Food Addiction: A cost-effective treatment proposal within a developing country context

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by

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ABSTRACT

This study explores the possible efficacy of a low carbohydrate and high fat nutritional intervention (LCHF) as a treatment possibility aiming to improve the ability of self-control and regulation in the context of carbohydrate-addiction.

The study first outlines why increased simple carbohydrate consumption has been implicated as a risk-factor in numerous chronic conditions, and then explores the possibility that a reduction of such consumption could lower general medical expenditure in the healthcare sector of already overburdened institutions, especially in developing countries like South Africa. Since the neurobiological evidence for food addiction is compelling, this study investigates the impact of a low carbohydrate and high fat eating (LCHF) regimen by measuring the change in the severity of addictive behaviour in relation to a reduced carbohydrate consumption. Results indicate that a LCHF nutritional intervention lessened addictive behaviour after just 30 days, resulting in a statistically significant decrease in addiction symptoms from day 1 to day 30. The weight and BMI values of the participants recorded at the end of the study showed a reduction from those obtained during the pre-treatment stage, and the self-perceived 'feeling in control' also improved in all participants after the intervention.

The introduction of a LCHF nutritional intervention presents a relatively cost-effective treatment and preventative measure to combat carbohydrate over-consumption and its numerous health complications, and it is therefore hoped that the positive findings of this study will foster further research, using larger samples, into this type of nutritional intervention against addictive eating behaviour.

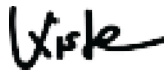
Keywords: low carbohydrate, high fat nutritional intervention, LCHF, addiction severity, carbohydrate over- consumption, chronic conditions, cost-effective treatment intervention, preventative treatment, Yale Food Addiction Scale, YFAS, diabetes, carbohydrate addiction, sugar, inflammation, Banting, ketogenic, depression, addiction, feeling in control, psychology, non-alcoholic fatty liver disease (NAFLD), obesity, overburdened healthcare, metabolic syndrome, stress, immune response, gut microbiome

DECLARATION

I declare that

"Food Addiction: a cost-effective treatment proposal within a developing country context" is my own work and that all the sources that I have used or quoted have been indicated and acknowledged by means of complete references.

I further declare that I have not previously submitted this work, or part of it, for examination at Unisa for another qualification or at any other higher education institution.



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CHAPTER 1
GENERAL OVERVIEW

1.1 INTRODUCTION

The ability to regulate one's behaviour effectively is relevant in many aspects of daily life, such as purchase decisions, sexual behaviour and the consumption of 'unhealthy' food. Behaviour, or rather the choice thereof, is strongly influenced by several explicit as well as implicit factors. Unbeknown to many, in a case of food consumption and choice, the common denominator might not be 'poor self-regulation' or a lack of discipline regarding conscious decision-making processes, but rather a form of addictive behaviour triggered by the overconsumption of a substance (Avena, Rada, & Hoebel, 2008; Gearhardt, Davis, Kuschner, & Brownell, 2011).

This study deals in large with the physiological process of digestion - what happens when we are unable to process nutrients effectively on the account of the foods we eat, and why we continue to eat them despite the harm they cause us. It investigates the repercussions of carbohydrate overconsumption in relation to obesity and proposes a way to curb the addictive behaviour and accompanying malaise by switching the bodies 'main fuel source' of carbohydrates to be dominated by fats.

Carbohydrate consumption appears to play a bigger role than previously assumed in disease progression. Low carbohydrate/high fat (LCHF) diet regimens have been investigated and successfully implemented in therapeutic environments at length to heal or aid the healing process of numerous conditions such as diabetes, auto-immune disorders, epilepsy, neuro-

degenerative conditions, dementia as well as psychopathological states such as schizophrenia, depression, and bipolar disorder (Hession, Rolland, Kulkarni, Wise, & Broom, 2009).

This study concentrates on establishing the positive attributes attainable by a nutritional change regarding addictive behaviour. The hypothesis that a main change in the central nervous system's fuel source improves the ability of self-control and regulation was investigated using an experimental pretest-posttest one-group design with a selected number of self-confessed 'carbohydrate over-consumers'. The aim of the study was to demonstrate the efficacy of a low carbohydrate high fat (LCHF) nutritional intervention (spanning over one month involving 30 participants). It was postulated that the participants will exhibit improved addictive-behaviour scores on the Yale Food Addiction Questionnaire after the 30-day period, as well as a self-perceived improvement in their 'feeling in control' with regard to their eating behaviour.

1.2 PROBLEM STATEMENT AND RESEARCH AIMS

1.2.1 The potential influence of a low carbohydrate, high fat nutritional intervention on addictive behaviour

The study will try to establish whether a LCHF nutritional intervention has a positive effect on addictive behaviour. The research is based on a small convenience sample (i.e. no random selection), and did not make use of a control group; it is therefore best classified as only quasi-experimental. Nevertheless, if the intervention does prove to be effective, the results will provide tentative support for a hypothesis that a LCHF regimen leads to an improvement in the ability to exercise self-control against addictive eating behaviour, and the study could therefore make a contribution to the existing research paradigm that investigates addictive behaviour in relation to carbohydrate consumption.

While the physical benefits of LCHF diets are starting to be recognised, all potential benefits have neither been fully established, nor have they influenced the main healthcare sector substantially. The increasing societal and financial burdens associated with disorders such as: diabetes, auto-immune disease, neuro-degenerative diseases, dementia as well as psychopathological disturbances could benefit immensely through the introduction of a nutritional intervention as proposed in this research (Leung, Carlsson, Colditz, & Chang, 2017).

Education on the importance of the right nutrition coupled with a revised nutritional regimen could help to address several stifling problem areas within the healthcare system, empowering both patient and provider - whilst being a cost- effective solution in a developing country context. Future research directions such as investigating changes within an individual's microbiome after a nutritional intervention could be initiated in support of the study findings.

1.3 MOTIVATION FOR CONDUCTING THE STUDY

As the available literature and areas of study regarding nutrition and its impact on physiological and psychological health expand, the question arises: 'Why does the public not know about it and where is the appropriate education?'

South Africa, as does the rest of the World, still follows a high carbohydrate, low fat nutritional approach despite vast indicators of the detrimental health consequences visible in the rise in cancer, auto-immune disease, dementia and neurological ailments, diabetes and obesity (Dehghan et al., 2017). Not only do these 'new-age' illnesses debilitate the afflicted and affect the next generation to come, they damage the economy and place immense pressure on an already strained healthcare system. Psychopathological behaviours arising

from food-addictions have not been considered at full length and are treated in no relation to the possibility of there being an addiction. Countless pharmaceutical measures are being developed and sold at high cost not only to the healthcare distributors, but also to the public - along with numerous side-effects impacting on the individual, the families, and society. Connections from carbohydrate-addiction to schizophrenia, depression, bipolar disorder, obsessive-compulsive behaviour and neurological pathologies have been established (Oriach, Robertson, Stanton, Cryan, & Dinan, 2016; Thornley, Russell, & Kydd, 2011), yet there is no sanctioned source of information made available to people enabling them to make better choices or to educate themselves freely regarding their body and nutrition. It is the most feasible option to take for a developing country such as South Africa.

I have been following the developments in nutrition and psychological as well as physiological health for over a decade and believe that a change in consciousness of the governing bodies is highly overdue. To offer practical and cost-effective treatment options for the 'epidemic obesity/carbohydrate addiction' to come, it is vital to understand that there is something like a 'food-addiction', what it entails, how far reaching the consequences are, and how it could be managed.

This study proposes a 'treatment option' (and possible diagnostic measure) concerning carbohydrate addiction, which I believe to be the core element of not only physiological stress on our organism but also of detrimental psychological consequence. Stress leads to a systemic communication breakdown which we call disease. What if we knew that a main stress factor could be alleviated by a nutritional intervention as opposed to a lifelong prescription of costly drugs and a still 'untimely' death?

This study starts with an overview of food and its addictive properties, elaborating on carbohydrate consumption in detail. Obesity is explained along the paradigm of it being an addictive, inflammatory disease, whereby obesity as an addiction plays a significant role

within a developing country context (Poobalan & Aucott, 2016). In this study the possibility is investigated that a simple nutritional intervention can be used to control this addiction, because this would not only provide a significant new obesity treatment regimen, but would also suggest that obesity complications and consequences such as diabetes might be averted if the cause of the addiction is identified, considered as such and treated accordingly. The study assesses carbohydrate consumption in relation to addictive behaviour and feeling in control by employing a widely-used scale of food addiction measurement to ascertain whether what is assumed to be the underlying cause of the addiction, namely carbohydrates, and the restricted consumption of such, does affect the self-perceived behaviour and feeling of control of the subjects enrolled for this study.

A discussion of methodologies follows as well as an elaboration on the study outcomes and the consequent interpretation by the researcher.

CHAPTER 2
THEORETICAL FRAMEWORK AND LITERATURE DISCUSSION

2.1 FOOD: CAN IT BE ADDICTIVE?

Obesity continues to grow as a major health risk to the global public (Mokdad, Marks, Stroup, & Gerberding, 2004). Sixty five percent of the world's population live in countries where excess weight and obesity kills more people than underweight-related factors (Paoli, 2014). "Worldwide obesity has nearly tripled since 1975 and in 2016, more than 1.9 billion adults, 18 years and older, were overweight. Of these over 650 million were obese. 39% of adults aged 18 years and over were overweight in 2016, and 13% were obese" (World Health Organisation [WHO], 2017). Overweight and obesity are seemingly linked to more deaths worldwide than underweight (World Health Organization [WHO], 2017).

From an evolutionary perspective, it is in the best interest of humans to have an inherent desire for food for survival. When a desire turns into an unhealthy dependence on palatable food or drink, it interferes with overall well-being and health. The US Department of Agriculture established that per capita soft-drink consumption has increased by almost five hundred percent in the past fifty years (Putnam & Allhouse,1999). By way of example, already 16 years ago research revealed that a high-fructose intake, the common sweetener in sodas and juices, reduces circulating insulin and leptin levels, and contributes to increased body weight (Ma et al., 2015), but high-fructose corn syrup is still a major constituent in any Western diet. Achieving sustainable weight loss with conventional treatments therefore remains a challenge and widespread prevention efforts may only have limited long-term success. Society's strong motivation to lose weight combined with the tremendous amount of

energy and resources spent on the 'obesity epidemic' suggest that the problem of obesity is not driven by lack of motivation or effort. Evocative research from the addiction and nutrition fields have recently uncovered similarities in patterns of food intake and consumption of drug abuse" (Gearhardt, Corbin, & Brownell, 2009, p. 430). Sugar feeds sugar: its intake may lead to an increased number of, and affinity for, opioid receptors, which in turn leads to further ingestion of sugar contributing to obesity (Avena, Rada, & Hoebel, 2008).

Certain foods or additives in food appear to trigger an addictive process, explaining why people experience difficulties in adhering to healthier food choices. As suggested, considerable evidence that food and drugs of abuse exploit similar pathways in the brain such as the dopamine and opiate systems (Hoebel et al., 1999; Nieto, Wilson, Cupo, Roques, & Noble, 2002) has been collected for decades. The release of extracellular dopamine in the nucleus accumbens (Volkow, Fowler, Wang, & Goldstein, 2002) increases with the food or drug. Positron emission tomographic imaging studies have shown that both obese and drug dependent individuals have significantly lower dopamine receptor levels (Wang et al., 2001). Flint et al. (2014) contend that in humans, obesity and substance dependence are associated with parallel neural mechanisms, and that some types of disordered eating associated with obesity are marked by behaviours commonly associated with addiction, namely the loss of control over consumption and continued use despite negative consequences.

There are many similarities between food and drug consumption. "Pleasurable food activates the brain through fast sensory signals and through slow ingestion process, such as increasing glucose in the brain" (Gearhardt et al., 2009, p. 430). The implication is that since glucose is the primary fuel source employed by most 'brains', reward signals for glucose reception should be investigated in addictive eating research. In a later study Gearhardt et al., (2011) found that similar patterns of neural activation are implicated in addictive-like eating behaviour and substance dependence: elevated activation in reward circuitry in response to

food cues and reduced activation of inhibitory regions in response to food intake.

The experimental question whether something like sugar can be a substance of abuse, leading to a natural form of addiction, was studied by Avena, Rada and Hoebel extensively in 2008. Since neural systems that evolve to motivate and reinforce foraging and food intake also underlie drug seeking and self-administration, any substance repeatedly causing the release of DA (Dopamine) or reducing DA reuptake at terminals via these circuits may be a candidate for abuse. A variety of foods can release DA in the NAc (Nucleus Accumbens core), including lab chow, sugar, saccharin, and corn oil. Extracellular DA decreases in reaction to drug withdrawal. Since the behavioural symptoms of withdrawal from dopaminergic drugs are less well defined than those observed during withdrawal from opiates, it may be easier to discern the signs of withdrawal when using foods that release both DA and opioids. Avena, Rada, and Hoebel (2008) found that sugar is one such food, and their reviewed evidence shows that intermittent access to sugar can lead to behavioural and neurochemical changes resembling the effects of a substance of abuse (Avena, Rada, & Hoebel, 2008). In this study, sugar 'dependency' was operationally defined by tests for bingeing, withdrawal signs, craving and cross-sensitization to amphetamine and alcohol (Avena, Rada, & Hoebel, 2008).

Simple carbohydrates translate to sugar. Fortuna (2010) reinforces the idea that carbohydrate craving may really be a serotonin or tryptophan craving. Serotonin (5-HT) is the primary antidepressant neurotransmitter which also plays a critical role in pain modulation. Serotonin also regulates sleep and the circadian rhythm. Many individuals crave 'comfort foods' in the evening. Foods such as cookies and milk, ice cream and milk chocolate elevate serotonin levels lifting mood, modulating pain, and help in the disengagement from the activities of the day (Fortuna, 2010).

Certain types of food disrupt and even misalign our communication channels,

preventing us from making the 'right' choices while initiating 'cravings' because they produce a 'maltuning' of the body. This indicates that our self-regulatory ability regarding food consumption is anything but 'self' regulatory.

The evidence for certain food's addictive properties lies in the biological realm. As soon as a particular food has the ability to manipulate neurotransmitter pathways, or a continued intake changes hormonal functioning, self-regulation is impeded.

The gut has been termed the 'second brain' by many scientists. This is not surprising, considering that the GI system is the primary gateway by which the external environment interacts with the body and that the intestinal mucosa contains ~100 million neurons, of which 90% conduct to the brain, whereas only 10% relay from the brain (Runow, 2011). Human life is dependent upon an intricate balance of minerals, water, organic molecules, and high energy bonds (Lamb, 2012), and it is characterised by cellular organization, growth and metabolism, reproduction and heredity. It is therefore not surprising that any stressors compromising the homeostasis of this dynamic balance could upset the body.

Since the status of the gut and liver has a profound effect on the functioning of the brain (Vasquez, 2010), affecting either would implicate the immune system by disrupting cellular signalling capacity and the delivery of necessary fuel or hormonal requirements, thus placing the system in a state of constant stress. If acute stress turns into constant stress, several systems collapse, leading to cell degeneration/ death or mutation. "Stress has been associated with many health conditions including cardiovascular disease, gastrointestinal disease and hormonal dysfunction" (Tatum, *Textbook of Functional Medicine*, 2010, p. 138). Different stressors elicit different patterns of activation of the sympathetic, nervous and adrenomedullary hormonal systems. If the consumption or over-consumption of a substance acts as a stressor, and the intake of such cannot be regulated as assumed, the notion of an addiction classification arises.

Stress elicits the activation of inflammatory pathways, normally used as a host defence response. Recent data on the microbiota and its interaction with food and obesity brought new hypothetical mechanisms for the obesity/diet relationship with inflammation. "Psychological and/ or circadian rhythm disturbances, may likewise contribute to the raise of oxidative/ inflammatory status" (Monteiro & Azevedo, 2010, para. 1). This is termed 'metabolic syndrome'. Increasing incidence of the metabolic syndrome all over the world accompanies the adoption of the modern Western lifestyle (Wilsgaard & Jacobsen, 2007). The main downside of this lifestyle is that it leads to increased stress (psychological, long-term and continuous), an excessively positive energy balance (high energy intake and low physical activity), low-quality food (both high 'bad' fat and energy dense, while poor in micronutrients), and a disruption of chronobiology. "An acute disturbance in any of the physiological regulatory systems evokes reactions that tend to re-establish equilibrium. When the stimuli, even of moderate magnitude, tend to be repetitive or chronic, change and allostasis in one system impact on the other, and vicious cycles are created and reinforced" (Monteiro & Azevedo, 2010, para. 7).

An important role in regulating the 'interaction' between host and its 'inhabitants' has the human gut as graphically discribed here: "Not only is the gastro-intestinal tract the recipient of massive amounts of 'external information' in the form of nutrients, toxicants, and allergens that weigh more than 700 kg per year, but the gastrointestinal tract is also a reservoir for the several hundred species and subspecies of yeast, bacteria and other microbes with the potential to modify hepatic function (e.g., detoxification) and overall health (e.g. immune response) by numerous mechanisms and with positive effects or negative consequences" (Liska & Lukaczer, *Textbook of Functional Medicine,* 2010, p. 100).

