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Raymond, K.-L., & Lovell, G. (2015). Food Addiction Symptomology, Impulsivity, Mood, and Body Mass Index in People with Type Two Diabetes. *Appetite*, 95, 383–389.

<https://doi.org/10.1016/j.appet.2015.07.030>

Document Type: Accepted Version

Link to Published Version: <https://doi.org/10.1016/j.appet.2015.07.030>

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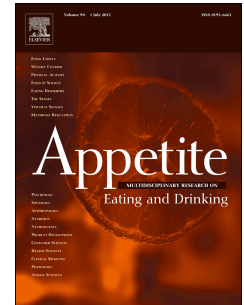
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Accepted Manuscript

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PII: S0195-6663(15)00347-5

DOI: [10.1016/j.appet.2015.07.030](https://doi.org/10.1016/j.appet.2015.07.030)

Reference: APPET 2652

To appear in: *Appetite*

Received Date: 23 April 2015

Revised Date: 3 July 2015

Accepted Date: 27 July 2015

Please cite this article as: Raymond K.-L. & Lovell G.P., Food Addiction Symptomology, Impulsivity, Mood, and Body Mass Index in People with Type Two Diabetes, *Appetite* (2015), doi: 10.1016/j.appet.2015.07.030.

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Food Addiction Symptomology, Impulsivity, Mood, and Body Mass Index in People with Type Two Diabetes

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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, $M_{BMI} = 37.6 \text{ kg/m}^2$, $SD_{BMI} = 8.0 \text{ kg/m}^2$) 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 FA classification group also had a significantly higher percentage of obese participants, $\chi^2(2) = 87.1$, $p < .001$, $\phi = .511$. Utilising a cross-sectional design to predict BMI, significant forward stepwise multiple regression demonstrated that FA ($\beta = .386$) and impulsivity (non-planning) ($\beta = .286$) 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 routinely screened, and if present, treated via addiction models rather than purely attempting to treat the potential consequences of FA.

Keywords

Obesity; Food addiction; Impulsivity; Type 2 diabetes.

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3 Food Addiction Symptomology, Impulsivity, Mood, and Body Mass Index in People with Type

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Two Diabetes

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, $M_{BMI} = 37.6 \text{ kg/m}^2$, $SD_{BMI} = 8.0 \text{ kg/m}^2$) 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 FA classification group also had a significantly higher percentage of obese participants, $\chi^2(2) = 87.1$, $p < .001$, $phi = .511$. Utilising a cross-sectional design to predict BMI, significant forward stepwise multiple regression demonstrated that FA ($\beta = .386$) and impulsivity (non-planning) ($\beta = .286$) 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.

Keywords

Obesity; Food addiction; Impulsivity; Type 2 diabetes.

Food Addiction Symptomology, Impulsivity, Mood, and Body Mass Index (BMI) in People with Type Two Diabetes

Diabetes mellitus has become a serious “global epidemic” with the International Diabetes Federation (IDF, 2013) stating that universally 382 million people had diabetes, with a further 175 million undiagnosed cases believed to exist. Furthermore, the IDF reports that the number of people with diabetes continues to climb at an alarming rate, predicting that by 2030 the number of global diagnoses will increase to 592 million. Australian statistics have shown an expanding rate of diabetes diagnoses increasing from 2.4% in 1995 to 3.8% in 2007/2008 (Australian Bureau of Statistics, ABS, 2011). Similarly, in North America the IDF (2013) estimated 27% undiagnosed cases of people with diabetes; a further 52 million people living with diabetes and an approximate prevalence rate of 7.9%. Furthermore, the European diabetes population is estimated at 52 million plus 33% undiagnosed cases of diabetes and a prevalence rate of 7.9% (IDF, 2013). Although type 2 diabetes (t2d) in Australia has primarily occurred in adults over 45, in recent years there has been an increase in the number of diagnoses in younger populations (Australian Diabetes Council; ADC, 2014). Similar trends have also been reported in Canada, Africa, and South East Asia (IDF, 2013). Associated with these increasing rates of t2d are a number of co-morbidity complications such as kidney disease, cancer, vision impairment, fatty liver disease, limb amputations, infertility, and heart disease (O’Connell, 2011). The current and predicted incidence rates of diabetes represents a huge global, physical, and economic cost to society. In 2013 there were 5.1 million associated deaths reported, with a minimum estimated health care cost at \$548 billion (US) and rising as the number of diagnoses increase.

