Food Addiction, BMI, Psychological Well-being and Impulsivity among People with Diabetes

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Karren-Lee Raymond
Food Addiction, BMI, Psychological Well-being and Impulsivity among People with Diabetes

Karren-Lee Raymond

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University of the Sunshine Coast

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"If I have seen further, it is by standing on the shoulders of giants."

(Isaac Newton, 1675)
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Food Addiction, BMI, Psychological Well-being and Impulsivity among People with Diabetes

Declaration of Originality

The work contained in this thesis has not previously been submitted for a degree or a diploma at any other higher education institute. To the best of my knowledge and belief, this thesis contains no material published or written by another person except where due reference is made.

Name: Karren-Lee Raymond

Signed: ____________________________________________

Date: September, 2017
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The dramatic increase in the availability of processed foods has substantially changed the contemporary food environment. Simultaneously, the nutrient density and quality of these foods have declined, contributing to the exponential increase in the prevalence of obesity and nutrition-related chronic diseases. Consequently, the populace percentages of diabetes have surged in tandem with this rise in obesity rates. Despite prolific research into obesity and type 2 diabetes (t2d), current treatment has been unable to stem the upsurge of this epidemic.

Food addiction (FA) is a contemporary concept challenging traditional methods in the treatment of obesity and related medical complications. Simply directing a person predisposed to FA to lose weight in order to decrease their diabetes symptomology is unlikely to be effective for everyone, which may also explain why current treatment modalities have had limited effect.

The aim of this dissertation, which encompasses two research phases, was to investigate the phenomenon of FA among a t2d cohort. The first phase incorporated two studies to examine how FA, impulsivity, and psychological distress relate to Body Mass Index (BMI) among people with t2d. The second phase incorporated two further studies which aimed to examine FA using the Yale Food Addiction Scale (YFAS; Gearhardt, Corbin & Brownell, 2009) symptom count with reference to the Diagnostic and Statistical Manual of Mental Disorders (DSM) severity level classifications (mild, moderate, severe) and Substance Use Disorders (SUD; American Psychological Association, 2013).
Studies one and two (phase 1) highlighted FA was a stronger predictor of impulsivity, depression, anxiety and stress than BMI. Study three and four (phase 2) verified four severity classifications for diagnosing FA (non FA, mild FA; moderate FA, severe FA) in line with the DSM-5 severity indicators.

Unlike diagnosing SUDs, the YFAS binary approach to diagnosing FA does not adequately capture important variations in FA severity. Investigating and introducing a graduated severity approach to the assessment of FA can aid in identifying potential FA in the early stages (i.e., mild or moderate FA symptomology). Consequently, it can help minimise risk factors associated with FA and hinder the progression to severe FA symptomology, whilst preventing other complications, including diabetes, hypertension, heart disease, and stroke.

In moving forward, the nomenclature of FA represents a limitation in current understanding and application of an addiction model to diagnose and treat FA. To address inconsistencies in the terms used to explain FA, I have proposed ‘FA’ be further researched and operationalised to align with DSM-5 SUDs such as alcohol use disorder (AUD). I have proposed the term ‘processed food use disorder (PFUD)’ be considered for further research in a later edition of the DSM-5, and the term ‘processed food addiction (PFA)’ be the consistent term of choice. Findings of this research have universal significance both in terms of theoretical and therapeutic implications, and applications for initiatives and approaches to combat obesity and potentially serious health problems with importance to t2d.
CHAPTER ONE

1. General Introduction

Worldwide, rates of obesity continue to soar, with the World Health Organisation (WHO) estimating 1.9 billion adults worldwide are overweight, and over 600 million people are obese (WHO, 2016). Indeed, there has been a shift in direction where obesity is now a more serious risk to global health than being underweight (International Diabetes Federation; IDF, 2015). Even more alarming, in 2014, over 40 million children under the age of five years were overweight or obese. The global obesity epidemic’s principal causes stems from an increase in the ingestion of processed foods that are calorie dense, coupled with a decrease in physical activity brought about by sedentary lifestyles, changing modes of transportation, and the way modes of work have changed for many occupations (Lifshitz & Lifshitz, 2014). Lustig (2016) has argued convincingly that the chronic consumption of easily digested carbohydrates leads simultaneously to obesity and diabetes, with obesity presenting first in most individuals. These factors have contributed to the increase in obesity rates and related non-communicable diseases such as cardiovascular diseases, some cancers, and diabetes (IDF, 2015).

There is a plethora of research linking diabetes and obesity (IDF 2015), with diabetes continuing to dominate as a cause of disability and death globally (Global Burden of Diseases, 2015). The global picture of diabetes, particularly type 2 diabetes (t2d) and its many complications, strongly suggests that diabetes is one of the largest population health challenges facing the 21st century (IDF, 2015). The International
Diabetes Federation (IDF) 7th edition of the “Atlas” (IDF, 2015) reported that the number of people with diabetes continues to climb at an alarming rate, and predicting that by 2040, the number of diagnoses will increase to 642 million globally. Australian statistics have shown a similar increase in the rate of diabetes diagnoses to that reported from other first-world world countries, increasing from 2.4% in 1995 to 3.8% in 2007/2008 (Australian Bureau of Statistics, ABS, 2011).

Traditional and recent interventions including simple methods to complicated and overlapping treatments, such as nutritional education and lifestyle modifications, appear to be not working for many (Marks, 2015b). At the individual level, increasing community access to a healthier way of life and food choices, plus engaging in more active lifestyles appear to not be sustainable (WHO, 2016). The food industry and governmental authority has also had a significant role in promoting reduced processed food consumption, by limiting the marketing and produce of processed foods targeting children and adolescents (Nestle, 2013). Nevertheless, an obesogenic environment is becoming more the norm of society today.

Despite extensive research, public health education initiatives, and social awareness campaigns to address the prevalence of obesity and t2d, these overlapping epidemics continue to increase globally with no reprieve forthcoming. Fildes et al., (2015) in a large population-based sample, investigated the likelihood of overweight individuals attaining normal weight between 2004 – 2014. Results indicated that current non-surgical obesity treatment strategies were inadequate when maintaining weight loss. For the majority of overweight patients, the probability of sustaining weight loss was very low. Furthermore, Mann et al., (2007) reviewed the long-term effects of calorie-restricting diets to investigate the efficacy of dieting as an effective treatment for obesity. In their consideration of over ten studies, up to two thirds of individuals who dieted regained more weight then what they had
lost. Mann et al’s research posits and highlights the misleading notion that diets are an effective long-term solution in combating the obesity epidemic, and subsequently associated complications. This highlights the need for additional treatment and management approaches for long-term weight control outcomes in people who are obese.

Unfortunately, societies search for effective obesity strategies, and individuals’ attempts to reduce weight such as dieting have been disturbingly resistant to known treatment approaches, as evidenced by current and ever-increasing obesity rates (Mann et al., 2007; Stevens et al., 2012). This may be due to interventions not addressing underlying causes. Effective interventions for tackling these epidemics may be found outside primary care (Fildes et al., 2015). A potential explanation is the food addiction (FA) concept which has been implicated empirically as having strong associations with Body Mass Index (BMI; Flint et al., 2014).

Although somewhat controversial (Ziauddeen, Farooqi, & Fletcher, 2012), research suggests that the FA concept shares many similarities to other Substance Use Disorders (SUDs) (Ifland et al., 2009; Volkow & Wise, 2005). Studies have demonstrated that the brain responds to food in a similar manner to the way it responds to drugs, as both these substances activate neural reward circuits (Blumenthal, & Gold, 2010; Volkow, Wang, Tomasi, & Baler, 2013; Volkow & Wise, 2005). Repeatedly activating these circuits results in the development of a preference for the particular stimulus and can lead to neurobiological adaptation, eventuating in the behaviour becoming compulsive, further reducing the individual’s control over their food or drug consumption (Potenza, 2014; Volkow & Wise, 2005).

Both human and animal models show drugs and palatable foods appear to initiate the same mesolimbic dopamine reward system in the brain (Avena, Rada, & Hoebel, 2009; Small, Jones-Gotman, & Dagher, 2003). Additionally, deficiencies in dopamine
receptors (D2) as observed among people who are addicted to drugs, have also been found in obese individuals (Johnson & Kenny, 2010; Noble et al., 1994; Wang et al., 2001). However, ongoing scientific research continues to investigate whether these neuronal findings are the cause of drug abuse, or the consequences. In other words, is there a preexisting condition that leads to an overindulgence on processed foods, or would it be possible that the answer lies in the repeated overstimulation of the neuronal system that causes the substance abuse.

The Yale Food Addiction Scale (YFAS; Gearhardt, Corbin, & Brownell, 2009) designed to recognise FA symptoms in line with the DSM-IV substance dependence criteria, is typically used as the standard measure of FA. The YFAS has been translated and released in other languages, including German and French versions (Bégin et al., 2012; Meule, Vögele, & Kübler, 2012). It has also been modified to a short form – mYFAS - and adapted for children (Gearhardt, Roberto, Seamans, Corbin, & Brownell, 2012).

The YFAS (Gearhardt et al., 2009) was the only validated test battery for assessing addictive like eating behaviour at the time of this dissertation. Since the inception of the DSM-5 (APA, 2013), a new version the YFAS 2.0 (Gearhardt, Corbin, & Brownell, 2016) has been developed and validated to maintain consistency with the current diagnostic understanding of addiction, and reflect changes to the substance-related and addictive disorders (SRAD; previously labelled substance use disorders; APA, 2013) section released in the DSM-5 (APA, 2013). In the developmental stages of the YFAS 2.0 a comparison study was undertaken (N = 209). Both interpretations of the YFAS were similarly associated with an increase in BMI, binge eating and continual weight loss and weight gain (Gearhardt et al., 2016). Regarding this dissertation, data collection for Studies 3 and 4 and their submission for publication were completed before the YFAS 2.0 was validated and published. While the YFAS 2.0 (Gearhardt et al., 2016) does achieve a similar graduated
approach to FA that studies 3 and 4 promote and demonstrate, this test battery is still evolving in terms of empirical validation and reliability when compared to the extensive validation of the YFAS. As such the YFAS which is still the most utilised test battery for FA, can be employed to select severity indicator cut-offs that have been arbitrarily chosen. While the YFAS 2.0 is a clear development in advancing the diagnosis of PFA, further research similar to Study 3 and 4 is still absent in the literature.

As this thesis developed, it became apparent that the binary approach utilised by the YFAS (Gearhardt et al., 2009) was limited in its lack of capacity to classify FA based on severity level. The YFAS (Gearhardt et al., 2009) has two scoring options which are provided to discern if a respondent meets a criterion for FA diagnosis or not. However, this blunt diagnosis suggests a one-size-fits-all approach and may not be suitable across different FA severity levels. Introducing and investigating a graduated approach for the diagnosis of FA (i.e., mild, moderate or severe) could help identify specific treatment options depending on the severity level of the diagnosis. This would mean a different diagnosis and treatment plan to address the individual’s specific pathology.

The consumption of highly palatable foods is a contributing factor to the development of obesity and other maladies. While there is a myriad of research, campaigns, laws, customs and literature based on the healthy mind-body principles, to date, they have not been effective in addressing a continued global rise in obesity (Marks, 2015a). Additionally, weight loss surgery, an invasive treatment option, has risen with inconsistent outcomes (Abilés et al., 2010; Kissler, & Settmacher, 2013). Research has shown a strong association, and substantial metabolic similarity, between obesity and diabetes, resulting in the term “diabesity” (Youssef & Mahmoud, 2011, p. 28). This suggests there are potentially causal physiological processes or mechanisms that are common to both phenomena (Youssef & Mahmoud, 2011). As such, obesity is a key risk factor for the development of t2d. Therefore, understanding what
mechanisms may place an individual at risk of becoming obese is important for understanding what mechanism place an individual at risk of developing t2d. Therefore, understanding how FA may relate to obesity in people with t2d is particularly relevant for this population, as obesity is a key contributor to the extent and complications of their t2d disease. Clearly a better understanding of the mechanisms underlying problematic eating behaviors, including FA, in people with t2d is warranted (O’Connell, 2011; Wang et al., 2001). An individual’s free choice and self-will is not negated by the notion of addiction, if there is a significant relationship between FA and t2d, this could provide an awareness as to why a subgroup of individuals are forced to continually battle the tyranny of dieting, and the consequences of obesity (Taylor, Curtis, & Davis, 2010). Furthermore, exploring the possibility of utilising a graduated approach for the diagnosis of FA, may aid in clinical diagnoses and in turn effective treatment plans to better manage both conditions.

The available body of information linking BMI, impulsivity, psychological distress and other addictions continues to grow (Hooper, Doehler, Jankowski, & Tomek, 2012; Murphy, Stojek, & MacKillop, 2014). However, there exists a large gap in the literature investigating FA among people with t2d, BMI, impulsivity and psychological distress. A greater understanding of these relationships could help determine whether behavioural, neurological, and pharmacological interventions that target FA increase the likelihood of losing weight and alleviating diabetes symptoms. Thus, screening for FA symptomology could improve weight management efforts with this population, and in turn potentially lessen the burden of preventable diabetes-related illnesses.

The primary objective of this research was to investigate the phenomenon of FA, or more accurately, processed food addiction (PFA) among diabetes cohorts. The research comprised of two main phases. The first phase incorporated two studies to examine how FA, impulsivity, and psychological distress related to BMI among people
Study 1 aimed to investigate (i) the percentage of people with t2d who meet FA criteria based on the YFAS (Gearhardt et al., 2009), (ii) whether BMI differed significantly between individuals with FA and individuals without FA as classified by the YFAS, and (iii) how FA, impulsivity, and psychological distress (depression, anxiety and stress), related to BMI in people with t2d.

Study 2 aimed to assess, (i) the associations between FA and psychological distress among people with t2d, with the primary aim of determining whether depression, anxiety, and stress scores differed significantly between participants who met a FA criterion and those who did not, (ii) compare the cross-sectional predictive contributions of FA and BMI to depression, anxiety, and stress scores, while controlling for covariation between depression, anxiety, and stress.

The second phase incorporated two further studies, which aimed to examine FA using the YFAS symptom count with reference to the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) severity level classifications (mild, moderate, severe) and Substance Use Disorder (SUD; American Psychological Association, 2013). Specifically, Study 3 evaluated a graduated severity level approach to FA classification against BMI (kg/m²) classifications (World Health Organisations; WHO 2013). I examined BMI against the YFAS symptom count with reference to current DSM-5 based diagnostic specifiers for SUDs (APA, 2013) to classify individuals’ FA symptomology severity. Specifically, I investigated which YFAS symptomologies should be grouped together to form four graduated FA classification groups in regard to BMI. I hypothesised BMI would significantly increase from non-FA, to mild- FA, to moderate- FA, to severe-FA participant groups.

Study 4 extended Study 3 by looking at psychological distress (depression, anxiety and stress) using the same graduated severity level approach to FA, against
the YFAS symptom count adopting current DSM-5 (APA, 2013) based diagnostic specifiers. Specifically, Study 4 investigated which YFAS symptomologies should be grouped together to form four graduated FA classification groups in regard to psychological distress; exploring whether the same symptom counts for each of the four severity groups held true with psychological distress (depression, anxiety and stress). It was hypothesised psychological distress would significantly increase serially from non-FA, to mild-FA, to moderate-FA, to severe-FA participant groups. Lastly, the research aimed to show from Studies 3 and 4 that, FA symptomology can be classified according to a graduated severity level approach, which differs from the current FA binary approach. This suite of four studies aimed to explore the validity of a new theoretical model – food addiction – for understanding obesity in the context of diabetes, based on addiction treatment models used in the diagnosis and management of other addictions. Should the new model prove valid, this research suggests new avenues for the prevention and treatment of obesity in the context of diabetes. Food addiction is a contemporary concept challenging traditional methods (e.g., educational models, Cognitive Behaviour Therapy (CBT), individual or group therapies) in the treatment of obesity and related medical complications. Simply directing a person predisposed to FA to lose weight in order to decrease their diabetes symptomology is unlikely to be effective for everyone. This approach may also explain why current treatment modalities have limited effect in many individuals.

1.1 Specific aims for this dissertation

Study 1 research objectives:

1. Determine what percentage of a t2d sample fulfil a classification of FA, based on the YFAS.
2. Investigate how does BMI differ significantly between food addicts and non-food addicts as classified by the YFAS.

3. Examine how does FA, impulsivity (non-planning, motor, attentional), depression, anxiety and stress relate to BMI in people with t2d.

**Study 2 research objectives:**

4. Explore how does depression, anxiety, and stress scores significantly differ between participant’s who meet a FA criterion and those that did not.

5. Investigate the cross-sectional predictive contributions of FA and BMI to depression, anxiety, and stress scores.

**Study 3 research objectives:**

6. Explore a graduated severity level approach to FA classification, against BMI classifications, then investigate differences in BMI against the YFAS count, by adopting current DSM-5 based diagnostic specifiers for SUDs (APA, 2013) to classify individuals’ FA symptomology severity.

7. Examine which YFAS symptomologies should be grouped together to form four graduated FA classification groups to demonstrate BMI will increase significantly from non-FA, to mild-FA, to moderate-FA, to severe-FA participant groups.

**Study 4 research objectives:**

8. Continue to explore a graduated severity level approach to FA classification, examining psychological distress (depression, anxiety and stress) against the YFAS symptom count by adopting current DSM-5 based diagnostic specifiers for SUDs (APA, 2013) to classify individuals’ FA symptomology severity.

9. Examine which YFAS symptomologies should be grouped together to form
four graduated FA classification groups in regard to psychological stress, to
demonstrate psychological distress increases significantly from non-FA to
mild-FA, to moderate- FA, to severe-FA participants groups.

10. Demonstrate from studies three and four, FA symptomology can be classified
according to a graduated severity level approach, which differs from the
current FA binary approach, that classifies a person as either a food addict or
not.

The findings of these studies have broad significance both in terms of theoretical
and therapeutic implications, and applications for initiatives and approaches to combat
obesity. The current binary approach to PFA diagnosis, unlike diagnosing SUDs (APA,
2013), does not adequately capture the important variations in FA severity. Investigating
and introducing a graduated severity approach to the assessment of FA can aid in
identifying potential FA in the early stages (i.e., mild or moderate FA symptomology),
which may help minimise risk factors associated with FA and prevent the progression to
severe FA, as well as other complications, including diabetes, hypertension, heart
disease, and stroke.
2. Review of the Literature

This chapter outlines the key issues, concepts and literature underpinning the present research. First, this chapter provides a description of diabetes and considers its association with obesity. Research evidence regarding the factors contributing to obesity, including FA, is then presented and critically analysed. The potential significance of FA in understanding, preventing, and treating obesity are also discussed.

2.1 Diabetes

Diabetes has historical underpinnings, and was once known as a rare disease. The literal meaning of diabetes is ‘to pass through’, meaning the passing of liquids through the body (Brand-Miller, Foster-Powell, Colagiuri, & Barclay, 2007). Today, diabetes is by no means a rare disease, it is now known as the epidemic of the 21st century and is one of the largest and fastest growing health crises globally (Diabetes Australia, 2016). In addition to the 415 million people estimated to have a diabetes diagnosis, over 318 million people have pre-diabetes (the term used to describe impaired glucose tolerance and/or impaired fasting glucose), meaning that a person’s blood glucose levels are somewhere between normal and a diabetes diagnosis, and with a potentially high risk of developing diabetes (IDF, 2015). Table 1 shows the global prevalence (top ten countries) of diabetes and the estimated numbers of people who will be affected by this chronic disease in the next 25 years.
Table 1  
*Top ten countries/territories for number of people with diabetes (20-79 years), 2015 and 2040.*

<table>
<thead>
<tr>
<th>Rank</th>
<th>Country/territory</th>
<th>Number of people with diabetes 2015</th>
<th>Rank</th>
<th>Country/territory</th>
<th>Number of people with diabetes 2040</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>China</td>
<td>109.6 million (99.6-133.4)</td>
<td>1</td>
<td>China</td>
<td>150.7 million (138.0-179.4)</td>
</tr>
<tr>
<td>2</td>
<td>India</td>
<td>69.2 million (56.2-84.8)</td>
<td>2</td>
<td>India</td>
<td>123.5 million (99.1-150.3)</td>
</tr>
<tr>
<td>3</td>
<td>USA</td>
<td>29.3 million (27.6-30.9)</td>
<td>3</td>
<td>USA</td>
<td>35.1 million (33.0-37.2)</td>
</tr>
<tr>
<td>4</td>
<td>Brazil</td>
<td>14.3 million (12.9-15.8)</td>
<td>4</td>
<td>Brazil</td>
<td>23.3 million (21.0-25.9)</td>
</tr>
<tr>
<td>5</td>
<td>Russian Federation</td>
<td>12.1 million (6.2-17.0)</td>
<td>5</td>
<td>Mexico</td>
<td>20.6 million (11.4-24.7)</td>
</tr>
<tr>
<td>6</td>
<td>Mexico</td>
<td>11.5 million (6.2-13.7)</td>
<td>6</td>
<td>Indonesia</td>
<td>16.2 million (14.3-17.7)</td>
</tr>
<tr>
<td>7</td>
<td>Indonesia</td>
<td>10.0 million (8.7-10.9)</td>
<td>7</td>
<td>Egypt</td>
<td>15.1 million (7.3-17.3)</td>
</tr>
<tr>
<td>8</td>
<td>Egypt</td>
<td>7.8 million (3.8-9.0)</td>
<td>8</td>
<td>Pakistan</td>
<td>14.4 million (10.6-20.4)</td>
</tr>
<tr>
<td>9</td>
<td>Japan</td>
<td>7.2 million (6.1-9.6)</td>
<td>9</td>
<td>Bangladesh</td>
<td>13.6 million (10.7-24.6)</td>
</tr>
<tr>
<td>10</td>
<td>Bangladesh</td>
<td>7.1 million (5.3-12.0)</td>
<td>10</td>
<td>Russian Federation</td>
<td>12.4 million (6.4-17.1)</td>
</tr>
</tbody>
</table>

*Source(s):* IDF Atlas, 2015

Diabetes mellitus is the term given to several different conditions that affect how an individual’s body utilises food for energy. If the body cannot maintain healthy levels of glucose in the blood, glucose (a type of sugar) builds up causing health problems linked to diabetes. (American Diabetes Association, ADA, 2014; Diabetes Australia, 2016). Diabetes
is classified into three main diagnoses. In type 1 diabetes, the pancreas produces minimal or no insulin, and daily insulin injections are needed to compensate for this deficiency. In type 2 diabetes (t2d), the pancreas does not completely cease the production of insulin; however, the body is unable to maintain blood glucose levels within the normal range because the body’s cells are impaired and as such do not respond positively to insulin (Diabetes Australia, 2016; O’Connell, 2011). The third type of diabetes, gestational diabetes, develops during pregnancy in women with no previous diagnosis. Typically, this classification of diabetes may resolve itself after the mother has given birth; however, a woman who has had gestational diabetes, has an increased risk of developing t2d post-partum (Diabetes Australia, 2016). The medical condition of prediabetes is typically defined as when an individuals’ blood glucose levels are somewhere above normal but below diabetes thresholds (ADA, 2015; Tabak, Herder, Rathmann, Brunner & Kivimake, 2012). (Table 2).