Compromised mucosal integrity of the gut due to injury from antigens, infection, systemic inflammation (disrupted microbiota as present in the metabolic syndrome for

example), or toxicants (e.g. alcohol or anti-inflammatory drugs, sugar, simple & processed carbohydrates) increases the absorption of potentially harmful substances normally excluded when mucosal integrity has not been breached. Considering that a substance or the over-consumption of this substance has the potential to substantially alter the gut microbiome, that is the mucosa/ intestinal flora, it is time to consider its addictive capacity and what can be done about it practically. The intake and over-consumption of carbohydrates (specifically gluten containing), has thus far been linked to a disrupted microbiome and a compromised intestinal barrier (Fasano, 2011), raising the level of systemic inflammation and initiating a spectrum of possible physiological, psychological and neurodegenerative malaise such as autoimmunity, cancer, rheumatoid arthritis, food allergies, asthma, eczema, celiac disease, inflammatory bowel disease, cystic fibrosis (Fasano, 2011), autism (Finegold, 2011) obesity (Ridaura et al., 2013), Type 2 diabetes (Qin et al., 2012), depression (Hsiao et al., 2013; Almond, 2013), behavioural/ mood disorders such as excessive anxiety (Hsiao et al., 2013; Foster & Neufeld, 2013), ADHD (Hsiao et al., 2013; Harding, Judah, & Gant, 2003), Parkinson's, dementia (Crane et al., 2013), Alzheimer's, neurodegenerative diseases in general (Hill, Bhattacharjee, Pogue, & Lukiw, 2014), as well as Crohn's disease (Schaubeck et al., 2015).

Most of the confusion sets in upon naming the actual culprit: Carbohydrates? Simple or complex? Fat, which type? Starches or Sugars? Sugar in this context may not be taken too literally. The term usually evokes images of white cubes, of which spoonfuls are fed into coffee, or of powdery sugar added to baked goods. It is quite the contrary - literally anything edible converts into or contains sugar in some form or colour. To keep that in perspective, a 12-ounce can of Coca-Cola contains the equivalent of nearly 10 teaspoons of sugar (coca-colaproductfacts, 2017). Processed and refined foodstuffs - albeit named or categorised differently - usually translate into a definite sugar-cube amount. Additionally, most foods

have a form of sweetener or sugar added (amongst flavour enhancers such as Monosodium glutamate) over and above to encourage and ensure ongoing high and rapid consumption (Mattes & Popkin, 2009; Nestle, 2013).

The term 'simple' carbohydrate translates into simple sugars. Sugars are found in a variety of natural food sources including fruit, vegetables and milk, lending food a sweet and desirable taste. Depending on how carbohydrate molecules are linked, the sugars can be categorised as single sugars (monosaccharides), which include glucose, fructose and galactose, or double sugars (disaccharides), which include sucrose (table sugar), lactose and maltose. The two monosaccharides to be followed most closely are glucose (the predominant energy source used by our body) and fructose. Otherwise we are dealing with disaccharides (two-sugars, e.g. table sugar) and polysaccharides, literally meaning many sugars. Polysaccharides are parted into indigestible carbohydrates (fibre both soluble and insoluble) and digestible ones, known as starch. Following the controversy of simple versus complex carbohydrates, how often is it stipulated that complex carbs are good for us? Can 'many sugars' really be?

The process by which our cells accept and utilise glucose is elaborate. A sugar molecule must be allowed into the cell (to be utilised as an energy source) by the hormone insulin, which is produced by the pancreas. Its role is to transport glucose from the blood stream into muscle, fat, and liver cells. Once it has arrived, it can be utilised as fuel (Lehninger, Nelson, & Cox, 2008).

Healthy cells have a high sensitivity to insulin. "But when the cells are constantly exposed to high levels of insulin as a result of persistent intake of glucose (much of which is caused by the overconsumption of hyper-processed foods filled with refined sugars that spike insulin levels beyond a healthy limit), our cells adapt by reducing the number of receptors on their surface that respond to insulin" (Perlmutter, 2013, p. 40). Our cells desensitize

themselves to insulin, in turn causing insulin resistance - allowing the cells to ignore the insulin and fail to retrieve glucose from the blood. In response, the pancreas pumps out more insulin, turning into a recurrent circle eventually culminating in 'system communication errors' such as Diabetes Type 2 (Lehninger, Nelson & Cox, 2008). Sugar in the blood presents a myriad of problems. As a toxin (now an unusable foreign substance) sugar inflicts damage and allows inflammation to run rampant in the body.

"The rise in dietary fructose consumption, primarily from sugar sweetened beverages (SSB), is at the forefront of interest and controversy from a public health perspective. Fructose, likened to addictive drugs and reviled as a scourge of the modern diet, has been implicated as a unique modifiable dietary risk factor for the rise of obesity, diabetes, cardiovascular disease, and recently NAFLD" (non-alcoholic fatty liver disease) (Ma et al., 2015, p. 469).

The DSM-V has replaced the DSM-IV's catchall diagnosis of 'substance dependence', with the diagnosis of 'substance-use' disorder. A substance-use disorder, as the DSM-V will define it, is "a maladaptive pattern of substance-use leading to clinically significant impairment or distress, as manifested by two (or more) of the listed criteria occurring within a 12-month period" (see Appendix 1).

Irrefutable evidence points to the overconsumption of carbohydrates resulting in excess body weight, significantly increasing the chances of cognitive decline (Spyridaki, Avgoustinaki & Margioris, 2016), loss of brain tissue (Wang et al., 2001) and various physio-psychological impairments ranging from depression (Luppino et al., 2010; Sánchez-Villegas et al., 2009)) to dementia (Wang et al., 2001; Tucker et al., 1990). For the purpose of this study, it is suggested that this qualifies as significant impairment or distress. As outlined in Chapter 2, several listed DSM-V criteria (Appendix 1) qualify regarding carbohydrate over-consumption.

The inherent challenges of studying excess sugar consumption and in particular fructose as a single dietary nutrient in humans include inaccurate assessment of carbohydrate consumption, inability to isolate fructose from other dietary carbohydrates and the current lack of well-designed clinical studies. The process in this study aims to follow the 'complete' carbohydrate intake in its reduction to an absolute minimum.

Note: According to the etymology of the word 'drug' it most likely referred to 'food' -From Middle English drogge("medicine"), from Middle French drogue ("cure, pharmaceutical product"), from Old French drogue, drocque("tincture, pharmaceutical product"), from Middle Dutch or Middle Low German droge, as in droge vate("dry vats, dry barrels"), mistaking droge for the contents, which were wontedly dried herbs, plants or wares. Droge comes from Middle Dutch drōghe ("dry"), from Old Dutch drōgi ("dry"), from Proto-Germanic *draugiz ("dry, hard"). Cognate with English dry, Dutch droog ("dry"), German trocken ("dry").

"Food is the oldest drug." (Hippocrates).

2.2 CARBOHYDRATES: HOW CAN THEY BE BAD?

Stafstrom and Jong (2012) established that it is possible that a final common neurometabolic pathway might be influenced by a variety of dietary interventions. The most notable example of a dietary treatment with proven efficacy against a neurological condition is the high-fat, low- carbohydrate ketogenic diet (KD) used in patients with medically intractable epilepsy. There is now compelling evidence that its efficacy is likely related to the normalization of aberrant energy metabolism (Paoli, Bianco, Grimaldi, Lodi & Bosco, 2013; Volek, Noakes,

& Phinney, 2015; Kossoff & Hartman, 2012; Riccio, 2011). The notion that many neurological conditions are linked pathophysiologically to energy dysregulation is providing a common research and experimental therapeutics platform, from which the course of several neurological diseases could be favourably influenced by dietary means.

Humans are programmed to exist at caloric excess (Higginson, McNamara, & Houston, 2016). Nutritional scientists and diets have counselled on maintaining a caloric balance for centuries. Metabolic studies, however, have shown an enormous variance between people and how they process calories. The firmicute/ bacteroidetes bacteria ratio is cautiously considered as an 'obesity biomarker', while keeping in mind that the relationship between metabolic syndrome, obesity, nutrition, and the microbiota is complex and multifactorial (Holmes, Li, Marchesi, & Nicholson, 2012). Firmicutes bacteria are exceptionally apt at extracting calories from food, increasing caloric absorption. Bactereoidetes break down bulky plant starches and fibres into shorter fatty acid molecules for energy consumption. A higher ratio of firmicutes in relation to bacteroidetes therefore implies higher caloric absorption leading to weight gain (Ley et al., 2005). Two paediatric studies collected faecal samples from infants 3 to 12 months old, prospectively following them up for seven to ten years. Results showed that children that became obese initially presented lower numbers of bacterioidetes, bifidobacteria and higher numbers of staphylococcus aureus (Kalliomaki, Collado, Salminen, & Isolauri, 2008).

Comparative analyses of human and other animal gut microbiomes have revealed that specific bacterial phyla and species differ between healthy individuals and those diagnosed with obesity and/or type 2 diabetes. "High-throughput methods have facilitated the identification of novel candidate bacteria and, most importantly, metabolic functions that might be associated with obesity and type 2 diabetes" (Cani, 2013, p. 381). A large study done by Turnbaugh et al. (2009) with 154 subjects (adult female monozygotic and dizygotic

twin pairs concordant for leanness or obesity, and their mothers) showed that while family members share the human intestinal microbiome, it remains specific for each individual. It is of interest that there was a comparable degree of co-variation between adult monozygotic and dizygotic twin pairs, which the researchers interpreted as suggestive of there being no genetic inheritance. Obesity in this study was also associated with decreased bacterial diversity (Turnbaugh et al., 2009).

While obesity can be triggered by a change in microbiota, it can also be influenced positively by such a change. Duseja and Chawla determined in 2013 that obesity might be affected by the gut microbiota through energy harvesting and fat storage by the bacteria. Small intestinal bacterial overgrowth is responsible for endotoxaemia, systemic inflammation, and its consequences including obesity and non-alcoholic fatty liver disease (NAFLD).

The relationship between gut microbiota and NAFLD is also dependent on altered choline and bile acid metabolism and endogenous alcohol production by gut bacteria. Further evidence comes from studies showing the usefulness of probiotics in animals and patients with NAFLD linking gut microbiota with obesity and NAFLD (Duseja & Chawla, 2013).

As a Harvard study (De Filippo et al., 2010) determined in 2010, Western Guts were dominated by firmicutes, whereas the African's (based on faecal analysis from rural African children) harboured more bacteroidetes, their obesity/ weight ratio reflecting accordingly.

Research conducted in 2006 by Turnbaugh and his team of researchers documented that obese individuals had 20 percent more firmicutes compared to normal-weight individuals and close to 90 percent fewer bacteroidetes (as cited in De Filippo et al., 2010), (Turnbaugh et al., 2006, 2009). In early 2015, the American Journal of Clinical Nutrition published a study demonstrating that higher levels of firmicutes change our genetic expression, noting that this precurses obesity, diabetes, cardiovascular disease and inflammation (O'Malley &

Stotz, 2011). It is conceivable that an altered microbiome, dominated by the wrong bacteria, not only 'makes us fat', but makes us 'want to get fat'. As Turnbaugh et al. (2009) have established, obesity presents different bacterial genes and metabolic pathways.

In 2007, Cani (2013) discovered that a high-fat diet profoundly affects gut microbiota. Long- term ingestion of a high-fat diet (14 weeks) induced a significant decrease in the family enterobacteriaceae and in bacteroides. Interestingly, the administration of bacteroides abolished the diet-induced immune and metabolic disorders associated with gut microbiota modifications in obese mice.

Turnbaugh et al. already established in 2009 that by switching humanized mice from a low- fat plant rich diet to a high-fat, high-sugar diet, the microbiota changed in just 24 hours. Western diet fed humanized mice became obese, and the phenotype could be transmitted to other mice by transplanting their gut microbiota to germ-free recipients. Hence, a 30 days' nutritional intervention appears to be feasible to induce a quick bacterial change.

In a striking result, Cani (2013) also found that obese mice treated with prebiotics had improved metabolic phenotypes (e.g. decreased metabolic endotoxaemia, glucose intolerance, improved leptin sensitivity and lipid metabolism) that were associated with a bloom in proteobacteria. Together, these studies suggest that specific phyla and/or genera might be increased or decreased during high-fat diet-induced metabolic disorders.

Conversely, the over-consumption of refined carbohydrates and simple sugar achieve the opposite. While our DNA has remained relatively stable over the course of human history, our microbiome appears to have experienced dramatic changes in response to the modern, western lifestyle. The Sonnenburgs highlight (Sonnenburg & Sonnenburg, 2014) that the western diet, being low in plant fibre and high in refined carbohydrates, lacks the fuel for good gut bacteria (being deficient in microbiota-accessible carbohydrates), resulting in

fewer microbes and beneficial by-products (ketone bodies) normally produced by gut bacteria by metabolising or fermenting the food. The 'microbial self' is being starved, and two key factors in chronic illness, namely that the gut bacteria produced help control inflammation as well as the immune system's response, fall by the wayside. The possibility of western microbiota being dysbiotic is contemplated, predisposing individuals to a variety of diseases.

Daulatzai (2015) established that an alteration in normal commensal gut microbiome with an increase in pathogenic microbes, impacts on homeostasis and health. Dysbiosis (an alteration in normal commensal gut microbiome with an increase in pathogenic microbes) causes gut inflammation, diarrhea, constipation, visceral hypersensitivity, abdominal pain, dysfunctional metabolic state, and peripheral immune and neuro-immune communication (communication between the immune system and the nervous system). "The above pathophysiological substrate and dysbiosis are underpinned by dysfunctional bidirectional 'Gut-Brain Axis' pathway. Pathogenic gut microbiota is known to up-regulate gut- and systemic inflammation; they enhance energy harvest, cause obesity, insulin resistance, and dysfunctional vago-vagal gut-brain axis" (Daulatzai, 2015, pp. 110-131).

Conceivably, the above cascade of pathologies may promote various pathophysiological mechanisms, neuroinflammation, and cognitive dysfunction, and it is clear that dysbiosis, gut inflammation, and chronic dyshomeostasis are of great clinical relevance. Thus, immune-mediated gut and extra-gut dysfunctions, due to gluten sensitivity and 'wrong' carbohydrate consumption for example, should be one of the focal points in the treatment of any inflammatory, aberrant energy metabolism indicative of disease progression.

Different foods feed and ensure the survival (if transient or not) of certain bacteria in the gut. Specific bacteria ensure their survival by triggering cravings and feelings of want as well as hunger, themselves targeting certain foods and/or expelling various toxins once the environment becomes 'inhospitable'. Judging by the influence an unbalanced microbiome

will exert over bodily responses, functions and inflammatory pathways, an inability to judge on suitable food choices seems inevitable - the mind and body are 'fremdgesteuert' - a loss of rationally induced control occurs (Alcock, 2014).

As Abu-Shanab and Quigley (2010) state, several mechanisms may explain the potential steatogenic and pro-inflammatory effect of intestinal microbiota. It promotes an increase in free fatty acid uptake and production by the liver. On the other hand, an increase in lipopolysaccharides (LPS), through activation of Toll-like receptor 4 (TLR-4) inflammatory signalling cascade, leads to insulin resistance and TNF-mediated inflammation. Note that tumour necrosis factors, or the TNF family, refer to a group of cytokines that can cause cell death/ apoptosis (Abu-Shanab & Quigley, 2010). An increase in lipopolysaccharides (LPS) - a combination of lipid and sugars and a major component of the outer membrane of certain bacteria - also leads to hepatic fibrogenesis. Fibrogenesis again is associated with many chronic liver diseases (Abu-Shanab & Quigley, 2010).

Highly processed foods which are usually high in simple carbohydrates and sugars, also contain high levels of LPS, which is classified as an 'endotoxin' because it induces a violent inflammatory response upon entering the bloodstream (Hrncir, Stepankova, Kozakova, Hudcovic, & Tlaskalova-Hogenova, 2008). In animal models, LPS is used to instantly create inflammation in laboratory studies (Hrncir et al., 2008). In case of an uncompromised mucosal integrity, LPS is blocked from entering the bloodstream. If the gut lining becomes permeable because of imbalanced bacterial growth and therefore an increased production of toxins (which is fuelled by the intake of processed carbohydrates) LPS goes into circulation, instigating an inflammatory cascade. M. Kahn (2011) established that animals injected bodily with LPS developed elevated levels of beta-amyloid (protein strongly implicated in plaque formation related to Alzheimer's disease) in their memory centre, the hippocampus. Studies have clearly demonstrated that mice receiving LPS injections in the

abdomen experience severe memory problems (Kahn, 2011). The LPS levels in Alzheimer patients are up to threefold higher than in healthy controls (Zhang et al., 2013).