Obesity

Modifiable risk factors implicated in the development of t2d include sedentary lifestyles, labour saving mechanical devices, and an increased adoption of Western lifestyles (high sugar, high fat diets). Other reported contributors emphasise treating the disease 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 & Ravissin 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, & Zimmet, 2005; IDF, 2013; WHO, 2013). WHO (2013) categorises a person as being overweight if their BMI index is 25-29.99 kg/m², or obese with a BMI of ≥ 30 kg/m².

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 (Fincane 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, 2015). With regard to Australian data, the prevalence of 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 (Fincane 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 & Gold, 2012; O'Connell, 2011; Swinburn, Sacks & Ravissin et al., 2009).

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 & Aronne 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, & Alison, 2003). According to recent theory, research has implied that stress is associated with increased cortisol levels (Vicennati, Pasqui, Cavazza, Pagotto, & Pasquali, 2009) which can affect appetite (Sinha & Jastreboff, 2013) and eating behaviour (Mouchacca et al., 2013). This has shown to be associated with an increased likelihood of obesity (Chen & Qian, 2012; Mouchaca, Abbott & Ball, 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, Faith, & Alison, 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

1 within 5 years (Finn, 2010). Blaine's (2008), meta-analysis (N = 33,000) also supports a causal
2 link between depression and obesity; depressed participants at baseline were observed as being
3 nearly twice more likely to be obese or gain weight at follow-up than non-depressed participants.
4 As well as depression and stress, anxiety has also been observed to be positively associated with
5 increasing BMI (Eyres, Turner et al., 2014; Kivimäki et al., 2009; Zhao et al., 2009). However
6 the relationship between anxiety and obesity is complex, for example gender is again implicated
7 as a moderating factor.

8 The personality trait of impulsivity has gained research attention due to the reported role
9 of impulse control in the self-regulation of certain behaviours, including the moderation of food
10 consumption (Hoffman, Friese, & Roefs, 2009). Having low impulse control is likely to reduce
11 an individual's ability to regulate the type and amounts of food that they consume, potentially
12 leading to the onset of obesity and the development of other secondary related illnesses
13 (Weygandt et al., 2013). This premise is supported by numerous links between high impulsivity
14 and excessive consumption of substances such as drugs, alcohol, nicotine, and caffeine, along
15 with other addictions (de Wit, 2008). More recently impulsivity has been recognised as a multi-
16 dimensional construct and as such not a singular trait (Muele, 2013). Consequently, Meule
17 (2013) advocates the use of subscales when analysing the relationship between impulsivity and
18 eating behaviours. Meule's (2013) literature review concluded that only attentional impulsivity
19 was consistently related to overeating, and only a weak relationship was reported between
20 impulsivity (non-planning) and overeating. Murphy, Stojek, and MacKillop (2013) proposed that
21 impulsivity indirectly effects BMI due to its influence on addictive eating behaviours, which is
22 consistent with recent conceptualisations of food addiction (FA).

23 **Food Addiction**

24 Many individuals' attempts to reduce weight have been consistently resistant to
25 treatments, possibly due to interventions not addressing underlying causes. It has been proposed
26 that problematic eating behaviours and addiction to refined food symptomology have clear
27 similarities to the DSM-IV criteria for substance use disorders (Corsica & Pelchat, 2010; Corwin
28 & Grigson, 2009; Ifland et al., 2009). Although somewhat controversial, recent research supports
29 the concept of FA and that it shares many similarities in the behavioural symptomology and
30 neurochemical mechanisms underlying other addictions (Gearhardt et al., 2013; Meule, 2013).
31 According to Volkow and Wise (2005), neurological responses to foods are similar to those

1 associated with addictive drugs (e.g., cocaine) in that both these substances activate reward
2 circuits. Repeatedly activating these circuits results in the development of a preference for the
3 particular stimulus and can lead to neurobiological adaptation, eventuating in the behaviour
4 becoming compulsive, further reducing the individual's control over their food or drug
5 consumption (Volkow & Wise, 2005).

6 Lenoir, Serre, Cantin, and Ahmed (2007) found that addiction to the sweetness of sugar,
7 surpassed the cocaine reward in cocaine-addicted rats as they consistently chose sucrose over
8 cocaine. Similar to addiction studies, deficiencies in dopamine receptors (D_2) have been found in
9 obese individuals (Noble et al., 1994; Wang et al., 2001). Therefore, although the consumption of
10 highly palatable foods is a contributing factor to the development of obesity and other maladies,
11 the addictive nature of these substances suggests that controlling ingestion is not a simple
12 process. Consequently, reducing rates of obesity and thus t2d requires a better understanding of
13 the mechanisms underlying problematic eating behaviours (O'Connell, 2011; Wang et al., 2001).
14 That is not to say an individual's role of free choice and self-will is negated by the notion of
15 addiction, however exploring this hypothesis of FA may provide an awareness as to why a
16 subgroup of individuals continue to unsuccessfully restrain their eating and experience elevated
17 BMI (Taylor, Curtis, & Davis, 2010). Although there is a large body of previous research
18 examining relationships between impulsivity and other addictions, there is a dearth of research
19 examining the relationship between FA, impulsivity, and obesity; especially for special
20 populations at risk of unhealthily high BMI such as those with t2d. A greater understanding of
21 these relationships could help to determine whether behavioural and pharmacological
22 interventions targeting FA and impulsivity could potentially successfully facilitate weight
23 management, decreasing obesity, and in turn alleviating t2d symptomology for individuals at
24 particular risk.