There are several blood tests used to diagnose Diabetes Mellitus: i) a fasting glucose (sugar) test where the patient fasts for a minimum of eight hours, such as drinking or eating overnight; ii) random glucose testing taken during the day; iii) an oral glucose tolerance test (OGTT) where a patient who has fasted drinks a sugary liquid and then has a blood test at one hour and then two hours later; iv) the HbA1c blood test, which provides information about a person’s average levels of blood glucose, (blood sugar) and is reported as a percentage. The higher the percentage, the higher an individual’s blood glucose levels have been over the past three months. Those individual’s presenting with a high-risk diagnosis are clearly classifiable as prediabetic, with a ten-fold increase progressing from prediabetes to a t2d diagnosis (O’Connell, 2011). The term ‘diabetogenic environment,’ (Brand-Miller, Foster-Powell, Colagiuri, & Barclay, 2007) meaning our Westernised diet and sedentary lifestyles, are what researchers infer as a trigger, and a major risk factor for prediabetes (ADA, 2015).

Individuals who are overweight especially regarding waist measurements, are three times
higher at risk of developing t2d than people with a normal BMI (Brand-Miller, Foster-Powell, Colagiuri, & Barclay, 2007).

Table 2

Diabetes Classification

The HbA1c test levels used to diagnose type 2 diabetes and prediabetes

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>A1C Level (percentages)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>Below 5.7%</td>
</tr>
<tr>
<td>Type 2 Diabetes</td>
<td>6.5% or above</td>
</tr>
<tr>
<td>Prediabetes</td>
<td>5.7% - 6.4%</td>
</tr>
</tbody>
</table>

*Note. Any test for diagnosis of diabetes requires confirmation with a second measurement, unless there are clear symptoms of diabetes (ADA, 2014).

Australian statistics have shown a similar increase in the rate of diabetes diagnoses to those reported from other first-world countries, increasing from 2.4% in 1995 to 3.8% in 2007/2008 (Australian Bureau of Statistics, ABS, 2011). Increased rates of diabetes were attributed to being overweight, genetic influences, ageing, and ethnicity. In a large-scale study of Australian Indigenous communities, the diagnosis of t2d was more than three times higher when compared to a non-indigenous Australian sample (ABS, 2006; Burrow & Ride, 2016).

Overall, diabetes mellitus represents a huge health and economic cost to society, contributing to 5.1 million deaths in 2013 worldwide. The IDF (2013) reported, in 2013, health expenditure related to diabetes cost a minimum of 548 billion United States Dollars (approximately 615 million Australian Dollars). These costs will continue to rise as the number of diagnoses increase.
2.2 Type 2 Diabetes

The IDF (2015) estimates that over 190 million people are oblivious to having diabetes world-wide and were suggesting t2d classification makes up the majority of these cases. Type 2 diabetes has primarily occurred in adults over 45 years of age; however, in recent years, there has been an increase in the number of diagnoses in younger populations (Australian Diabetes Council (ADC), 2014). Data suggested the prevalence of t2d in high-income countries is higher than developing countries, however, there is a trend demonstrating some middle-income countries have a higher prevalence of t2d than richer countries (WHO, 2016).

Both the factors contributing to individual t2d onset, and societal level concerns vary. These factors include, an escalation of labour saving mechanical devices, increased processed food consumption, adopting sedentary Westernised lifestyles, greater emphasis on managing t2d with drugs rather than promoting a lifestyle change, and considerable attention investigating treatments rather cures (ADC, 2014; O’Connell, 2011). Furthermore, diagnosed t2d is associated with a range of serious health complications, including, coronary heart disease, cardiovascular disease, toxic shock (from limb amputation), rheumatoid arthritis, kidney disease, non-alcoholic fatty liver disease, and diabetic retinopathy, (ABS, 2011; American Diabetes Association, (ADA), 2014; ADC, 2014; Canadian Diabetes Association, 2017; IDF, 2013; O’Connell, 2011).

Research continues to show that the increasing prevalence of obesity is a major factor contributing to the escalating incidence of t2d (Eckel, Grundy, & Zimmet, 2005; IDF, 2013; WHO, 2013). As a consequence of being overweight, the body’s resistance to and production of insulin are inhibited (Kahn, & Flier, 2000) thus, reiterating the absolute necessity of screening and diagnosis of t2d in those who are overweight. The earlier a
diagnosis is established, followed by the implementation of a treatment and management plan, the greater the chances are of averting further complications.

2.3 Obesity

Of the many antecedents of t2d, previous research has clearly implicated obesity as a major risk factor. The “obesity epidemic” (James, Leach, Kalamara & Shayeghi, 2001; Wang, Beydoun, Liang, Caballero, & Kumanyika, 2008) continues to grow at an alarming rate, with Australia exhibiting an extremely high prevalence of obesity (ABS, 2011). Numerous intervention strategies in the past five years have aimed to address obesity, high body weight, disordered eating, dieting and associated issues (Marks, 2015a). Despite these extensive and exhaustive efforts, an obesogenic environment continues to dominate the 21st century (Marks, 2015b).

In Australia, the prevalence of people aged 18 years and over who were classified as being overweight or obese rose from 56.3% in 1995 to 61.2% in 2007-08 (ABS, 2011). This trend continues to increase as the latest National Health Survey 2014-2015, (ABS, 2014-2015) showed over 11 million Australians (63.4%) aged 18 years and over were overweight or obese. Furthermore, the survey demonstrated overweight and obesity rates continued to rise as a person ages, with approximately four in five males 45 years and over being overweight or obese, while two in three women of the same age were overweight or obese. (ABS, 2015). (Table 3).
Table 3
Persons aged 18 years and over – Proportion of males and females overweight or obese, 1995 to 2014-2015 in Australia.

<table>
<thead>
<tr>
<th>Age Group (years)</th>
<th>Males % 1994</th>
<th>Males % 2014-2015</th>
<th>Increase %</th>
<th>Females % 1994</th>
<th>Females % 2014-2015</th>
<th>Increase %</th>
</tr>
</thead>
<tbody>
<tr>
<td>18-24</td>
<td>36.7</td>
<td>43.8</td>
<td>7.1</td>
<td>26.3</td>
<td>33.3</td>
<td>6.0</td>
</tr>
<tr>
<td>25-34</td>
<td>58.8</td>
<td>62.5</td>
<td>3.7</td>
<td>38.2</td>
<td>42.5</td>
<td>4.3</td>
</tr>
<tr>
<td>35-44</td>
<td>66.4</td>
<td>74.3</td>
<td>7.9</td>
<td>45.5</td>
<td>58.1</td>
<td>12.6</td>
</tr>
<tr>
<td>45-54</td>
<td>76.9</td>
<td>79.8</td>
<td>2.9</td>
<td>57.3</td>
<td>61.9</td>
<td>4.6</td>
</tr>
<tr>
<td>55-64</td>
<td>76.2</td>
<td>81.8</td>
<td>5.6</td>
<td>68.5</td>
<td>68.2</td>
<td>-0.3</td>
</tr>
<tr>
<td>65-74</td>
<td>73.1</td>
<td>79.9</td>
<td>6.8</td>
<td>63.8</td>
<td>68.8</td>
<td>5.0</td>
</tr>
<tr>
<td>75+</td>
<td>66.1</td>
<td>73.2</td>
<td>7.1</td>
<td>59.8</td>
<td>65.6</td>
<td>5.8</td>
</tr>
</tbody>
</table>

Source(s): National Health Survey: First results 2014-2015

On a global scale, in the 1970s and 1980s, most developed and higher income countries began to see an increase in obesity (Swinburne et al., 2011). In the years that followed, the majority of mid and lower income countries also began to see a surge in the rates of obesity (Popkin, Conde, Hou, & Monteiro, 2006). Depending on the source, it is reported that, in 1980, 6 percent of adults were overweight or obese, and by 2008, this figure doubled to approximately 12 percent. Of greater concern is the number of children under 5 years of age who were overweight or obese in 2013, which was approximately 42 million (Stevens et al., 2012). The prevalence of obesity globally by 2050 is predicted to be approximately 55 percent of adults and 25 percent of children reflecting the obesogenic environment as a significant psychosocial problem reaching pandemic levels, particularly across Australia, the United States, United Kingdom and the majority of Europe. Despite
efforts around the world to search for a solution, thus far, no country has been able to produce a public health model to abate this crisis (Swinburne et al., 2011).

The WHO (2013) categorises a person as being overweight if they have a BMI of $\geq 25-29.99$ kg/m$^2$, and as obese if a BMI is $\geq 30$ kg/m$^2$. BMI is calculated by dividing a person’s weight in kilograms by the square of his or her height in metres (kg/m$^2$). Of great concern is the finding that the rates of people with higher BMI scores (35+) have almost doubled between 1995 (5%) and 2011-12 (9.6%); WHO, 2013). Approximately 3.4 million adults die annually from being overweight or obese (WHO, 2014). Obesity research has demonstrated a causal relationship between obesity and numerous other diseases such as: diabetes, cardiovascular disease, non-alcoholic fatty liver, hypertension, gallstones, sexual impotence, sleep apnoea, osteoarthritis and some cancers (Grundy et al., 2005; Liberopoulos, Mikhailidis, & Elisaf, 2005).

A wide range of traditional and contemporary obesity prevention strategies have been explored and implemented over the last fifty plus years including public health and educational initiatives, counselling, diet and exercise plans, engaging family and friends, and support groups, attending nationally and internationally “slimmer’s groups” and weight loss programs, bariatric surgery and control methods namely self-induced vomiting, compulsive exercising and starvation (Cade & O’Connell, 1991; Fairburn & Cooper, 1982; Han, Lawlor, & Kimm, 2010; Kissler & Settmacher, 2013). Despite society’s immense economical expenditure on trying to halt the rise in obesity, and the amount of publications on this topic, little progress has been made in combatting the ever-growing obesogenic environment (Brownell & Gold, 2012; Dobbs et al., 2014; Marks, 2015b).

In response to this global crisis, WHO (2013) published an action plan to end the rising diabetes rates and obesity crisis as part of a vision: ‘A world free of the avoidable burden of non-communicable diseases’. With six objectives and nine voluntary global
targets, Objective 3 proposes to reduce four modifiable risk factors: tobacco, physical inactivity, harmful use of alcohol, and unhealthy diet. The primary aims were to ‘reduce these four modifiable risk factors for noncommunicable diseases, and address the underlying social determinants through creation of health promoting environments’. Albeit, if compared to the historical battle that took place in combatting the nicotine epidemic, which took over half a decade to reduce smoking rates and tobacco related diseases, (Brownell & Warner, 2009) then, only time will tell if a global - action plan - can start to address “globesity” which continues to increase globally (WHO, 2003).

2.4 Psychological and physiological factors

Some of the most widely researched psychological and physiological factors that have been found to contribute to obesity include stress, depression, and anxiety (Chen & Qian, 2012; Eyres, Turner, Nowson, & Torres, 2014; Stunkard, Faith, & Alison, 2003). The literature indicates stress is associated with increased cortisol levels (Vicennati, Pasqui, Cavazza, Pagotto, & Pasquali, 2009), affecting appetite (Sinha & Jastreboff, 2013) and eating behaviour (Mouchacca, Abbott & Ball, 2013), which, in turn, has been associated with an increased likelihood of obesity (Chen & Qian, 2012). Chronic stress has also been found to have a role in the development of depression, anxiety, and addiction (Foss & Dyrstad, 2011).

Depression is identified based on the presentation of a prolonged and sustained depressed mood (Ingram, 2009). Sufferers of depression frequently report symptoms such as anhedonia, weight gain or loss, psychomotor agitation or retardation, sleep disturbances, fatigue, feelings of guilt or worthlessness, and reduced concentration (Ingram, 2009). Studies have demonstrated strong links between depression and obesity, with a majority of females suffering from depression and experiencing weight gain (Stunkard, Faith, & Alison, 2003).
A meta-analysis conducted by de Wit et al. (2010), which included 17 depression and obesity studies (total N = 204,507), demonstrated a significant relationship between depression and obesity. Notably, gender differences were found with females suffering from depression more likely to be obese than males. Blaine (2008) performed a similar meta-analysis, with 16 longitudinal studies (N = 33,000) relating to depression and weight control. The meta-analysis demonstrated people who were depressed at baseline measurement were 1.8 times more likely to be obese or have experienced weight gain at follow-up, than non-depressed individuals, with a significant population effect size of 1.47 (95% CI; 1.16-1.85).

Contrary to the findings of de Wit et al. (2010), Blaine (2008) found no overall gender differences between males and females. However, when the analysis was modified to include only adolescent samples, females with depression were more likely to have become obese or experienced weight gain at follow-up than males. Despite this finding, Blaine (2008) recommended cautions when interpreting these findings due to the small number of male samples.

Although the relationship between depression and obesity has been consistently found, there has not always been consensus about the direction of causality, or whether the relationship is bidirectional. Finn (2010) conducted a longitudinal study, which demonstrated average weight women (BMI <25) who were depressed at baseline had a 54% more chance of developing obesity than females who were not depressed. In support of a non-bidirectional relationship, Finn (2010) found only 27% of the overweight or obese females were more likely to develop depression within five years compared with normal weight women.

Furthermore, a systematic review and meta-analysis by Luppino et al., (2010) demonstrated that obesity at baseline increased the risk of depression at follow-up, as did
being overweight at baseline. Depression was also found to be predictive of participants being overweight or obese at follow-up. Results also demonstrated obese people had a 55% higher risk of developing depression over time, whereas the risk of people with depression becoming obese was 58% (Luppino et al., 2010). Although directional relationships were found between depression and obesity and obesity and depression, both Finn (2010) and Luppino et al., (2010) found baseline depression presented more risk of developing obesity than the converse.

Anxiety relates to an individual experiencing excessive fear is due to an overestimation of the true extent of the danger present in the environment, or a situation, or in anticipation of future threats (APA, 2013; Oltmanns & Emery, 2007). Anxiety typically entails dysphoria and somatic symptoms, which may lead to the avoidance of feared places and or situations (APA, 2013; Colman, 2003). A positive relationship between anxiety and obesity has frequently been found (Eyres, Turner, Nowson, & Torres, 2014).

Scott et al. (2008) utilised data from the World Mental Health Surveys and self-reported BMI to examine the relationships between anxiety and obesity. Results demonstrated a pooled odds ratio of 1.2 was found for anxiety and obesity, whereas severe obesity resulted in an odds ratio of 1.5. Further analyses differentiated between gender, with results exhibiting a significant relationship between anxiety and obesity but only in females with a pooled odds ratio of 1.3 (CI: 1.1-1.5, p <.05) compared to males 0.9 (CI: 0.8- 1.1, ns). A similar meta-analysis conducted by Gariepy, Nitka, and Schmitz (2010) also found a positive relationship between anxiety and obesity. The pooled odds ratio from cross-sectional studies was 1.4 (CI: 1.2-1.6) demonstrating a moderate level of empirical evidence. However, in contrast to the findings of Scott et al., (2008), Gariepy et al., (2010) found in a subgroup analyses a positive relationship
between anxiety and obesity in both males and females. Gender, one of many variables including age and socio-economic status, is often identified in obesity and anxiety studies as a cofounding variable (Gariepy et al., (2010). While both studies found a relationship between obesity and anxiety, there was not a consensus on whether gender influenced this relationship or not.

Moreover, there is continuing debate and research on the comorbidity of anxiety and depression (Anderson, Cohen, Naumoba, & Must, 2006; Nutt, 2004; Rivas-Vazquez, 2004). Bitsika and Sharpley (2012) investigated this bidirectional relationship among university students and found a comorbid relationship between anxiety and depression for over one-third of the participants, further suggesting that the inclusion of this comorbidity may be paramount when analysing obesity and psychological distress.

Taken together, the psychological actors of stress, depression and anxiety have all been strongly associated with t2d (Al-Amer, Sobeh, Zayed, & Al-domi, 2011; Trento et al., 2011). Kaur, Tee, Ariaratnam, Krishnapillai, and China (2013) investigated these factors among 2,508 Malaysians with t2d, finding the prevalence of stress, depression, and anxiety to be 12.5 %, 11.5 % and 30.5 % respectively. Being mindful that research generally observes anxiety as the more dominant factor over depression in people with t2d (Tovilla-Zárate et. al., 2012), Meigs, (2002) examined the prevalence of depression and anxiety among people with td (N = 820). The results demonstrated that the rates for depression and anxiety were 48.27% and 55.10% respectively.

Similarly, Collins, Corcorant, and Perry (2008) examined the prevalence of anxiety and depression symptoms in patients with diabetes, and highlighted the prevalence of depression and anxiety in people with t2d was more than double what would be expected based on general population estimates. Kaur et al. (2013)
expounded the importance of routine screening for depression and anxiety during outpatient visits to ensure a more holistic approach in the treatment of people with T2D, which appears to be a void in general practice areas (Tovilla-Zárate et. al., 2012).

Impulsivity is a multi-faceted construct that relates to an individual’s ability to inhibit impulses, drives or temptations (Colman, 2003; Tice, Bratslavsky, & Baumeister, 2001). Furthermore, there is continuing empirical investigations on how impulsivity – as a personality trait – is linked to a variety of behaviours (Barratt, 1959; Mitchell & Potenza, 2014; Patton et al., 1995). Indeed, a certain level of impulsivity can be useful in situations where rapid decisions are required (Crews & Boettinger, 2009). Generally speaking, impulsive behaviour demonstrates a lack of foresight and planning, and may provide short-term pleasurable rewards. However, impulsivity can prevent the attainment of longer-term goals and/or result in negative consequences (Braet, Claus, Verbeken, & Van Vlierberghe, 2007).

The trait impulsivity is included in every major personality model (Hofmann, Friese, & Wiers, 2008) and features prominently as a diagnostic criterion for a myriad of psychopathological disorders detailed in the Diagnostic and Statistical Manual for Mental Disorders (DSM). It is associated with a number of health and social issues, including substance abuse and misuse (e.g., drugs, alcohol, nicotine, caffeine, compulsive gambling), Borderline Personality Disorder (Herbert et al., 2016), crime, domestic violence, and obesity (Binge Eating Disorder; BED) (de Wit, 2008; Marazziti, Baroni, Picchetti, & Catena dell’Osso, 2011; Tice, Bratslavsky, & Baumeister, 2001; Whiteside & Lynam, 2001; DSM-IV.TR 2004; DSM-5, 2013).

Hofmann, Friese and Roefs (2009) argued that many health-related problems can be formulated as a conflict between impulsivity and reasoning, dictating a need to restrain behaviour. Consequently, impulse control is considered to play a major role in
the self-regulation of eating behaviour (Meule, 2013). In a study by Hofmann et al. (2009), three factors of impulse control (executive control, inhibition, and affect regulation) were measured to examine their role in candy consumption in female university students. Results demonstrated executive attention and inhibitory control significantly moderated the impact of automatic affective reactions with regards to candy consumption. Of late, there has been a diverse array of literature exploring impulsivity and dietary associations among children, adolescents, and adults, including, eating disorders, emotional eating, overeating, food and addiction, BMI (including underweight, normal and overweight samples), and neurological mechanisms unpinning the over ingestion of processed foods (Nederkoorn, Dassen, Franken Resch, & Houben, 2015; Meule, Hofmann, Weghuber, & Blechert, 2016).

However, there is a dearth of literature specifically exploring links between the underlying factors of t2d and the multi-faceted trait of impulsivity. Exploring this dual combination appears to be pertinent especially regarding the challenges in achieving daily optimal Glycemic control. Considering and investigating the multiple facets of impulsivity and its various links to the many and diverse components of health such as t2d and substance abuse appears to be critical for advancing knowledge, and providing insight into further developments of targeted diagnosis, treatment and prevention.

2.5 Food addiction and obesity

The history of addiction is replete with questions of when to make a distinction, and when to include new theories and or models into the substance abuse pantheon. In the early 1930’s alcoholism began to be considered as an allergy (Alcoholics Anonymous, 1988). In 1956 the American Medical Association had accepted alcoholism as a disease that required treatment (Schneider, 1978). Moving forward, in the 1980s and 1990s, nicotine, cocaine and marijuana (Eysenck, 1991; Musto, 1991), and more
recently gambling, sex and the internet have been topical in the addiction arena (APA, 2013; Müller, Dreier, Beutel, & Wölfling, 2016)

Until the last decade, the science of FA and any accompanying theories were limited in number and scope with little hard evidence to validate the FA concept (Brownell & Gold, 2012). As far back as the 1950s, Randolph (1956) recognised that a specific adaptation to one or more regularly consumed foods to which a person is highly sensitive produced a common pattern of symptoms descriptively similar to those of the addictive processes. Randolph (1956) suggested those addictive eating patterns could be observed when ingesting corn, wheat, coffee, milk, eggs, and potatoes. Current research has extended and refined the concept of FA stimulating further scientific interest and debate (Schulte et al., 2015).

The FA concept has become progressively prominent, especially among the lay population of today (Brownell & Gold, 2012). However, within the scientific literature, similar to any new theories and concepts being conceived, the notion of FA continues to be a somewhat controversial field of study (Ziauddeen, Farooqi, & Fletcher, 2012). As such, there are many questions still being debated concerning the FA concept, including what is meant by the term FA, and does it include all foods, some foods, something in certain foods, or the behaviours and patterns in and around the act of eating? Over the last decade there has been a surge in research implying that an addiction-like process may underpin a person’s inability to regulate their food intake (Bégin et al., 2012; Blumenthal, & Gold, 2010; Burmeister, Hinman, Koball, Hoffman, & Carels, 2013; Moodie, 2013; Schulte, Joyner, Potenza, Grilo, & Gearhardt, 2015).

While FA has been portrayed as a problem arising from a lack of willpower or gluttony (Ifland, Shephard, & Wright, 2012), more recent evidence has provided neurobiological, psychological, and behavioural support for the existence of FA
Although FA has not been included in the DSM-5, binge-eating disorder (BED), which shares many similarities with the concept of FA, has been included. The diagnostic criteria for BED includes symptoms such as eating an amount of food larger than what most people would eat, experiencing a lack of control over eating throughout the bingeing period, eating large amounts of food despite not feeling physically hungry, and experiencing marked distress related to the binge-eating behaviour (APA, 2013). The diagnostic category of BED is based on the assumption that the symptoms are simply behavioural in nature, rather than a complex interplay between behavioural, psychological, and biochemical factors. Ifland et al. (2009) expounds that the concept of FA is more complex than simply a behavioural issue, and the behavioural symptoms along with brain mechanisms intrinsic to FA are more consistent with other SUDs. Based on this premise, it is reasonable that individuals with FA symptomology could be diagnosed as having “other (or unknown) substance use disorder,” as defined in the DSM-5.

