e.g. wording such as ‘sizzling steak’, TFEQ-Disinhibition subscale), which may confound subsequent performance. However, it has to be acknowledged that this may confound the long-term reliability of the TFEQ, and future studies might be useful in optimizing the timespan between completing the TFEQ and the experimental session, without any possible priming confounds, whilst maintaining the test-retest reliability of the TFEQ. In defence of the long-term reliability of the TFEQ, Bond, McDowell & Wilkinson (2001) demonstrated an ‘impressively large’ 12-month test-retest reliability of the TFEQ. Nonetheless, long-term reliability is an issue that would benefit from further examination.

In terms of the general use of the TFEQ in our laboratory, and specifically in our thesis, there were no alternatives considered. For example, TFEQ is the primary eating attitudes measure that is used across the body of appetite literature, with great support for its relationship to eating behaviour of interest. It is this body of work and historical use of the measure which allows for the promotion of continued use of the measure in experimental research. Nonetheless, it would be advisable to accompany the TFEQ with other measures of either disordered eating (e.g. the Dutch Eating Behaviour Questionnaire, DEBQ, van Strien et al, 1986), or to attempt to examine further the factor structure and reliability of the measure itself.

Sample populations throughout the experimental chapters

A second limitation to the studies conducted in this thesis lies in the recruitment of and sample of participants used. Although participants were screened to meet the standard appetite exclusion criteria (medication, smoking status etc), there was no selective recruitment with regards to TFEQ distributions. As discussed, if examining the interaction between experimental conditions and both TFEQ-D

and TFEQ-R, there is potential for a difficulty detecting sensitive effects with group sizes becoming smaller due to the segmentation of TFEQ-D and TFEQ-R high and low for each condition. I would therefore suggest that future studies may negate this limitation by recruiting extreme scorers in TFEQ-D and TFEQ-R in order to maximize the ability to detect any effects of TFEQ variables. In terms of any other recruitment limitations, only females were recruited, and recent research, Greenwood et al (2014) demonstrated interestingly that the interaction between restraint and impulsivity on overeating behaviour was only significant in the male participants of their sample. Although I am aware that participant recruitment time and experimental finances are not infinite, it would be interesting to include a male selection of the experimental sample to negate and examine any gender differences, or more specifically to examine any gender/impulsivity interactions. Finally in terms of the sample population studies throughout this thesis, the sample suffers from being relatively homogenous. That is to say that participants were all female, normal BMI, broadly Caucasian undergraduate students at the University of Sussex, mostly within the ages of 18-23. As discussed in the first paragraph of the 'future experiments' section below, a more heterogeneous sample (particularly with regard to BMI or weight status) might have allowed for the detection of more sensitive effects, and in turn may have allowed for the examination of more extreme TFEQ scorers, particularly with regard to a normal vs. overweight participant interaction with TFEQ variables on impulsivity measures. I would recommend that future work may utilise community-sampling methods (as opposed to purely university sampling) in order to achieve a broader representation of ages across participants, and may provide a broader distribution of subjects to examine the theoretical framework in question.

Power

Given the inability to replicate the results of experiment 1 or the work of Yeomans & Brace (2015) successfully, in hindsight it is clear that experiments 3 and 4 of this thesis suffer from a lack of power. For example, the findings in experiment 1 may not be representative of the true effect (e.g. a type 1 error), or

may have produced larger effects than would be expected if replication attempts of the same sample size were conducted. However, given the effect size that was reported, experiments 3 and 4's sample sizes were calculated (and based on study designs conducted similarly in our laboratory, Ridley-Siebert et al, 2015), but more conditions were added without an increase in the overall sample size of the study. This in hindsight meant that once comparisons were made between experimental condition, TFEQ-D and TFEQ-R (4x2x2), individual cell sizes were underpowered and not equipped with the participant numbers to enable detection of any effects if they were present. It would have been advisable in this situation not to rely on the results of an original, unreplicated study as a sample size estimator, and it would have possibly been more effective to have conducted experiment 3 and experiment 4 with cell sizes at least the same as experiment 1, which would have meant at least including 50 participants for each additional two experimental conditions (an additional 200 participants across experiment 3 and experiment 4). In terms of the practicalities of this, this would not have been possible in terms of both time and research resources, but an ideal scenario would have ensured at least matched cell sizes with experiment 1, whether the effect size demonstrated in that study was unrepresentative of the true effect size or otherwise.