25 In response to the saliency of obesity in terms of current trends in t2d and limited
26 previous research, this current study was designed to gain a better understanding of how FA and
27 impulsivity (non-planning, motor, attentional) relate to BMI in a t2d sample. To this end, three
28 specific research objectives were investigated. Firstly, what percentage of a t2d sample fulfil a
29 classification of FA based on the Yale Food Addiction Scale (YFAS; Gearhardt, Corbin and
30 Brownell, 2009). Secondly, whether BMI significantly differs between people classified by the
31 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.

Method

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/m², $SD_{\text{BMI}} = 8.0$ kg/m², BMI range = 18.9 – 58.6 kg/m²). 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 (≥ 18 -24.99 kg/m²); 11% were categorised as overweight (≥ 25 -29.99 kg/m²), and 79% were categorised as obese (≥ 30 kg/m²).

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 Impulsivity Scale (BIS-II), the Yale Food Addiction Scale (YFAS), and demographic questions.

Measures

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 – never*) to 3 (*Applied to me very much, or most of the time – 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, as well as satisfactory

convergent validity when compared to other measures of depression and anxiety (Henry & Crawford, 2005).

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-Peña, 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).

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 – 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. 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).

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.

Table 1

Dependent variables' descriptive statistics and intercorrelations

Measure	α	M	SD	1	2	3	4	5	6	7
1. BMI(kg/m ²)	-	37.6	8.0	-						
2. DASS-21 A	.90	8.8	5.2	.53**	-					
3. DASS-21 S	.91	10.6	4.9	.48**	.85**	-				
4. DASS-21 D	.94	11.2	5.7	.53**	.83**	.86**	-			
5. YFAS	.98	4.7	2.2	.58**	.78**	.76**	.79**	-		
6. BIS-II attentional	.74	21.5	4.1	.52**	.78**	.76**	.75**	.80**	-	
7. BIS-II motor	.64	25.7	4.1	.44**	.65**	.63**	.60**	.70**	.73**	-
8. BIS-II non-planning	.69	29.0	4.8	.55**	.65**	.62**	.66**	.68**	.75**	.64**

Note. ** $p < .001$

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).

Table 2

Percentage incidence of food addiction symptom count for current t2d sample and Gearhardt et al.'s (2009) non-t2d sample

Symptom	Gearhardt et al.'s. (2009) non-t2d sample	Current t2d sample
Substance taken in larger amount and for longer period than intended.	21.7%	35.6%
Persistent desire or repeated unsuccessful attempt to quit.	71.3%	97.6%
Much time/activity to obtain, use, recover.	24.0%	50.9%
Important social, occupational, or recreation activities given up or reduced.	10.3%	66.5%
Use continues despite knowledge of adverse consequences.	28.3%	76.3%
Tolerance (marked increase in amount; marked decrease in effect).	13.5%	75.7%
Characteristic withdrawal symptoms; substance taken to relieve withdrawal.	16.3%	72.8%
Clinically significant impairment.	14.0%	71.9%

BMI Differences

Independent *t*-test compared revealed that participants classified as meeting FA classification had significantly higher BMI ($M = 40.4 \text{ kg/m}^2$, $SD = 6.7 \text{ kg/m}^2$; $n = 236$) than those that did not meet the criteria ($M = 30.3 \text{ kg/m}^2$, $SD = 6.5 \text{ kg/m}^2$; $n = 98$, $t(332) = 12.11$, $p < .001$. The effect size for this difference was considered large, $d = 1.47$ (Cohen, 1988). Cross tabulation analysis of BMI classification groups (normal: 18.50-24.99 kg/m^2 ; overweight: 25-29.99 kg/m^2 ; and obese $\geq 30 \text{ kg/m}^2$) by FA classification was significant, $\chi^2(2) = 87.1$, $p < .001$, $\phi = .511$, with a large effect (Cohen 1988). As shown in Figure 1, a far greater percentage of the FA classification group were categorised as obese ($\text{BMI} \geq 30 \text{ kg/m}^2$) than the non FA group.