2.6 Biochemical and Neurological Basis of Food Addiction

The biochemical foundation of FA is evidenced by the findings of Noble et al. (1994), who are credited with the discovery that the dopamine receptor D2 gene associated with alcoholism was also associated with FA. Noble et al. (1994) conducted a DNA analysis of 73 obese patients (BMI ≥ 28) who were drug and alcohol free but regularly binged on refined carbohydrates. Results showed that 64.3% of participants who binged on carbohydrates carried the DRD2 (dopamine receptor D2 gene) A1+ allele, compared to only 21.1% of participants who preferred to binge on other foods (fats, proteins, or food in general).

Furthermore, having one or a combination of the three phenotypic factors, which
included parental history of obesity (one or both parents), onset of obesity (adolescent or adult), and carbohydrate preference, were associated with an increased likelihood of having the A1+ allelic classification. Noble et al. (1994) suggested that the increased expression of symptoms associated with the prevalence of the A1+ allele might be associated with a decrease in the functioning of the DRD2 gene. Thus, individuals with the A1+ allele polymorphism are more likely to have a deficiency in dopamine.

Positron Emission Tomography (PET), a powerful tool for in vivo imaging investigations of human brain function, has been utilised to uncover the neurological basis of FA. Wang et al. (2001) examined the dopaminergic system in an attempt to understand the way in which it modulates food reward via the meso-limbic circuit of the brain. Results revealed brain dopamine D2 receptor availability was significantly lower in participants classified as obese (BMI 40+) than participants in the control group. The BMI of obese participants also had a negative relationship with measures of D2, indicating an association between low D2 receptor density and obesity – that is, the higher the participants’ BMI, the lower their number of D2 receptors. Although there is growing evidence to support the role of genetics in food addiction (Davis et al., 2013), the evidence is somewhat equivocal (Cornelis et al., 2016) and importantly, it is not based on samples of people with t2d.

Animal studies have also been employed in the FA research field. For example, Lenoir, Serre, Cantin and Ahmed (2007) examined whether a preference for saccharine (artificial sweetener) would develop in rats already addicted to cocaine. Cocaine addicted rats were exposed to a choice of saccharine or cocaine with results showing 94% of the rats consistently preferring the sweeter taste of the saccharine than the cocaine. Even when Lenoir et al. (2007) increased the dosage of cocaine fourfold, the rats continued to
prefer saccharine, demonstrating saccharine to be more addictive than cocaine in rats. Furthermore, Lenior et al. (2007) suggested that despite saccharine being much less effective in inducing presynaptic dopamine in the ventral striatum than cocaine, consumption of saccharine may produce a more intense postsynaptic dopamine signal than cocaine. Investigating the predisposition for sugar addiction, Avena, Rada and Hoebel (2008) used a series of models to investigate patterns and or behaviours in sugar addiction. Rodents were first exposed to a palatable substance, then denied access to it. The substance was then provided, but on a restricted basis. This pattern of restricting and exposure advanced bingeing and changes in neural networks, implying potential addictive behaviours akin to those induced by addictive substances was occurring. This line of research is suggestive that FA is more about the way the substance is ingested (restrict then overeat or binge) that appears to generate addiction-like practices (Avena, Rada & Hoebel, 2009; Pelchant, 2009; Wojicki, Robers & Corwin, 2006).

More recently, human studies have demonstrated FA as a valid concept. Pedram et al., (2013) assessed the prevalence of FA contributing to obesity in the general population, with results highlighting the prevalence of FA increased with participants’ obesity status. Gearhardt, White, Masheb, and Grilo (2013) researched the role of FA (as assessed by the YFAS) in 96 obese patients with BED, who were seeking treatment for binge-eating and obesity in a clinical setting. Findings demonstrated 41.5% met the YFAS diagnostic threshold of ≥ 3 symptoms (7 symptoms in total) and had clinically significant impairment/distress present in the past 12 months. Of the 41.5% of participants who met the diagnostic threshold, all of them reported experiencing the symptom of being “unable to cut down or stop.” Similarly, an earlier study found approximately 50% of obese BED patients seeking treatment reported similar symptomatology and characteristics in line with the processes of addiction (Gearhardt,
Based on the findings of Wang et al. (2001), Lenoir et al. (2007), Ifland et al. (2009), and Gearhardt et al. (2013), there appears to be empirical evidence to support the concept that FA shares similarities with other disorders of addiction. As such, BED’s focus on behavioural components does not adequately cover the concept of FA and its behavioural, neurochemical, and genetic aspects. Accordingly, it is perhaps more appropriate to consider FA as a substance use disorder, and individuals who are considered to have FA may actually meet the diagnostic criteria for other (or unknown) substance-related disorders as described by the DSM-5 (APA, 2013). Food addiction includes diagnostic criteria based on the symptomology of craving, tolerance, and withdrawal (APA, 2013), which are consistent with the FA criteria described and measured in the YFAS (Gearhardt et al., 2009).

Despite the genetic (Davis et al., 2013; Noble, 1994), neurological (Volkow, Wang, Tomasi & Baler, 2013; Wang, 2008), and biochemical (Avena et al., 2012) theories that have been put forward to explain FA, Ziauddeen, Farooqi, and Fletcher (2012) criticised previous findings relating to FA, their links to obesity, and the theory that FA is analogous to addiction. While agreeing that the clinical model of FA (based on the DSM-IV-TR criteria of addiction) showed some clinical validity, Ziauddeen et al. (2012) questioned the applicability of the FA model in non-clinical samples, as past research has focussed on neurobiological models and clinical populations. Two important questions further posed by Ziauddeen et al. (2012) related to whether people can actually become “food addicts”, and whether there are particular foods that are “addictive” and, therefore, akin to drugs of abuse. Ifland et al. (2009) suggested a distinction between refined and unrefined foods that may answer the question of whether particular foods are inherently “addictive”. However, Ziauddeen et al. (2012) argued
this categorisation is too broad and ambiguous, and that FA is based on paralleling the
behavioural dependence criteria of disordered eating.

In response to Ziauddeen et al’s (2012) criticisms, Avena, Gearhardt, Gold, Wang, and Potenza (2012) agreed that not everyone who is overweight presents with FA symptomology, but highlighted that dismissing this model prematurely may in fact negate empirical evidence that supports the concept of a FA model (Blumenthal & Gold, 2010; Corsica & Pelchat, 2010; Corwin & Grigson, 2009; Davis et al., 2011; Frascella, Potenza, Brown, & Childress, 2010; Holden, 2001). Avena et al. (2012) postulated this impact would be dire if it were dismissed on the premise that the notion of FA has limited empirical evidence.

Despite the criticism and literature that are generally a natural part of investigative fields, research continues to demonstrate the FA model to be a valid concept for continued investigation. Gearhardt et al. (2012) found approximately fivefold higher rates of FA in obese people with BED, and findings by Davis et al. (2011) demonstrated FA has similar symptoms to that of a typical individual who abuses drugs. As such, individuals may be classified as having psycho-behavioural characteristics accompanying clinical symptomology. Later research continues to reiterate these findings regarding a FA concept. Burmeister, Hinman, Koball, Hoffmann, and Carels (2013) noted that there has been an overlap between FA and other ways of conceptualizing disordered eating - especially BED - where strong relationships between binge eating and FA were observed along with parallel relationships between the constructs of distress and poor weight loss. Current considerations regarding FA (Schutle, Joyner, Potenza, Grilo, Gearhardt, 2015) convey that FA has many facets of addiction, suggesting strongly that cautionary procedures needed to be in place to avoid the repeated mistakes that hindered the recognition of
tobacco use as a substance use disorder.

As with all empirical research, it takes critical analyses and the cultivation of a collegial atmosphere in bringing about valid and reliable evidence to move forward in any neurobiopsychosocial field. While FA research is not as far advanced as other SUDs, nevertheless this genre of addiction medicine continues to progress along similar pathways of prevention, diagnosis, and treatment analogous to SUDs.

Investigating a FA concept and applying an addiction model, specifically, applying processed food addiction to problematic eating and the associated consequences, may lead to novel and more effective intervention approaches. Examining individual manifestations that may increase the risk of processed food addiction via a graduated approach to FA classifications may help discern differences in addictive processes contributing to problematic eating. As such, a graduated approach to FA classifications can facilitate individualised treatment care and management, focusing on what would be best for the particular person based on their FA symptom count.

The present research was designed in response to current trends in the study of the saliency of obesity and psychological well-being in a subgroup of people whose diagnosis and treatment is largely based on BMI levels. Thus, this dissertation aimed to gain a better understanding of the factors implicated in the development of obesity and poor mental health in a t2d cohort. Although there is a vast amount of research on the relationships between impulsivity and other addictions, there is a lack of studies examining the relationships between FA, psychological distress, impulsivity and obesity among diabetes cohorts. A greater understanding of these relationships could help determine whether behavioural, neurological, and pharmacological interventions that target psychological distress, impulsivity, and FA increase the likelihood of losing weight and alleviating diabetes symptomology in particular individuals.
CHAPTER THREE – PHASE 1

STUDY 1

This study relates to research objectives 1-3, has been completed and published in *Appetite* (impact factor 2.691)

**Food addiction symptomology, impulsivity, mood, and body mass index in people with type 2 diabetes.**

Karren-Lee Raymond* and Geoff P. Lovell

Faculty of Arts and Social Science, University of the Sunshine Coast, Maroochydore, Australia


Available from:

[https://www.researchgate.net/profile/Geoff_Lovell/publication/280602439_Food_Addiction_Symptomology_Impulsivity_Mood_and_Body_Mass_Index_in_People_with_Type_Two_Diabetes/links/5657afb208aefe619b1f3320.pdf](https://www.researchgate.net/profile/Geoff_Lovell/publication/280602439_Food_Addiction_Symptomology_Impulsivity_Mood_and_Body_Mass_Index_in_People_with_Type_Two_Diabetes/links/5657afb208aefe619b1f3320.pdf)

All authors were responsible for research design and methodology; K-LR contribution was 75%. K-LR was solely responsible for data collection. K-LR lead the data analysis, and GPL provided feedback on analysis and interpretation. K-LR’s contribution 75%. K-LR wrote the manuscript; GPL reviewed and provided feedback on the final manuscript.
Food addiction symptomology, impulsivity, mood, and body mass index in people with type two diabetes

Karren-Lee Raymond, Geoff P. Lovell

School of Social Sciences, University of the Sunshine Coast, Maroochydore, Australia

**Abstract**

This research explored how food addiction (FA) and impulsivity (non-planning, motor, and attentional) relate to body mass index (BMI) in a sample of people with type 2 diabetes (T2d). Participants with T2d (N = 334, M_age = 41.0, SD_age = 9.5, 66% female, M_weight = 83.6 kg/m², SD_weight = 8.0 kg/m²) completed an online survey including the Depression Anxiety Stress Scale (DASS-21), the Barratt Impulsiveness Scale (BIS-II), and the Yale Food Addiction Scale (YFAS). Results demonstrated that over 70% of the sample with T2d met the YFAS criteria for FA. Results also demonstrated that participants classified as FA had significantly higher BMI, t (332) = 12.11, p < .001. The food addict classification group also had a significantly higher percentage of obese participants, χ² (2) = 87.1, p < .001, ϕ = .511. Utilizing a cross-sectional design to predict BMI, significant forward stepwise multiple regression demonstrated that FA (β = .286) and impulsivity (non-planning) (β = .286) were significant predictors. In combination FA and impulsivity (non-planning) significantly explained 38% of BMI variance; however depression, anxiety, and stress did not significantly improve the model. These results suggest FA and impulsivity (non-planning) are more salient cross-sectional predictors of BMI in people with T2d, than indices of depression, anxiety, stress, and impulsivity (motor and attentional). These results, implicating FA in the development of obesity, have important ramifications for potential future treatment methods of T2d where FA symptomology could be routinely screened, and if present, treated via addiction models rather than purely attempting to treat the potential consequences of FA.

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with drugs rather than promoting a healthy lifestyle, and an increasingly commonly ‘toxic’ environment: easily accessible, high calorie, nutrient poor, inexpensive, and attractive foods (Swinburn, Sacks, & Ravussin, 2009; WHO, 2015). While acknowledging that there are many interrelated biopsychosocial factors that contribute to the development of t2d, research continues to affirm that obesity is a major modifiable risk factor contributing to the escalating incidence of t2d (Eckel, Grundy, & Zimet, 2005; IDF, 2015; WHO, 2013). WHO (2013) categorises a person as being overweight if their BMI index is \(25 \leq 29.99 \text{ kg/m}^2\), or obese with a BMI \(\geq 30 \text{ kg/m}^2\).

The link between obesity and t2d is of great concern given reports of an ‘obesity epidemic’. Recent global reports estimated that in 2008 approximately one-and-a-half-billion adults were overweight, with an additional half-a-billion adults being obese (Finucane et al., 2011), and that the number of people with higher BMI scores (\(>35\)) have almost doubled between 1995 and 2012 (WHO, 2013). Likewise, international childhood obesity (under 18 years) continues to surge and it is estimated that globally over 42 million children under the age of 5 are obese (WHO, 2013). With regard to Australian data, the prevalence among people aged 18 years and over in Australia who were classified as being overweight or obese rose from 56.3% in 1995 to 61.2% in 2008 (Finucane et al., 2011). Despite these growing trends in obesity, thus far no country has been able to produce a public health model to abate this crisis (Brownell & Cold, 2012; O’Connell, 2011; Swinburn et al., 2009).

2. Factors associated with the development of obesity

Although obesity is generally considered to be caused by excessive food consumption, in reality the aetiology is much more complicated and includes interaction between genetics, ethnicity, and medication side effects, as well as environmental, psychological, economic, social, and political factors (Saelens & Daniels, 2003; Wright & Aронne, 2012). Some of the mostly extensive researched psychological factors found to contribute to obesity include stress, depression, and anxiety (Chen & Qian, 2012; Eyres, Turner, Nowson, & Torres, 2014; Stunkard, Faith, & Allison, 2003). According to recent theory, research has implied that stress is associated with increased cortisol levels (Visceni, Pasqui, Cavazza, Pagotto, & Pasquali, 2009) which can affect appetite (Sinha & Jastreboff, 2013) and eating behaviour (Mouchacca, Abbott, & Ball, 2013). This has shown to be associated with an increased likelihood of obesity (Chen & Qian, 2012; Mouchacca et al., 2013). Research also suggests strong links between depression and obesity; although these results have often been found to differ as a function of gender with depressed females gaining weight, whilst their male counterparts more often lose weight (Stunkard et al., 2003).

Moreover, a recent longitudinal study showed that women with depression were 54% more likely to develop obesity than females who were not depressed (Finn, 2010). In support of a causal relationship where depression leads to obesity, only 27% of overweight or obese females were more likely to develop depression than normal weight women within 5 years (Finn, 2010). Blaine’s (2008) meta-analysis (N=33,000) also supports a causal link between depression and obesity; depressed participants at baseline were observed as being nearly twice more likely to be obese or gain weight at follow-up than non-depressed participants. As well as depression and stress, anxiety has also been observed to be positively associated with increasing BMI (Eyres et al., 2014; Kivimaki et al., 2009; Zhao et al., 2009). However the relationship between anxiety and obesity is complex, for example gender is again implicated as a moderating factor.

The personality trait of impulsivity has gained research attention due to the reported role of impulse control in the self-regulation of certain behaviours, including the moderation of food consumption (Hofmann, Friese, & Roefs, 2009). Having low impulse control is likely to reduce an individual’s ability to regulate the type and amounts of food that they consume, potentially leading to the onset of obesity and the development of other secondary related illnesses (Weygandt et al., 2013). This premise is supported by numerous links between high impulsivity and excessive consumption of substances such as drugs, alcohol, nicotine, and caffeine, along with other addictions (de Wit, 2008). More recently impulsivity has been recognised as a multi-dimensional construct and as such not a singular trait (Muele, 2013). Consequently, Meule (2013) advocates the use of subscales when analysing the relationship between impulsivity and eating behaviours. Muele’s (2013) literature review concluded that only attentional impulsivity was consistently related to overeating, and only a weak relationship was reported between impulsivity (non-planning) and overeating. Murphy, Stoje, and Mackillop (2013) proposed that impulsivity indirectly effects BMI due to its influence on addictive eating behaviours, which is consistent with recent conceptualisations of food addiction (FA).

3. Food addiction

Many individuals’ attempts to reduce weight have been consistently resistant to treatments, possibly due to interventions not addressing underlying causes. It has been proposed that problematic eating behaviours and addiction to refined food symptomatology have clear similarities to the DSM-IV criteria for substance use disorders (Corsica & Pelchat, 2010; Corwin & Grigson, 2009; Ifland et al., 2009). Although somewhat controversial, recent research supports the concept of FA and that it shares many similarities in the behavioural symptomology and neurochemical mechanisms underlying other addictions (Gearhardt, White, Masheb, & Grilo, 2013; Meule, 2013). According to Volkow and Wise (2005), neurological responses to foods are similar to those associated with addictive drugs (e.g., cocaine) in that both these substances activate reward circuits. Repeatedly activating these circuits results in the development of a preference for the particular stimulus and can lead to neurobiological adaptation, eventuating in the behaviour becoming compulsive, further reducing the individual’s control over their food or drug consumption (Volkow & Wise, 2005).

Lenoir, Serre, Cantin, and Ahmed (2007) found that addiction to the sweetness of sugar, surpassed the cocaine reward in cocaine-addicted rats as they consistently chose sucrose over cocaine. Similar to addiction studies, deficiencies in dopamine receptors (D2) have been found in obese individuals (Noble et al., 1994; Wang et al., 2001). Therefore, although the consumption of highly palatable foods is a contributing factor to the development of obesity and other maladies, the addictive nature of these sub-stances suggests that controlling ingestion is not a simple process. Consequently, reducing rates of obesity and thus t2d requires a better understanding of the mechanisms underlying problematic eating behaviours (O’Connell, 2011; Wang et al., 2001). That is not to say an individual’s role of free choice and self-will is negated by the notion of addiction, however exploring this hypothesis of FA may provide an awareness as to why a subgroup of individuals continue to unsuccessfully restrain their eating and experience elevated BMI (Taylor, Curtis, & Davis, 2010). Although there is a large body of previous research examining relationships between impulsivity and other addictions, there is a dearth of research examining the relationship between FA, impulsivity, and obesity; especially for special populations at risk of unhealthy BMI such as those with t2d. A greater understanding of these
relationships could help to determine whether behavioural and pharmacological interventions targeting FA and impulsivity could potentially successfully facilitate weight management, decreasing obesity, and in turn alleviating t2d symptomology for individuals at particular risk.

In response to the saliency of obesity in terms of current trends in t2d and limited previous research, this current study was designed to gain a better understanding of how FA and impulsivity (non-planning, motor, attentional) relate to BMI in a t2d sample. To this end, three specific research objectives were investigated. Firstly, what percentage of a t2d sample fulfil a classification of FA based on the Yale Food Addiction Scale (YFAS; Gearhardt, Corbin, & Brownell, 2009). Secondly, whether BMI significantly differs between people classified by the YFAS as being food addicts and non-food addicts. Lastly, how FA, impulsivity (non-planning, motor, attentional), depression, anxiety, and stress relate to BMI in people with t2d, while controlling for covariation between the assessed variables.

4. Method

4.1 Participants

Three hundred and thirty four participants with t2d completed an on-line survey (males 34.4%, $M_{\text{age}}$ 41.0 yrs, SD$_{\text{age}}$ 9.5, $M_{\text{BMI}}$ 37.6 kg/m$^2$, SD$_{\text{BMI}}$ 8.4 kg/m$^2$, BMI range 18.0 to 58.6 kg/m$^2$). Participants were only eligible if over 18 years of age and with a current t2d diagnosis from a healthcare professional. Of the 384 participants that started the survey 24 withdrew immediately after the informed consent, another 15 withdrew during the survey, four did not have t2d, and a further five did not respond to the diabetes diagnosis question; thus the completion rate was 87%. Participants were recruited internationally through a variety of on-line diabetes related websites and newsletters, including both Diabetes Australia and Diabetes UK. Forty-seven percent of participants were from Australia, 25% from the USA, 23% from the UK, 2% from New Zealand, with remaining participants from Germany, Ireland, Malaysia, Norway, and Spain. Seven percent of the participants were in the normal BMI category ($218624.99$ kg/m$^2$); 11% were categorised as overweight ($225629.99$ kg/m$^2$) and 79% were categorised as obese ($23030$ kg/m$^2$).

4.2 Procedure

Following institutional ethical approval and indication of informed consent, participants completed an online survey set on the SurveyMonkey platform. The survey included the 21 item version of the Depression Anxiety Stress Scale (DASS-21), the Barratt impulsiveness Scale (BIS-II), the Yale Food Addiction Scale (YFAS), and demographic questions.

4.3 Measures

4.3.1 Depression Anxiety Stress Scale (DASS-21)

Depression, anxiety, and stress were measured by the DASS-21 (Lovibond & Lovibond, 1995). The DASS-21 is scored on a four-point Likert scale with scores from 0 (Did not apply to me at all or almost never) to 3 (Applied to me very much, or most of the time or almost always). The range of possible scores for each subscale is 0-21, where a higher score represents a higher level of depression, anxiety, or stress. The DASS-21 has shown adequate internal consistency ($\alpha$ .93), discriminant validity when compared to other measures of depression and anxiety (Henry & Crawford, 2005).

4.3.2 The Barratt Impulsiveness Scale (BIS-II)

The 30 item BIS-11 (Patton, Stanford, & Barratt, 1995) was used to measure impulsivity and the three subscales of attentional impulsiveness (e.g., I don’t “pay attention.”), motor impulsiveness (e.g., “I do things without thinking”), and non-planning (e.g., “I am more interested in the present than the future”). The BIS-11 is scored on a four-point Likert scale with scores from 1 (rarely/never) to 4 (almost always/always). The sum of the scores is the raw impulsiveness measure and the three second order factors are broken down to measure specific aspects of impulsivity. Possible scores for the entire scale range from 30 to 120 where a higher score reflects a higher level of impulsivity. The BIS-11 has shown reliability and validity (Carrillo-de-la-Pena, Otero, & Romero, 1993), and exhibited good internal consistency for the entire scale ($\alpha$ .83), as have the second order factors: attentional impulsiveness, $\alpha$ .74, motor impulsiveness, $\alpha$ .69, and non-planning impulsiveness, $\alpha$ .72 (Patton et al., 1995).