The way bacterial cultures are fed and nurtured plays a vital part in inflammatory pathways as well as in the mucosal gut integrity. Ingredients such as gluten and sugar not only contribute to elevated insulin levels (Zhang & Zhang, 2013), dysfunctional leptin signalling and a permeable gut lining (Fasano, 2011), but also contribute significantly to the reduction of bacterial diversity of the gut lining. Gluten is found in the most unexpected products, ranging from ice cream to face cream, but also forms a staple part of the Western diet with its inseparable companion simple carbohydrates: bread, cereal, pasta, baked goods, pastries - in short, anything involving grains and used in white flour.

Increasing studies are confirming the link between gluten sensitivity and neurological dysfunction (Vojdani, O'bryan, & Kellermann, 2008). A disrupted microbiome not only implies inflammation but compromised brain function. Dr James M Hill investigated the level of production of brain chemicals such as GABA (gamma-amino butyric acid, amino acid serving as a neurotransmitter in the central nervous system), glutamate (vital neurotransmitter produced by gut bacteria) and BDNF (brain-derived neurotrophic factor, involved in neurogenesis), in relation to a direct reflection of gut bacteria (Hill, Bhattacharjee, Pogue, & Lukiw, 2014).

Behavioural changes observed in mice could be proportionately calculated in the volume of chemicals measured (Hill et al., 2014). GABA regulates anxiety and is in large part secreted by a strain of bifidobacteria (Bested, Logan, & Selhub, 2013). Anxiety is a common trigger to gastrointestinal disorders rooted in inflammation. Behavioural deficits such as anxiety and depression have been attributed to a lack of GABA and glutamate. Antidepressants of today for example, work by increasing the availability of the neurotransmitter serotonin, yet the precursor tryptophan is regulated by gut bacteria

(bifidobacterium infantis) (Jenkins, Nguyen, Polglaze, & Bertrand, 2016).

Good microbes influence the environment in the body, contributing to good health by producing certain chemicals affecting the health of the brain and the nervous system. They also determine the fortitude of the gut wall. Additionally, they produce vitamins that are essential to brain health, including Vitamin B12. A B12 deficiency is not only a risk-factor for dementia, but also for depression (Desbonnet, Garrett, Clarke, Bienenstock, & Dinan, 2008). Without a healthy microbiome, humans not only lack vital minerals and vitamins, but cannot gain the health benefits from polyphenols (vital for the detoxification process) from the foods eaten.

Inflammatory processes regulated by gut bacteria will assault the mitochondria. Mitochondria control the fate of the cell, determining whether it will live or die (Hoffman et al., 2013). Increased inflammation implies increased risk for inflammatory disorders such as Parkinson's, Multiple Sclerosis and Alzheimer's (Almond, 2013). Healthy fats such as omega-3 fatty acids sustain brain function, prevent cognitive decline and are potentially beneficial in Alzheimer's. The Western lifestyle still advocates a low fat, high carbohydrate approach.

Bazan (2016) states that omega-3 fatty acids, particularly DHA, clearly play a key role in neuroinflammation and Alzheimer's disease. It is not known how omega-3 fatty acids interact with the gut microbiome, but as established, there is a definite exchange between gut microbes and the immune system nurturing intestinal homeostasis.

Healthy fats such as omega fatty acids are not only crucial for intestinal homeostasis and the repression of inflammation, but necessary for survival. Omega-3 and omega-6 are polyunsaturated fatty acids (PUFAs) that play critical role in human health and must be provided by food. In the brain, PUFAs are precursors of endocannabinoids (Bosch-Bouju & Layé, 2016), which act as neuromodulators for a variety of processes, including motor

learning, appetite, and pain sensation, among other vital cognitive and physical processes. Bosch-Bouju and Layé (2016), list the functions of PUFAs in the brain in the following way:

A) Synaptic effects of PUFAs. PUFAs influence synaptic function. As structural elements of plasma membranes, PUFAs can modulate the dynamic of membranes and thus the functionality and traffic of trans-membrane and membrane-associated proteins. Firstly, these proteins are numerous at both pre-and post-synapses receptors, ion channels, transporters, and are essential for the synapse function. Secondly, PUFAs and/or their derivatives are agonists of receptors with synaptic functions and thirdly, PUFAs are precursors of endocannabinoids, which are lipid mediators with essential functions in neurotransmission and synaptic plasticity.

B) Role of PUFAs in neurogenesis and neuroprotection. DHA (docosahexaenoic acid) has positive effects on neuronal survival and neurogenesis. Synaptamide, an endocannabinoid derivative of DHA, plays an important role in cellular growing and in the differentiation of the brain during development. Neuroprotectin D1 (NPD1) as another derivative from DHA, protects against neuronal death by triggering the synthesis of anti-apoptotic proteins. DHA stimulates neuronal survival by inducing the synthesis of BDNF (brain-derived neurotrophic factor).

C) Role of PUFAs in neuroinflammation. A diet rich in DHA in humans is associated with a decreased risk of developing neurological disorders with an inflammatory component, such as Alzheimer's disease or depression. In animal models, Bosch-Bouju and Layé's (2016) laboratory demonstrated that neuroinflammatory processes are over-activated in the brain of mice fed a diet deficient for omega-3 PUFAs. Conversely, omega-3 PUFA brain-

enrichment protects against the deleterious effects of inflammation on cognitive performance.

Endocannabinoids receptors are mostly present in the adipose tissue, immune system, musculoskeletal system, gonads and cardiovascular system. These compartments are also regulated by dietary PUFAs (Bosch-Bouju & Layé, 2016). Dietary PUFAs appeared as homeostatic regulators of endocannabinoids. According to Bosch-Bouju and Layé (2016), it has been shown that a diet rich in omega-3 PUFAs leads to weight loss, in parallel to a decrease of AEA (anandamide) and 2-AG (2-arachidonylglycerol). Interestingly, a high fat diet rich in omega-3 PUFAs does not induce weight gain, while a low-fat diet rich in omega-6 PUFAs increases weight gain. This evidence suggests that dietary PUFAs act on fat formation and thus on weight gain via the endocannabinoid system. Inflammation is another component of obesity that can be modulated by endocannabinoids and PUFAs. Endocannabinoids are homeostatic regulators of the immune system, and their oxidised metabolites (directly derived from PUFAs) can have a direct role in inflammation (Chiurchiù, Battistini, & Maccarrone, 2015). Omega-6 PUFAs, such as ARA (arachidonic acid), are metabolized in pro-inflammatory derivatives while omega-3 PUFAs, such as DHA and EPA (eicosapentaenoic acid), are metabolized in anti-inflammatory and pro-resolution derivatives. PUFAs play thus a central role in the immune response of the organism.

D) PUFA-endocannabinoid interactions in mood and anxiety disorders. As Bazan (2016) determined, the higher the levels of inflammatory markers, the higher the level of depression. PUFAs appear to be determinants for the regulation of mood and anxiety disorders. In humans, the risk of developing depression is associated with low content of omega-3 PUFAs in the diet (Appleton, Rogers, & Ness, 2010), and patients with mood and/or anxiety disorders have lower levels of omega-3 PUFAs in the blood and in the brain compared to healthy subjects. In humans, it is established that patients suffering from mood

and anxiety disorders have lower levels of endocannabinoids in blood (Hill, Miller, Carrier, Gorzalka, & Hillard, 2009). The supplementation with omega-3 PUFAs would therefore constitute an interesting strategy for the prevention and treatment of mood and anxiety disorders, in particular because of the low side-effects to be expected compared to pharmacological agents and its cost effectiveness.

E) PUFAs / endocannabinoids interactions in neurodegenerative diseases. In neurodegenerative disease (e.g. Parkinsosn, Alzheimers), endocannabinoids play a protective role by decreasing the oxidative stress. From Bosch-Bouju and Layé's (2016) perspective, it is interesting to note that both PUFAs and endocannabinoids could interfere with the development of the disease by dampening neuroinflammation and oxidative stress.

Bosch-Bouju and Layé's (2016) conclude that dietary omega-6/3 PUFAs appear to be potent modulators and homeostatic regulators of endocannabinoids in the brain. The most promising hypothesis they sought to explore is that dietary PUFAs could switch the system from 'bad' (omega-6-derived) endocannabinoids to 'good' (omega-3-derived) endocannabinoids. Besides Bosch-Boujou and many others, science shows that a Mediterranean-type diet, rich in anti-inflammatory fats and proteins, correlates positively with lowered rates of depression (Sánchez-Villegas et al., 2009), whereas a diet high in carbohydrates and sugars initiates an 'inflammatory microbiome'.

Fructose, for example, increases circulating LPS by 40% (Bested, Logan, & Selhub, 2013). High-fructose corn syrup largely represents 42% of all caloric sweeteners. Most people will be erroneously content with seeing 'low fat' or 'no-sugar added' on their label, a health paradigm stipulated to this day by governments and health practitioners. An open discussion about healthy versus unhealthy fat is in dire need and not to be evaded for much

longer. Thus, Nakamura et al., (2016, p. 1515) state in this regard: “Carbohydrate intake below 50% of total energy with higher intakes of vegetable protein and mono-and-poly-unsaturated fatty acids, and lower intakes of saturated fatty acids may be favourable for reducing cardiometabolic risk factors.”

2.3 OBESITY: AN ADDICTIVE INFLAMMATORY DISEASE?

Not only do obese people have excessive amounts of firmicute bacteria (Filippo et al., 2010), they also tend to lack a diversity of bacteria (Gerritsen, Smidt, Rijkers, & de Vos, 2011). In addition, obesity is associated with an increased production of cytokines (pro-inflammatory chemicals) (Lumeng, & Saltiel, 2011). Largely stemming from the fat tissue, itself, fat cells are more like organ - releasing hormones and inflammatory substances. Visceral fat, often pronounced in obese people, activates signalling molecules capable of interfering with the body's normal hormonal dynamics (Yang et al., 2010). Fat-generated cytokines are found at elevated levels in all inflammatory conditions, ranging from arthritis and autoimmune disorders, to dementia and heart disease. Having a high CRP (c-reactive protein) level is correlated with a three-fold risk of dementia (Schmidt et al., 2010). Obesity is associated with increased inflammatory markers. It is correlated with a 55% increased risk of depression. Depression in turn is associated with a 58% increased chance of developing obesity (Luppino et al., 2010). Comfort eating, as seen in depression, triggers excess weight, in turn causing microbiome changes, enforcing excess carbohydrate consumption and cravings.

A waist-to-hip-ratio therefore implies more than just a belly-circumference. The bigger the belly, the smaller the hippocampus (memory centre of the brain) (Debette et al., 2010). Researchers also found a higher risk for small strokes, themselves being associated with a decline in brain function. If an excess of body fat predicts neurological disorders by increasing inflammation, obesity presents a risk factor for brain disease.

As obesity is the outcome of a metabolic dysfunction, the issue of blood sugar control is of the essence. When a cell is continuously exposed to excessive amounts of insulin

through an ongoing presence of glucose (on account of the over-consumption of carbohydrates), it reduces the number of insulin-responsive receptors on its surface, and insulin resistance is set (Qin et al., 2012). Since the American Diabetes Association recommended a 60-70% part calorie consumption from carbohydrates in 1994, diabetes cases doubled between 1997 and 2007. From 1980 to 2011, the figures have tripled (National Diabetes Statistics Report 2014 [CDC], retrieved 2015).

Obese people are blamed for their weight issues, seemingly unable to restrain themselves from eating foods associated with weight gain. The Western diet, high in carbohydrates, refined sugars and processed fat, predisposes the gut for obesity.

Mice that acquired bacteria from obese women have been shown to grow fat, whereas mice implanted with microbes from thin women stayed lean. When both mice shared the same cage, allowing the fat mice to acquire some microbes from the lean (mostly through consuming faeces), both mice remained lean. This type of experiment (Turnbaugh et al., 2006; Turnbaugh, Bäckhed, Fulton, & Gordon, 2008) was taken further by transferring bacteria strains from lean mice to those destined to be obese. The mice developed a healthy weight. This provides some evidence of a cause-effect pathway to obesity.

Turnbaugh et al. (2008) have shown that when humanised mice are fed a Westernised diet, mice with obese-type microbes become obese despite being exposed to their lean co-inhabitants. An unhealthy diet prevents ‘lean’ bacteria from having a positive impact.

Although there are more human studies needed, evidence to date warrants further investigation into the connection between obesity- inflammatory pathways and addiction. Bäckhed et al. (2004) clearly showed that conventionally raised mice had a 42% higher body fat as well as hepatic triglyceride content than germ-free mice even though conventionally raised mice had a lower caloric consumption overall. This supports the role of gut microbiota in nutritional absorption, germ-free mice with transplanted gut microbiota from

conventionally raised mice produced a 57% increase in body fat within two weeks. Certain gut bacteria can ferment complex carbohydrates, which are not digested by mammalian enzymes. Short-chain fatty acids (SCFAs), which are digested products of complex carbohydrates, account for 10% of dairy energy intake and stimulate de novo lipogenesis (the enzymatic pathway for converting dietary carbohydrate (CHO) into fat). Thus, gut microbiota contributes to the development of conditions such as NAFLD (Bäckhed et al., 2004).

In addition to nutritional absorption and energy storage, the gut microbiota is a source of Toll-like receptor (TLR) ligands, which induce inflammation under certain conditions. As outlined in Chapter 1.2, a compositional change of gut microbiota can increase the amount of TLR ligands delivered to the liver. TLR ligands can stimulate liver cells to produce pro-inflammatory cytokines. The liver has a high tolerance to TLR ligands (although bacterial components are potent TLR ligands) as hepatic cells express minimal TLRs in a normal liver (Miura, 2014). On the contrary, TLR signalling is activated and downstream molecules are increased in NAFLD because the tolerance has been disrupted. Altered gut microbiota and increased gut permeability are potential causes of the breakdown of tolerance. Circulating bacterial components and hepatic TLR expression are increased in human NAFLD patients as well as in animal models. To date, TLR2, TLR4, TLR5, and TLR9 have been shown to be associated with the pathogenesis of NAFLD (Miura, 2014).

The gut-liver axis has attracted much interest, particularly regarding the pathogenesis of NAFLD, as a change in gut microbiota alters nutritional absorption and storage (Miura, 2014). Non-alcoholic fatty liver disease is the most prevalent chronic liver disease in Western countries. NAFLD affects all spheres of society, especially the poorest and least educated. In a study involving overweight/ obese subjects screened by ultrasound, and including those with fatty liver/ hepatomegaly, Kruger et al. (2010) established that insulin resistance was the universal factor present upon investigating the disease prevalence in the Western Cape, South

Africa, whereby overweight/ obese subjects were screened by ultrasound and those with fatty liver/hepatomegaly were included. Paruk, Pirie, Motala, and Kolawole (2011) discovered a high prevalence of liver function test abnormalities in patients with type 2 diabetes enrolled in their study, particularly so in the morbidly obese subjects (South Africa). This is comparable with the reported prevalence in the Western world. Lipid abnormalities were more frequent in the group with liver enzyme derangements (Paruk, et al., 2011).

Younossi et al. (2016) did a meta-analysis that clearly showed that NAFLD is not only a disease of the obese, but is typically associated with metabolic dysfunction. It should be noted that metabolic dysfunction as a founding cause of systemic inflammation extends over and above the classification 'obese'. Younossi et al. (2016) conclude however, that metabolic comorbidities associated with NAFLD included obesity (51.34%; 95% CI: 41.38-61.20), type 2 diabetes (22.51%; 95% CI: 17.92-27.89), hyperlipidaemia (69.16%; 95% CI: 49.91-83.46%), hypertension (39.34%; 95% CI: 33.15-45.88), and metabolic syndrome (42.54%; 95% CI: 30.06-56.05). In a closing comment, Younossi et al. (2016) voice their concern that as the global epidemic of obesity fuels metabolic conditions, the clinical and economic burden of NAFLD will become enormous.

Considering that the liver is a vital organ of vertebrates, playing a major role in the metabolism responsible for the regulation of glycogen storage, the decomposition of red blood cells, plasma protein synthesis, hormone production and detoxification, NAFLD is to be avoided. The liver is an accessory digestive gland in producing bile, the alkaline compound aiding in digestion via the emulsification of lipids. The livers' highly specialised tissue regulates a wide variety of high-volume biochemical reactions, including the synthesis and breakdown of small and complex molecules, many of which are necessary for normal vital functions. NAFLD is only one of the diseases implicated by obesity and the compulsive overconsumption of carbohydrates.

NAFLD causes impaired nutritional absorption and storage. It is but one failing element in a cascade of systemic breakdowns. Overconsumption of carbohydrates is implicated in NAFLD as well as obesity, and simple carbohydrates are implicated in causing overconsumption. Obesity as an addictive inflammatory disease has become a stark reality.