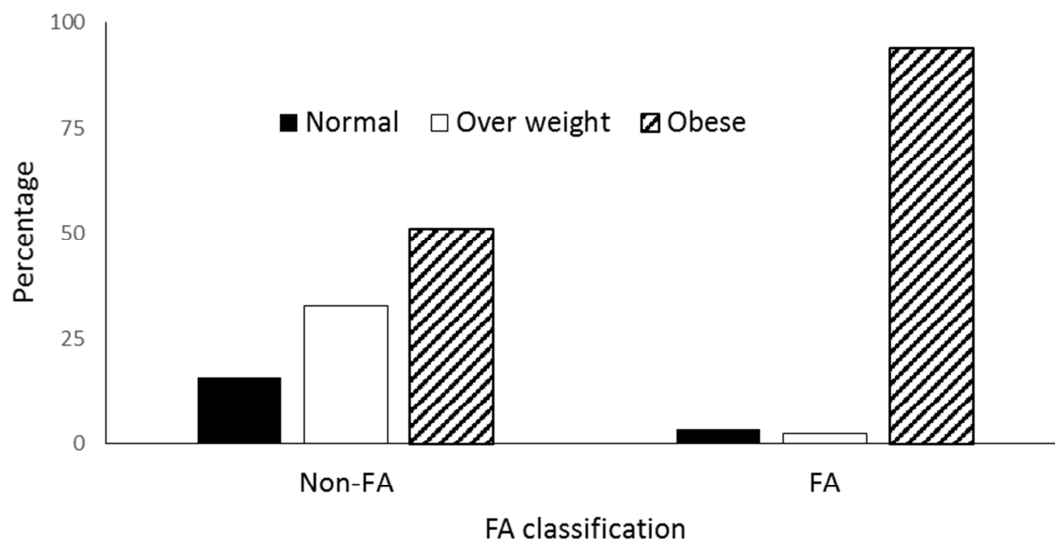


Figure 1. Frequency of BMI classifications by FA symptomology classification

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 symptomology and impulsivity (non-planning), accounted for a significant 38% of the variance in BMI scores in our sample of participants with t2d, $R^2 = .381$, adjusted $R^2 = .377$, $F(2,331) = 101.73$, $p < .001$. Cohen's f^2 (.62) indicated this effect to be large (Cohen, 1988). FA symptomology made the biggest significant unique contribution to BMI ($\beta = .386$), followed by impulsivity (non-planning) ($\beta = .286$). Following FA symptomology 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).

Table 3

Stepwise (forward) Multiple Regression Summary Statistics for BMI Scores in People with Type Two Diabetes

Model	<i>B</i>	<i>SE B</i>	[95% CI]	β	<i>t</i>	<i>Partial r</i>	<i>p</i>	VIF
Included								
YFAS symptomology	1.38	.21	[.96, 1.79]	.39*	6.56	.34	< .001	1.848
BIS non-planning	.48	.10	[.28, .67]	.29*	4.87	.26	< .001	1.848
Excluded								
DASS-21 D				.09	1.3	.07	.212	.351
DASS-21 A				.10	1.4	.08	.150	.362
DASS-21 S				.04	.51	.03	.610	2.458
BIS-II attentional				-.02	-.24	-.01	.813	3.625
BIS-II motor				-.02	-.34	-.02	.732	2.176

Note: N = 334. CI = confidence interval.

$R^2 = .336$ for Step 1, $\Delta R^2 = .044$ for Step 2 ($p < .001$). * $p < .001$.

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 (Kivimäke 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 symptomology. This discovery is novel and compares to reports of 11.4% (Gearhardt et al., 2009) and 19.9% (Pursey et al., 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

1 withdrawal, and clinically significant impairment. Furthermore, almost all the participants with
2 t2d in our sample reported unsuccessful attempts in reducing their intake of sweet, starchy, salty,
3 and fatty foods/drinks. These results demonstrate an association between FA symptomology and
4 t2d diagnosis.

5 Research objective two examined BMI differences between participants with t2d who met
6 the YFAS criteria for FA and those who did not. The first finding demonstrated a large and
7 significant difference in mean BMI between those that met the criteria in this sample for FA, and
8 those who did not. Secondly, utilizing WHO obesity classifications (WHO, 2014), the group
9 classified as meeting FA criteria comprised of significantly more obese individuals. This
10 suggests that meeting the YFAS criteria for FA is implicated with being obese; an important
11 modifiable risk factor for t2d.