4.3.3 Yale Food Addiction Scale (YFAS)

The YFAS is a self-report 25 item scale designed to measure FA symptomology over the past 12 months (Gearhardt et al. 2009). The YFAS measure focuses on operationalising addictive eating behaviours, which parallel the symptomology of substance dependence criteria, listed in the DSM-IV e TR (American Psychiatric Association; APA, 2000). The seven food criteria include: 1) substance taken in larger amount and for longer period than intended; 2) persistent desire or repeated unsuccessful attempts to quit; 3) much time/activity to obtain, use, and recover; 4) important social, occupational, or recreation activities given up or reduced; 5) use continues despite knowledge of adverse consequences (e.g., failure to fulfill role obligation, use when physically hazardous); 6) tolerance (marked increase in amount; marked decrease in effect); and 7) characteristic withdrawal symptoms; substance taken to relieve withdrawal. In our research both of the YFAS scoring options were used; the dichotomous FA addiction diagnosis or classification version and the continuous version providing a FA symptom count. The continuous score is the total number of symptoms endorsed and ranges from 0 to 7. For a classification of FA to be inferred, respondents needed to have experienced three or more symptoms over the past year, as well as meeting the “clinically significant impairment” criterion. The YFAS has been found to exhibit good internal reliability ($\alpha$ .86), along with good convergent and divergent reliability (Gearhardt et al., 2009), and is considered to be an appropriate tool for assessing eating behaviours (Brunault, Ballon, Gaillard, Reveillere, & Courtois, 2014).

5. Results

SPSS (version 22; SPSS Inc., Chicago, IL, USA) was used for statistical analyses. Data screening was conducted to ensure that appropriate assumptions were met. The internal consistency of all scales and subscales were analyzed and found to be satisfactory. Dependent variables’ descriptive statistics, intercorrelations, and Cronbach alphas are reported in Table 1.

5.1 Food addiction symptomology

In total 70.7% of our t2d sample met the criteria for FA as measured by the YFAS (based on the DSM-IV criteria for substance dependence). This compares to 11.6% reported for non-diabetic samples (Gearhardt et al. 2009) and Pursey, Stanwell, Gearhardt, Collins, and Burrows's (2014) 19.9% weighted mean prevalence. As shown in Table 2, the current sample demonstrated substantially higher YFAS symptom counts than the general ‘norm’ non-t2d samples (Gearhardt et al. 2009).
were; tolerance withdrawal, variables are correlated, as all variance in BMI (see Table 3). While multicollinearity is always a concern when motor, and attentional), depression, anxiety, and stress made on dependent unique contributions of FA, impulsivity (non-planning, than the non FA group.

5.3. **BMI differences**

Independent t-test revealed that participants classified as meeting FA classification had significantly higher BMI ($M_{\text{FA}}$ 40.4 kg/m$^2$; $SD_{\text{FA}}$ 6.7 kg/m$^2$; $n_{\text{FA}}$ 246) than those that did not meet the criteria ($M_{\text{NonFA}}$ 30.3 kg/m$^2$, $SD_{\text{NonFA}}$ 6.5 kg/m$^2$; $n_{\text{NonFA}}$ 98), t (332) 12.11, p < .001. The effect size for this difference was considered large, $d$ $\approx$ 4.7 (Cohen, 1988). Cross tabulation analysis of BMI classification groups (normal: 18.5 to 24.99 kg/m$^2$; overweight: 25 to 29.99 kg/m$^2$; and obese 30 kg/m$^2$) by FA classification was significant, $\chi^2$ (2) 87.1, p < .001, phi .511, with a large effect (Cohen, 1988). As shown in Fig. 1, a far greater percentage of the FA classification group were categorised as obese (BMI 250 kg/m$^2$) than the non FA group.

53. **Predictors of BMI**

Adopting a cross-sectional predictive design, forward stepwise multiple regression analysis was calculated to evaluate the independent unique contributions of FA, impulsivity (non-planning, motor, and attentional), depression, anxiety, and stress made on BMI (see Table 3). While multicollinearity is always a concern when variables are correlated, as all variance inflation factor (VIF) scores were well below 10 with tolerance statistics all above 2, we confidently conclude that there were no collinearity within our data (see Field, 2013). In combination, FA symptomatology and impulsivity (non-planning), accounted for a significant 38% of the variance in BMI scores in our sample of participants with t2d, $R^2_{\text{FA, Impulsivity}} = .381$, adjusted $R^2_{\text{FA, Impulsivity}} = .377$, F (2,331) 101.73, p < .001. Cohen's $f^2$ (.62) indicated this effect to be large (Cohen, 1988). FA symptomatology made the biggest significant unique contribution to BMI ($b_{\text{FA}}$ .868), followed by impulsivity (non-planning) (.428). Following FA symptomatology and impulsivity (non-planning); depression, anxiety, stress, impulsivity (attentional), and impulsivity (motor) were not significant predictors of BMI and did not significantly improve the predictability of the model (see Table 3).

6. Discussion

Despite the recognition that obesity is a growing global epidemic, further research is needed to clarify the underlying factors contributing to the ongoing development of this crisis. Although endogenous and exogenous factors are implicated in the development of obesity, our research focussed on internal psychological factors. Three factors which have gained considerable attention in their relationship with obesity are depression, anxiety, and stress (Kivimäki et al. 2009); whilst two further emerging concepts as potential contributors to the development of obesity are FA and impulsivity. The current study sought to examine how FA and impulsivity (non-planning, motor, and attentional) relate to BMI in a t2d sample, whilst also considering depression, anxiety, and stress.

Results of the first research objective demonstrated that a substantial proportion of our sample of people with t2d, over 70%, met the criteria for FA symptomatology. This discovery is novel and compares to reports of 11.4% (Gearhardt et al., 2009) and 19.9% (Pursey, Stanwell, Gearhardt, Collins, & Burrows, 2014) of people in the general population. Symptomology showing the greatest disparity between the t2d population and the general population described by Gearhardt et al. (2009) were; tolerance withdrawal, and clinically significant impairment. Furthermore, almost all the participants with t2d in our sample reported unsuccessful attempts in reducing their intake of sweet, starchy, salty, and fatty foods/
drinks. These results demonstrate an association between FA symptomology and type 2 diabetes.

Research objective two examined BMI differences between participants with type 2 diabetes who met the YFAS criteria for FA and those who did not. The first finding demonstrated a large and significant difference in mean BMI between those that met the criteria in this sample for FA, and those who did not. Second, utilizing WHO obesity classifications (WHO, 2014), the group classified as meeting FA criteria comprised of significantly more obese individuals. This suggests that meeting the YFAS criteria for FA is implicated with being obese; an important mediating risk factor for type 2 diabetes.

The third research objective indicated that the variables of FA, impulsivity (attentional, motor and non-planning), depression, anxiety, and stress were all significantly related to BMI, however, FA and non-planning impulsivity demonstrated the strongest relationships with BMI. Furthermore, the significant multiple regression analysis model of FA and non-planning impulsivity predicting BMI (accounting for 38% of BMI variance) was not significantly improved by the inclusion of depression, anxiety, stress, impulsivity (motor and attentional). While there was evidence that multicollinearity was not present in the data, because type 2 diabetes is heavily confounded with morbid obesity caution is needed in the interpretation that type 2 diabetes and FA are directly associated. Although there was a strong correlation between FA and non-planning impulsivity, the finding that impulsivity (non-planning) added to the predictive ability of the model suggests that impulsivity (non-planning) is oblique to FA. Meule’s review of literature (2013) detailed numerous studies which found relationships between attentional impulsivity and overeating, but not between impulsivity and BMI. This is in contrast to the findings of our study, where BMI had a strong positive relationship with impulsivity. With regards to an explanation of this difference, we suggest this be related to the particular characteristics of the samples drawn. We suggest that there may be differences across special populations, further supporting the need for additional research in this area. Moreover, the discovery that FA and non-planning impulsivity were stronger predictors of BMI, and, that their inclusion in this model negated the need to consider depression, anxiety, and stress are novel findings.

These current results, implicating FA in the development of obesity, have important ramifications for potential future treatments of type 2 diabetes. Addiction has additional facets than simply being unwilling or lacking the willpower to abstain or reduce the use of addictive substances. Consequently, the 70% of our sample with type 2 diabetes who met the criteria for FA may not be benefiting from current obesity treatments and interventions; historically generally focused on encouraging dietary and lifestyle changes, and in some cases the use of pharmacological treatments and gastric surgery. Simply instructing a food addict to change their diet without considering the underlying addictive elements is unlikely to be successful at reducing or stopping their use of the addictive substance (Ifland et al., 2009; Burmeister, Hinman, Koball, Coffmann, & Carels, 2013). Accordingly, it may be more productive and constructive to routinely screen people with type 2 diabetes for FA symptomology, and if present, focus treatments on FA, rather than purely attempting to treat the potential consequences of FA (ie obesity, and type 2 diabetes). Moreover, it is important to contemplate the possibility that FA caused the obesity leading to the onset of type 2 diabetes, hence, potentially screening for FA symptomology could improve weight management efforts with this population, which in turn could potentially lessen the burden of preventable type 2 diabetes related illnesses on the healthcare system as well as potential suffering for the individual.

Previous theories and traditional methods including psychosocial models, individual or group therapy, and CBT in the treatment of obesity and eating disorders are now being challenged with additional contemporary theories and concepts such as FA. Recent research approaches to FA have included: animal models (Avena, Rada, & Hoebel, 2009); biochemical models (Volkow & Wise, 2005; Wang et al., 2001); neurological theories (Blumenthal & Gold, 2010; Wang, Volkow, Thanos, & Fowler, 2009); standardised test batteries (YFAS, Gearhardt et al., 2009), and addiction models (Ifland et al., 2009). These recent avenues of research have produced empirical findings supporting the concept of FA. This convergence of knowledgeable and empirical assessment of FA as a theory establishes greater scientific evidence that FA is also within the chemical dependency domain.

Former and continued research paves the way for suggesting that there are greater risks in treating just the obesity aspect and not looking at the whole picture; while with some individual success, traditional approaches to the treatment of obesity have agreeably had limited impact on the world obesity epidemic; especially in certain special populations. In particular, our study demonstrated a significant number of people with type 2 diabetes also had high symptomology of FA; further investigations regarding the relationship between FA and BMI with non-diabetic and prediabetic samples could shed further light, and aid in determining the significance of the FA association with the type 2 diabetes population.

This study has limitations. Firstly, findings of cross sectional studies are associations and do not infer causality. The second limitation pertains to the use of self-report measures. As many of our participants were overweight; having to provide information relating to their weight and height may have been somewhat confronting, which may have led to an underestimation of body weight (Conner Gorber, Tremblay, Moher, & Gorber, 2007; Fairburn & Beglin, 1994) as well as socially biased distortions of other assessed variables. Another consideration of this research is related to gender differences with a larger number of female participants than male participants. Forthcoming research focused upon identifying any potential gender differences would appear well.
warranted. The lack of recorded race or cultural background data also presents as a limitation; future research should include such valuables in subsequent analyses. A further consideration relates to the lack of data available relating to participants’ use of medication. Our sample had extremely high levels of depression and anxiety. One commonly used form of pharmacological treatments for depression and anxiety are tricyclic antidepressants (van Reedt Dortland et al., 2013). Metabolic syndrome abnormalities have been found to be associated with tricyclic antidepressant use, hence if participants were taking tricyclic antidepressant it could have affected BMI results (van Reedt Dortland, Gillay, van Veen, Zitman, & Penninx 2010). Lastly if participant were taking specific weight loss medications, this may also have had some influence on this research.

7. Conclusions

The results of the current investigation compliment prior research within the FA realm, adding substantially new factors to be explored and considered in helping to further understanding obesity, especially in people with t2d. The results of the current investigation have shown a very large percentage of people with t2d reporting high levels of FA symptomology. A large difference in BMI between people with t2d who were classified as food addicts and non-food addicts was also observed. This research clearly implicates FA in elevated BMI and consequently the risk of developing t2d. Almost three quarters of our sample of people with t2d met the criteria for FA; much higher than the percentage of FA reported in the non-diabetic populations (Pedram et al., 2013). Based on this finding alone, we argue that it is important for healthcare professionals to address and routinely screen for FA symptomology using tools such as the YFAS, when consulting people with t2d. Simply telling people who meet the criteria for FA to lose weight, in order to decrease their t2d symptomology, is unlikely to be widely effective and may explain why current treatment methods have had limited effect. We hope that this research may provide insight into a previously unexplored link between FA symptomology and BMI in people with t2d, leading to improved treatment outcomes globally.

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References


CHAPTER FOUR – PHASE 1
STUDY 2

This study relates to research objectives 4 and 5, has been completed and published in *Journal of Diabetes and Its Complications*, (impact factor 3.005)

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**Food addiction associations with psychological distress among people with type 2 diabetes.**

Karren-Lee Raymond* and Geoff P. Lovell

Faulty of Arts and Social Science, University of the Sunshine Coast, Maroochydore, Australia


Available from:

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**STUDY 2.**

All authors were responsible for research design and methodology; K-LR contribution was 75%. K-LR was solely responsible for data collection. K-LR lead the data analysis, and GPL provided feedback on analysis and interpretation. K-LR’s contribution 75%. K-LR wrote the manuscript; GPL reviewed and provided feedback on the final manuscript.
Food addiction associations with psychological distress among people with type 2 diabetes

Karren-Lee Raymond *, Geoff P. Lovell 1
Faculty of Arts and Social Sciences, University of the Sunshine Coast, Maroochydore, Australia

1. Introduction

Type 2 diabetes is a major international health crisis with its associated high morbidity and mortality rates. Reflecting an increase in awareness that people with t2d are confronted with a range of biopsychosocial challenges, recent research has documented strong associations between t2d and poor mental health (Bener, Al-Hamaq, & Dafeelah, 2011; Mezuk, Eaton, Albrecht, & Golden, 2008). While the direction of a causal relationship between having t2d and experiencing poor mental health, such as depression, is still somewhat contentious, it is likely to be a bidirectional relationship. Higher levels of depression, anxiety, and stress associated with t2d not only present in themselves as poor health, but may also increase the risk of potential diabetes complications. Such potential complications may include increased insulin resistance (Sinha & Jastreboff, 2013) or increased risk of cognitive decline and Alzheimer’s disease (Feinkohl, Price, Strachan, & Frier, 2015). Additional complications of t2d with depression stem from the reported higher incidences of unhealthy behaviours, such as smoking, physical inactivity, unhealthy diet, and poor glycaemic management compliance (Lin et al., 2004). Indeed, dating back to 1864 Thomas Willis – a British Physician, implied that diabetes ensued from ‘sadness or long sorrow and other depressions and disorders’ (Willis, 1791).

While increased psychological distress appears to be associated with t2d, current empirically evidenced understandings of why people with t2d have such poor psychological well-being is somewhat limited (Rubin & Peyrot, 2002). Much of the previous research in this area has focused on biological and obesity related explanations of poor mental health (e.g., variations in glycaemic levels, neurological alterations in metabolism of serotonin and norepinephrine, trophic agent changes, biochemical changes associated with increased dyslipidemia, and stigmatisation of obesity) (Lustman et al., 2000; Miller, Maletic, & Raison, 2009; Sutin & Terracciano, 2013; Valabhji & Elkeles, 2003). Indeed, research continues to demonstrate that obesity is one of the strongest predictors of depression in t2d samples, as well as non-type 2 diabetes populations, with risk of depression being 20% to 50% higher among obese individuals than normal weight individuals (Mather, Cox, Enns, & Sareen, 2009; Simon et al., 2006; Svenningsson, Björkelund, Marklund, & Gedda, 2012). Furthermore, these risks are reported to be even higher for extremely obese individuals. (Abilés et al., 2010; Petry, Barry, Pietrzak, & Wagner, 2008).

Despite the gains that such biological and obesity orientated research have made to the understanding of psychological distress in populations with t2d, we are still presented with large variances in...
psychological distress that remain unaccounted for. This gap in our knowledge compromises the design of evidence based interventions to provide effective care to ameliorate psychological distress associated with t2d. This highlights the need to consider new approaches to the understanding of psychological well-being in persons with t2d.

1.1. Food addiction model

One such approach to explaining the high levels of depression, anxiety, and stress in people with t2d is to consider an addiction model, or, more specifically, a ‘food addiction’ (FA) model (Corwin & Grigson, 2009). The concept of FA parallels that of substance abuse in terms of mechanism and associated negative consequences characterised by the classic symptoms of addiction: tolerance and withdrawal (Ball & Brownell, 2009). Food addiction behaviours associated with highly processed and hyperpalatable foods (typically refined carbohydrates, sweeteners, fats, and processed foods) have been shown to mimic the DSM-IV–TR, (American Psychiatric Association, 2000, 4th ed., text rev.) criteria for substance use disorders (Corsica & Pelchat, 2010; Gearhardt, Davis, Kuschner, & Brownell, 2011). It should also be noted that there has been reported overlap between FA and other ways of conceptualizing disordered eating; especially binge eating disorder where strong relationships between binge eating and FA as well as parallel relationships between these constructs and distress, poor weight loss, have been observed (Burmeister, Hinman, Koball, Hoffman, & Carels, 2013).

While the FA model has been a contentious topic (Ziauddeen, Farooqi, & Fletcher, 2012), evidence suggests it has many similarities in the neurochemical mechanisms and behavioural symptomology underlying other substances of abuse (Gearhardt, White, Masheb, & Grilo, 2013; Volkow, Wang, Tomasi, & Baler, 2013). Of key importance to t2d and a FA model, substance abuse has been linked to psychological distress and obesity (Martins & Gorelick, 2011; Tolliver & Anton, 2015), both of which separately and together have been linked to t2d (Furuya, Hayashino, Tsuji, Ishii, & Fukuhara, 2013; Kaur, Tee, Ariaratnam, Krishnapillai, & China, 2013; Svenningsson et al., 2012).

Previously measuring a person’s FA symptomology or eating related problems has been challenging. Issues have been encountered such as appraising whether an addictive element applies to disordered eating (Schulte, Joyner, Potenza, Grilo, & Gearhardt, 2015). Currently, the most generally accepted measure for assessing FA is the Yale Food Addiction Scale (YFAS; Gearhardt, Corbin, & Brownell, 2009). The YFAS was introduced as a valid and reliable self-report measurement for the assessment of FA symptomology paralleling the DSM-IV-TR Substance Disorder criteria (Gearhardt et al., 2009; Muele & Gearhardt, 2014). Pedram et al. (2013) utilised the YFAS investigating the degree that the FA framework contributed to obesity among a general populous. Results demonstrated that FA was a key player; over 80% of food addicted people were overweight/obese suggesting the FA phenomenon is significantly contributing to the fast-growing obeseogenic climate.

1.2. Substance abuse, psychological distress, and type 2 diabetes

Earlier research has shown a relationship between various forms of substance use disorders and psychological distress (Martins & Gorelick, 2011; Pettinati, O’Brien, & Dundon, 2013; Tolliver & Anton, 2015). Results from the large scale National Epidemiologic Survey on Alcohol and Related Conditions (NESARC), covering the co-morbidity of DSM-IV substance use disorders and nine separate mood and anxiety disorders (n = 43,093), found that 60 percent of the participants seeking treatment for any drug use disorder in the past year had a mood disorder and 43 percent presented with at least one anxiety disorder (Grant et al., 2004). Additionally, a recent literature review demonstrated over one-third of alcohol dependence was associated with other mental illnesses (Klimkiewicz, Klimkiewicz, Jakubczyk, Kieres-Salomofski, & Wojnar, 2015). Martins and Gorelick (2011) highlighted substance use over the lifespan was higher in participants with psychiatric illness than in participants without a disorder.

Specifically, nicotine dependence and active smoking have been recognised among people with t2d (Pan, Wang, Talaei, Hu, & Wu, 2015; Willi, Bodenman, Ghali, Faris, & Cornuz, 2007) and mood disorders including depression was shown to be of greater intensity amongst nicotine addicts than non-smokers (Pietras, Witusik, Panek, Szemraj, & Górski, 2011). Furthermore, recent research indicates that people with t2d and with a diagnosed substance abuse disorder are twice as likely to have a psychiatric mood disorder (Wu et al., 2015). While Wu et al. (2015) research didn’t consider FA, their conclusions do provide further evidence that people with t2d and FA symptomology are likely to have an increased risk of psychological distress.

Taken together previous research evidences high associations between psychological distress and substance abuse. Although research has examined the relationship between alcohol, tobacco, and drug substance abuse with psychological distress among people with t2d, no research has considered the association between psychological distress and a FA model in this population. This is despite recent research demonstrating that over 70% of people with t2d met a FA criteria (Raymond & Lovell, 2015). Hence the current study aimed to assess the relationship between a FA model and psychological distress among a t2d sample. Specifically, two research objectives were approached: firstly, whether depression, anxiety, and stress scores significantly differed between participants who met a FA criterion and those that did not. Secondly, to compare the cross-sectional predictive contributions to depression, anxiety, and stress scores made by FA and body mass index (BMI), while controlling for covariation between the assessed variables. Positive findings to support such hypotheses would have substantial implications for the design of interventions to provide care for people with t2d and the enhancement of their psychological well-being.

2. Methods

This study was approved by the University of the Sunshine Coast Ethics committee.

2.1. Participants

An online survey set on the SurveyMonkey platform recruited 381 participants. Twenty-four participants withdrew immediately after the informed consent section, 15 participants withdrew during the survey, nine participants did not confirm that they had a current t2d diagnosis, leaving a total of 334 participants. The final sample included 221 females (65.6%) and 110 males (32.9%; five participants did not indicate their gender). Ages ranged from 27 to 81 years (M = 58.0, SD = 9.5). Data from this sample have been published previously which demonstrated over 70% of people with t2d met the YFAS criteria for FA, and highlighted the FA classification group had significantly higher BMI scores. Furthermore FA and impulsivity (non-planning) were significant predictors of BMI (Raymond & Lovell, 2015).

The majority of participants were recruited from Australia (47%), with the remaining 53% participants from: USA (25.1%), UK (23.1%), New Zealand (1.8%), Germany (0.6%), Ireland (0.3%), Malaysia (0.6%), Norway (0.3%), and Spain (0.3%), with 0.6% not reporting their domicile. Participants’ weight ranged from 46 – 184 kg, Mweight = 106.88 kg, SDweight = 25.59 kg; Height ranged from 147 – 194 cm, Mheight = 168.66 cm, SDheight = 9.06 cm; BMI ranged from 18.9 – 58.6 kg/m², MBMI = 37.6 kg/m², SDBMI = 7.99. Seven percent of the participants were in the normal BMI category.
(≥ 18-24.99 kg/m²), 11% were categorised as overweight (≥25-29.99 kg/m²), and 79% were categorised as obese (≥30 kg/m²).