It has been shown that the gut itself can communicate with the brain and influence behaviour (Neufeld, Kang, Bienenstock, & Foster, 2011). Researchers compared the behaviour of mice previously stripped of microbes and the behaviour of normal mice, where bacteria-free mice displayed reduced levels of BDNF (Brain-derived neurotrophic factor) and higher levels of the stress-hormone cortisol, displaying more risk-taking behaviour. By 'switching' a mouse's gut bacteria with another, it was found that behaviour could be significantly altered (Bercik et al., 2011). Shy mice became outgoing and bold mice became apprehensive. Microbiota appear to determine behaviour.

Changes in the gut affect the brain's response to negativity, predetermining emotional response and well-being, as was established in a small human-based study conducted by Tillisch et al. (2013). Gut bacteria may thus even affect a person's perception of the world around them.

If signals sent from the intestine to the brain can be modulated by dietary change, new strategies could be found to prevent and treat mental, neurological as well as digestive disorders. By altering the gut flora not only could the metabolism be positively affected, but also the brain function overall. Achieving optimal weight and mental well-being appears to depend on whether 'fat' microbes or 'non-fat' microbes are being harboured.

In a study of twins by Ridaura et al. (2013), gut bacteria were transferred from an obese human twin into the gastrointestinal tract of slender mice, and the mice grew fat. Conversely, when bacteria from the slender twin were implanted into lean mice, they stayed lean while eating a healthy diet. Oriach and colleagues (2016) conclude that this indicates

that there is now "…strong evidence for the role of the commensal gut microbiota in brain function and behaviour. Many potential pathways are involved in this bidirectional communication between the gut microbiota and the brain such as immune mechanisms, the vagus nerve and microbial neurometabolite production" (Oriach, et al., 2016, p. 25).

Oriarch et al. (2016) clearly see the association between dysbiosis of gut microbial function and behavioural and neurophysical deficits, noting that research focused on developing novel therapeutic strategies to treat psychiatric disorders by targeting the gut microbiota is rapidly growing. According to Oriarch et al. (2016), numerous factors can influence the gut microbiota composition such as health status, mode of birth delivery and genetics. They consider diet to be among the most crucial factors impacting on the human gut microbiota from infancy to old age: "Thus, dietary interventions may have the potential to modulate psychiatric symptoms associated with gut brain axis dysfunction. Further clinical and in vivo studies are needed to better understand the mechanisms underlying the link between nutrition, gut microbiota and control of behaviour and mental health" (Oriach, et al., 2016, p. 25).

Along psychological terms, according to Zilberter (2012), carbohydrate bias (or preference) in the brain's control of energy homeostasis reveals itself in several well-known ways including the phenomena termed 'positive reward,' 'hedonism', 'wanting', 'liking', and so forth. The 'sweet-addiction', comparable by magnitude with alcohol addiction and drug addictions, is well documented. Gold, Frost-Pineda and Jacobs (2003) argued that a deficit in 'reward' is coupled with sugar cravings in obesity and this coupling is common for sugar, cocaine, and heroin addictions.

Sugar results in a "reward dysfunction associated with drug addiction and compulsive eating, including continued consumption despite receipt of electric shocks" (Gearhardt et al., 2012, pp. 657-58). According to Corsica and Pelchat (2010, p. 167), "high concentrations of

sugar, refined carbohydrates [...] are addictive substances, and that foods containing these ingredients are consumed in a manner consistent with generally understood concepts of addictive behaviour found in the DSM-IV-TR." Although external stimuli such as environmental cues can modulate food intake/seeking through learning mechanisms, the abuse potential of carbohydrates appears to defeat rational thought. Increased stress, for example, could trigger a higher carbohydrate demand.

If the number of firmicutes can be reduced by dietary intervention, multiple risk factors are lowered. An increase in bacteroidetes will lead to a decrease of gut permeability, reducing inflammatory disease. Once the gut microbiota is stabilised, it seems that the addiction will not predominate physiologically - consequently setting the stage for mental/psychological support and counselling. Further exploration is warranted in what is now termed 'compulsive over-eating' (or binge drinking of alcoholic substances) as the single most common cause of obesity, excess weight and the resultant metabolic dysfunction.

Spyridaki, Avgoustinaki, and Margioris (2016) sum up that obesity provokes chronic low grade inflammation, which contributes to the development of insulin resistance. According to Spyridaki et al. (2016), this ends up in diabetes mellitus (DM), atherosclerosis, hyperlipidaemia, polycystic ovary syndrome, and so forth. In addition to these metabolic problems, obese individuals experience a progressive decline of their cognitive faculties. "This decline may be a consequence of their metabolic disturbances, that is due to insulin resistance, diabetes-associate hyper- and hypoglycemias, atherogenesis of their vascular endothelium, hyperlipidemias, hypertension, etc., or due to a direct effect of obesity-provoked chronic low grade inflammation. On the other hand, a decline of cognition leads to poor life style choices which accelerate weight gain in a self-accelerating cycle" (Spyridaki, et al., 2016, p. 169).

NOTE: In a recent study, Vrieze et al. (2012) have shown that subjects with metabolic syndrome treated with feacal enema harvested from a lean healthy donor exhibited an improved insulin sensitivity, which lasted for up to 6 weeks.

2.4 OBESITY AS AN ADDICTION: IN A DEVELOPING COUNTRY CONTEXT

Nutritional factors are neglected for several reasons. Much of the literature on nutritional treatments has yet to evolve beyond the early stages of scientific investigation. Physicians learn so little about nutritional medicine during their training that they feel too uninformed to include it in their practices. Sub-optimal nutrition is generally believed to be rare in industrialized societies - even though up to 50% of the population may fail to ingest the recommended dietary allowance for one or more vitamins or minerals. In the context of a worryingly rapid increase in obesity and obesity-related diseases in low- and middle-income countries including South Africa, Kimani- Murage (2013) established that both undernutrition-and obesity-related diseases contribute substantially to the burden of disease overall.

Kimani-Murage (2013) states that South Africa has undergone a complex health transition due to its historical background, being characterised by nearly half a century of Apartheid, high levels of HIV/AIDS over the past few decades, the recent rapid economic and social transition as well as urbanisation. She sees South Africa as being characterised by high levels of persisting undernutrition among the Black population potentially due to high levels of food insecurity reported at the household level (Kimani-Murage, 2013). At the same time, Kimani-Murage (2013) also acknowledges that a rapid nutrition transition has been experienced with a marked shift from staple foods towards an energy dense diet occurring

alongside urbanisation.

“High levels of physical inactivity and sedentary lifestyles have also been associated with the nutrition transition in several studies in South Africa. This has resulted in a high prevalence of overweight and obesity among adults, particularly women; for example, 55% of adult women are either overweight or obese, with a consequent high disease burden of non-communicable diseases” (Kimani-Murage, 2013, p. 194).

Although the substantial risk for metabolic disease in adolescent girls is of great public health concern (as chronic diseases associated with obesity are already contributing markedly to the burden of disease in this community and other parts of South Africa among adults), education fails to recognise the importance of nutritional direction.

In regard to behavioural syndromes, nutritional factors are neglected, in part, because marginal nutritional deficiencies are not believed to affect behaviour despite growing evidence to suggest that the belief may be false. For example, subtle neuropsychological impairment has been documented by EEG recordings of older subjects in association with any of several marginal nutritional deficiencies. Tucker et al. (1990) and Werbach (1995) identify a whole list of associations in inspecting everything from vitamin and mineral deficiencies to metal intoxications and food-sensitivities.

Werbach’s (1995) article clarifies that nutritional influences play a part so big regarding self-regulation that it cannot be ignored or disregarded:

"Despite the relative paucity of scientific evidence from controlled studies, [...] studies suggest that attention to nutritional factors may reduce overaggressive behaviors and the devastation resulting from them. Those clues, plus the safety of most nutritional interventions, argue that a nutritional approach should be considered in the treatment of the aggressive behavioral syndrome." (Werbach, 1995)

Oriach et al. (2016) conclude that diet-induced gut microbiota modifications may be

associated with brain dysfunction, behavioural and metabolic deficiencies.

"The emerging evidence of a microbiota-gut brain axis dys-regulation in certain neuropsychiatric disorders warrants further clinical and in vivo studies to investigate gut microbiota-targeted interventions as novel therapeutic strategies. Indeed, dietary interventions to treat dysfunction of the gut brain axis may pose potential as therapeutic strategies for psychiatric disorders" (Oriach, et al., 2016, p. 36). As an example, according to Oriarch et al., (2016), a diet deficient in magnesium, increases depressive-like behaviour and alters the gut microbiota, suggesting that magnesium deficiency could be a mediator of the behavioural effects through an altered gut microbiota. The era of commercial availability of true 'psychobiotics' might not be far away.

Whether the self-regulation is implicitly or explicitly influenced, fact is, that no form of self-regulation can take place without considering the addiction potential of the substance(s) consumed first and what influence it has on the brain, the gut and the overall state of mind.

The MODE model (Friese, Hofmann, & Wänke, 2008) suggests that behaviour is predominantly influenced by controlled processes only if a person is sufficiently motivated to engage in deliberate reasoning and has the necessary resources to do so, such as time and cognitive capacity. If either motivation or capacity are missing, associative processes assume a larger role and behaviour will be influenced by more attitudes that are automatically activated.

Whilst recognition is given to the fact that nutrition also influences the efficiency of educational programmes, influencing a child's cognitive development, most programmes amount to the alleviation of short term hunger only - and due to financial constraints and lack of support, lack of motivation or lack of the necessary resources, the ability to reason independently remains limited. The necessary level of understanding to initiate change is not

reached.

Even though schools are uniquely positioned to promote healthy eating behaviours and attitudes among children, approximately 25% of all children in developing countries are vitamin A deficient, whilst other nutrients most likely to be deficient in school-aged children are reportedly iron and iodine, with prevalence rates of the latter between 35% and 70% (Nhlapo, Lues, Kativu, & Groenewald, 2015). Additionally, nutritional guidelines adhered to support the implementation of grains and 'meat alternatives' (Nhlapo et al., 2015), which stand to be questioned. By replacing just 30% of the daily recommended meat and dairy intake with plant based sources, estimated intakes of zink, thiamin (vitamin B1), vitamins A (retinol) and B12 (cobalamin), and calcium, can go below recommendations (Seves, Verkaik-Kloosterman, Biesbroek, & Temme, 2017).

Also, a lack of proper storage practices potentially exposes foodstuffs to surrounding elements hindering the preservation of the quality of nutrients. As Cassim (2010) states: "The prevalence of overweight and obesity, particularly in children, raises serious attention to its causes and possible interventions." (Cassim, 2010, p. 181) addresses the global evidence based on the issue, exploring policy interventions practised in the more developed world as well as the local challenges in South Africa, which falls into the category of countries without statutory regulation of food marketing to children. Section 15(1) of the Foodstuffs, Cosmetics and Disinfectants Act 54 of 1972, Clause 52(2)(e) of the South African Government Gazette No.30075 prohibits advertising of foods "not regarded as part of a healthy diet and healthy lifestyle" to children under the age of 16 (Cassim, 2010, p. 184). This allows the food and beverage industry to resort to self-regulatory measures. "While industry commitment to the self-regulatory scheme continues across the globe, reservations are expressed at industry's ability to act in the interests of children since it is held that the goals of public health are fundamentally in conflict with the economic objective of business" (Cassim, 2010, p. 184).

2.4.1 What does the South African Government stipulate as healthy?

The South African revised food-based dietary guidelines (FBDG) released in 2013 advocate the consumption of a daily diet containing a "variety of foods" (Gibney, & Vorster, 2001). These dietary guidelines stand to be challenged. Considering what has been established about the human microbiome, the dietary advice - if prompt and simple in its delivery - in no way provides for a healthy microbiome. The alleviation of food choice being an informed choice is inexcusable being in direct contrast with the MODE model. Please consider this short excerpt in context:

"The FBDGs consist of 10 short, clear and simple messages which have been tested for comprehension, appropriateness and applicability in consumer groups of different ethnic backgrounds in both rural and urban areas" (Nutrition Society of South Africa [NSSA], 2013). The NSSA guidelines are as stipulated:

1. Enjoy a variety of foods.

Author's note: Variety needs to be specified, and food groups explained to allow for an informed choice.

2. Be active. Make starchy foods the basis of most meals.

Author's note: Starchy foods are insulinaemic foods, spiking the insulin level and only providing short- term satiety (Phy, 2015) therefore translate to simple carbohydrates

3. Eat plenty of vegetables and fruit every day.

Author's note: Plenty of fruit implies plenty of fructose processed by the liver immediately as available sugar, influencing insulin levels, contributing to excess weight (Elliott, Keim, Stern, Teff, Havel, (2002)

4. Eat dry beans, peas, lentils and soya regularly.

Author's note: Bioavailability, bioactivity and health effects of dietary phytoestrogens (mimicked by soya for example) strongly determine the intestinal bacteria of each individual (Landete et al., 2015)

5. Have milk, maas cheese or yoghurt every day

Author's note: Pasteurised dairy products are increasingly identified as inflammation markers (Rajilić-Stojanović et al., 2015).

6. Fish, chicken, lean meat or eggs can be eaten daily

Author's note: It should be pointed out, that free-range animals and pasture reared (not-grain fed) animals are the best choice, and that anti-biotic use needs to be considered - otherwise full nutritional benefits cannot be enjoyed. Advocating lean when it is a known fact, that high fat (LC/HFD) impacts positively on human health, is questionable to say the least (Pivovarova et al., 2015).

7. Eat fats sparingly. Choose vegetable oils rather than hard fats.

Author's note: This is crucially misleading and wrong. The choice should be based on saturated fatty acids versus trans-fatty acids; monounsaturated fatty acids versus polyunsaturated fatty acids and needs to be explained accordingly (Vučić et al., 2015). Fat is vital in the efficient uptake of vitamins (A, D, E, K) (Lehninger, Nelson, & Cox, 2008) and the digestion of macro-nutrients.

8. Use salt and foods high in salt sparingly.

9. Drink lots of clean, safe water.

10. Use sugar and drinks high in sugar sparingly.

Author's note: Sugar should be avoided and drinks/foods using sweeteners should be included & highlighted. Artificial sweeteners induce glucose intolerance by altering the gut microbiota (see Suez et al., 2014).

If people are kept in ignorance about the workings of their own body, they are restrained from making the right choice as the necessary reasoning is lacking.

High food prices, alongside growing inflation, increasingly restrict food choices (Schönfeldt, Hall, & Bester, 2013). "Unfortunately, even when the most basic and low-cost food items are selected to make up a recommended daily diet, the associated costs are well out of reach of poor individuals residing in South Africa" (Schönfeldt et al., 2013, p. 226). Food-based dietary guidelines alone therefore have little relevance in such circumstances where financial means limit food choice. Alternative interventions are required to equip the economically disadvantaged to follow recommended healthy diets and improve individual food intake and nutritional security.

If motivation and or capacity are inevitably influenced by the physical state of the person in question, it would not be unreasonable to assume that the physical side needs to be grounded before motivation or cognition can be adjusted. Motivational questions appear to be a luxury rather than of essence within the South African context - straining the importance of cost-effective ways to deal with the issues at hand. In regard to the consequences of long-term 'carbohydrate abuse', however, it seems a minor expense to improve on education and basic food supply in comparison to the looming costs of chronic disease implicated by carbohydrate-overconsumption. Although food and drugs of abuse act on the same central networks (Blumenthal, & Gold, 2010), food consumption is also regulated by peripheral signalling systems, adding to the complexity of understanding how the body regulates eating. Before treating pathological eating habits, it is therefore vital to consider and be aware of the possible path-effect causeways in the analysis process.

Pathological patterns of food consumption such as consistent overeating, binge eating, stress-induced binge eating and emotional eating bear a striking resemblance to substance-use

disorders (Blumenthal, 2010). They refer to studies of women who report on carbohydrate cravings whereby carbohydrates seem to have an almost medicinal value in women who crave them. Their findings support the conclusion that carbohydrates have an abuse potential: Over time, women who crave carbohydrates develop an increased preference for this type of food (desensitization) and tolerance to the food's ability to ameliorate dysphoria. Advertisements do their utmost to help trigger such food cravings.

Most people consume more food when stressed (Blumenthal, & Gold, 2010), demonstrating a preference for high-carbohydrate foods. Just as stress predisposes a drug addict to relapse, it is a significant cause of failure in dieters. Low levels or non-existent levels of self- regulation seem to have less and less to do with how disciplined or strong willed you are.

Upon investigating alcohol abuse in Coloured Western Cape communities in South Africa, Lesch and Adams (2016) established that knowledge is limited about the social and contextual factors that perpetuate this problem. Inspecting alcohol discourses of committed couples from one community led to the discovery of a lack of identification with problematic drinking of the participants. It is pointed out that the local alcohol policy and intervention efforts to address normative drinking discourses and practices in this research community are limited and insufficient. Alcohol translates into simple carbohydrates. "Contemporary research has shown that a high number of alcohol-dependent and other drug dependent individuals have a sweet preference, specifically for foods with a high sucrose concentration. Moreover, both human and animal studies have demonstrated that in some brains the consumption of sugar-rich foods or drinks primes the release of euphoric endorphins and dopamine within the nucleus accumbens (NAc), in a manner similar to some drugs of abuse" (Fortuna, 2010, p. 147).