The lack of statistical power exhibited in experiments 3 and 4 would have made it more difficult to detect any of the potentially sensitive effects of condition and TFEQ-variables on impulsivity outcomes. Future work would take a broader view of experimental research conducted in order to base sample size and power calculations by ensuring that cell size differences are negated between studies (if practically and economically possible), rather than relying on experiments that are yet to be face replication attempts to base experiment sample sizes.

An alternative paradigm

Throughout this thesis, the paradigm of cue-exposure leading to greater impulsivity was explored, and to some extent supported in experiment 1. The meta-analysis presented in this thesis and also previous work from our

laboratory (Yeomans & Brace, 2015) demonstrated to some degree the modulation of discounting hypothetical monetary reward using cue exposure in individuals high in TFEQ-Disinhibition. However, in subsequent chapters of this thesis (experiments 3 and 4), there was no evidence found for the modulated nature of behavioural impulsivity. It is posited here that the theoretical model has been adequately tested using the appropriate design and measures used, with steps taken throughout each study to address methodological shortcomings of the previous experiment in order to show visible progress in the examination of the experimental and theoretical framework, but without being able to replicate experiment 1 or previous work from our laboratory.

I feel that this thesis demonstrated progression in the methodology necessary to examine the original posited theoretical framework, building on experiment 1's preload vs. no preload design to experiment 3, where a stringently piloted additional experimental preload was conceived, enabling the examination of which characteristics might be pertinent in driving the modulation of impulsivity (hedonic value, perceived calories, anticipation of reward). From here, Experiment 4 removed any possible confounds of satiety, which was suggested as an unmeasured bias in experiment 3, by using an established conditioning procedure. It is felt that although the experiments presented in this thesis failed to produce significant findings, with the exception of experiment 1, the theoretical model was examined with sufficiently constructed experimental methods showing progression, albeit in hindsight with a lack of statistical power.

This inability to replicate may well have been due to a lack of power (discussed above), but another explanation should also be considered, not the theoretical model originally posited, but an alternative; one in which impulsivity is just one risk or contributing factor to overeating, not necessarily a moderated behavioural system which leads to overeating. For example, research has suggested a wealth of contributing factors which may increase risk of overeating or weight gain, such as genetic factors (Lyon & Hirschhorn, 2006, Felsted et al, 2010), eating attitudes (Stunkard & Messick, 1985), or facets of impulsivity. It may be that impulsivity is just one contributing factor, which makes up the

overall picture of risk factors that leads to overeating, rather than impulsivity itself being modulated.

This may explain why there has been a great deal of mixed findings not only in this thesis, but also generally across the appetite literature with impulsivity and eating behaviour. It may be more useful in future research to examine not the relationship between cue exposure the modulation of impulsivity, but rather the relationship between impulsivity and eating behaviour in combination with other risk factors (e.g. external eating, Kakoschke et al, 2015) to understand the variance accounted for respectively in eating behaviour. The original theoretical model posited in the introduction chapter posited that there may be a pathway between cue exposure and modulated behavioural impulsivity, which might be moderated by TFEQ-disinhibition. However the experimental evidence presented in this thesis fails to support that idea, and we must accept that the role of impulsivity is most likely one that is a risk factor in overeating as presented in other frameworks (e.g. Mela, 2006), and possibly necessarily a pathway between cue/prime/preload exposure and overeating behaviour.