3. Procedure

Following institutional ethical approval and confirmation of informed consent, participants over 18 years of age with formalised diagnosis of t2d from a healthcare professional completed the online survey. Participants completed a self-report questionnaire, which included demographic questions regarding t2d status, age, gender, country of residence, and two separate test batteries: the Depression Anxiety Stress Scale (DASS-21) and the YFAS. Participant recruitment was achieved via Facebook, placing advertisements and flyers on message boards in medical practices and gymnasiums, and diabetes websites. Diabetes Australia placed a research advertisement on their website for the duration of the research, and Diabetes UK included an advertisement in their magazine. Potential participants received a Research Project Information Sheet detailing the aims of the research, which entailed exploring FA and psychological distress associations in a t2d sample.

4. Data collection

4.1. Assessment of Depression, anxiety and stress

The Depression Anxiety Stress Scale (DASS-21) was utilised to measure depression, anxiety, and stress, (Lovibond & Lovibond, 1995). The DASS-21 is scored on a four-point scale with scores from 0 (Did not apply to me at all – never) to 3 (Applied to me very much, or most of the time – almost always). The range of possible scores for each of the three 7-item subscales is 0-21, where a higher score represents a higher level of depression, anxiety, or stress. The DASS-21 has shown adequate internal consistency (α = .93), discriminant validity, as well as satisfactory convergent validity when compared to other measures of depression and anxiety (Henry & Crawford, 2005).

4.2. Assessment of food addiction symptomology

The Yale Food Addiction Scale was utilised to assess FA symptomology, a self-report 25-item scale designed to measure FA symptomology over the past 12 months (Gearhardt et al., 2009). The YFAS measure focuses on operationalising addictive eating behaviours, which parallel the symptomology of substance dependence criteria, listed in the DSM-IV-TR (American Psychiatric Association; APA, 2000). The seven food criteria include: 1) substance taken in larger amount and for longer period than intended; 2) persistent desire or repeated unsuccessful attempt to quit; 3) much time/activity to obtain, use, and recover; 4) important social, occupational, or recreation activities given up or reduced; 5) use continues despite knowledge of adverse consequences (e.g., failure to fulfil role obligation, use when physically hazardous); 6) tolerance (marked increase in amount; marked decrease in effect); and 7) characteristic withdrawal symptoms; substance taken to relieve withdrawal. The YFAS scoring provides a dichotomous FA addiction classification and a continuous FA symptom count score. The continuous score is the total number of symptoms endorsed and ranges from 0 to 7. For a classification of FA to be inferred, respondents needed to have experienced three or more symptoms over the past year, as well as meeting the “clinically significant impairment” criterion. The YFAS has been found to exhibit good internal reliability (α = .86), along with good convergent and divergent reliability (Brunault, Ballon, Gaillard, Réveillère, & Courtois, 2014), and is considered to be an appropriate tool for assessing eating behaviours (Muele & Gearhardt, 2014).

4.3. Data analysis and statistical methods

Descriptive statistics - One-sample t-tests were employed to compare the depression, anxiety, and stress scores of participants against Crawford, Cayley, Lovibond, Wilson, and Hartley’s (2011) Australian general adult population norm scores. Research objective one (employing the YFAS) was investigated by a one-way between-groups multivariate analysis of variance (MANOVA) examining the dependent variables of depression, anxiety, and stress with FA criteria (2 levels): i) meeting the criterion for FA diagnosis; ii) not meeting the FA criterion for diagnosis representing the independent variable. Research objective two (to explore the variables of BMI and FA as significant predictors of depression, anxiety and stress scores in people with t2d) was investigated by three multiple regression analyses.

5. Results

SPSS (version 20; SPSS Inc., Chicago, IL, USA) was used for statistical analyses. Data screening was conducted to ensure that appropriate assumptions were met with no serious violations noted for any of the reported analyses. The internal consistency of all scales and subscales was analyzed. Sample statistics, variables’ descriptive statistics, and intercorrelations are reported in Table 1.

5.1. Descriptive statistics

One-sample t-tests were used to compare the depression, anxiety, and stress scores of our sample of participants with t2d against Crawford et al.’s (2011) Australian general adult population norms. Our sample of people with t2d had significantly and meaningfully higher scores on each of the DASS-21 subscales than Crawford et al.’s (2011) Australian norms: depression, t (333) = 27.59 p b .001, d = 2.30 (Cohen, 1988); anxiety, t (333) = 24.56 p b .001, d = 2.56 (Cohen, 1988); and stress, t (333) = 25.04 p b .001, d = 2.74 (Cohen, 1988). As shown in Table 2, approximately three quarters of the sample had higher than normal scores across the depression, anxiety, and stress subscales. Overall, the anxiety subscale demonstrated the most elevated scores, with almost half of the sample having scores in the extremely severe range. Similarly, well over a third of the sample had scores in the extremely severe range for the depression subscale.

5.2. Depression, Anxiety, and Stress Differences Between Food Addiction Classifications

Research objective 1 examined whether depression, anxiety, and stress scores significantly differed between people with t2d who met the criterion for FA and people with t2d who did not meet this criterion. To assess this, a one-way between-groups MANOVA was performed on the dependent variables of depression, anxiety, and stress with FA classification (meeting the criterion for FA diagnosis or not) representing the independent variable. Results of the MANOVA demonstrated a main effect of FA, F (3, 330) = 135.53, b.001, Wilks

<table>
<thead>
<tr>
<th>Measure</th>
<th>M</th>
<th>SD</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. BMI (kg/m²)</td>
<td>-</td>
<td>37.6</td>
<td>8.0</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2. DASS-21 A</td>
<td>.90</td>
<td>8.8</td>
<td>5.2</td>
<td>.53**</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>3. DASS-21 S</td>
<td>.91</td>
<td>10.6</td>
<td>4.9</td>
<td>.48**</td>
<td>.89**</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>4. DASS-21 D</td>
<td>.94</td>
<td>11.2</td>
<td>5.7</td>
<td>.53**</td>
<td>.83**</td>
<td>.86**</td>
<td>-</td>
</tr>
<tr>
<td>5. YFAS</td>
<td>.98</td>
<td>4.7</td>
<td>2.2</td>
<td>.50**</td>
<td>.78**</td>
<td>.76**</td>
<td>.79**</td>
</tr>
</tbody>
</table>

Note. ** p b .001
Table 2
Percentage of Sample of People with Type 2 Diabetes Classified by DASS-21 Severity Categories.

<table>
<thead>
<tr>
<th>Classification</th>
<th>Depression (%)</th>
<th>Anxiety (%)</th>
<th>Stress (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>18.6%</td>
<td>21.5%</td>
<td>26.9%</td>
</tr>
<tr>
<td>Mild</td>
<td>5.2%</td>
<td>9.1%</td>
<td>8.2%</td>
</tr>
<tr>
<td>Moderate</td>
<td>11.9%</td>
<td>7.3%</td>
<td>26.0%</td>
</tr>
<tr>
<td>Severe</td>
<td>22.9%</td>
<td>13.0%</td>
<td>29.9%</td>
</tr>
<tr>
<td>Extremely severe</td>
<td>41.5%</td>
<td>49.1%</td>
<td>9.1%</td>
</tr>
</tbody>
</table>

Lambda = .45, $n^2_f = .55$, with this being a large effect size (Cohen, 1988). Subsequent univariate tests of the same design demonstrated that those meeting the criterion for FA diagnosis had significantly higher levels of depression, $F(1, 332) = 336.29, p < .001, \eta^2_p = .50$; anxiety, $F(1, 332) = 336.86, p < .001, \eta^2_p = .50$; and stress, $F(1, 332) = 282.52, p < .001, \eta^2_p = .46$, than those who did not meet the criterion. As shown in Fig. 1, the mean scores tripled for anxiety, and significantly doubled for depression and stress when compared to those in the sample whom did not meet the FA criteria.

5.3. Predictors of Depression, Anxiety and Stress

Objective 2 aimed to determine what extent BMI and FA symptomology were significant cross-sectional predictors of depression, anxiety, and stress scores among people with t2d. To estimate the proportion of variance in depression, anxiety, and stress scores that could be accounted for by the predictor variables of BMI and FA, three separate simultaneous multiple regression analyses were performed. While multicollinearity is always a concern when variables are correlated, as all variance inflation factor (VIF) scores were well below 10 with tolerance statistics all above 2, we confidently conclude that there were no collinearity within our data.

5.3.1. Depression

Results of the multiple regression demonstrated that the overall model was significant and that in combination, BMI and FA accounted for a 63% of the variation in participants’ depression scores, $R^2 = .63$, adjusted $R^2 = .62$, $F(2, 331) = 276.22, p < .001$. This represented a large effect size. Food addiction made a substantially larger unique contribution to depression scores than BMI, with 35% of the unique variation in depression scores being explained by FA ($\beta = .73, t = 17.57, p < .05, sr^2 = 35\%$) as compared to 1% of the variation being explained by BMI ($\beta = .10, t = 2.53, p < .05, sr^2 = 1\%$).

5.3.2. Anxiety

Results of the multiple regression showed that the overall model for anxiety was significant, demonstrating that BMI and FA in combination accounted for 62% of the variance in participants’ anxiety scores, $R^2 = .62$, adjusted $R^2 = .62$, $F(2, 331) = 267.05, p < .001$. An overall large effect size was observed. FA was again observed to make a far larger unique contribution to explaining variation in anxiety scores than BMI, with 34% of the unique variation in anxiety scores being explained by FA ($\beta = .72, t = 17.18, p < .001, sr^2 = 34\%$) and 1% of the unique variation explained by BMI ($\beta = .11, t = 2.64, p < .01, sr^2 = 1\%$).

5.3.3. Stress

The multiple regression demonstrated that, in combination, BMI and FA significantly predicted stress scores, together accounting for 58% of the variance in stress scores, $R^2 = .58$, adjusted $R^2 = .57$, $F(2, 331) = 225.43, p < .001$. An overall large effect size was observed. Moreover, 34% of the unique variation in stress scores was explained by FA ($\beta = .72, t = 16.39, p < .001, sr^2 = 34\%$), while BMI failed to account for a significant proportion of unique variance in stress scores ($\beta = .07, t = 1.49, p = .138, sr^2 < 1\%$).

6. Discussion

This study aimed to assess the association between FA model and psychological distress in a t2d sample. The results supported those already evidenced in previous research that individuals with t2d are at substantial risk of heightened depression, anxiety, and stress (Bener et al., 2011). Our data demonstrated that three quarters of the participants had depression, anxiety, and stress levels beyond the normal range. With regard to our study, novel yet significant results relating to the first research objective, showed that people with t2d who met the YFAS criterion for FA reported significantly higher depression, anxiety, and stress scores than people who did not meet the criterion. The results relating to the second research objective highlight the potential importance of FA in the etiology of psychological distress in people with t2d and the unique contribution of FA to predicting depression, anxiety, and stress scores was substantially larger than that made by BMI (which accounted for around 1% of the unique variation in scores once FA was factored in). This novel finding suggests that implementing a FA model in treatment, prior to or in tandem with BMI and other treatment frameworks for t2d, may well produce substantially greater effects upon individuals’ psychological well-being outcomes.

The current study’s results mirror findings regarding other substances of abuse. The relationship between increased psychological distress and FA symptomatology found in our study expands on the already existing empirical evidence base typically demonstrating co-occurring substance use disorders and psychological distress (Martins & Gorelick, 2011; Tolliver & Anton, 2015). Moreover, this finding stimulates and justifies advocacy for clinical research regarding the routine screening of people with t2d for FA symptomatology, implementing standardised self-report instruments such as the YFAS (Gearhardt et al., 2009; Schulte et al., 2015). Furthermore, this development and implementation of programs to ameliorate psychological distress in people with t2d may consider adopting a FA model which involves a recovery treatment plan; for example Sheppard (Sheppard, 2000). Likewise, potential care interventions to alleviate psychological distress and obesity based on a FA addiction model also borrow from our colleagues working with traditional addictions. Accepted foundations of addiction interventions include the Minnesota Model (Lefever, 2000), outpatient treatments, and...
employing a FA model when treating psychological distress among people with t2d.

Acknowledgments

We would like to acknowledge Diabetes Australia and Diabetes UK for their valuable support with participant recruitment.

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CHAPTER FIVE – PHASE 2
STUDY 3

This study relates to research objectives 6 and 7, has been completed and
published in Journal of Health Psychology,
(impact factor 2.010)

A graduated food addiction classification approach significantly
differentiates obesity among people with type 2 diabetes.

Karren-Lee Raymond, Lee Kannis-Dyman and Geoff P. Lovell

Faculty of Arts and Social Science, University of the Sunshine Coast, Maroochydore, Australia


Available from:


All authors were responsible for research design and methodology; K-LR contribution was 75%. K-LR was solely responsible for data collection. K-LR lead the data analysis, and GPL provided feedback on analysis and interpretation. K-LR’s contribution 75%. K-LR wrote the manuscript; GPL and LK-D reviewed and provided feedback on the final manuscript.
A graduated food addiction classification approach significantly differentiates obesity among people with type 2 diabetes

Karren-Lee Raymond, Lee Kannis-Dyman and Geoff P Lovell

Abstract
This study examined a graduated severity level approach to food addiction classification against associations with World Health Organization obesity classifications (body mass index, kg/m²) among 408 people with type 2 diabetes. A survey including the Yale Food Addiction Scale and several demographic questions demonstrated four distinct Yale Food Addiction Scale symptom severity groups (in line with Diagnostic and Statistical Manual of Mental Disorders (5th ed.) severity indicators): non-food addiction, mild food addiction, moderate food addiction and severe food addiction. Analysis of variance with post hoc tests demonstrated each severity classification group was significantly different in body mass index, with each grouping being associated with increased World Health Organization obesity classifications. These findings have implications for diagnosing food addiction and implementing treatment and prevention methodologies of obesity among people with type 2 diabetes.

Keywords
food addiction, obesity, processed food use disorder, severity levels, type 2 diabetes

Introduction
Obesity is a global challenge faced by most developed nations as well as many under developed nations. It was estimated that in 2008 approximately one-and-a-half-billion adults were overweight (body mass index (BMI) $\geq 25$ kg/m²), with an additional half-a-billion adults obese (Finucane et al., 2011). While acknowledging that there are many interrelated biopsychosocial factors that contribute to the development of poor health, research consistently affirms obesity as major modifiable risk factor contributing to an escalating incidence of a range of health conditions including hypertension, respiratory complications, heart disease and stroke, and type 2 diabetes (t2d) (WHO, 2013).

The University of the Sunshine Coast, Australia

Corresponding author:
Karren-Lee Raymond, School of Social Sciences, The University of the Sunshine Coast, Locked bag 4 Maroochydore, QLD 4558, Australia.
Email: karren-lee.raymond@research.usc.edu.au
Obesity

Marks (2015b), in his editorial to the Journal of Health Psychology’s special issue on food diets and dieting, highlights the importance of answers to some basic questions including: what is causing the obesity epidemic, and what can be done about it? Obesity has often been stigmatised (Puhl and Heuer, 2009) and stereotype associated with weakness, lack of willpower or simply gluttony without a biological foundation in today’s society (Harper and Carels, 2014). Fortunately, and of late, there has been a greater acceptance that obesity is much more complex with biological, social and psychological aspects including interaction between genetics, ethnicity, medication side effects, as well as environmental, psychological, economic, social and political determinants (Saelens and Daniels, 2003; Wright and Aronne, 2012). Despite these advances in our understanding and the many strategies and methodologies proposed to abate the obesity pandemic (Swinburn et al., 2011), ‘progress has been frustratingly slow’ (Marks, 2015b: 471). Clearly, if obesity prevention is to be approached in ways other than via predicted food and water shortages, policy level as well as individual level developments are needed. Specifically focussing upon person level approaches, recent research addressing food, diets and dieting are enhancing our understanding, knowledge, insight and direction as to how to tackle this obesity epidemic (Brown et al., 2015; Buchanan and Sheffield, 2015; Calder and Mussap, 2015; Marks, 2015a; Schulte et al., 2015b).

Particularly in the last decade, food addiction (FA) as a valid contributor to obesity (Gearhardt et al., 2013b; Lerma-Cabrera et al., 2016; Raymond and Lovell, 2015) has gained both momentum and recognition as having societal impacts similar to other forms of substance abuse (Avena et al., 2009; Meule and Gearhardt, 2014). However, it is important to note that currently there is little data to evidence a causal relationship between FA and obesity.

FA and substances of abuse

Although somewhat controversial (Ziauddeen et al., 2012), evidence suggests FA is emerging as a distinct construct with behavioural, biological and neurological similarities to alcohol abuse and drug dependence (Blumenthal and Gold, 2010; Noble et al., 1994; Potenza, 2014). Davis and Carter (2009) demonstrated parallels in clinical and behavioural symptoms between compulsive overeating and drug addiction. According to Volkow and Wise (2005), neurological responses to foods are similar to those associated with addictive drugs (e.g. cocaine) in that both these substances activate neural reward circuits. Repeatedly activating these circuits results in the development of a preference for the particular stimulus and can lead to neurobiological adaptation, eventuating in the behaviour becoming compulsive, further reducing the individual’s control over their food or drug consumption.

The practical application of the concept of FA would appear substantial. Although the consumption of highly palatable foods is the key contributing factor to the development of obesity and other complications, the addictive nature of such substances suggests that controlling ingestion is not a simple process (Burmeister et al., 2013). Traditional treatment options for obesity founded on education, diet control coupled with a systemic exercise regime may achieve effective long-term weight loss. However, an individual with an FA diagnosis may find implementing such approaches do not deliver longevity in effective treatment outcomes as only the symptoms of the disease, not the cause (addiction), are treated. Thus, addiction-based treatment approaches for a food addict are more likely to achieve favourable long-term outcomes.

Yale Food Addiction Scale, DSM-IV-TR substance dependence criteria and the DSM-5

A tool widely adopted for FA assessment is the Yale Food Addiction Scale (YFAS), (Gearhardt
et al., 2009). While the design of YFAS was based on the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text rev.; DSM-IV-TR) Substance Dependence criteria (American Psychiatric Association (APA), 2000), it is still consistent with many aspects of the *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; DSM-5) Other (or Unknown) Substance Use Disorder (APA, 2013). The diagnostic criterion for Other (or Unknown) Substance Use Disorder is the problematic pattern of use that leads to clinically significant impairment or distress, occurring within a 12-month period. Similarly, the current YFAS classification of FA requires use that causes clinically significant impairment or distress and at least three symptoms based on the DSM list. The YFAS has shown to be a valid and reliable measure (Pursey et al., 2016), predictive of BMI and general psychological distress (Raymond and Lovell, 2015, 2016). The YFAS has also been used internationally (Meule and Gearhardt, 2014), having been successfully translated into various languages (Meule et al., 2012). A short form has been developed (the Modified Yale Food Addiction Scale, mYFAS; Flint et al., 2014) as well as children’s version (YFAS-C; Gearhardt et al., 2013a).

Despite the evidenced utility of the YFAS and that it already lends itself to providing a symptom count score, the YFAS as typical within the FA domain, usually categorises one either as a food addict or not. We suggest that such a binary approach to the diagnosis of FA limits specificity of treatment and is not consistent with diagnosis of other substance use disorders. With regard to the current inconsistency between FA classification and the current DSM-5 (APA, 2013), while FA is not specifically listed, the Other (or Unknown) Substance Use Disorder (as do other substance use disorders such as alcohol) does specify levels of severity (Mild: presence of 2–3 symptoms; Moderate: presence of 4–5 symptoms; and Severe: presence of 6 or more symptoms). Fundamentally, the current dichotomous approach to FA diagnosis ignores differences in the severity of symptoms recognised in contemporary diagnoses of other substance use disorders. As such, individuals diagnosed with FA could receive the same treatments, while the severity of their symptoms presentation may substantially vary. The result of this blunt diagnosis, potentially being a one-size-fits-all approach, is that treatment is unlikely to be appropriate across the entire range of FA severities. For example, an abstinence approach is unlikely to be necessary or appropriate for someone with mild FA symptomatology, but would be more appropriate for someone with a severe FA diagnosis with secondary complications.

Based on the demonstrated utility of the FA construct in developing theoretical explanations and practical therapeutic approaches to help ameliorate obesogenic challenges at population and individual levels, this research examined the validity of a graduated approach to FA classifications. Adopting current DSM-5-based diagnostic specifiers to classify individuals’ FA symptomatology severity (mild, moderate and severe), we predicted that if such a classification system was valid that in populations shown to exhibit high levels of FA such as those with t2d, there would be significant differences in BMI between FA severity classification groups. Specifically, that BMI will significantly increase from non-food addiction, to mild FA, to moderate FA, to severe FA participant groups. Findings from this research would have global significance both in terms of theoretical and therapeutic implications, and applications for initiatives and approaches to help combat obesity.

**Method**

**Participants**

A total of 408 adults professionally diagnosed with t2d completed an online survey set on the SurveyMonkey platform. Of the 484 participants who started the survey, 17 withdrew immediately after the informed consent section, 10 withdrew during the survey, 45 did not confirm they had a current t2d diagnosis and 4 did not provide height and weight data. The final sample of 408 participants included 270 females (66.2%) and 133 males (32.6%; five participants
did not indicate their gender). Ages ranged from 22 to 81 years (\(M_{\text{age}}=56.2, SD_{\text{age}}=9.3\)).

Participants were recruited through advertisements and flyers at Australian medical centres, through social media including Facebook, Twitter and online diabetes media sites, and letters sent out to medical practitioners internationally including Diabetes Australia and Diabetes UK. Of the final sample, 48.2 per cent from Australia with the remaining 52.8 per cent participants from the United States (25.7%), the United Kingdom (22.5%), New Zealand (1.7%) and other (1.7%; including Germany, Ireland, Malaysia, Norway and Spain); 0.5 per cent did not report their domicile. Participants’ height, weight and BMI are shown in Table 1. One participant (0.2%) had an underweight BMI (16.13kg/m\(^2\)), 9.1 per cent of the participants were in the normal BMI category (≥18–24.99kg/m\(^2\)), 13.7 per cent were categorised as overweight (≥25–29.99kg/m\(^2\)) and 77.0 per cent were categorised as obese (≥30kg/m\(^2\)).

### Results

Statistical analyses were conducted using SPSS (version 22; SPSS Inc.). Data screening indicated that all appropriate assumptions were met.

The mean YFAS symptom count for our sample was 4.7 (SD = 2.2) with symptom count being significantly and meaningfully positively correlated with BMI (kg/m\(^2\)), \(r = .598, p < .001\). In terms of the percentage of participants that reported various YFAS symptom counts: 2.2 per cent had no FA symptoms, 13.2 per cent endorsed 1 symptom, 7.1 per cent endorsed 2 symptoms, 4.4 per cent endorsed 3 symptoms, 8.8 per cent endorsed 4 symptoms, 15.2 per cent endorsed 5 symptoms, 22.5 per cent endorsed 6 symptoms and 26.5 per cent endorsed 7 symptoms.