12 The third research objective indicated that the variables of FA, impulsivity (attentional,
13 motor and non-planning), depression, anxiety, and stress were all significantly related to BMI,
14 however, FA and non-planning impulsivity demonstrated the strongest relationships with BMI.
15 Furthermore, the significant multiple regression analysis model of FA and non-planning
16 impulsivity predicting BMI (accounting for 38% of BMI variance) was not significantly
17 improved by the inclusion of depression, anxiety, stress, impulsivity (motor and attentional).
18 While there was evidence that multicollinearity was not present in the data, because t2d diagnosis
19 is heavily confounded with morbid obesity caution is needed in the interpretation that t2d
20 diagnosis and FA are directly associated. Although there was a strong correlation between the FA
21 and non-planning impulsivity, the finding that impulsivity (non-planning) added to the predictive
22 ability of the model suggests that impulsivity (non-planning) is oblique to FA. Meule's review of
23 literature (2013) detailed numerous studies which found relationships between attentional
24 impulsivity and overeating, but not between impulsivity and BMI. This is in contrast to the
25 findings of our study, where BMI had a strong positive relationship with impulsivity. With
26 regards to an explanation of this difference, we suggest this be related to the particular
27 characteristics of the samples drawn. We suggest that there may be differences across special
28 populations, further supporting the need for additional research in this area. Moreover, the
29 discovery that FA and non-planning impulsivity were stronger predictors of BMI, and, that their
30 inclusion in this model negated the need to consider depression, anxiety, and stress are novel
31 findings.

1 These current results, implicating FA in the development of obesity, have important
2 ramifications for potential future treatments of t2d. Addiction has additional facets than simply
3 being unwilling or lacking the willpower to abstain or reduce the use of addictive substances.
4 Consequently, the 70% of our sample with t2d who met the criteria for FA may not be benefiting
5 from current obesity treatments and interventions; historically generally focused on encouraging
6 dietary and lifestyle changes, and in some cases the use of pharmacological treatments and
7 gastric surgery. Simply instructing a food addict to change their diet without considering the
8 underlying addictive elements is unlikely to be successful at reducing or stopping their use of the
9 addictive substance (Ifland, et al, 2009; Burmeister, Hinman, Koball, Goffmann, & Carels,
10 t2d for FA symptomology, and if present, focus treatments on FA, rather than purely attempting
11 to treat the potential consequences of FA (ie obesity, and t2d). Moreover, it is important to
12 contemplate the possibility that FA caused the obesity leading to the onset of t2d, hence,
13 potentially screening for FA symptomology could improve weight management efforts with this
14 population, which in turn could potentially lessen the burden of preventable t2d related illnesses
15 on the health care system as well as potential suffering for the individual.

17 Previous theories and traditional methods including psychosocial models, individual or
18 group therapy, and CBT in the treatment of obesity and eating disorders are now being
19 challenged with additional contemporary theories and concepts such as FA. Recent research
20 approaches to FA have included: animal models (Avena, Rada, & Hoebel, 2009); biochemical
21 models (Volkow et al., 2012; Wang et al., 2014); neurological theories (Blumenthal & Gold,
22 2010; Wang, Volkow, Thanos & Fowler, 2009); standardised test batteries (YFAS; Gearhardt et
23 al., 2009), and addiction models (Ifland et al., 2009). These recent avenues of research have
24 produced empirical findings supporting the concept of FA. This convergence of knowledgeable
25 and empirical assessment of FA as a theory establishes greater scientific evidence that FA is also
26 within the chemical dependency domain.

27 Former and continued research paves the way for suggesting that there are greater risks in
28 treating just the obesity aspect and not looking at the whole picture; while with some individual
29 success, traditional approaches to the treatment of obesity have agreeably had limited impact on
30 the world obesity epidemic, especially in certain special populations. In particular, our study
31 demonstrated a significant number of people with t2d also had high symptomology of FA;

further investigations regarding the relationship between FA and BMI with nondiabetic and prediabetic samples could shed further light, and aid in determining the significance of the FA association with the t2d 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 (Connor 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 analysis. 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, Giltay, van Veen, & Zitman, 2010). Lastly if participant were taking specific weight loss medications, this may also have had some influence on this research.

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.

Acknowledgments

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

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Food Addiction Symptomology, Impulsivity, Mood, and Body Mass Index in People with
Type Two Diabetes

HIGHLIGHTS

- Obesity is a modifiable risk factor for type 2 diabetes (t2d).
- Results showed a link between obesity and food addiction (FA) in a sample of t2d.
- Over 70% of our t2d sample met criteria for classification of FA.
- Those classified as FA had significantly higher body mass index (BMI).
- FA and impulsivity significantly predicted BMI.