The neurobiological pathways of drug and "sugar addiction" involve similar neural

receptors, neurotransmitters, and hedonic regions in the brain. There appears to be cross sensitization between sugar addiction and narcotic dependence in some individuals. Moreover, it has been observed that the biological children of alcoholic parents, particularly alcoholic fathers, are at greater risk to have a strong sweet preference, which may manifest in form of an eating disorder (Fortuna, 2010). "In the last two decades' research has noted that specific genes may underlie the sweet preference in alcohol- and drug-dependent individuals, as well as in biological children of paternal alcoholics. There also appears to be some common genetic markers between alcohol dependence, bulimia, and obesity, such as the A1allele gene and the dopamine 2 receptor gene" (Fortuna, 2010, p. 147).

Levels of foetal alcohol syndrome (FAS) in South Africa are the highest ever recorded (Parry, 2005). As Parry (2005) states more than ten years ago: "Roughly one in four adult males and one in 10 adult females experience symptoms of alcohol related problems, and almost one in four high school students report binge-drinking in the previous month; that is, drinking five or more drinks on one or more days." (Parry, 2005, p. 426)

If an understanding were facilitated of the addictive potential of simple carbohydrates and what food as well as beverage groups are representative of this, social discourse and acceptance levels could be influenced positively.

As in many African countries, studies in South Africa have indicated that heavier bodies among females are preferred even during adolescence, particularly in rural settings (Kimani-Murage, 2013). Thus, the fact that the average household income of the poor in South Africa equips many households to procure mainly low-cost staple foods such as maize meal porridge, with limited added variety, enforces addictive behaviour. An open discourse on the repercussions of the addictive potential of simple carbohydrates would induce change even on a cultural level - but unless a factual understanding is reached within a contextual setting,

little progress can be initiated. A lack of identification with the existing problem will persist and escalate over generations, as being overweight is socially accepted (and even aspired to) and synonymous to an image of desirability. Understanding the forces that sustain hedonic eating is essential to developing and implementing treatment and management strategies that address the root causes of the obesity epidemic. As Blumenthal and Gold (2010) outline, the conduction of PET and fMRI studies over decades have enabled the detailing of changes that routinely occur in human addicts, where neurobiological advances in modelling tobacco smoking, alcohol, and other drug addictions have enabled clinical scientists to study the brain systems of interest in humans. The provision of these neurobiological insights has changed physician acceptance of the importance of brain change in the addiction process (Blumenthal, & Gold, 2010).

The neurobiological evidence for food addiction is compelling, and the dependence as conceptualized with respect to alcohol and other drugs of abuse as a fundamental behavioural disorder will hopefully initiate public and elected leaders to become aware of food triggering an addictive process, which should be used to inform the public.

The sole responsibility in changing eating habits or educating consumers does not have to lie with the state alone. Taking the tobacco industry as an example: Public health and treatment professionals developed legal and taxation strategies aimed at delaying use, decreasing harm, and increasing protection from second-hand exposure to tobacco. New insights were utilised to provide replacement, detoxification, anti-craving and relapsing medications. Education about the harmful effects of tobacco are not to be missed anymore: every packet carries an educational warning.

If hyper palatable, processed foods are capable of triggering an addictive process, the negative impact of any addictive potential associated with these foods should not be enhanced by their cheapness, accessibility and exceptional marketing. Amongst increasing

the public health burden, they minimise the consumer awareness of their potential to cause harm.

South Africa is faced with two paradigms in terms of curbing addictive behaviour - the unsurmountable problem of poverty coupled with the healthcare system's blatant ignorance of, as well as its inaccessibility to, two thirds of South Africa's population. Overall then we are faced with the possibility of:

A) Changing nutritional paradigms to change behaviour and improve overall physical well-being, or

B) Attempting to change the healthcare system overall (e. g. by making chronic medication more affordable and accessible while concentrating on symptom alleviation rather than tackling the root of the problem).

Which allows for a more 'functional' and self-sustaining society? Which is financially more viable? Stakeholder interests need to be considered, regulatory philosophy must be challenged and regulatory tools revised. Several issues need to be addressed before any decision is made, as Gearhardt, Robert and Ashe state, "... whether taxes or bans on products change consumer behavior or are an unnecessary burden and intrusion on consumers; whether a reduction of farm subsidies and sugar programs (e.g. sugar tax as introduced recently) effectively reduce sugar consumption; and whether class action litigation furthers public health objectives by changing the production and marketing of sugar products and the consumption habits of consumers." (Gearhardt, Roberts, & Ashe, 2013, p. 49).

Just as the tobacco industry was forced to educate consumers as well as pay special excise duties, value-added taxes and sales taxes, so could the food industry be coerced into supporting education and paying extra taxes to fund education, make nutritionally beneficial substrates more available and in turn, non-beneficial foods costlier.

According to Lambert and Kolbe-Alexander (2013), over 50% of South African adult women and 30% of adult men are either overweight or obese, and nearly half of all adults are insufficiently active, with major increases in obesity-associated healthcare expenditures since 1980.

An incentivised programme is of the essence, substantiated by fact and initiated by the powers that be. Proof of nutritional intervention influencing consequent behaviour potentially curbing addictive behaviour could initiate a change in directional guidelines starting at school level, assuring a brighter future for all at minimal cost while assuring long-term viability.

2.5 ADDICTION: AND THE KETOGENIC/ LOW CARB & HIGH FAT 'DIET'

To tackle the problem of an addiction we must accept that there is one. While the culprit is identifiable as carbohydrates (simple sugars, e.g. starches) - it is widely used as our primary fuel source. Why is that so?

As Zilberter (2012) points out, macronutrients do play a crucial role in determining a diet's behavioural and metabolic consequences. But which ones do we need? And how can you swap one fuel primary for the other? In the case of the ketogenic diet, a first reaction is an outcry of: How can a high fat diet be good for you? The reluctance to accept current research proving the 'fat makes you fat' hypothesis wrong, deters practitioners as well as patients. Bazzano et al. (2014) have shown a low-carbohydrate diet to be more effective for weight loss and cardiovascular risk factor reduction than a low-fat diet. Therefore, restricting carbohydrate intake may be an option for persons not only seeking to lose weight but also reduce cardiovascular risk factors. According to Dehghan et al. (2017) high carbohydrate intake was associated with a higher risk of total mortality, whereas total fat and individual

types of fat were related to lower total mortality. They suggest that global dietary guidelines should be reconsidered in light of these findings. Fat isn't just fat, there is good and bad.

The type of fat ingested plays an important role: "Low-carbohydrate diets have been avoided because of the high-fat nature of the diets and the predicted associated hypercholesterolemia. However, serum lipids generally improve with the low-carbohydrate diet, especially the triglyceride and HDL measurements. In sharp contrast, high-carbohydrate diets, which reduce high-density lipoprotein (HDL) cholesterol and raise triglyceride levels, exacerbate the metabolic manifestations of the insulin resistance syndrome" (Manninen, 2004, p. 9). As Manninen's (2004) findings reveal, all fats raise HDL cholesterol, whereby the relative potency of fatty acid classes in raising HDL cholesterol is from saturated to monounsaturated and then polyunsaturated. Thus Manninen (2004) concludes that replacement of total fat (of any fatty acid distribution) with carbohydrates results in significant reductions in HDL cholesterol. "Indeed, recent studies of carbohydrate intake and its relationship to the development of CHD and type-2 diabetes have been rather revealing, showing that an increase in carbohydrate intake is related to increases in both conditions" (Manninen, 2004, p. 9). The discussion on healthy versus unhealthy fats exceeds the scope of this study - it is, however - an important player in the constellation of the gut microbiome and its functioning. Stipanuk's (2006) elaboration on the biochemical, physiological, & molecular processes of fat ingestion underline and support Manninen's findings.

Since overweight individuals generally prefer highly processed foods containing simple sugars rather than complex carbohydrates, a low-fat diet could actually encourage the consumption of sugars and refined carbohydrates. Additionally, adherence of overweight individuals to the conventionally accepted high carbohydrate/low fat nutrition is problematic as the majority have been shown to have dietary preferences for foods with a rich processed fat content (Paoli, 2014). As a consequence of the debatable efficacy of these diets and the

afore-mentioned ‘addiction- factor’ of carbohydrates (Freedman, King & Kennedy, 2001), a very low carbohydrate/ ketogenic diet is the regimen of choice in this study. Not only does it minimise the carbohydrate intake, but the ketogenically inclined diet could introduce a beneficial metabolic condition named ‘ketosis’ by Hans Krebs, which is not to be confused with the pathological diabetic 'ketoacidosis' (Krebs, 1966).

As the central nervous system cannot use fatty acids as an energy source (unlike glucose, they do not cross the blood-brain barrier), glucose is ordinarily the sole fuel for the human brain (Hartman et al., 2007). After 3-4 days of fasting or a very low carbohydrate diet the central nervous system needs an alternative energy source, which is derived from an overproduction of acetyl-COA, leading to the production of so-called ketone bodies (KB) (Paoli, 2014). This is called ketogenesis and occurs principally in the liver. The liver produces ketone bodies, but is unable to utilise them. Since Ketone bodies and glucose have similar kinetics, the dependence of velocity on substrate [i.e. ketone bodies and glucose] can be described for many enzyme-catalyzed reactions by the Michaelis-Menten equation (Lehninger, Nelson, & Cox, 2008). For the glucose transport to the brain, ketone bodies begin to be utilised as an energy source by the CNS (central nervous system) when they reach a certain concentration (about 4mmol/L) (Veech, 2004). The Kreb’s Cycle or glucogenesis is explained in biochemistry textbooks such as Nelson and Cox (2008). The CNS efficiently uses these molecules for energy in place of glucose.

There is not only strong supportive evidence that the use of ketogenic diets (KD) in weight loss therapy is effective, but also that KD lead to a natural reduction in appetite due to a higher satiety effect of proteins and influences on appetite control hormones (Westerterp, 2009). Other hypothesized mechanisms of the KD’s weight loss, as summarised by Paoli (2014, p. 2097), are:

“- A possible direct appetite suppressant action by ketone bodies

- Reduction in lipogenesis and increased lipolysis

- Greater metabolic efficiency in consuming fats highlighted by the reduction in resting respiratory quotient

- Increased metabolic cost of glucogenesis and the thermic effect of proteins"

The KD not only has beneficial effects on fat and weight loss, but, as Paoli, Bianco, Grimaldi, Lodi, and Bosco (2013) have suggested, ketones may protect from cognitive impairment caused by weight gain and obesity. Although subjects may complain of lethargy during the first 3-5 days of the 'fuel-switch', the effect passes rapidly and subjects subsequently report an improved mood (Paoli, 2014). The beneficial effects of low carbohydrates are not just a function of weight loss per se but also improve glycaemic control, haemoglobin A1c, and lipid markers, which more often than not lead to reduced use or withdrawal of insulin or other medications (Paoli, 2014).

El-Mallakh and Pasketti already observed in 2001 that several anticonvulsant interventions such as KD may improve outcome in mood disorders (El-Mallakh, & Paskitti, 2001). Furthermore, beneficial changes in brain-energy profile were noted in subjects on the ketogenic diet. This is important since global cerebral hypometabolism is a characteristic of the brains of depressed or manic individuals. "The extracellular changes that occur in ketosis would be expected to decrease intracellular sodium concentrations, a common property of all effective mood stabilizers. Trials of the KD in relapse prevention of bipolar mood episodes are warranted" (El-Mallakh, & Paskitti, 2000, p. 724).

Ketogenic/ LCHF Diets have been extremely successful in epilepsy treatment, cancer and neurodegenerative diseases (Paoli, 2014). Important for this study however, are Paoli's findings on the Yo-Yo effect. He demonstrates that two brief periods of KD separated by longer periods of maintenance on a Mediterranean diet, led to successful long term weight

loss and improvements in health risk factors without weight regain effect (Paoli et al., 2013). Despite 'loosening' the parameters of the KD regimen while maintaining a high fat, low carbohydrate content, no fall-backs were observed. A large study undertaken in Europe demonstrated that an increase in protein content and a reduction in the glycaemic index led to better maintenance of weight loss without differences regarding adverse effects (Paoli, 2014).

It needs to be understood, that, as with any other addictions, the addictive substance is not to be re-introduced. Essentially this implies that a KD or any form of low carbohydrate diet for that matter, is not a 'diet' - an intermittent, passing phase - but the start of the rest of the patient's life. Most low-carbohydrate, paleolithic forms of nutrition are introduced on a 30-day trial run: cutting out all foods with allergic potential (e.g. wheat, gluten, dairy) to allow the body a degree of re-sensitization. The body needs time to reset and rid itself of potential irritants and inflammation, before healing and sensing can even take place. A full thirty days of no cheats, slips, or "special occasions" is required, because: "Only a small amount of any inflammatory foods breaks the healing cycle—one bite of pizza, one splash of milk in the coffee, one lick of the spoon mixing the batter within the 30-day period and you've broken the "reset" button, requiring you to start over again." (*The Whole30 Program*, Retrieved from http://whole30.com/whole30-program- rules, September 2016).

Carbohydrate over-consumption is to be seen and treated as an addiction. Building on addiction neuroscience models, neuroscience studies have demonstrated avid self-administration of glucose, fructose, and junk food in laboratory animals with corresponding changes in addiction-relevant neurotransmitters and systems. The potential role of the KD in depression has been studied in the forced choice model of depression in rats, which led to a beneficial effect similar to that afforded by conventional antidepressants (Murphy, Likhodii, Nylen, & Burnham, 2004).

Human neuroimaging studies, also using methods developed over decades of studying

human alcoholics and addicts, have suggested that hedonic food can act like a traditional drug of abuse, causing brain changes almost indistinguishable from those produced by drugs (Blumenthal & Gold, 2010).

"Studies have shown somato sensory cortex and other neurobiological changes that make losing weight much more difficult. In the absence of consistently effective lifestyle or pharmacological interventions that address these root causes, families are increasingly turning to invasive and expensive bariatric surgical treatments, including gastric bypass and gastric banding, to help themselves and their children lose weight. These procedures can yield dramatic weight loss, but also have significant, well known, and potential side-effects" (Blumenthal & Gold, 2010, p. 364).

Psychosocial outcomes of excess weight and obesity and the attendant complications, place an ever increasing, non-manageable burden on healthcare systems and society. The limitations of current treatments should be compelling enough to consider a cost-effective (long-term), self-empowering, non-medicating and extremely promising approach. A KD (or extremely low-carb) diet could not only offer a perspective on the 'obesity' problem but potentially offer a way out of the carbohydrate addiction.

Indirectly speaking, the KD/ LCHF would heighten the subjects sense of self-efficacy. The construct of self-efficacy, or 'believing that one can', relates to the power of positive thinking. When subjects think in a self-efficacious manner they believe themselves capable of doing something effectively. Self-efficacy is defined as a person's judgement about his or her capability to successfully perform a particular task. Judgements such as these would relate to level of performance expected, the strength or certainty of those attainment beliefs, and the generality of those beliefs to other related tasks or domains (Bandura, Freeman, & Lightsey, 1999).

Successful weight-loss and the ability to control the amounts you eat will not only make the subject look more according to societies 'expected norms', it will make a change from addiction to one of conscious choice. The KD/low carb diet may pave the way for successful implementation by working through the mechanism of a primary 'fuel-change', allowing the body to function and heal itself properly, lessening appetite and cravings, elevating mood and leading to a greater sense of self-efficacy, coupled with a renewed sense of self physically and psychologically.

While the perceptions on low carbohydrate eating regimens are shifting along with Tim Noakes scientific discoveries and legal battles (Harcombe & Noakes, 2016) on the subject in South Africa, the researcher has opted to adhere to the guidelines of a low carbohydrate high fat nutritional plan excluding dairy (Banting heavily relies on dairy as a staple source of healthy fat). Since most chronic inflammatory diseases (e.g., obesity, diabetes) as well as allergic diseases are strongly influenced by nutrition, the metabolism of food being intimately associated with inflammatory processes (Hotamisligil, 2006), the researcher has chosen to avoid unnecessary complications in form of allergens and unexpected food reactions accountable to processed dairy products.

The relatively high concentration of dietary antigens in cow milk for example has raised concern and some scientists are convinced that dairy products are a major cause in the development of chronic inflammatory disorders and autoimmune diseases (Melnik, 2009). As Bordoni et al. (2017) state, the properties of the foods investigated in many human nutritional trials often lack documentation, making an objective evaluation of any clinical outcome very difficult. While narrowing the gap between food science and nutritional science is ideal, this study focuses on the impact on addiction severity that a LCHF intervention can have and merely excludes potential allergens, which could have influenced the outcome of the study. The nutritional regimen used in this paper recognises the potential inflammatory properties

dairy has been documented to have, but does not seek to demonstrate or detect such.