To provide an indication of which numbers of YFAS symptomatologies should be grouped together to form a graduated FA classification, BMI was plotted against YFAS FA symptom count (see Figure 1); furthermore, a one-way analysis of variance (ANOVA) was employed to assess differences in BMI between the various YFAS symptom count groups. Results to the ANOVA revealed a significant YFAS symptom count effect with large effect size (Cohen, 1988), \(F(7, 400) = 33.357, p < .001, \eta^2_p = .369\). Fisher’s least significant difference (LSD) post hoc tests demonstrated that the 0 and 1 YFAS symptomatology over the last 12 months, according to the eight criteria for substance dependence listed in the DSM-IV-TR (e.g. tolerance, withdrawal, loss of control, clinically significant impairment or distress). The YFAS provides two scoring choices: a dichotomous FA addiction classification (either a food addict or not) and a continuous FA symptomatology score (0–7). To infer an FA classification, participants needed to have experienced three or more symptoms over the past 12 months, as well as meeting the clinically significant impairment criterion. The YFAS has been found to exhibit good internal reliability (\(\alpha = .86\)) and good convergent and divergent reliability (Brunault et al., 2014). For our current sample, internal reliability \(\alpha = .94\).

### Measures

**YFAS.** The YFAS (Gearhardt et al., 2009) is a 25-item self-report scale designed to measure

### Table 1. Sample descriptives (\(N = 408; 32.6\%\) males).  

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Mean</th>
<th>SD</th>
<th>Range</th>
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</thead>
<tbody>
<tr>
<td>Weight (kg)</td>
<td>108.3</td>
<td>23.9</td>
<td>46–200</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>172.5</td>
<td>9.8</td>
<td>154–210</td>
</tr>
<tr>
<td>BMI (kg/m(^2))</td>
<td>36.5</td>
<td>7.9</td>
<td>16.1–60.8</td>
</tr>
</tbody>
</table>

SD: standard deviation; BMI: body mass index.
Figure 1. Mean and standard deviation BMI of participants with T2D grouped by YFAS symptom count overlaid with proposed FA severity groupings (non-food addiction, mild FA, moderate FA and severe FA).

Symptom count groups were not significantly different to each other with regard to BMI, but were significantly lower than all the higher symptom count groups except for the 2 symptoms group. The 3 and 4 symptom count groups were not significantly different to each other, but had significantly higher BMI compared to the 0 and 1 groups and significantly lower BMI compared to the 5, 6 and 7 groups. The 5, 6 and 7 symptom count groups also appeared to cluster together, not being significantly different to each other, but were significantly higher in BMI compared to all the lower symptom count groups. While the plot in Figure 1 and these post hoc results suggest that the 0 and 1, the 3 and 4, and the 5, 6 and 7 symptom count groups clustered together, the 2 symptom group overlapped in terms of BMI with both the 1 and 3 YFAS symptom groups as demonstrated by a lack of significant differences.

Based on the current DSM-5 diagnostic criteria for Other (or Unknown) Substance Use Disorder and how FA symptomatology groups clustered together on the plot of BMI against FA (Figure 1), we tested a four FA severity classification solution (see Figure 2). Those participants with 0 or 1 YFAS symptom were grouped and termed non-food addicts (n = 63; 15.4% of the sample). Participants with 2 symptoms were classed as having mild FA (n = 29; 7.1% of the sample). Those participants with 3 or 4 symptoms were grouped as having moderate FA (n = 54; 13.2% of the sample). Those with 5 or more YFAS symptoms were termed to having severe FA (n = 262; 64.2% of the sample).

To test the cross-sectional predictive validity of this classification, it was hypothesised that there would be significant differences in BMI between each of the FA severity groups. Results of one-way ANOVA on BMI scores by FA severity group (non-food addiction, mild FA, moderate FA and severe FA) demonstrated a significant group effect with a large effect size (Cohen, 1988), \(F(3, 404) = 40.382, p < .001, \eta^2_p = .360\) (see Figure 2). Fisher’s LSD post hoc tests confirmed that each of the FA severity groups were significantly different to each other with BMI increasing with FA severity.

To test the convergent validity of the proposed FA severity classification, each severity grouping was compared to WHO BMI obesity classifications (WHO, 2000). The non-food addict severity group’s mean presented in the middle of the overweight/pre-obese classification, the mild FA group mean fell within the
obese class I classification, the moderate FA group mean was only slightly lower (0.71 kg/m²) than the obese class II classification threshold and the severe FA group mean BMI was on the cut-off for class III obesity (see Figure 2).

Discussion

A binary approach to the assessment of FA presents an inherent challenge for the diagnosis and treatment of FA, as a broad FA diagnosis is unlikely to adequately capture the important variations in FA severity that exist among people across the range of FA symptomatology. The primary purpose of this research was to investigate and potentially validate a graduated approach to FA severity diagnoses, thus addressing this critical gap in FA literature. To our knowledge, this research is the first to propose and quantitatively test DSM-5 (APA, 2013)-based diagnostic specifiers (mild, moderate and severe) in classifying a person’s FA severity levels.

Similar to previous research (e.g. Bégin et al., 2012; Pedram et al., 2013; Raymond and Lovell, 2015), our data demonstrated that FA symptomatology was strongly associated with BMI. This observation is particularly critical for this sample of people with t2d, as increased BMI and obesity is a specific risk factor for developing t2d and complications associated with t2d. Therefore, a better developed understanding of FA in people with t2d as well as other populations, including how to classify different levels of FA severity, is important for the design of efficacious treatments to help control obesity and in turn manage t2d risk and complications.

In our development and evaluation of a graduated FA severity diagnosis, we performed two sets of analyses. The first analysis where FA symptom count was plotted against BMI clearly showed that increased BMI was associated with increased YFAS symptom count, but more importantly that several coherent clusters of FA symptomatology count were apparent. However, while our groupings of FA symptomologies into non-food addiction (0–1 YFAS FA symptom), moderate FA (3 and 4 symptoms) and severe FA (5, 6 and 7 symptoms) were clearly and significantly different, there was a lack of significant difference in BMI between those participants endorsing 2 symptoms and those endorsing 1 or 3 YFAS FA symptoms. This presented a challenge in deciding how to classify participants with 2 symptoms; either into the non-food addiction classification or include them with those of 3 and 4 symptoms? In reconciling this issue, we considered the overall plot of YFAS FA symptom count against BMI (Figure 1), the original YFAS FA classification system and the DSM-5. The original YFAS classification required for an FA classification to be inferred, the individual endorse at least 3 FA symptoms. We therefore felt that including the 2 symptoms with the 3 and 4 symptoms groupings was not desirable as was at odds with the YFAS. We then considered including the participants endorsing 2 YFAS symptoms with those endorsing 0 and 1; the non-food addict grouping. While this solution did have attraction, we considered that the distribution of BMI against FA symptom count did not strongly support this approach as the BMI of participants endorsing 0 and 1 symptoms were so homogeneous and visually different to those with 3 FA symptoms. We then turned to the DSM-5 severity descriptors for Other (or Unknown) Substance Use Disorder (as well as other substance use disorders such as alcohol and cocaine), where 4 levels of severity are considered. Finally, based on the observed distribution of BMI against YFAS symptom count, the original YFAS FA classification system and the DSM-5, we elected to keep the 2 YFAS symptom count group separate from the non-food addiction group as well as from the 3 and 4 symptom grouping. In line with the DSM-5 nomenclature for Other (or Unknown) Substance Use Disorder, such as alcohol, opioids and stimulants (APA, 2013), we then termed the 2 symptom participants as having mild FA, those with 3 or 4 symptoms as moderate FA and those with 5, 6 or 7 symptoms as having severe FA. We acknowledge that the mild FA group only represented 7 per cent of the...
sample and that further research should consider the validity of this grouping.

To evaluate this proposed FA severity descriptors, in the second stage of analysis, we tested its cross-sectional predictivity for BMI. The results showed that the proposed FA severity descriptors were associated with significantly different levels of BMI. Furthermore, the proposed descriptors demonstrated a level of convergent validity with WHO (2000) BMI obesity classifications. The non-food addiction grouping \( (M_{\text{BMI}}=25.6 \, \text{kg/m}^2) \) were indicated as overweight/pre-obese. The mild FA grouping \( (M_{\text{BMI}}=30.4 \, \text{kg/m}^2) \) were indicated as being obese class I. The moderate FA \( (M_{\text{BMI}}=34.3 \, \text{kg/m}^2) \) and severe FA \( (M_{\text{BMI}}=39.8 \, \text{kg/m}^2) \) classifications were each on the cusps of obese class II and obese class III classification, respectively. These findings provide further support for the validity of our proposed graduated FA severity classification.

This research has limitations. As data were collected via self-report, there is the potential of social desirability bias. It is worthy of note that previous research has suggested in samples such as this where the majority of participants were overweight, that underestimation of body weight is more likely than overestimates (Connor Gorber et al., 2007). Therefore, the observed effects may actually be larger than reported, that is, the actual relationship between FA and obesity may in fact be stronger than which we reported. There is also the limitation related to the relatively small number of participants in the mild FA severity classification. Furthermore, as all participants had professionally diagnosed t2d, there is the question of how these findings may generalise to other populations without t2d. Similarly, additional research is needed that considers possible confounding variables including the use of medication and cultural differences. Additionally, this research only considered obesity (via the proxy of BMI which also has known limitations); future research should consider the validity of this proposed FA severity classification using other variables known to be associated with FA.

Our proposed four-category FA classification system is based on the assumption that all symptoms listed on the YFAS are equal. It could be likely that a person could have four of the most benign symptoms while another person has two of the most severe symptoms. Hence, it may be that a person’s specific symptoms and the severity of these specific symptoms have a greater impact upon the FA extent than simply their number of FA symptoms. This would be a fruitful avenue of future research.

We also suggest that the term FA may also represent a limitation; ‘processed food use disorder’ may be more preferable for theoretical reasons as well as being more in line with DSM-5’s reference to disorders, for example, alcohol use disorder and tobacco use disorder. The ambiguity and misnomer of the term FA has been discussed in respect to substance abuse, suggesting the term may need to be changed especially when considering DSM-5 substance-related disorders (Hebebrand et al., 2014). Moreover, there are numerous articles that use different terms to describe FA as a substance use disorder: high fat, high sugar (HFHS) foods (Pivarunas and Conner, 2015), refined foods (Ifland et al., 2009), processed foods (Murphy et al., 2014), highly processed foods (Schulte et al., 2015a) and ultra-processed foods (Moodie et al., 2013). The consistent adoption and use of the term processed food use disorder would help prevent potential confusion in the operationalisation of terms in this important research and applied topic area.

**Conclusion**

While currently there is a lack of longitudinal FA literature, we proposed that if FA does progress analogous to other Substance Use Disorders, diagnosing FA in the early stages, that is, the mild or moderate FA, is likely to have implications for preventing the development of severe FA and associated risk of other chronic diseases such as diabetes, hypertension, respiratory complications, heart disease and stroke. Thus, a dichotomous approach to FA diagnosis is likely to lack the required sensitivity to facilitate early intervention. Similarly, it can lead to the assumption that all individuals diagnosed
with FA are the same and should be given the same treatment. Based on our interpretation of the data collected from people with t2d, we conclude that FA severity can be validly classified as non-food addiction (0–1 YFAS symptom), mild FA (2 YFAS symptoms), moderate FA (3–4 YFAS symptoms) and severe FA (5–7 YFAS symptoms). We argue that this proposed graduated severity approach to FA has important theoretical and practical implications.

Declaration of conflicting interests

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This study relates to research objectives 8 - 10, has been completed and published in *Diabetes Research and Clinical Practice*, (impact factor 3.667)

A Graduated Food Addiction Classifications Approach Significantly Differentiates Depression, Anxiety and Stress among People with Type 2 Diabetes.

Karren-Lee Raymond, Lee Kannis-Dymand and Geoff P. Lovell


All authors were responsible for research design and methodology; K-LR contribution was 75%. K-LR was solely responsible for data collection. K-LR lead the data analysis, and GPL provided feedback on analysis and interpretation. K-LR’s contribution 75%. K-LR wrote the manuscript; GPL and LK-D reviewed and provided feedback on the final manuscript.
A graduated food addiction classifications approach significantly differentiates depression, anxiety and stress among people with type 2 diabetes

Karren-Lee Raymond *, Lee Kannis-Dymand, Geoff P. Lovell

School of Social Sciences, University of the Sunshine Coast, Maroochydore, Australia

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**A B S T R A C T**

**Aims:** To examine differences in depression, anxiety, and stress across people with type 2 diabetes mellitus (t2d) classified according to a four level processed food addiction (PFA) severity indicator dichotomy.

**Methods:** Four hundred and eight participants with a t2d diagnoses completed an online survey including the Yale Food Addiction Scale (YFAS) and the DASS-21. Based on YFAS symptom counts participants were classified as either: non-PFA; mild-PFA; moderate-PFA; or severe-PFA.

**Results:** Multivariate, $k = 0.422$, $F(9, 978.51) = 46.286$, $p < 0.001$, $r^2 = 0.250$, and univariate analyses of variance demonstrated that depression $F(3, 408) = 159.891$, $p < 0.001$, $r^2 = 0.543$, anxiety $F(3, 408) = 127.419$, $p < 0.001$, $r^2 = 0.486$, and stress scores $F(3, 408) = 129.714$, $p < 0.001$, $r^2 = 0.491$, significantly and meaningfully increased from one PFA classification level to the next. Furthermore, the proportion of participants with more severe classifications of depression $\chi^2 (12) = 297.820$, $p < 0.001$, anxiety $\chi^2 (12) = 271.805$, $p < 0.001$, and stress $\chi^2 (12) = 240.875$, $p < 0.001$, were significantly higher in the more severe PFA groupings.

**Conclusion:** For people with t2d, PFA is an important and meaningful associate of depression, anxiety, and stress, and that the adopted four level PFA severity indicator dichotomy is valid and useful.

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1. Introduction

Mood disorders, also known as affective disorders, are prevalent among people with type 2 diabetes mellitus (t2d) [1,2] and frequently co-exist with a cluster of other medical complications including obesity, hypertension, cancer, and heart disease [3]. Mood disorders have been strongly associated with Substance Use Disorders (SUDs), implying interactions between both maladies, which affect the course and diagnosis of both [4,5]. Together and separately, mood disorders and SUDs have been widely linked to a range of chronic conditions, including t2d [6]. Clinical evidence of SUDs and other mental illnesses have been found among people with t2d, implying multi-co-morbidity [6].

* Corresponding author at: School of Social Sciences, University of the Sunshine Coast, Locked bag 4, Maroochydore, Qld 4558, Australia.
E-mail addresses: karren-lee.raymond@research.usc.edu.au (K.-L. Raymond), lkannisd@usc.edu.au (L. Kannis-Dymand), glovell@usc.edu.au (G.P. Lovell).
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1.1. **Type 2 diabetes and psychological distress**

Diabetes prevalence worldwide continues to snowball at an alarming rate; it is recognised as the world’s fastest growing chronic condition [7–9]. Depression has shown to be more prevalent in people diagnosed with t2d than in the general population [10,11] and is often analogous with anxiety [12,13]. Research demonstrates that moderate to severe depression and anxiety among people with t2d is more than double what would be expected in the general population [14,15]. Furthermore, developing t2d creates additional stress due to the long-term prognosis of the disease and the potential complications associated with this illness [16].

1.2. **Psychological distress and Substance use disorders**

Research has shown associations between mood disturbances and SUDs [17]. Sinha [18] reviewed empirical evidence demonstrating that chronic and acute stress were strongly associated with increased desires to abuse addictive substances. Moreover, Lai et al. [19] performed a systematic review and meta-analysis of research published between 1990 and 2014 investigating prevalence rates of SUDs comorbid with mood and anxiety disorders; the strongest statistical relationships linked illicit drug use and major depression, illicit drug use and anxiety disorder, alcohol use and major depression, and alcohol use and any anxiety disorder. On an international scale, data from the Global Burden of Diseases, Injuries, and Risk Factors Study [20] summarised fatal, non-fatal, and total burden for 11 classes of mental diseases and SUDs, in regards to changes in burden since 1990. Internationally, mental diseases and SUDs contributed to over seven percent of the global burden of disease trumping other diseases such as tuberculosis, HIV/AIDS, and transportation accidents [3]. Clearly, there is strong evidence that psychological distress and SUDs are linked and associated with substantial economic burden and personal distress.

1.3. **Type 2 diabetes, psychological distress, and Substance use disorders**

Hitherto, empirical evidence has mainly focussed on dyadof diseases such as mental health and t2d, or mental health and SUDs [21]. Previously people with multiple health problems, multimorbidity, were somewhat overlooked in medical research [22]. However, research attention particularly linking primary healthcare and multimorbidity, has increased over the last two decades [21–25]. Despite such positive developments, research concerning the interplay of t2d, mental health, and SUDs has been limited. Research that has adopted this broader scope specific to t2d for example, Tann et al. [26] has made important observations. Tann et al.’s [26] research with American Indians and Alaskan Natives suggests that the multimorbidity of alcohol abuse, depression, and diabetes have a much larger combined effect than they do individually (OR = 10.95; 95% CI = 2.98–40.32) when compared to a White population. Similarly, Wu et al. [6] demonstrated that among a t2d cohort, participants with a SUD (alcohol, tobacco, or drug use) were more likely to have a mood (OR = 2.229; 95% CI = 2.016–2.465) or anxiety diagnosis (OR = 1.857; 95% CI = 1.656–2.081).

Recent research has further progressed, linking food addiction (FA) (comparable to a DSM-5 SUD) with t2d psychological distress [27]. Empirical attention in the last decade continues to advance and support FA as a distinct construct with neurobiological, behavioural, and psychological criteria analogous to alcohol and drug abuse disorders [28,29]. Ifland et al. [30] suggests ‘refined foods’ are analogous to the manufacturing of addictive drugs using procedures including concentration and extraction, distillation, and crystallisation. Likewise, processed foods are refined and manufactured and because of this refining process are no longer natural. Moreover, ‘food addiction’ has had many and varied labels including; chocolate addiction [31], processed food addiction [27,30,32–34], eating addiction [35], problem with highly processed foods [36], physically and psychologically dependent on high fat, high sugar (HFHS) foods [37], and ultra-processed foods addiction [38]. From this point forward we have adopted the term ‘processed food addiction’ (PFA) to facilitate greater research direction and consistency.

1.4. **Processed food addiction**

Processed food addiction has previously been strongly associated with body mass index (BMI) in people with t2d [27,39,40], a key risk factor for the development and complications of t2d. Raymond and Lovell [2] demonstrated multimorbidity among those participants with t2d meeting a FA criteria [41] with higher depression, anxiety, and stress scores compared to those that did not. Also, worthy of note was that FA symptomatology accounted for a greater proportion of depression, anxiety, and stress scores (35%, 34%, 34% respectively) than participants’ BMI (<1%).

Recently, Raymond et al. [27] focussing on BMI in a sample of people with t2d, introduced and tested severity specifiers for PFA in line with the DSM-5 nomenclature for other (or Unknown) SUDs, such as alcohol, opioids and stimulants [42]. Conclusions included that a 4 level classification of PFA based on YFAS symptoms count was valid: none and one YFAS symptom = non-PFA; two symptoms = mild-PFA; three or four symptoms = moderate-PFA; and five, six, or seven symptoms = severe-PFA. Results also demonstrated the four severity indicators aligned with WHO BMI obesity classifications: the more severe the PFA classification, the more obese the WHO BMI classification.

The impetus for this current research was the consideration that previous binary approaches to PFA (either one has PFA or one does not), and the lack of a graduated measure of PFA symptomatology analogous to Other Substance Use Disorders [42] limited theoretical understanding and practical treatment of PFA. Based on the demonstrated relevance of psychological distress and PFA for people with t2d, we investigated depression, anxiety, and stress across people with t2d of different PFA classifications. Specifically, we hypothesised that there would be significant differences between PFA severity groups’ (non-PFA, mild-PFA, moderate-PFA and severe-PFA) psychological distress (depression, anxiety and stress) scores. Not only would findings of this research have
important implications for the further development of theoretical understandings of PFA and psychological distress associated in people with t2d, but also in terms of informing addiction orientated practical interventions to ameliorate psychological distress in this specific population.

2. Method

2.1. Participants and procedure

As part of a larger study, following institutional ethical approval, participants over 18 years of age with a current medical practitioner diagnosis of t2d were recruited on a volunteer basis to take part in an online survey set on the SurveyMonkey platform. The survey involved participants providing basic demographic information including t2d diagnosis, age, gender, domicile, height, and weight) along with self-report measures of PFA (YFAS) [41], depression, anxiety, and stress (DASS-21) [43]. Participants’ informed consent was indicated by completion of the survey. Recruitment was achieved through advertisements and flyers at Australian medical centres, social media (including Facebook and Twitter), and online diabetes media sites (Diabetes Australian and Diabetes UK), as well as letters sent out to medical practitioners. Data regarding participants’ demographics and BMI have already been published in Raymond et al. [27].

Of the 484 participants that started the survey, 17 withdrew immediately after informed consent, 10 withdrew during the survey, 45 did not confirm they had a current t2d diagnosis, and four did not provide height and weight data. Of the final sample (N = 408), 48.2% from were from Australia with the remaining 52.8% participants from: USA (25.7%), UK (22.5%), New Zealand (1.7%), and other (1.7%; including Germany, Ireland, Malaysia, Norway, and Spain); 0.5% did not report their domicile. One participant (0.2%) had an overweight BMI (16.1 kg/m²), 9.1% of the participants were in the normal BMI category (2:18–24.9 kg/m²), 13.7% were categorised as overweight (2:25–29.9 kg/m²), and 77.0% were categorised as obese (2:30 kg/m²). Participants’ age, height, weight and BMI are shown in Table 1.

2.2. Measures

2.2.1. Depression, Anxiety and Stress Scale (DASS-21), short version

The DASS-21 [43] is a 21-item (short-form version) self-report measure yielding three scales of seven items each to measure depression, anxiety or stress (psychological distress). Utilising a 4-point scale: 0 = “Did not apply to me at all”; 1 = “Applied to me to some degree, or some of the time”; 2 = “Applied to me to a considerable degree or a good part of the time”; or 3 = “Applied to me very much, or most of the time” participants determined how much each statement applied to them over the past seven days. The range of possible scores for each of the three subscales is 0–21, where a higher score represents a higher level of depression, anxiety or stress. Totalling up the scores from each subscale has shown to be reflective of general psychological distress [44]. The DASS-21 has demonstrated to be an adequate measure of depression, anxiety, stress and psychological distress with reliable Cronbach’s alpha internal consistencies of 0.88, 0.082, 0.90, 0.93 respectively; compared to other measures of depression and anxiety the DASS-21 has exhibited satisfactory convergent and discriminate validity [44].