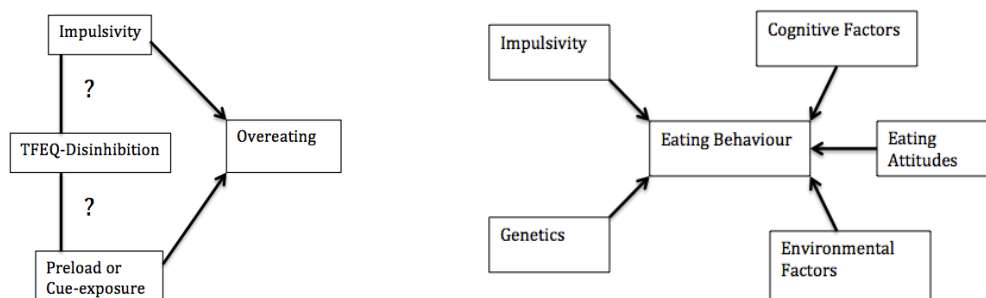


Figure 7: a) original model examined in this thesis, b) Revised model, proposing impulsivity as just one of many factors Influencing eating behaviour.

7.3 Future Experiments

As discussed, there has been some inconsistency in the findings from this thesis, in relation to the degree to which cue exposure induced significant changes in measures of behavioural impulsivity, the relationship between TFEQ scores and impulsivity tasks, and the relationship between RRV and impulsivity. Therefore future experiments may look to accentuate the differences between experimental groups and TFEQ interaction groups. A first proposed experiment would use the same methodology as Experiment 3 in this thesis, implementing 4 experimental preload conditions: a hedonic preload, a non-hedonic preload, a no consumption anticipation condition, and a no preload or anticipation condition. I suggest here that although the experiment 3 preload pilot provided the appropriate preloads (a non-hedonic rated as less hedonic than the hedonic preload, but not perceived as less healthy), when checked in the experiment itself, this was not replicated, therefore a pilot should be conducted between preloads, in order to prevent participants from making their evaluations of the preloads relative to others that they have previously evaluated. Following the successful pilot, I would suggest to be selective with recruitment regarding TFEQ scores, and to ensure that TFEQ questionnaires are completed in a short space of time from the experimental session in order to control for the possibly unstable nature of TFEQ across time. For example, in order to examine the interactive role of TFEQ variables, participants should be recruited that meet the top and bottom 33% of each of the restraint and disinhibition groups, which would allow for a greater sensitivity to detect these effects. I also suggest here that a post-preload measure of hunger should also be taken to statistically control for the role of satiety's dampening effects on reward activation. As also noted in Experiment 3, the anticipation condition was not statistically distinguishable in impulsivity outcomes to the experimental or control condition. I proposed in that chapter that we couldn't be sure what exactly the participants were expecting, as at no point were they exposed directly to the preload. I would suggest that in this future study, the sight and olfactory cues of the milkshake preload should be presented in the anticipation condition to control to some extent what reward those participants are anticipating. From these controls, and this extended piloting and recruitment strategy, it should maximize the chances that a detectable effect will be demonstrated, aiding our understanding of the mechanisms involved in reward consumption, exposure, and impulsivity. Given the only

significant effects shown in Experiment 1 and in the work of this thesis, I would recommend a sample size of at least 50 per experimental condition if looking to examine also the interaction of TFEQ-D and condition, however should the further interaction of TFEQ-R be included, it is likely that sample size requirements would be much greater due to the sensitive nature of these effects.