Tim Noakes, together with his co-author Harcombe, managed to sway professional opinion within academic circles based on scientific evidence. Hopefully this study adds a little to the movement forward in nutritional science and the debunking of many opinions and beliefs built on nutritional industry aims and gains. Although every researcher or scientist is probably driven by their own interests and beliefs, personal opinions should be honestly acknowledged and at the same time be avoided in scientific methodology.

2.6 RESEARCH PROBLEM

The research problem explored in this study was whether a low carbohydrate, high fat nutritional intervention would have a positive effect on addictive eating behaviour. This research problem was investigated using three research questions, which are set out further down, and a small convenience sample of participants.

2.6.1 Measurement need identification and research questions.

The main research aim was to establish whether a nutritional intervention had a positive effect on the weight maintenance of the participants. A pretest-posttest research design was used, and the respective BMI measurements, waist-to-hip ratios, and weight of the participants were tracked to determine if the intervention had a positive physical effect on weight and body composition. The main research assumption was that a shift in body composition would indicate that the reduction of carbohydrates initiated a ‘controlled’ form of eating and thus prevented weight gain.

Research question one was therefore defined as:

RQ1: Research Question 1

Is there a measurable difference in body composition before and after the intervention?

This entailed two sub-questions:

Is there a measurable difference in the weights of the participants before and after the intervention?

Is there a measurable difference in the BMI of the participants before and after the intervention? Additionally, waist-to-hip ratios were taken before and after the intervention, to establish if there had been a change in body composition.

Since the study's main concern is that of addiction, rather than weight loss, an assessment of the addiction severity from before and after the intervention was essential. Participants were assessed according to the YFAS guidelines and questionnaires to answer research question 2:

RQ2: Research Question 2

Is there a measurable difference in addiction severity before and after the intervention?

In support of the YFAS assessment and the definitive physical measurements it was deemed useful to introduce a third indicatory measurement which, albeit subjective and reported by self- observation of the participants alone, rendered a reflection by the participants on the impact of the intervention. In this part of the research the participants were asked to report on their feeling of 'being in control' of their eating behaviour, and an increased feeling of

control was seen as acting in support of feeling less addicted. This leads to research question number 3:

RQ3: Research Question 3

Is there a noticeable self-perceived improvement in 'feeling in control' after the intervention?

The results reflected in combination of all three questions were considered to be indicative of the following possible outcomes:

Whether there was a difference in body composition from before and after the intervention. If so, was it in support of the hypothesis (no weight gain/no change in BMI) or not (weight gain/ change in BMI)?

Whether there was a measurable difference in addiction severity before and after the intervention. If so, was it in support of the hypothesis (less addiction severity) or not (feel more addicted)?

Whether the participants felt a difference in control after the intervention. If so, was it in support of the hypothesis (feeling more in control) or not (feeling less in control)?

CHAPTER 3
METHODOLOGY

3.1 RESEARCH DESIGN

A quasi-experimental one sample, pretest-posttest design was used to determine whether a change to a LCHF diet could have a significant effect in curbing the addictive eating behaviour manifested by the participants in the pre-intervention phase.

A sample could not be selected at random, as the participants had to have a clean medical bill whilst showing concern regarding their lack of control over their eating behaviour. Since the independent variable, namely the intervention, involves a change in the participant's nutritional regimen with the aim of establishing a change from before and after (less addiction) the intervention, and the intention was not to compare the proposed eating regimen with the outcome of another regimen, a control group was not employed. The dependent variables (measured before and after the intervention) were analysed in three ways to determine if the posttest measurements were indicative of less addiction. Thus the results were analysed to determine if there was:

1. A change in body composition with regard to the variables weight, waist-hip ratio and BMI from before and after the intervention.
2. A measurable difference in addiction severity reflected in the posttest scores obtained by the participants on the YFAS instrument.
3. A measurable difference in feeling in control by the participants as measured on a simple (0-3) scale.

The treatment was governed by the researcher's instructions to the participants on how to eat

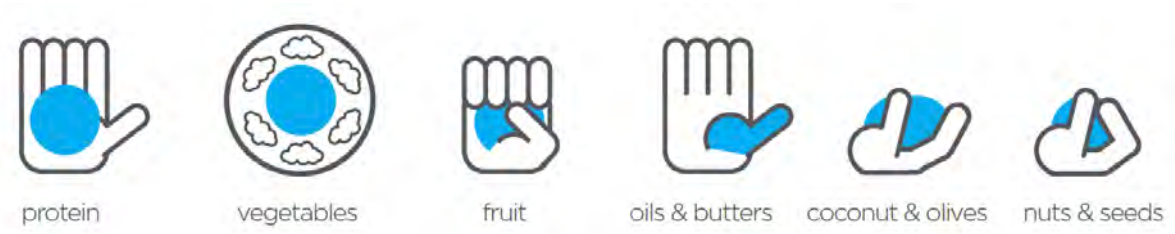
and cook according to a LCHF meal plan. The meal plan was developed by the writer in conjunction with a nutritional expert, and the LCHF meal plan was introduced to each participant personally. To find a suitable definition of individualised portion size, hand measurements were referred to as portion size indicators (Figure 1).

Ratios are similar when it comes to macronutrient ratios. We can thrive on a very large array of macronutrient intake, but the estimated optimal intake is at around 20% carbohydrates, 65% fat and 15% protein by caloric intake, not weight (The Question of Macronutrient Ratios | Paleo Leap, 2017).

Being easy to understand and practical to translate (e.g. the protein content of your meal should fit into a portion of your hand, see fig. 1-6), it was thankfully adopted by participants, who generally noted an aversion to weighing food portions.

Figure 3.1.1

Illustration of food group portions



The LCHF plan offered each participant an introduction, a general guideline and a FAQ section with the most commonly encountered issues in a low carb intervention. It explains the reasoning behind the intervention (called a 30-day reset), what it may do, how it works and why it is thought of as beneficial in various disease conditions - while it is to be regarded as a lifestyle change rather than a diet. The LCHF meal planner clearly stipulated

what food to eat and not to eat, what to do in moderation and what can be seen as being allowed in liberal consumption. It also supplied the participants with eating lists according to food groups called a core food plan, whilst educating on food additives and ready-made foods. Additionally, participants were supplied with two hand-outs outlining the different terminologies used for 'hidden sugars' and the kinds of things a pantry should and should not contain, to enable self-motivated decisions and facilitate understanding (Appendices 6, 7, 8).

Any participant not adhering to the LCHF regimen was asked to discontinue out of their own volition. Knowing that any 'lifestyle' intervention such as the LCHF eating regimen can be very disruptive, the LCHF Plan anticipates situations such as going for dinner, what to do when exercising and NOT to use a scale, all in light of reducing anxiety and stress of the participants. Overall, the plans were received very well because of their ease of use, practicality and readily adoptable information.

While full control could be expected upon having participants willingly enrol in the programme, and counselling was made available by the nutritional expert and the researcher during the time of the intervention, the control over what participants ate and did not eat remained the patient's responsibility alone. Educational intro sessions (90min) elaborating on the causal factors underlying the treatment were held before the intervention and all participants attended. Participants could withdraw from the study at any stage. As stipulated, the participants were assessed before and after the nutritional intervention to enable answering the following research questions:

RQ 1

Is there a measurable difference in body composition before and after the intervention?

Considering the biological background of carbohydrate addiction and the physiological means of digestion and fuel usage, a switch in the main energy source can be observed both physically and mentally. Physical observations consisted of two measurable components in answer to two sub-questions:

A) Did the participants BMI change from before and after the intervention?

B) Did the participants weight change from before and after the intervention?

The participants were weighed and their waist to hip ratio was recorded before the start of the intervention and after.

RQ 2

Is there a measurable difference in addiction severity before and after the intervention?

To increase the cost-effectiveness of the study, it was opted to utilise a questionnaire to ascertain the addiction severity. The Yale Food Addiction Scale was used as an assessment tool before and after the intervention to investigate whether there was any change in the addiction symptom severity from before and after the intervention.

RQ 3

Is there a noticeable self-perceived improvement in 'feeling in control' after the intervention?

A qualitative survey assessing the 'feeling more in control' was performed by asking all participants to judge this on a scale from 0-3 after the intervention via e-mail. Subjective experiences were also recorded in writing following voluntary talk and question sessions

after the intervention, assessing the general sense of well-being and the ability to adhere to the nutritional regimen in support of an expected physiological and psychological change. The experience reports did not contribute to the statistical evaluation.

3.2 SAMPLING

A sample of 32 participants were recruited with the aid of online/ word by mouth adverts and by approaching potential clients from the ThinkFood clinic (also via website) for the study. Participants were selected amongst the South African population based on a self-confessed inability to adhere to healthy food-choices or harbouring a weight concern. Potential participants qualified as being suitable based on self-confessions such as 'eating is out of control' and 'wanting to lose weight but being unable to' (loss of control).

Participants were informed of the outcome, procedure, nutritional treatment regimen and aim of the study, namely to determine a severity score on the YFAS addiction measure while determining physiological as well as psychological changes. Eligible participants were between ages 18 and 80 with a BMI of at least 18.5. They had to have a 'clean bill of health', not have any currently diagnosed inflammatory, chronic condition or suffer from any other debilitating state, mentally or physically. They were asked to testify to not taking insulin or Coumadin (warfarin). Participants could not have kidney stones, heart failure, angina or liver cirrhosis. They had to be willing to provide bodily measurements and to fill out questionnaires before and after the intervention. Racial and ethnic minorities were encouraged to apply for this study.

3.3 PROCEDURE

All participants were pre-screened to determine a satisfactory health profile before the start of the intervention. All participants partook in the YFAS questionnaires (before and after), completed the physical assessment (waist to hip ratio & body weight) and indicated a qualitative 'feeling in control' measurement after the assessment (online via e-mail). Some handed in additional 'daily diary' notes on the mental and physical well-being as well as any notable changes of interest observed.

The participants filled in the relevant YFAS questionnaire before and after the intervention (see Appendix 3). Only fully completed questionnaires were accepted. The completion of the questionnaire was explained in detail and assistance in completion (i.e. facilitating the understanding of a question as well as necessary translation) was available at all times. A nutritionist representative of the current understanding of a low carb high fat regimen provided the nutritional intervention guidelines and plans. Due to financial uncertainty and future usage, the questionnaire had to be based on the English language. However, if it had become evident during the selection/ pre-screening of the candidates that language proficiency and/or literacy could be a problem, necessary measures such as fully translated study questionnaires would have been considered. All participants were asked to contact the researcher in case of any language difficulties or problems of understanding.

The completed forms, measurements and other data are confidential and remain accessible to the researcher only. There was a 100% compliance by the participants to the low carbohydrate, high fat diet (LCHF), and therefore subjective observations could be made about various changes in the participants that were possibly due to the intervention (e.g. weight loss, general sense of well-being, ability to adhere to the nutritional regimen). This was in addition to the actual data that were collected during the course of the research.

3.4 DATA ANALYSIS PROCEDURES

3.4.1 RQ1 Measuring differences in body composition before and after the intervention

Physical measurements were manually taken by every participant and recorded as part of the YFAS questionnaire. Group means and BMIs were calculated and recorded. Although the study aimed at reflecting a change on addictive behaviour rather than to pacify weight loss or bodily image concerns, the researcher considered the possibility of participants 'falsifying' their information to improve their self-representation. Since the researcher met all participants before and after the study, a gross falsification of values would presumably have been noticeable to the researcher based on a simple visual comparison of the participant's physical appearance before and after the intervention.

3.4.2 RQ2 Measuring differences in addiction severity before and after the intervention

The YFAS criterion, group means, and standard deviations before and after the intervention were calculated according to the YFAS Instruction Sheet (Appendix 5), using the YFAS Excel formula as stipulated by Gearhardt, Corbin, & Brownell (2009). After computing the cut-offs, the questions were summed up under each substance dependence criterion (e.g. Tolerance, Withdrawal, Clinical Significance, etc.).

According to Gearhardt, Corbin and Brownell (2009), the YFAS exhibits adequate internal reliability and shows good convergent validity with measures of similar instruments as well as good discriminant validity relative to related but dissimilar instruments. The YFAS

predicted binge-eating behaviour above and beyond existing measures of eating pathology, demonstrating incremental validity. “It is a sound tool for identifying eating patterns that are similar to behaviours seen in classic areas of addiction” (Gearhardt et al., 2009, p. 430), which is highly relevant in the case of carbohydrate addiction. Flint et al. (2014) have established that the YFSA is a psychometrically sound tool to identify food addiction, it translates the substance-dependence diagnostic criteria to apply to the consumption of highly palatable foods. “Elevated scores on the YFAS have been linked to more frequent binge-eating episodes, elevated impulsivity, increased depression, higher rates of craving, reduced weight loss in response to treatment, and elevated weight regain after bariatric surgery” (Flint et al., 2014, p. 578).

The YFAS can return a dichotomous outcome for a ‘food addiction’ diagnosis (or not), but can also exhibit a continuous outcome. Whereas the scoring instructions according to Gearhardt et al (2009) attach no significance to the continuous scores within the framework of the YFAS, an inclusion and evaluation of the continuous outcome score was decided upon by the researcher. Since the premise of the study was not to ascertain a persistent addiction in participants, but rather to establish whether an improvement in addictive symptoms had occurred from before and after the intervention in a small population sample based on the premise of a ‘carbohydrate addiction’, the continuous scores of symptoms were seen as being of considerable significance. Some questions could be seen successful in minimising ‘addictive behaviour’ only in the reverse scores in context of this study and other questions were questionable in their application on account of their overt emphasis on extreme eating pathologies (see detailed elaboration in INSTRUMENTS, 3.5.5).

Some researchers (e.g. Gearhardt, Corbin, & Brownwell, 2009) maintain that a symptom count should be employed in community samples when the ‘food addiction’ diagnosis presumably has a relatively low prevalence in this population. Whilst candidates

with a tendency towards 'weight issues' or a self-perception of 'uncontrolled' eating where sought out, none of the participants came from a community identified as having overt eating disorders or weight issues, nor were they classified according to any other 'eating disorder' scale such as binge-eating (Gearhardt, Corbin, & Brownell, 2009). Also, the YFAS measures a single trait whereby most items load onto that factor (Meule, Heckel, & Kubler, 2012). While the study employs a 7-point answer scale (5-point subscale), considering the use of continuous scoring in addition has three potential justifications. Firstly, it may be argued that 'food-addiction' is not an all-or-nothing attribute. Secondly, some individuals might be ignored by counting only extreme answers beyond a certain threshold. Finally, dichotomizing continuous variables could lead to a loss of information, or add errors of discreteness to the measurement error in the original scales (Cohen, 1983). Adding error to measurement scores would result in scores with lower reliability, therefore lower validity.

Dichotomous scoring may have some disadvantages compared to continuous scoring such as the reliability of the scores from the dichotomous scoring procedure being lower than that of the scores from the continuous scoring procedure (Cohen, 1983).

To score the continuous version of the scale, all the scores were added up for each of the criteria (e.g. Tolerance, Withdrawal, Use Despite Negative Consequence). The scores ranged from 0 to 7 (0 symptoms to 7 symptoms) when all questions were included (except for the 'primer questions', 17, 18, 23) before and after the intervention.

To score the dichotomous version (addicted versus not being addicted), a variable (determining addiction or non-addiction from an average of all symptom counts from before and after the intervention per individual) was computed in which clinical significance was equal to 1 (items 15 or 16 =1), and the symptom count was bigger than 3. This rendered either a 0 or 1 score. If the score for the criterion was > 1, then the criterion was recorded as met, and scored as 1. If the score equalled 0, then the criteria were not met. Dichotomous and

continuous scoring was applied to minimise error, enabling internal reliability score assessments and allowing for a reflection of all symptom counts. The results of test one and test two were compared on an individual and group average basis.

Finally, the reliability was determined using the Cronbach's alpha measure of internal consistency. The Cronbach alpha correlation coefficient was calculated before and after the intervention including questions 4, 22, 23, 24, 25 (all questions pertaining to symptom 2). In support of the argument on questioning the ambiguity and applicability of the questions within the study context and premise (see Appendices 12-16) the same procedure was applied to determine the relevant Cronbach alphas without questions 4, 22, 23, 24, 25 (all questions pertaining to symptom 2). The statistical program 'R' was used for all statistical calculations (see appendices 17).

3.4.3 RQ3 Measuring self-perceived improvement in 'feeling in control' after the intervention

Following the intervention, all participants who had successfully completed all requirements were asked to assess their sense of 'feeling in control' in regard to their eating behaviour by rating their sense of improvement in their 'feeling in control' on a scale of 0-3, whereby 0 implied no improvement, 1 a slight improvement, 2 a noticeable improvement and 3 a significant improvement. The group mean was calculated (see Appendix 11).