2.2.2. Yale Food Addiction Scale (YFAS)

Process Food Addiction severity was determined by employing the YFAS [41], a 25-item self-report scale designed to measure FA over the past 12 months to clearly discern between persons with and without addictive eating behaviours. The YFAS is based on the seven substance abuse criteria in the DSM-IV [45]. The YFAS has shown good internal consistency (α = 0.86) [41] and psychometric properties have also been reflected in a German version [46], a French version [28], and a version for children (YFAS-C) [47]. Participants’ severity indicators of PFA were based on YFAS symptoms count: non-PFA = 0 or 1 symptom; mild-PFA = 2 symptoms; moderate-PFA = 3 or 4 symptoms; and severe-PFA = 5 or more symptoms [27].

2.3. Data analysis

Statistical analyses were conducted using SPSS (version 24; SPSS Inc., Chicago, IL, USA).

3. Results

Data were screened to ensure compliance with appropriate assumptions. Scales’ and subscales’ internal consistency were analyzed via Cronbach alpha calculations; all were found to be satisfactory. Dependent variables’ descriptive statistics, intercorrelations, and Cronbach alphas are reported in Table 2.

3.1. Psychological distress differences

One-way multivariate analysis of variance with PFA classification as the independent variable and DASS-21 depression, anxiety, and stress as the multiple dependent variables, returned a significant omnibus PFA classification main effect with large effect size, η² = 0.422, F(9,978.51) = 46.286, p < 0.001, η² = 0.250. Subsequent univariate tests of the same design demonstrated significant PFA classification main effects with large effects for each of the dependant variables: depression F (3, 408) = 159.891, p < 0.001, η² = 0.543; anxiety F(3, 408) = 127.419, p < 0.001, η² = 0.486; and stress F(3, 408) = 129.714, p < 0.001, η² = 0.491. Post hoc Fisher’s least significant difference (LSD) tests showed that each of DASS-21 measurescores

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Table 1 – Sample descriptives (N = 408; 32.6% males).

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Mean</th>
<th>SD</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>41</td>
<td>8.9</td>
<td>18–64</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>108.3</td>
<td>23.9</td>
<td>46–200</td>
</tr>
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<td>Height (cm)</td>
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<td>BMI (kg/m²)</td>
<td>36.5</td>
<td>7.9</td>
<td>16.1–60.8</td>
</tr>
</tbody>
</table>
significantly increased from one PFA classification participant grouping to the next (see Fig. 1).

3.2. Depression, anxiety, and stress severity indicators

As shown in Table 3, approximately three quarters of participants with t2d categorised as non-PFA had normal levels of depression, anxiety, and stress. For both depression and anxiety, the percentage of participants with more severe psychological distress classifications systematically increased with increasing PFA classifications; approximately 60% of participants with severe PFA had extremely severe depression, and 70% had extremely severe anxiety. Stress followed a similar pattern, with just less than half the participants with severe PFA having moderate levels of stress and 34.4% with severe levels of stress.

Table 2 – Dependent variables’ descriptive statistics and intercorrelations.

<table>
<thead>
<tr>
<th>Measure</th>
<th>Cronbach alpha</th>
<th>M</th>
<th>SD</th>
<th>DASS-21 Depression</th>
<th>DASS-21 Anxiety</th>
<th>DASS-21 Stress</th>
</tr>
</thead>
<tbody>
<tr>
<td>YFAS</td>
<td>0.941</td>
<td>4.72</td>
<td>2.19</td>
<td>0.753</td>
<td>0.754</td>
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</tr>
<tr>
<td>DASS-21 D</td>
<td>0.939</td>
<td>11.28</td>
<td>5.66</td>
<td>–</td>
<td>0.821</td>
<td>0.851</td>
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<tr>
<td>DASS-21 A</td>
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<td>8.82</td>
<td>5.12</td>
<td>–</td>
<td>–</td>
<td>0.847</td>
</tr>
<tr>
<td>DASS-21 S</td>
<td>0.908</td>
<td>10.79</td>
<td>4.75</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
</tbody>
</table>

*p < 0.001.

4. Discussion

Our aim in this study was to test the hypothesis that for people with t2d, PFA severity classifications (non-PFA, mild-PFA, moderate-PFA, and severe-PFA) would significantly differ on depression, anxiety, and stress, with higher PFA severity indicators having higher scores. Findings supported our hypothesis demonstrating that participants with more severe PFA had significantly and meaningfully higher levels of depression, anxiety, and stress. Furthermore, the majority of those participants who met the severe-PFA classification (5 or more symptoms) had extremely severe levels of depression and anxiety (59% and 70%, respectively). A similar pattern of results was observed for PFA severity and stress, although to a more modest degree. Participants in this sample without PFA were more likely to have normal stress levels and people with severe PFA were more likely to have moderate stress levels.

Results of this study corroborate previous correlational findings for people with t2d, demonstrating a relationship between PFA and depression, anxiety, and stress [2]. Importantly, this current research has extended the current body of knowledge by exploring how psychological distress varies across previously published PFA severity indicator classifications, with important implications for treatment and prevention methodologies. Additionally, in a previous study it was acknowledged that the mild-PFA group (2 symptom count) only represented 7% of the sample [27] and the meaningfulness of this classification was questioned. In this current study, the presence of statistically significant and meaningful differences in depression, anxiety, and stress between the mild-PFA and non-PFA, as well as moderate-PFA, do provide additional evidence that this PFA classification does appear valid and useful; further confirming the proposed four level dichotomy.

4.1. Limitations and future research

This study has limitations. As a cross-sectional study, we cannot make any definitive conclusions regarding a causal relationship between psychological distress and PFA. Using self-report assessments may have questionable reliability and validity, and be limited in determining a clinical diagnosis. Self-report measures may increase the likelihood of participants providing inaccurate information, especially when asking sensitive details such as a person’s body weight, due to past stigmatisation with obesity and body image [48]. However, while acknowledging these potential limitations, previous research using similar methodologies have produced valid and meaningful results. Despite all participants reporting a current health professionals’ diagnosis of t2d, we did not collect objective indices of t2d status such as HbA1C. These data were not collected as anecdotal evidence suggests that the majority of Australian’s with t2d do not know their HbA1C scores and that only 7% of a US sample of people with t2d were fully adherent to the ADA-recommended A1C testing.

Fig. 1 – Mean and standard deviation depression, anxiety, and stress DASS-21 scores of PFA severity participant classification groupings.
In terms of implications for practice, based on our findings we understand as well as application have been compromised behaviours related to the ingestion of high calorie foods. We context of uncontrolled food consumption and addictive by the inconsistent use of terms to describe addiction in the our extensive online searches of literature regarding associa -tion and treatment planning, screening of PFA severity should be conducted. Based on results of this screening, those individuals seen to have high levels of PFA could then be treated, alongside current t2d treatment modalities, with addiction specific based intervention methods. This would appear as a likely efficacious modus operandi for people who as yet have been unsuccessful in managing their psychological distress and obesity through traditional approaches. This novel approach to symptomologies in this population represents a fruitful avenue for future research, both in clinical and in practice among people with t2d.

4.2. Practical implications

In terms of implications for practice, based on our findings we are proposing that for a proportion of people with t2d, interventions that merely focus on addressing the symptoms of psychological distress, as well as obesity, without addressing the underlying disease of addiction, are unlikely to resolve the primary pathology. We argue in the early stages of the diagnosis and treatment planning, screening of PFA severity should be conducted. Based on results of this screening, those individuals seen to have high levels of PFA could then be treated, alongside current t2d treatment modalities, with addiction specific based intervention methods. This would appear as a likely efficacious modus operandi for people who as yet have been unsuccessful in managing their psychological distress and obesity through traditional approaches. This novel approach to symptomologies in this population represents a fruitful avenue for future research, both in clinical and in practice among people with t2d.

Table 3 – Percentage of sample of people with type 2 diabetes classified by PFA severity. Presenting with normal, mild, moderate, severe or extremely severe depression, anxiety, and stress DASS-21 severities.

<table>
<thead>
<tr>
<th>DASS-21 severity category</th>
<th>Normal</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
<th>Extremely severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depression’</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-PFA</td>
<td>77.8</td>
<td>4.8</td>
<td>11.1</td>
<td>1.6</td>
<td>4.8</td>
</tr>
<tr>
<td>Mild-PFA</td>
<td>37.9</td>
<td>27.6</td>
<td>10.3</td>
<td>10.3</td>
<td>13.8</td>
</tr>
<tr>
<td>Moderate-PFA</td>
<td>12.5</td>
<td>11.5</td>
<td>26.9</td>
<td>25.0</td>
<td>19.2</td>
</tr>
<tr>
<td>Severe-PFA</td>
<td>1.2</td>
<td>0.8</td>
<td>10.4</td>
<td>29.0</td>
<td>58.7</td>
</tr>
<tr>
<td>Anxiety’</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-PFA</td>
<td>77.8</td>
<td>6.3</td>
<td>12.7</td>
<td>0</td>
<td>3.2</td>
</tr>
<tr>
<td>Mild-PFA</td>
<td>51.7</td>
<td>20.7</td>
<td>17.2</td>
<td>3.4</td>
<td>6.9</td>
</tr>
<tr>
<td>Moderate-PFA</td>
<td>18.5</td>
<td>11.1</td>
<td>29.6</td>
<td>14.8</td>
<td>25.9</td>
</tr>
<tr>
<td>Severe-PFA</td>
<td>2.7</td>
<td>2.7</td>
<td>8.8</td>
<td>16.2</td>
<td>69.6</td>
</tr>
<tr>
<td>Stress’</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-PFA</td>
<td>69.8</td>
<td>20.6</td>
<td>6.3</td>
<td>1.6</td>
<td>1.6</td>
</tr>
<tr>
<td>Mild-PFA</td>
<td>51.7</td>
<td>24.1</td>
<td>3.4</td>
<td>13.8</td>
<td>6.9</td>
</tr>
<tr>
<td>Moderate-PFA</td>
<td>18.5</td>
<td>29.6</td>
<td>40.7</td>
<td>7.4</td>
<td>3.7</td>
</tr>
<tr>
<td>Severe-PFA</td>
<td>1.1</td>
<td>9.5</td>
<td>47.3</td>
<td>34.4</td>
<td>7.6</td>
</tr>
</tbody>
</table>

Note: Significant Chi-squared test for independence: DASS-21 depression $\chi^2(12) = 297.820, p < 0.001$; DASS-21 anxiety $\chi^2(12) = 271.805, p < 0.001$; DASS-21 stress $\chi^2(12) = 240.875, p < 0.001$.

frequency in 1 year [49]. Based on the decision to reduce questionnaire fatigue, we did not attempt to collect HbA1C data. It is unknown whether these results are unique for people with t2d; no data or previous research were found through our extensive online searches of literature regarding associations between PFA and depression, anxiety, and stress in samples without t2d. As such, future research should consider how PFA and psychological distress relationships compare across different sample types.

In terms of research implications about furthering the knowledge base in this topic area, developments in current understanding as well as application have been compromised by the inconsistent use of terms to describe addiction in the context of uncontrolled food consumption and addictive behaviours related to the ingestion of high calorie foods. We encourage future discussions in the ‘PFA’ domain to reach better agreement on this universal term for the construct of food addiction, as well as obesity, without addressing the underlying disease of addiction, are unlikely to resolve the primary pathology. We argue in the early stages of the diagnosis and treatment planning, screening of PFA severity should be conducted. Based on results of this screening, those individuals seen to have high levels of PFA could then be treated, alongside current t2d treatment modalities, with addiction specific based intervention methods. This would appear as a likely efficacious modus operandi for people who as yet have been unsuccessful in managing their psychological distress and obesity through traditional approaches. This novel approach to symptomologies in this population represents a fruitful avenue for future research, both in clinical and in practice among people with t2d.

Acknowledgements

We would like to acknowledge Diabetes Australia and Diabetes UK for their valuable support with participant recruitment.

The authors declare that there is no conflict of interest.

REFERENCES


CHAPTER SEVEN

7. General Discussion of Results

This research program was undertaken in response to the continuing rise in obesity rates globally and its link to t2d, psychological distress and impulsivity, as well as the emerging research in the domain of FA, particularly its association with obesity. Although the relationships between depression, anxiety, and stress, on BMI has previously been established (Kivimäke et al., 2009), it has been more marked among people with chronic illnesses such as t2d then the general population. Investigating these relationships appears to potentially be quite important, especially when treating this chronic malady. This may ensure health professionals have the confidence, self-management skills, and ability to implement the most fitting treatments, which in turn may assure optimal disease control and prevention of secondary complications.

While empirical research has demonstrated a relationship between FA and BMI, and FA and impulsivity, (Bégin et al., 2012; Mulele, 2013), the evidence is scarce. This research project extended the existing evidence, by investigating the role (if any) FA may play in other factors such as psychological distress, (depression, anxiety and stress) among people diagnosed with t2d. The first phase of this research (Studies 1 and 2) examined the relationships between PFA, BMI, psychological distress and impulsivity in people with t2d. The second phase developed and tested a graduated approach to FA classification based on severity levels as per DSM-5 (APA, 2013) (Study 3 and 4). Results provided strong support for the hypotheses proposed for each study.

Study 1, which investigated how FA and impulsivity were associated with BMI
in a t2d cohort, demonstrated that a substantial proportion (70.7%) of people with t2d met the criteria for FA as described by Gearhardt et al. (2009). This figure is much higher than the 11.4% of people in the general population used to develop norms (Gearhardt et al., 2009), highlighting the problematic nature of diet-focused interventions in the treatment of t2d. Furthermore, 97.6% of people with t2d reported that they had unsuccessfully attempted to reduce their intake of sweet, starchy, salty, and fatty foods and drinks. With results showing large percentages of people with t2d meeting the criteria for FA, addressing FA could have important implications for the future prevention and treatment of t2d.

Additionally, Study 1 demonstrated people who met the criteria for FA displayed higher levels of impulsivity and BMI. This result is consistent with previous findings linking substance abuse with high impulsivity (Crews & Boettiger, 2009). Since the inception of this study, Murphy, Stojek, and MacKillop (2014) have published findings demonstrating strong positive relationships between impulsivity, FA and BMI. The strong relationship between impulsivity and FA observed in Study 1 and by Murphy et al. (2014) highlight the importance of developing a better understanding of how and why impulsivity is related to FA, and whether addressing impulsivity may be beneficial in improving the efficacy of treatments for FA. Furthermore, as the study by Murphy et al. (2014) was conducted with a non-clinical sample (university students), this relationship between FA and impulsivity is not only relevant to people with t2d, but also the general population.

Addiction has many more facets than simply lacking the willpower to abstain from or reduce the use of substances. Consequently, the 70.7% of people with t2d who met the criteria for FA might not be benefiting from current treatments, which often focus on encouraging dietary and lifestyle changes, and in some cases, pharmacological treatments. Simply telling a food addict to change their diet without considering the
underlying addictive elements, is potentially setting the person up for failure. Thus, the individual may unlikely be able to reduce or stop using the addictive substance on willpower alone. (Burmeister, Hinman, Koball, Goffmann, & Carels, 2013, Ifland et al., 2009). Therefore, it may be useful to routinely screen people with t2d for FA symptomology, and if present, focus primarily on treating FA, rather than treating solely the potential consequences of the FA (e.g., obesity and t2d).

Study 2 extended Study 1 by exploring FA and psychological distress among people with t2d. Findings relating to Study 2 were particularly startling given the extremely high levels of depression, anxiety and stress observed in this sample of people with t2d who also met a FA criteria, compared to those who did not meet the FA criteria. Accompanying this result, it was surprising to see that impulsivity explained over a third of the unique variances in depression, anxiety and stress scores, whilst BMI explained less than 1% of the unique variance scores. Thus, in line with the objectives of the first phase of this research program, Studies 1 and 2 provided empirical support, demonstrating that FA is related to BMI, impulsivity and psychological distress.

The second phase of this research dissertation responded to the current binary approach to PFA (either one is a processed food addict or one is not) and the lack of a graduated measure of PFA symptomatology analogous to Other Substance of Abuse Disorders (APA, 2013). The third study investigated and compared a graduated severity level approach to PFA classification against the depression, anxiety, and stress scores of the Australian general adult population (Crawford, Cayley, Lovibond, Wilson, & Hartley, 2011). Study 3 further supported the findings of Study 1, with results showing that as participants’ BMI increased, so too did their FA symptom count. This finding led to the applicability of a graduated FA criteria analogous to the DSM-5 SUDs (APA, 2013). After establishing four severity levels to diagnose FA - non-FA (0-1 symptom), mild-FA (2
symptoms), moderate- FA (3-4 symptoms) and severe-FA (5+ symptoms) their cross-sectional predictivity for BMI were tested. The proposed FA severity indicators were significantly associated with the different WHO BMI classifications (overweight/pre-obese, obese class I, obese class II, obese class III). As the severity level of a participant’s FA symptoms increased, so too did their BMI WHO obesity classification.

Study 4 built on Study 3 by testing the PFA classification among people with t2d against their depression, anxiety and stress levels. For both depression and anxiety, our findings illustrated that the percentage of participants with more severe psychological distress classifications increased systematically with PFA classifications; approximately 60% of participants with severe-PFA severity indicator, had extremely severe depression, and 70% had extremely severe anxiety. Stress followed a similar pattern; just less than half the participants with the severe-PFA severity indicator demonstrated moderate levels of stress, and fifteen percent with severe levels of stress. In contrast, those participants with t2d and categorised as non-PFA had normal levels of depression, anxiety and stress. Most notably, this study demonstrated that the majority of people with t2d meeting a severe-PFA classification (4+symptoms) also met the extremely severe group categories of depression and anxiety of the DASS-21 (Lovibond & Lovibond, 1995). Thus Study 4 supported the proposed PFA classifications based on the current DSM-5 diagnostic criteria for Other (or Unknown) Substance Use Disorder, (APA, 2013). The results of these 4 Studies have treatment implications and raise pertinent questions regarding the role PFA plays in the assessment, diagnosis and treatment of people with t2d. Albeit, there still remains a lack of consistency in how PFA is explored in the research community, and there is variable understanding about the nature of PFA among health professionals.

Processed food addiction continues to be a serious global health problem, and may
be one of the fundamental causes of disorders viewed in medicine as primary illnesses. Hence, limited understanding of PFA has meant that treatment efforts have focused on the secondary diseases and symptoms (e.g., obesity, depression, anxiety) associated with PFA. For example, if a patient has pneumonia, the clinician does not simply treat the high temperature and then send the patient home after his or her fever is down, and ask the patient to monitor his or her pneumonia. It appears that this approach is what could be applied to the treatment of obesity or t2d, taking care of the patients’ secondary symptoms, while stating to the patient, “Now your weight or blood sugar is normal, stick to your prescribed diet.” This research at the very least may help the medical community to gain improved understanding of PFA and recognition that there are alternative ways for diagnosis and treatment of this malady.

Not only does this research point to an important gap in the diagnosis and potential treatment of t2d, but also in the normal population showing signs of PFA symptomology. Hitherto, the current methods of treatments have been fixated on treating the symptoms of PFA through ‘control’ methods. For example: obesity and t2d (diets, exercise, education); a person’s craving and desire for processed foods, Incentive-Sensitization and the Role of Cues; depression, anxiety and stress, Rational Emotive Therapy, Mindfulness; impulsivity, Acceptance Commitment Therapy; eating behaviours and psychological habits, harm reduction models; and for negative effect, the Effect Regulation model of Substance abuse (Brownell & Gold, 2012; Cade & O’Connell, 2011; Chan & Woo, 2010; Hoffman, Friese, & Roefs, 2009; Schulte, Joiner, Schiestl & Gearhardt, 2017; WHO 2013; WHO, 2016). For people with t2d, checking glucose levels and administering insulin before and after most meals could be exceptionally challenging for patients with significant trouble controlling their food intake. Hence treatment and prevention methodologies to date appear to have had limited long-term effectiveness in ameliorating
the obesity epidemic and secondary complications.

This research has demonstrated that implementing an addiction model - PFA - analogous to SUDs, appears to be an efficacious avenue to consider. For example, at assessment if an individual’s difficulties with ingesting processed foods are seen to be consistent with this research formulation of PFA, rather than an eating disorder per se, this could have quite notable treatment implications. That is, for an individual who presents with food-related issues, rather than an eating disorder, the treatment may be best approached within an addiction model rather than with an eating disorder treatment protocol. Alternatively, it may be that treatment, even if an eating disorder protocol is utilised, incorporates aspects of an addiction treatment model in order to achieve more substantial and long-lasting treatment gains for the patient.

The severity level indicators have clinical applications in terms of informing the treating clinician about the type of intervention they may utilise. For example, someone with mild PFA symptomatology may not be a processed food addict, hence his or her treatment may differ from someone who is assessed to have severe PFA. Specifically, addressing PFA analogous to alcohol and other SUDs by applying an addiction model (goal of entire abstinence) may bring about consistent recovery, slow down the obesity epidemic and secondary complications that go hand-in-hand with PFA. Conversely, abstinence may not be a suitable treatment approach for a person who has a diagnosis of ‘mild-PFA’ (2 PFA symptoms), instead, effective CBT and behavioural interventions that focus on harm reduction (moderation of processed food intake) rather than a treatment goal of entire abstinence of the substance (i.e., processed foods) may be more appropriate (Schulte Joyner, Potenza, Grilo, & Gearhardt, 2015). Harm reduction invokes practices, policies and programs typically aiming to reduce negative health, social and economic consequences without the necessity of reducing the substance intake (Watson et al., 2017).
Outcomes would be most beneficial for the individual, their families and the broader society. Consequently, this research has important clinical implications for the training of health professionals who work with those experiencing food-related difficulties and distress.
CHAPTER EIGHT

8. General Conclusion

As this research program progressed, it became more apparent that the nomenclature describing ‘food addiction’ is a limitation in FA research. The term “food addiction” can be misleading, because it is not consuming food per se, but the excess consumption of processed foods that is the problem. In the current literature on FA, there is a myriad of terms to describe FA, including, addiction to processed foods (Murphy et al., 2014; Raymond, Kannis-Dyman & Lovell, 2016), ultra-processed foods (Moodie et al., 2013), refined foods (Ifland et al., 2009), highly processed foods (Schulte et al., 2015), high-fat high-sugar (HFHS) foods (Pivarunas & Connor, 2015) just to name a few. The misnomer and ambiguity of the term FA is a disadvantage with continued inconsistency across current research, potentially providing confusion in the operationalisation of a consistent nomenclature in this important research area, and this could cloud the pathway towards optimal interventions.

Considering the pattern of findings from these four studies, this research indicates that FA or PFA should be a candidate for inclusion in future editions of DSM-5. For example, the likely chapter would be ‘Conditions for Further Study’ which include conditions and criteria that are set forth to encourage future research but not as yet meant for clinical use. A further consideration may be the consistent adoption and use of the term ‘processed food addiction’ or moreover ‘processed food use disorder (PFUD). This is analogous to other substance abuse disorders currently in the DSM-5 (APA, 2013) including alcohol use disorder, or opioid use disorder, providing a segue to establishing ‘processed food addiction’, in future editions of the DSM-5.
This research program complements prior research within the FA realm, (Pursey, Collins, Stanwell, & Burrows, 2016; Schulte et al., 2015) contributing significantly to the exploration and consideration of new factors to help grow and mature understanding of the global obesity epidemic in people with t2d, and clearly with demonstrating the importance of PFA, psychological distress and impulsivity in BMI and, consequently, t2d. Almost three quarters of people with t2d in this sample met the criteria for PFA, which is 12 times higher than the percentage of PFA found in the non-diabetic population (Pedram et al., 2013).