In terms of the discounting paradigm that was implemented throughout this thesis, I feel that there is great scope for future experimentation. For example, I would be particularly keen to explore the possibility of an anchoring effect of longer-term delay points in the broader DDT literature: there still is no accepted single method for measuring or indeed analyzing DDT data. This would be relatively easy to conduct with 2 experimental groups. One group would be presented with a long term delay point (5 years as in Baumann & Odum, 2010) at the beginning of the randomized rotation of delay points, whereas the other group receives their monetary preference questions regarding the longest delay point as the last of their randomized delay points. Following the examination of the differences of each groups' area-under-the-curve, some useful insights into the role of the anchoring effect of long-term delay points would be achieved. This could be taken even further with the implementation of a preload vs. no preload condition (now a 2x2 design), which would allow us to examine the extent to which preload consumption (and possibly enhanced reward sensitivity) may lead to greater anchoring monetary preferences relatively to the longest term delay period. This would be theoretically driven by the idea that more impulsive discounting would be as a result of immediate gratification needs, whereas this experiment would allow us to determine or to gather information on the parameters involved in immediate vs. delayed gratification, and how these figures could be anchored relative to a long term delay point. This would allow us to examine the role that the choice of delay points may play in modulating choices, and although typically delay points are randomized, it might be possibly that an incongruently long delay point might have the potential to create systematically relative responses between differences in at what point they are presented between participants. It would also be fruitful in future experiments to examine the extent to which either the participants trust the experimenter, or believe that a long-term reward would actually be received relative to the probability of it being received in a shorter time period (e.g. £50 in 2 years, as opposed to £50 in 7 days). This would control for

any possible confound of the participants' temporal discounting preferences being biased by the belief of the probability of receiving the reward, as opposed to their actual abilities or preference to delay gratification.

Summary:

Although this thesis demonstrated a modulation of behavioural impulsivity through a milkshake preload consumption, further experiments attempts to replicate this finding and explore what pertinent characteristics of the preload were, and how impulsivity was modulated through the presentation of reward-conditioned cues were not successful. However, with the inclusion of these studies and studies using similar designs conducted previously in our laboratory, a meta-analysis demonstrated a consistent modulation of delayed discounting between those high in TFEQ-D who have consumed a preload or were exposed to reward related cues, and those high in TFEQ-D who did not. This thesis goes on to present novel methodological and theoretical insights, which lay the foundations for attempting to understand the modulation of behavioural impulsivity through preload consumption and rewarding cue exposure.

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Appendices:

Experiment 2 Regression Tables:

Table A.1 Standardized regression coefficients (β), R^2 , and change statistics from hierarchical regression model predicting total snack intake (kcal).

	<i>Step 1</i>	<i>Step 2</i>	<i>Step 3</i>
TFEQ-D	.045	.028	.039
TFEQ-R	.095	.098	.113
TFEQ-D x TFEQ-R	.027	.043	.050
BMI	-.103	-.057	-.030
DDT		-.153	-.189
GoStop Stop Latency		.235	.175
IST Mean Boxes		.132	.155
BAS			.148
BIS			-.259*
R^2			
F	.291	.702	1.120
ΔR^2	.020	.063	.079
ΔF	.291	.411	.418

* $p < .055$.

Note: BMI, body-mass-index. TFEQ-D/TFEQ-R, Three-Factor Eating Questionnaire Disinhibition/Restraint. DDT, Delay Discounting Task. IST, Information Sampling Task. BIS/BAS, Behavioural Inhibition/Activation Scale.

^aMeasured using total mouse-clicks in the food-reinforcement task.

Table A.2 Standardized regression coefficients (β), R^2 , and change statistics from hierarchical regression model predicting RRV food.

	<i>Step 1</i>	<i>Step 2</i>	<i>Step 3</i>
TFEQ-D	.206	.197	.186
TFEQ-R	.034	.039	.030
TFEQ-D x TFEQ-R	.036	.093	.093
BMI	-.080	-.115	-.106
RRVfood		.280	.241*
DDT		.176	.116
GoStop Stop Latency		-.085	-.130
IST Mean Boxes		.140	.149
BAS			.187
BIS			-.062
R^2	.047	.156	.186
F	.693	1.199	1.143
ΔR^2		.109	.129
ΔF		.506	.505

* $p < .09$.

Note: BMI, body-mass-index. RRV, reinforcing value of food task. TFEQ-D/TFEQ-R, Three-Factor Eating Questionnaire Disinhibition/Restraint. DDT, Delay Discounting Task. IST, Information Sampling Task. BIS/BAS, Behavioural Inhibition/Activation Scale.

^aMeasured using total mouse-clicks in the food-reinforcement task.