3.5 INSTRUMENTS

3.5.1 Choosing the Yale Food Addiction Scale (YFAS)

There is a current lack of psychometrically validated measurement tools in the field of

eating pathology. While there are a multitude of measures and approaches, ranging from neural co-relates of food addiction (monitoring brain activity or heart rate and salivation) to self-reported responses, the Yale Food Addiction Scale (YFAS) was selected as the most suitable measure in the context of this study. Since neural correlates and any form of neuro-imaging would have required a clinical setting and adequate financial funding, the researcher of this study opted to employ a widely implemented (e.g., Flint et al., 2014; Meule, Heckel and Kübler, 2012; Clark and Saules, 2013) and successfully validated measure such as the YFAS as stipulated by Gearhardt, Corbin and Brownell (2009). While there are several self-report measures broadly dealing with the topic of eating pathology such as the Forbush et al.'s 2013 Eating Pathology Symptoms Inventory (EPSI), the YFAS specifically targets or singles out addictive behaviour as a measurable outcome (Forbush et al., 2013, Forbush 2015).

Since this study aims at using the YFAS 'only' to validate the addictive symptoms while measuring the degree of severity from before and after a nutritional intervention to prove that the nutritional intervention has incurred change, wider measures of severe eating pathologies were not deemed appropriate. The subjects in the study were consciously chosen from the normal, average, and healthy population to show that addictive behaviour in regard to simple carbohydrate consumption co-exists in the lean as well as what is considered pathological, such as the obese population. The measure sought out in this study therefore had to be non-biased about pre-existent eating pathology, accepting the concept of the possibility of a food-addiction based on carbohydrate consumption without limiting the addiction to a certain food-group (such as sweets or red meat). Additional scales or measures such as the Difficulties in Emotion Regulation Scale (DERS), Emotional Overeating Questionnaire (EOQ), Beck Depression Inventory (BDI), and Eating Disorder Examination Questionnaire (EDE-Q) were not considered necessary, but could presumably form part of a

larger assessment battery. This study investigated the possible curbing influence of a nutritional change on addictive eating behaviour, while presupposing that simple carbohydrates invariably cause a form of such behaviour which is not necessarily measured by conventionally defined eating pathology markers (e.g., obese or bulimic or anorexic as suffering from a compulsive eating disorder), and yet is an eating problem common to all human beings of all shapes, sizes, colours and ages. Food addiction may be seen as part of the obesity framework, but essentially spans across all human beings basing their staple food intake on simple carbohydrates, and such 'substance dependence' can be seen as a behavioural disorder defined by the experience of the individual rather than his or her physiology. These behavioural indicators of food dependence corresponding to the criteria of substance dependence could be examined by the YFAS, which is therefore, albeit with reservation, the measure of choice in this study.

3.5.2 Evaluation of the Yale Food Addiction Scale (YFAS).

Converging research suggests phenomenological and neurobiological similarities between excessive food consumption and addictive behaviour in substance dependence. Meule, Heckel and Kübler (2012) report on the YFAS having been employed successfully as an assessment measure for addictive behaviour (having been administered to obese patients seeking bariatric surgery).

3.5.3 YFAS Measurement development.

The Yale Food Addiction Scale (YFAS) is a means to assess food addiction in accordance with DSM-IV criteria for substance dependence. It was assumed that although the

goal of the study is not to validate the actual addiction, the YFAS could serve as a tool to measure the degree of severity of addictive behaviour either subjectively perceived or displayed by the participants. The YFAS is composed of 25 questionnaire items that are used to assess diagnostic criteria for food addiction (Flint et al., 2014), and it translates the diagnostic criteria for substance dependence outlined in the Diagnostic and Statistical Manual for Mental Disorders-Text Revision-IV to apply to the consumption of high-fat and-sugar foods. The YFAS is meant to provide both a count of food addiction symptoms and a diagnosis of food addiction as scoring options. Its diagnostic threshold measure is based on the presence of 3 (of 7) addiction symptoms in addition to the presence of significant impairment or distress.

A combination of dichotomous and frequency scoring is used to capture diagnostic criteria. Frequency scoring is used to assess behaviour that could occur in non-carbohydrate addicts (i.e., per chance excess consumption, emotional eating), and dichotomous scoring is used for questions that would potentially relate to carbohydrate addiction behaviour (e. g. continue to consume carbohydrates even though it severely impacts daily functioning: tiredness, physical incapacity).

According to the instructions of the developers, continuous scoring was used on the researcher's discretion with a seven and a six symptom count to compare the internal validity upon necessary scoring adjustments before and after the intervention, whereby Cronbach's alpha coefficient was applied as an internal reliability measure.

3.6 RELIABILITY AND VALIDITY.

Researchers developed the continuous items in the YFAS scale based on existing measures of eating pathology (Gearhard et al., 2009). Exploratory factor analysis was used to determine

the factor structure of the instrument, and eleven diagnostic criteria were determined in this way.

Convergent validity was assessed by examining correlations between other well established predictors of eating pathology and the YFAS. Discriminant validity was assessed by examining correlations between well-validated measures of alcohol use and related problems. Incremental validity was investigated using multiple regression (Gearhardt et al., 2009).

3.6.1 Concerns regarding the YFAS and its application in this study

While the prevalence of each symptom in the validation of the Yale Food Addiction Scale has been developed to measure the existence of the diagnostic criteria as applied to eating behaviour, the use of several DSM-IV indicators of dependence has received some criticism. According to Gearhardt, Corbin and Brownell (2009), the scale questions fall under criteria resembling the symptoms for substance dependence according to the Diagnostic and Statistical Manual of Mental Disorders IV-R.

While the researcher of this paper has made some amendments based on question relevance and their applicability for the research in question, Gearhardt, Corbin and Brownell (2009) have structured the questions in the following way:

1) Substance taken in larger amount and for longer period than intended (thresh. 4)

Questions #1, #2, #3

2) Persistent desire or repeated unsuccessful attempt to quit

Questions #4, #22, # 24, #25

3) Much time/activity to obtain, use, recover

Questions #5, #6, #7

4) Important social, occupational, or recreational activities given up or reduced

Questions #8, #9, #10, #11

5) Use continues despite knowledge of adverse consequences (e.g., failure to fulfil role obligation, use when physically hazardous)

Question #19

6) Tolerance (marked increase in amount; marked decrease in effect)

Questions #20, #21

7) Characteristic withdrawal symptoms; substance taken to relieve withdrawal

Questions #12, #13, #14

8) Use causes clinically significant impairment

Questions #15, #16

In addition to general concerns regarding the DSM IV-R definitions, some questions were seen by the researcher as needing refinement but only in the follow-up questionnaire that was used after the intervention. Extra care (direct follow up questioning and continuous scoring) was taken to capture the participants' true intent in answering the questions.

A) Tolerance as an indicator could be argued as not being unique to addiction, but rather accompanying the frequent use of a substance regardless of the dependence symptoms. It is possible therefore, to exhibit signs of physical dependence on a substance without receiving a diagnosis of substance dependence (e.g. caffeine). Hence, the evidence of withdrawal of a substance is not associated with all addictive substances. The YFAS however, directly assesses the tolerance to foods that have addictive properties (Gearhardt, Corbin, & Brownell, 2009).

B) Loss of control is met in substance dependence if the consumption occurs in larger quantities or/and over a longer period of time than intended. Here, emphasis is placed on the subjective experience of control spanning over time. Such behaviour seems to be

captured within the criteria of Eating Disorder not otherwise specified (EDNOS). The Eating Disorder Examination (EDE) was used to generate DSM-IV diagnoses, but, according to Turner and Bryant-Waugh (2004), eating profile disorders need further investigation and their usefulness as a diagnosis. For the purpose of this study however, the subjective experience regarding loss of control is relative and specific to the participant's perception and meant to be so. If the participant felt that it was of concern in relation to their personal feeling of being in charge, the amount of time or what is seen as the 'norm' in quantity was irrelevant. One of the YFAS questions however, was possibly misleading in this context:

Question 4. Not eating certain types of food or cutting down on certain types of food is something I worry about

To eliminate or drastically cut down on sugar in any eating regimen, worry is called for. Each label must be inspected with care and food groups should be evaluated according to their carbohydrate type and content. A low carbohydrate nutritional intervention forces the participant to worry about cutting down on certain types of food per se. The question was perceived as 'two-folded', as the extent of experienced concern might even rise. Also, the subjective interpretation of the following questions placed into a different context (during and after the intervention) should be noted:

Question 22. I want to cut down or stop eating certain kinds of food.

Question 23. I have tried to cut down or stop eating certain kinds of food.

Question 24. I have been successful at cutting down or not eating certain kinds of food.

The low carbohydrate nutritional intervention forced the participants to cut down and stop eating certain types of foods as a prerequisite for the nutritional regimen. Hence the want to cut out or down on certain foods was not necessarily perceived as a negative in this case.

Question 25. How many times in the past year did you try to cut down or stop eating certain foods altogether?

Question 25 falls under the scoring of category symptom 2, which states:

2) Persistent desire or repeated unsuccessful attempt to quit

Whereby the question does not reflect 'an **unsuccessful** attempt to quit'.

As pertaining to question 22 a low carbohydrate nutritional intervention forces the participant to cut down and stop eating certain types of food as a prerequisite for the nutritional regimen. Hence the want to cut out or down on certain foods was not necessarily perceived as a negative in this case.

C) A great deal of time spent in activities necessary to obtain, use or recover.

This criterion could be seen to not apply equally to all addictive substances (e.g. Nicotine). Social attitudes and policies surrounding the substance play a big part in the definition of terms like addiction or over-use. High caloric foods are cheap, legal, easily accessible and socially acceptable while not showing evident signs of intoxication. The question relating to this criterion in the study, was, however, difficult because it could be perceived in two ways:

Question 7. I find that when certain foods are not available, I will go out of my way to obtain them. For example, I will drive to the store to purchase certain foods even though I have other options available to me at home.

A low carbohydrate nutritional intervention naturally forces the participant to search for particular kinds of foods and the appropriate sources. This implies taking extra care and time to frequent suppliers who have the correct ingredients on offer and selectively choose what is needed. An extended drive or search did therefore not appear unusual for any participant, who did not have a fresh fruit and vegetable vendor as well as a fresh protein source available in their imminent surrounds.

D) Giving up other activities

This criterion addresses the extent to which substance use impacts daily functioning. A low carbohydrate intervention requires adjustments to certain situations subjecting the participant to limited choice and unnecessary social pressure. This in turn could be interpreted as impacting daily functioning because avoiding certain substances or the inability to choose appropriate nourishment. Question 11 of the YFAS is therefore to be applied with caution. It should be noted that while altering environmental factors may result in a reduction of problems related to food, they also require an adjustment to the environment which may impact on time or social occasions.

Question 11. There have been times when I avoided professional or social situations be- cause I was not able to consume certain foods there.

A low carbohydrate nutritional intervention expects that the participant chooses professional or social situations wisely as not to be tempted or be forced into ordering inappropriate foods or consume alcohol because of limited choice and social pressure. A variety of venues and events were simply seen as unsuitable on account of their failure to offer any low carbohydrate foods or beverages.

E) Continued use despite physical or psychological problems

Continued high carbohydrate food consumption could be falling under excessive food consumption, which in case of the DSM-VI as well as the study, was found to be a valid criterion not to be questioned.

3.6.2 The YFAS in context

The threshold levels as stipulated by Gearhardt, Corbin and Brownell (2008), appear to be excessively high in their applicability to this study. Finding yourself ‘consuming certain

foods continuously though you are no longer hungry' (Question 2) for example, is not seen as unusual (YFAS) when recurring 2-3 times a week and is only counted as significant if occurring 4 or more times daily. The Cut-offs of the YFAS were developed for the continuous questions by examining scatterplots of the answers compared to Binge Eating scores, EAT-26 scores (see Appendix 4), and BMI. The extremity of binge eating or anorexic tendencies as a threshold measurement is considered excessive when investigating sugar addiction. Since this study did not concern itself with conventionally labelled eating pathologies, lower threshold levels were considered as possibly being more appropriate. Despite that, the levels were applied as stipulated by the YFAS, whereby the application of continuous scoring alongside dichotomous was established as being necessary for the sample applied.

The diagnostic performance of BMI measures diminishes as age increases. As stated by Romero-Corral et al., (2008) in men, BMI had a better correlation with lean mass than with body fat percentage, while in women BMI correlated better with body fat percentage than with lean mass. In their study on the accuracy of BMI measurements, within the intermediate range of BMI (25–29.9 kg m2), BMI failed to discriminate between body fat percentage and lean mass in both sexes. The proportion of lean body mass as opposed to body fat is a vital discriminant of physiological health and well-being, as well as a reliable disease progression marker. BMI as a measure might, in this case and in many others relating to diet and nutrition, not be an appropriate measurement to apply - especially in respect to establishing norms and thresholds.

Scores of the EAT-26 also rely heavily on the applicability of BMI measurements. Completing the EAT-26 yields a "referral index" based on three criteria: 1) the total score based on the answers to the EAT-26 questions; 2) answers to the behavioural questions related to eating symptoms and weight loss, and 3) the individual's body mass index (BMI)

calculated from their height and weight (Garner, Olmsted, Bohr and Garfinkel, 1982). The EAT-26 is a refinement of the original EAT-40 (first published in 1979) and used in one of the first studies to examine socio- cultural factors in the development and maintenance of eating disorders. Had the EAT-26 been employed as a sole measure in this study, similar interpretation discrepancies of the questions employed would have to be considered:

Question 8. Feel that others would prefer if I ate more

Question 20. Feel that others pressure me to eat

Eating along a low carbohydrate regimen eliminates most conventional foods and may cause the participant to be conspicuously different in eating behavior because he or she will not conform to social expectations.

Question 9. Take longer than others to eat my meals

Generally, high simple carbohydrate foods are accompanied by substances such as MSG or other added sugars to increase the consumption and speed thereof. A low-carbohydrate eater will take longer to eat their food and reach a level of satiety at finite point.

Question 7. Particularly avoid food with a high carbohydrate content (that is bread, rice, potatoes, etc.)

Question 10. Avoid foods with sugar in them

The point of the nutritional intervention is to avoid the consumption of sugar, not because a desire to lose weight but on account of striving to reach a healthy bodily constitution.

Question 23. Engage in dieting behaviour

Question 17. Eat diet foods

Both questions depend strongly on their interpretation. What this study names a nutritional intervention and a consequently necessary 'lifestyle change', will be perceived as a 'diet' or eating 'diet foods' on average, as the mainstream belief on 'healthy' and 'diet' rest on misconstrued principles of understanding and a general lack of education on the topic both

on a professional and non-professional level.

Question 3. Finding myself preoccupied with food

Question 21. Give too much time and thought to food

As in question 7 of the YFAS, a low carbohydrate nutritional intervention forces the participant naturally to search for particular kinds of foods and applicable sources. This implies taking extra care and time to frequent suppliers who have the correct ingredients on offer and selectively choose what is needed. This implies an extended drive or search for any participant, who does not have a fresh fruit and vegetable vendor as well as the applicable fresh protein source available in their imminent surrounds.

Since the aim of this study is to ascertain the possibility of a positive influence of a nutritional intervention on addiction and the questions of the EAT-26 are not answered directly, but form part of the back-bone of the YFAS ability to measure addiction, it was perceived as an adequate, if in need of change, part of the YFAS methodology.

3.6.3 Concerns on using self-report measures

Certain types of nutritional interventions such as lowering the carbohydrate in-take may cause withdrawal-like symptoms. Reducing sugar has been reported to cause symptoms such as headaches and fatigue (clinical experience of accompanying nutritionist). The reliance on self-reports could increase the likelihood of attributing experienced discomforts with withdrawal symptoms. This would not influence the study at hand, since it does not aim at characterising food withdrawal, but could impact on the self-report measures nonetheless.

In relation to diagnostic thresholds and clinically significant impairment or distress, a diagnosis of substance dependence is not given unless significant distress or impairment is present. Whereas several studies using self-report criteria for substance dependence fail to

address the issue of impairment and distress, inflating prevalence estimates, data from the YFAS highlights its importance.

Even though self-report measures require people to rely on their own insights and perceptions, there is little risk of participants unconsciously being influenced by a social desirability response in the case of this study. The researcher acknowledges though, that there is an undeniable risk of bias in respect to pleasing the experimenter with a self-report scale. Since carbohydrate consumption is a staple part of our diet though, the exclusion of such is an experimental outcome question rather than that of personal achievement or betterment of any participant.

3.6.4 Implications for the current study

The following questions forming part of the YFAS questionnaire were seen as needing refinement for future use in context of this study topic, while continuous scoring was applied in addition to determine the internal consistency of the following questions (addiction-like criteria symptom counts):

2) Persistent desire or repeated unsuccessful attempt to quit

Questions #4, #22, # 24, #25

whereby ‘unsuccessful’ is not reflected in the questions.