This finding alone, points to the need for health care professionals to routinely screen for PFA symptomology using tools such as the YFAS, when working with people with t2d. Consequently, future treatments may benefit from treating people who meet the criteria for PFA with PFA methods. Furthermore, this research program has highlighted the role impulsivity plays in PFA and obesity in people with t2d, thus future treatments of PFA may also benefit from the inclusion of current psychological treatments for impulsivity. It may also be beneficial to consider whether people with t2d, with high levels of psychological stress and addiction to processed foods, might benefit from an overall improved set of coping skills. Finally, findings in this research would be particularly advantageous as a pathway to undertake much more needed research in examining the role PFA plays within the type 2 diabetic community, and uncover the reasons why comorbidity occur in this population group.

In summary, these four studies have contributed the extant literature in the FA realm and demonstrated the significance of PFA among people with t2d – a subgroup of people that have not been examined in relation to PFA. Nevertheless, questions regarding ‘processed food addiction’ e.g., the nature or characteristics of PFA, the use of the term, and underlying causes continue to arise, paving the way for ongoing, and a healthy
outlook, for future research in this ever-growing important addiction field.

8.1. Future Research & Directions

The studies of this dissertation have built on each other to extend current knowledge and understanding of PFA, demonstrating that a pertinent area for future research would involve the evaluation of typical t2d treatments against current PFA treatments (stopping the ingestion of processed foods) in a sample of people with t2d.

Having shown that impulsivity was associated with PFA and BMI, it would be beneficial to develop a better understanding of the specific aspects of impulsivity (e.g., based on the subscales of the BIS-11) that related to PFA and BMI. Following from Studies 3 and 4, there is also potential for future research to investigate if severity categories of PFA diagnosis can also be mapped against levels of impulsivity. Specifically, whether an individual’s impulsivity levels would increase as his or her PFA severity indicator increases from mild, moderate to severe symptomology. Developing a better understanding of how impulsivity influences PFA and BMI may lead to the inclusion of impulsivity-related interventions being introduced in the treatment of PFA in people with t2d. Path analysis or structural equation modelling could be integral in developing a model to explain how these variables combine to influence BMI.

The inclusion of PFA treatment, and subsequently addressing the symptomology of impulsivity and psychological distress, could potentially reduce BMI and improve treatment outcomes in people with t2d. As psychological distress and impulsivity have been linked to numerous other addictions, it would be beneficial to examine the comorbidity of PFA with other substances of addiction in people with t2d, as a transdiagnostic phenomenon.

With regard to assessing PFA, the YFAS is currently the most utilised test battery for PFA. However, subsequent to data collection for this program of research, the YFAS
2.0 was published and validated. The YFAS 2.0 does contemporise the assessment of PFA as was developed to maintain consistency with the current diagnostic understanding of addiction, and reflect changes to the substance-related and addictive disorders (SRAD; previously labelled substance use disorders; APA, 2013) section released in the DSM-5 (APA, 2013). While the YFAS 2.0 has been successfully translated in to other languages, for example German (Meule, Müller, Gearhardt, & Bechert, 2017), further research is needed to clarify and confirm how a DSM-5 based understanding and assessment of PFA relates to factors such as psychological distress, BMI, and impulsivity.

Investigating the genetic contribution to food addiction and obesity in people with t2d appears to be a fruitful avenue for future research. The genetic component of addiction (specifically, the DRD2 and ANKK1 haplotypes) has been established with other SUDs, and recently has been implicated in food addiction (Davis et al., 2013; Ma et al., 2015). To recapitulate, the key outcome of this research was for the development of our understanding of the causes of obesity. Thus, investigating evidence that obesity, and as such t2d risk, is affected by a certain genetic that increase the likelihood of becoming addicted to highly processed refined foods, may open the door to new methods of preventing and treating obesity, based on models used in the management of other addictions. Such a development could have substantial implications for the community and individuals alike. Albeit, one of the limitations of this dissertation was the cross-sectional design; the direction of causality from FA to obesity and t2d could only be hypothesized but not tested. Without a clear indication of the mechanisms, including biological, that drive the observed strong relationships between FA, obesity, and t2d, there is only a weak evidence base for the adoption of addiction orientated therapies to combat the over ingesting of refined, processed foods and obesity, thus in-turn t2d symptom presence.
Further afield, there is a need to take this research program into a non-clinical sample and move beyond people with t2d - to pre-diabetes and non-diabetes samples - that is, examining the extent to which psychological distress and impulsivity are important predictors of BMI and PFA in the general population. Hence, research into whether PFA, impulsivity and psychological distress influence BMI in a nondiabetic sample could improve current understanding of the development of obesity and lead to better treatment outcomes.

In summary, based on my findings, I have demonstrated that for a proportion of people with t2d, interventions that merely focus on addressing the symptoms of psychological distress, as well as obesity, without addressing the underlying disease of addiction, are unlikely to resolve the primary pathology. I argue that treatments based on addiction models would be an efficacious modus operandi for people who as yet have been unsuccessful in managing their psychological distress and obesity through traditional approaches. This novel approach to symptomologies in this population represents a fruitful avenue for future research and practice among people with t2d, and with a view of investigating and implementing an addiction treatment model to the normal population showing mild, moderate or severe PFA symptomology.
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Weygandte, M., Mai, K., Dommes, E., Leupelt, V, Hackmack, K., Kahnt, T., Rothemund,


Appendices

Appendix A: Ethics Approval

22 December 2015

Michelle Searle
Director, Office of Research
Tel: +61 7 5459 4574
Email: humanethics@usc.edu.au
F25386

Mrs Karren-Lee Raymond
Dr Geoff Lovell
Dr Lee Kannis-Dymand
University of the Sunshine Coast

Dear Karren-Lee, Geoff and Lee

Expedited ethics approval for research project: Diabetes and Non-diabetes diagnoses, Psychological Distress, Self-control, Food addiction and BMI (S/15/860)

This letter is to confirm that on 22 December 2015, following review of the application for ethics approval of the above named research project, the Chairperson of the Human Research Ethics Committee of the University of the Sunshine Coast granted expedited ethics approval for the project.

The Human Research Ethics Committee will review the Chairperson’s grant of approval and the conditions of approval at its next meeting and, should there be any variation of the conditions of approval, you will be informed as soon as practicable.

The period of ethics approval is from 22 December 2015 to 22 June 2017. Could you please note that the ethics approval number for the project is HREC: S/15/860. This number should be quoted in your Research Project Information Sheet and in any written communication when you are recruiting participants.

The standard conditions of ethics approval are listed overleaf. If you have any queries in relation to this ethics approval or if you require further information please contact a Research Ethics Officer by email at humanethics@usc.edu.au or by telephone on +61 7 5459 4574 or 5430 2823.

I wish you well with the success of your project.

Yours sincerely

[Signature]

Michelle Searle
Director, Office of Research

[Address Information]
Appendix B: Research Project Information Sheet

Research Project – Information Sheet:

Ethics approval number: S/15/860

Diabetes Diagnoses, Non-diabetes diagnoses, Psychological Distress, Self-control, Food addiction and BMI.

Project overview
The International Diabetes Federation (IDF, 2013), states that globally 382 million people had diabetes in 2013. Similarly, the Australian Bureau of Statistics has reported that diabetes diagnoses have increased from 2.4% in 1995 to 3.8% in 2007/2008. Individuals presenting with a high-risk of developing the disease are clearly classifiable as pre-diabetic and are up to 10 times more likely to develop the disease than the general population. Lifestyle interventions have been shown to be effective in preventing the advancement of pre-diabetes to type 2 diabetic status, thus decreasing reliance on pharmaceutical intervention and burden of associated complications of the disease on the health care system. A growing body of scientific evidence reports increased risk of gestational diabetes among females who are overweight or obese compared with lean or normal weight women. Moreover many antecedents of diabetes research clearly implicate obesity as a major risk factor; the “obesity epidemic” continues to grow as a major health risk at a startling rate. While strong associations between Body Mass Index (BMI), type 2 diabetes, prediabetes, gestational diabetes and non-diabetes have been found, our understanding of how attitudes towards eating, self-control and psychological distress relate to BMI in a prediabetes, and gestational diabetes populations are limited. This research aims to help increase our understanding by determining the relationships between these factors among diabetes populations and non-diabetes populations.

Voluntary participation
Your participation in this project is voluntary. There are no consequences for choosing not to participate and you can withdraw at any time. Completion of the survey is accepted as an indication of your consent to participate in this project. Consent is also given for your data to be used in future research projects.
Participation criteria
To take part in this research you need to be 18 years of age or above, and be any of the following categories:

- Nondiabetic;
- prediabetes (formal diagnosis);
- prediabetes (non-formal diagnosis);
- have/had gestational diabetes (formal diagnosis);
- type 2 diabetes diagnosis;
- your BMI is above 25.99;
- your BMI is above 30.00; (you may tag more than one box).

Race / ethnicity are broken down into more distinct categories to investigate psychological distress, self-control, food addiction, BMI, diabetes and non-diabetes within different countries.

Research project details
Participation in this research will involve completing a questionnaire which will take approximately 20 minutes.

Confidentiality/anonymity
Your answers will remain anonymous as no identifying information will be collected during this research. The data will however be securely stored for five (5) years (data may also be kept longer than this) in accordance with the University of the Sunshine Coast (USC) Australian policy, and will only be accessible by the primary researcher and supervisor.

Risks
There are minimal risks associated with your participation with this project. However, participation does involve answering questions which relate to moods (relating to depression, anxiety and stress) and eating behaviour patterns (which include problem eating behaviours), and some people may experience moderate emotional discomfort as a consequence of this. If at any time you feel uncomfortable and do not wish to continue with this survey, you are under no obligations to complete the rest of the questions, and are free to withdraw at any time. If you are still experiencing any distress due to your participation, Australian participants can access Lifeline’s free counselling service 24 hours a day on 13 11 14. It is also suggested to arrange an appointment with one’s diabetes care GP or diabetes educator to discuss any issues/concerns. International participants may contact a mental/health professional in their area of residence.

Expected benefits
Participation will not provide any immediate benefits to you. However, information gained from this research may provide insight into factors which influence BMI in prediabetes, gestational diabetes and non-diabetes populations. Hopefully this research will have implications in future practice and treatment modalities.
Feedback
If you are interested in the results of this study please email: karren-lee.raymond@research.usc.edu.au for a summary of the research findings.

Disclosure of funding sources
A small amount of funding was received from the University of the Sunshine Coast.

Researchers’ details
Karren-Lee Raymond (Ph.D. Cand.), Chief Investigator Email: karren-lee.raymond@research.usc.edu.au
Dr. Geoff Lovell Ph.D., Principal Supervisor Email: glovell@usc.edu.au

Concerns about conduct of research
Should there be any concerns about the nature and/or conduct of this research project, please contact Dr. Geoff Lovell or Karren-Lee Raymond (contact details above). Alternatively you can contact the Chairperson of the Human Research Ethics Committee, the University of the Sunshine Coast, c/- The Research Ethics officer, Office of Research, University of the Sunshine Coast, Sippy Downs, 4558. Ph: (07) 5459 4574; or Fax: (07) 5430 1177.

Australian and International participants email: humanethics@usc.edu.au

The researchers (Karren-Lee Raymond, & Dr. Geoff Lovell) and the University of the Sunshine Coast would like to sincerely thank you for your participation in this research.
Appendix C:

Research Questionnaire Ethics Approval S/15/860
The relationships between mood, attitudes towards food, self control and Body Mass Index (BMI) in people with Type 2 Diabetes.

Project overview
The International Diabetes Federation (IDF, 2013), states that globally 362 million people had diabetes in 2013. It was estimated that by 2035 the number of diagnoses will increase to 592 million. Similarly, the Australian Bureau of Statistics has reported that diabetes diagnoses have increased from 2.4% in 1995 to 3.8% in 2007/2008. Most of this rise was attributed to the increase in Type 2 Diabetes, which accounts for 80-90% of diagnoses. While strong associations between BMI and Type 2 Diabetes have been found, our understanding of how mood, attitudes towards eating and self control relate to BMI are limited. To help increase our understanding, this research aims to determine the relationships between these factors in people with Type 2 Diabetes.

Voluntary participation
Your participation in this project is voluntary. There are no consequences for choosing not to participate. You can withdraw at any time. Completion of the survey is accepted as an indication of your consent to participate in this project.

Participation criteria
To take part in this research you need to be 18 years of age or above, and have been diagnosed with Type 2 Diabetes by a health professional.

Research project details
Participation in this research will involve completing a questionnaire which will take approximately 30 minutes.

Confidentiality/Anonymity
Your answers will remain anonymous as no personal information will be collected during this research. The data will however be securely stored for seven (7) years in accordance with the University of the Sunshine Coast (USC) Australian policy, and will only be accessible by the primary researcher and supervisor.

Risks
There are minimal risks associated with your participation with this project. However, participation does involve answering questions which relate to mood and eating behaviours, and some people may experience
mild emotional discomfort as a consequence of this. If at any time you feel uncomfortable and do not wish to continue with this survey, you are under no obligations to complete the rest of the questions, and are free to withdraw at any time. If you are still experiencing any distress due to your participation, Australian participants can access Lifeline’s free counselling service 24 hours a day on 13 11 14. International participants may contact a mental health professional in their area of residence.

**Expected benefits**

Participation will not provide any immediate benefits to you. However, information gained from this research may provide insight into factors which influence obesity in people with Type 2 Diabetes. Hopefully this research will have implications in future practice and treatment of Type 2 Diabetes.

**Feedback**

If you are interested in the results of this study, please email: K_R050@student.usc.edu.au for a summary of the research findings.

**Disclosure of funding sources**

A small amount of funding was received from the University of the Sunshine Coast.

**Researchers’ details**

Karena-Lee Raymond (research student)
Email: K_R050@student.usc.edu.au
Dr. Geoff Lovell (supervisor)
Faculty of Arts and Business
University of the Sunshine Coast
Phone: (07) 5456 5100
Email: glovell@usc.edu.au

**Concerns about conduct of research**

Should there be any concerns about the nature and/or conduct of this research project, please contact Dr. Geoff Lovell or Karena-Lee Raymond (contact details above). Alternatively you can contact the Chairperson of the Human Research Ethics Committee, the University of the Sunshine Coast, C/- The Research Ethics officer, Office of Research, University of the Sunshine Coast, Sippy Downs, 4558. Ph: (07) 5459 4574, or Fax (07) 5459 1177. 

Australia and International participants email: humeret@usc.edu.au

The researchers (Karena-Lee Raymond & Dr. Geoff Lovell) and the University of the Sunshine Coast would like to sincerely thank you for your participation in this research.

---

**1. I have read and understood the above Research Project – Information Sheet and agree to volunteer to take part in this research.**

- Agree
- Disagree
2. Have you been diagnosed with Type 2 Diabetes?
   - Yes
   - No

3. How old are you?

4. What is your gender?
   - Female
   - Male

5. Where do you live?
   - Australia
   - USA
   - UK
   - Other (please specify)

6. What is your height? (Remove shoes before measuring)
   - Feet and inches
   - Centimetres

7. What is your weight?
   - Pounds
   - Kilograms

8. What is your waist circumference?
   - Inches
   - Centimetres
9. Please read each statement and circle a number 0, 1, 2 or 3 which indicates how much the statement applied to you over the past week. There are no right or wrong answers. Do not spend too much time on any statement. The rating scale is as follows:

- 0 Did not apply to me at all - NEVER
- 1 Applied to me to some degree, or some of the time - SOMETIMES
- 2 Applied to me to a considerable degree, or a good part of the time - OFTEN
- 3 Applied to me very much, or most of the time - ALMOST ALWAYS

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<tr>
<td>I found it hard to wind down.</td>
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<td>I was aware of dryness of my mouth.</td>
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<td>I couldn’t seem to experience any positive feeling at all.</td>
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<td>I experienced breathing difficulty (e.g., excessively rapid breathing, breathlessness in the absence of physical exertion).</td>
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<td>I found it difficult to work up the initiative to do things.</td>
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<td>I tended to over-read to situations.</td>
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<td>I experienced trembling (e.g., in the hands).</td>
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<td>I felt that I was using a lot of nervous energy.</td>
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<td>I was worried about situations in which I might panic and make a fool of myself.</td>
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<td>I felt that I had nothing to look forward to.</td>
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<td>I found myself getting agitated.</td>
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<td>I found it difficult to relax.</td>
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<td>I felt down-hearted and blue.</td>
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<td>I was intolerant of anything that kept me from getting on with what I was doing.</td>
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<td>I felt I was close to panic.</td>
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<td>I was unable to become enthusiastic about anything.</td>
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<td>I felt I wasn’t worth much as a person.</td>
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<td>I felt that I was rather touchy.</td>
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<td>I was aware of the action of my heart in the absence of physical exertion (e.g., sense of heart rate increase, heart missing a beat).</td>
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<td>I felt scared without any good reason.</td>
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<td>I felt life was meaningless.</td>
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</table>
10. This survey asks about your eating habits in the past year. People sometimes have difficulty controlling their intake of certain foods such as:
- Sweets: like ice cream, chocolate, doughnuts, cookies, cake, candy.
- Starches: like white bread, rolls, pasta and rice.
- Salty snacks: like chips, pretzel, and crackers.
- Fatty foods: like steak, bacon, hamburgers, cheeseburgers, pizza, and French fries.
- Sugary drinks: like soda pop.

When the following questions ask about “CERTAIN FOODS” please think of ANY OTHER foods you have had a problem with in the past year.

**IN THE PAST 12 MONTHS:**

0 = Never

1 = Once a month

2 = 2 to 4 times a month

3 = 2 to 3 times a week

4 = 4 or more times or daily

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<tr>
<th>Item</th>
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<tr>
<td>I find that when I start eating foods, I end up eating much more than planned.</td>
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<td>I find myself continuing to consume certain foods even though I am no longer hungry.</td>
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<td>I eat to the point where I feel physically ill.</td>
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<td>Not eating certain types of food or cutting down on certain types of food is something I worry about.</td>
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<td>I spend a lot of time feeling sluggish or fatigued from overeating.</td>
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<td>I find myself constantly eating certain foods throughout the day.</td>
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<td>I find that when certain foods are not available, I will go out of my way to obtain them, e.g., I will drive to the store to purchase certain foods even though I have other options available to me at home.</td>
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<td>There have been times when I consumed certain foods so often or in such large quantities that I started to eat food instead of working, spending time with my family or friends, or engaging in other important activities or recreational activities I enjoy.</td>
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<tr>
<td>There have been times when I consumed certain foods so often or in such large quantities that I spent time dealing with negative feelings from overeating instead of working, spending time with my family or friends, or engaging in other important activities or recreational activities I enjoy.</td>
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<td>There have been times when I avoided professional or social situations where certain foods were available, because I was afraid I would overeat.</td>
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<td>There have been times when I avoided professional or social situations because I was not able to consume certain foods there.</td>
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<td>I have had withdrawal symptoms such as agitation, anxiety or other physical symptoms when I cut down or stopped eating certain foods. (Please do NOT include withdrawal symptoms caused by cutting down on caffeinated beverages such as soda pop, coffee, tea, energy drinks, etc.)</td>
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<tr>
<td>I have consumed certain foods to prevent feelings of anxiety, agitation, or other physical symptoms that were developing. (Please do NOT include withdrawal symptoms caused by cutting down on caffeinated beverages such as soda pop, coffee, tea, energy drinks, etc.)</td>
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<td>I have found that I have elevated desire for urges to consume certain foods when I cut down or stop eating them.</td>
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<td>My behaviour with respect to food and eating causes significant distress.</td>
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11. IN THE PAST 12 MONTHS:

- My food consumption has caused significant psychological problems such as depression, anxiety, self-harming, or guilt.
- My food consumption has caused significant physical problems or made a physical problem worse.
- I kept consuming the same types of food or the same amount of food even though I was having emotional and/or physical problems.
- Over time, I have found that I need to eat more and more to get the feeling I want, such as reduced negative emotions or increased pleasure.
- I have found that eating the same amount of food does not reduce my negative emotions or increase pleasurable feelings the way it used to.
- I want to cut down or stop eating certain kinds of food.
- I have tried to cut down or stop eating certain kinds of food.
- I have been successful at cutting down or not eating these kinds of foods.

12. How many times in the past year did you try to cut down or stop eating certain foods altogether?

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<th>1 time</th>
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<th>4 times</th>
<th>5 or more times</th>
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13. People differ in the ways they act and think in different situations. Read each statement and put a tick on the appropriate circle. Do not spend too much time on any statement. Answer quickly and honestly.

1 = Rarely / never
2 = Occasionally
3 = Often
4 = Almost always / always

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<tr>
<th>Statement</th>
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<tr>
<td>I plan tasks carefully.</td>
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<td>I do things without thinking.</td>
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<td>I make up my mind quickly.</td>
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<td>I am happy-go-lucky.</td>
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<td>I don’t ‘pay attention.’</td>
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<td>I have ‘racing’ thoughts.</td>
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<td>I plan trips well ahead of time.</td>
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<td>I am self-controlled.</td>
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<td>I concentrate easily.</td>
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<td>I save regularly.</td>
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<td>I “squirm” at plays or lectures.</td>
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<tr>
<td>I am a careful thinker.</td>
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<td>I plan for job security.</td>
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<td>I say things without thinking.</td>
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<td>I like to think about complex problems.</td>
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<td>I change jobs.</td>
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<td>I act ‘on impulse.’</td>
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<td>I get easily bored when solving thought problems.</td>
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<td>I act on the spur of the moment.</td>
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<td>I am a steady thinker.</td>
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<tr>
<td>I change residences.</td>
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<tr>
<td>I buy things on impulse.</td>
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<td>I can only think about one thing at a time.</td>
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<td>I change hobbies.</td>
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<td>I spend or charge more than I earn.</td>
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<td>I often have extraneous thoughts when thinking.</td>
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<td>I am more interested in the present than the future.</td>
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<td>I am restless at the theatre or lectures.</td>
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<td>I like puzzles.</td>
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<tr>
<td>I am future oriented.</td>
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</table>
Feedback
If you are interested in the results of this study please email K_R050@student.usc.edu.au for a summary of the research findings.

The researchers (Karren-Lea Raymond, & Dr. Geoff Lovell) and the University of the Sunshine Coast would like to sincerely thank you for your participation in this research.

Please click 'Done' to exit.