Whilst the questions where included in the dichotomous scoring format, the continuous scores reflect one score excluding any of the symptom counts relating to the above questions on account of their ambiguity and consequent potential to add scoring error, and one score including the score for comparative reasoning.

4) Important social, occupational, or recreational activities given up or reduced

Questions #8, #9, #10, #11

Question eleven could be regarded as a misrepresentative of the actual results (after)

on account of the participant's compliance to the nutritional regimen - which is to be seen positive as opposed to negative (in terms of personal perception). One participant answered in the affirmative before the intervention. Seven participants scored positively after the intervention. Upon closer inspection, it was found that all participants had understood the question within a positive, 'doing the right thing', framework: Full compliance was enabled by the participant's conscious decision to avoid certain venues or social situations. The question was withdrawn from the overall dichotomous score in the modified questionnaire example to reflect scores true to their meaning.

Due to the origin and aim of the existing YFAS measurement, specific questions were therefore applied with reservations regarding their applicability in the case of carbohydrate/sugar- addiction, whereby question 11 was omitted in a 'modified' follow up questionnaire to reflect the applicable interpretation (in dichotomous scores) of the participant's answers. Since the threshold levels of the existing YFAS were considered as being excessively high for the purpose of this study, continuous scores were essential for the interpretation of the study premise. Because the focus of the research was on the improvement of symptoms pertaining to carbohydrate addiction before and after intervention, reverse scoring was used for the calculation of the Cronbach alpha coefficient in the case of question 11.

3.7 ETHICAL CONSIDERATIONS

3.7.1 Informed consent and respect for autonomy.

Traditional legal and ethical norms for research involving human subjects require full disclosure of the nature of the research as well as the potential risks and benefits of participation (Lin, Owen, & Altman, 2004).

To serve the purpose of this study, the participants were informed of the outcome, the procedure, the nutritional treatment regimen and aim of the study, namely to determine a severity score on the YFAS addiction measure while determining physiological as well as psychological changes. Participation in the survey was voluntary and informed consent was obtained from all qualifying participants.

3.7.2 Data sharing and protection of privacy.

Participants were informed about the possible limits to privacy (e.g. data, survey responses, and/ or personally identifying information may be compromised in the event of a security breach or failure to follow protocol) and the uncertain nature of the risks. The researcher took all the necessary steps to keep data secure and private and to ensure that only the researcher had access to the data. Recorded data were not stored under the name of the participant, instead any results published in the study were stored anonymously based on aspects such as symptom count and addiction severity outcome only.

3.7.3 Invasiveness of sampling and minimizing risk.

Participants were fully cognizant of the data collection to be done and the intended measurement purpose. If they subsequently felt uncomfortable or unwilling to complete all questions or to supply the measurement required, they were informed that they could withdraw at any time. Assistance, if requested, was available at any stage by a nutritionist and the researcher could be contacted at any time via an online access.

3.7.4 Diversity of subjects and justice.

The researcher sought to maximize the racial and ethnic diversity of participants. However, concerns had to be raised about the potential to draw inappropriate conclusions or invalid correlations between variations in the addiction potential and race or ethnicity. These concerns were appropriately addressed and responsibly managed by participating researchers by clarifying, that the data reflected would not be interpreted in relation to race or ethnicity at all, but rather evaluated on an anonymous basis, focusing on a nameless symptom score.

The main goal of this research was to advance knowledge and science, and this was made clear to all the participants. The participants voluntarily consented to the research and, as already mentioned, could discontinue participation at any time.

3.7.5 General.

The research participants were protected and safe, with direct and immediate access to a nutritionist (online access) at all times. The researcher took all the necessary steps to avoid any harm, injury, or distress to the research subjects. The research was conducted in a responsible manner.

As outlined in the introduction, the research is relevant both to the overall health and developmental needs of the population of South Africa, and the individual needs of those who suffer from the disease and/ or concerns and subject matter of the study. The Yale Food Addiction Scale Questionnaire can be seen as valid scientific methodology, posing a high probability of providing answers for the specific research questions posed.

The research was managed and conducted by a suitably qualified principal investigator who is experienced in the field of health/nutritional research. It was ensured that research participants were well informed and able to make appropriate choices, which was undertaken with informed consent of the participants or their legal representatives.

It was ensured that participants' rights were respected, including but not limited to their rights to dignity, privacy, bodily integrity and equality, independently reviewed and inspected by an a registered research ethics committee (UNISA).

CHAPTER 4
RESULTS

4.1 SAMPLE DESCRIPTION AND SCORING PROCEDURE

This research was conceptualised as a repeated measures, quasi-experimental design. Measurements were obtained at the pre - and post-intervention stages using the YFAS scale, which consists of 8 subscores, and a total score for the two phases (pre-and post) of the study. A sample of 32 people enrolled for the study, and they had a mean age of 46.5 years (see Table 1).

Table 4.1.1

age and gender of participants

	Study ID	age	gender
	1	40	female
	2	40	female
	3	48	male
	4	33	female
	5	43	female
	6	40	female
	7	52	female
	8	41	male
	9	35	female
	10	74	female
	11	54	female
	12	37	female
	13	41	male
	14	37	female
	15	32	female
	16	27	male
	17	46	female
	18	60	female
	19	79	male
	20	50	male
	21	72	female
	22	75	female
	23	44	male
	24	52	female
	25	39	female
	26	30	female
	27	27	female
	28	34	female
	29	46	female
	30	68	female
Average age		**46.5**	
StDev age		**14.6**	
	min age	27	
	max age	79	
	female	23	76.67%
	male	7	23.33%

The sample comprised 25 females and 7 males, but two of the females terminated the trial month prematurely out of their own volition and were not included in the final scores. The remaining 30 participants were assessed based on the following considerations:
For each of the subparts of the scale (subscores) there is a positive and a negative score. The positive score was the most appropriate, because the researcher wanted to determine whether there was an increase in the positive dimension of the eating behaviour. The researcher assessed increase in positive scores for each subpart (subscale, subdimension), enabling an overall assessment of the means of 'positive' scores for the group of individuals in the post-test relative to their scores in the pre-test. To assess whether the data were consistent with an expectation in a positive score increase, a dependent t-test analysis was performed. The totals were considered (adding the positive scores for each participant to a total out of 8), and evaluated along all the participants to determine if there was an increase during the testing period. Due to the small sample size the statistical significance was set quite high (alpha < 0.1). Finally, effect sizes for the t-test were determined.

After analysing the totals, it was of interest to see if there was a statistically significant improvement for each subpart/ subquestion of the scale. Since the dependent variable in this case is dichotomous and not interval, the McNemar test was applied. Therefore 8 additional analyses were executed to evaluate the outcome relating to each subpart of the scale. Because doing multiple analyses require controlling for cumulative error, a Bonferonni correction was used. Two t- tests with BMI and height as the two dependent variables were done to determine if there was a statistically significant difference between the pre- and post-measurements of BMI and weight (see tables 4.2.2.1, 4.3.1.3).

4.2 IS THERE A MEASURABLE DIFFERENCE IN BODY COMPOSITION BEFORE AND AFTER THE INTERVENTION?

4.2.1 Difference in weight from before and after the intervention

To assess the overall mean in improvement on a physical scale, differences between the pretest and posttest weight as well as the reduction in waist-to-hip ratio were recorded. The mean average in weight for all 30 participants was 79.9kg before and 76.4kg (-3.5kg) after the intervention, and the male participant averaged a loss of 7.2kg, whereas the females obtained an average loss of 2.5kg. The participants as a group achieved an overall reduction in weight of 4.6% (see Table 4.2.1.1/2).

Table 4.2.1.1 *Average weight before/after intervention*

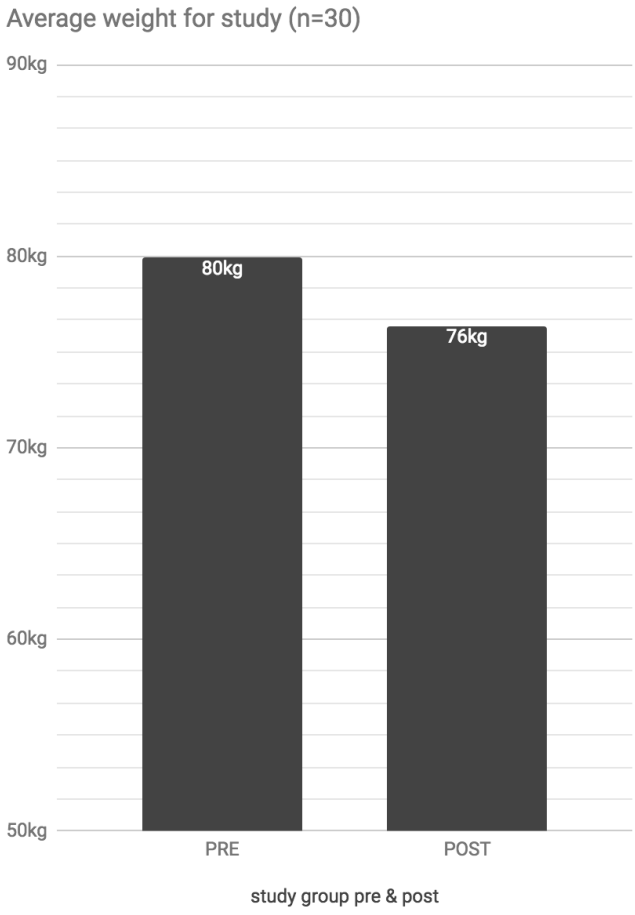


Table 4.2.1.2 *average weight loss after intervention and standard deviation*

Study ID	date	age	gender	weight	weight after	weight loss
Angie	02.07.2016	40	female	72	72	0.0
Anja	02.07.2016	40	female	87	83	4.0
Bill	02.07.2016	48	male	105	97	8.0
Candice	02.07.2016	33	female	64	64	0.0
Catherine	02.07.2016	43	female	63	61	2.0
Cindy	02.07.2016	40	female	121	115	6.0
Claudia	02.07.2016	52	female	62	59	3.0
David	15.8.2016	41	male	72	69	3.0
Heather	28.07.2016	35	female	71	70	1.0
Ingrid	02.07.2016	74	female	64	64	0.0
Jill	02.07.2016	54	female	63	60	3.0
Justine	02.07.2016	37	female	52	53	-1.0
Karl	28.07.2016	41	male	99	94	5.0
Lois	15.8.2016	37	female	67	67	0.0
Ltisch	30.6.2016	32	female	64	60	4.0
Mitch	30.6.2016	27	male	137	129	8.0
Nikki	02.07.2016	46	female	62	58	4.0
Pat	02.07.2016	60	female	77	72	5.0
Paul	02.07.2016	79	male	95	86	9.0
Rael	02.07.2016	50	male	95	85	10.0
Renate	02.07.2016	72	female	80	78	2.0
Renee	02.07.2016	75	female	92	88	4.0
Roland	18.08.2016	44	male	127	120	7.0
Sally Jane	02.07.2016	52	female	51	52	-1.0
Sam	15.6.2016	39	female	92	86	6.0
Sheena	02.07.2016	30	female	72	69	3.0
Stevie	30.6.2016	27	female	72	69	3.0
Tarryn	02.07.2016	34	female	60	59	1.0
Ulrike	02.07.2016	46	female	70	67	3.0
Valerie	02.07.2016	68	female	89	85	4.0
	standard deviation weight l			21.68	19.47	2.2
				average weight loss		**3.5**
				standard deviation weight		**2.9**

A dependent-samples, one-tailed t-test was performed to evaluate the effect of the intervention on the participants' weight. There was a statistically significant decrease in weight from day 1 (M= 79.9 kg, SD =21.68) to day 30 (M= 76.4 kg, SD =19.47), t (29) = 6.57, $p<0.05$). The decrease in weight between the two periods was 2.2.1. The eta squared statistic (0.60) indicated a large effect size according to Cohen's (1988, pp. 284-287) guidelines.

4.2.2 Difference in BMI from before and after the intervention

The mean in waist to hip ratio of all 30 participants stayed constant on 0.9 before and after the intervention, whereby the overall BMI changed from 27.3 to 26.1 (-1.2). Male BMI values changed from 30.6 before to 28.5 (-2.1) after, Female BMI values from 26.3 to 25.4 (-0.9) (see table 4.2.2.1/2).

Figure 4.2.2.1 *Average BMI before/after intervention*

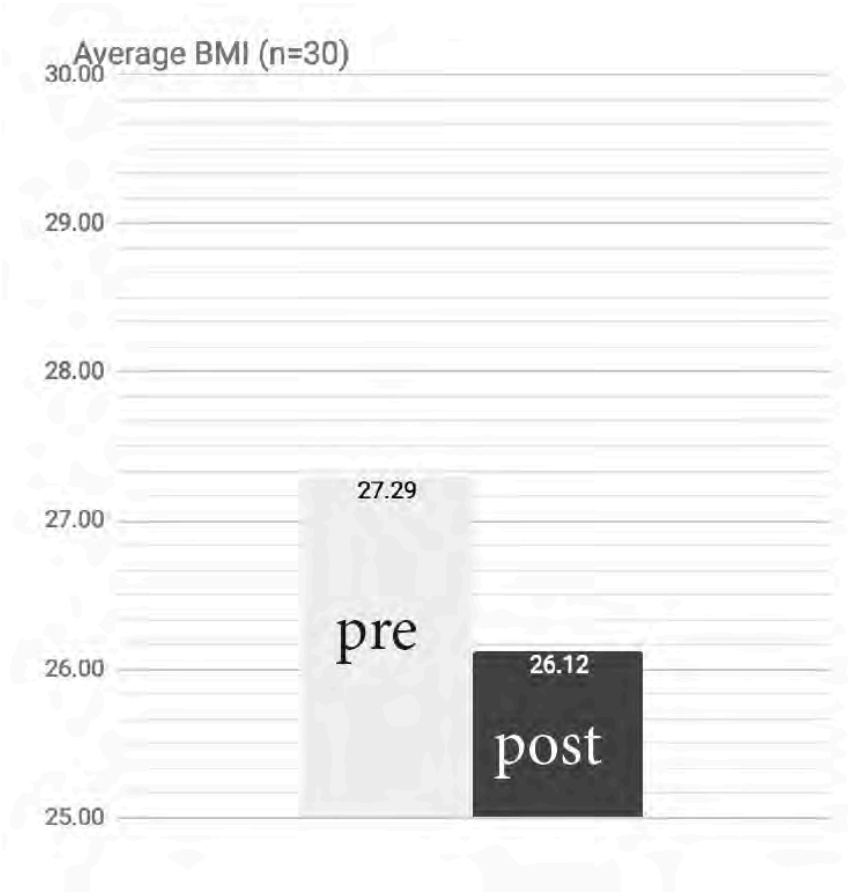


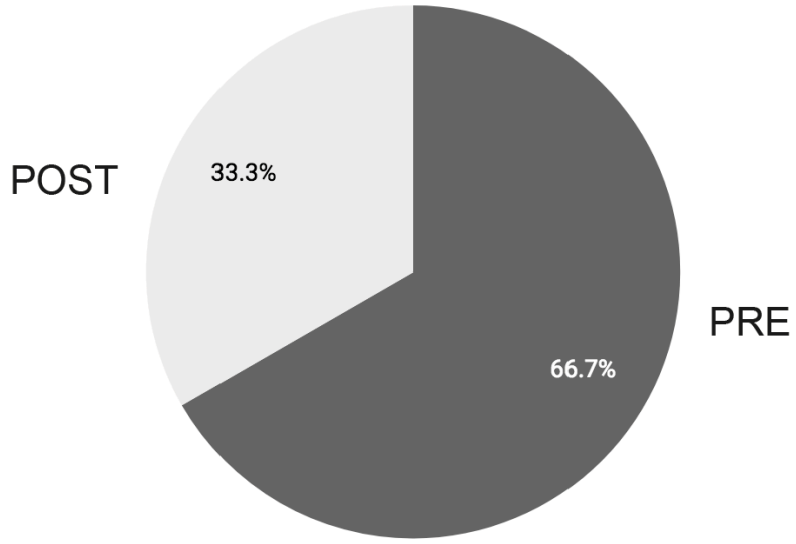
Table 4.2.2.2 *Average BMI reduction after intervention and standard deviation*

Study ID	date	age	gender	weight	weight after	weight loss	BMI	BMI after	BMI reduction
Angie	02.07.2016	40	female	72	72	0.0	25.8	25.8	0.0
Anja	02.07.2016	40	female	87	83	4.0	30.1	28.7	1.4
Bill	02.07.2016	48	male	105	97	8.0	31.4	29.0	2.4
Candice	02.07.2016	33	female	64	64	0.0	22.9	22.9	0.0
Catherine	02.07.2016	43	female	63	61	2.0	24.0	23.2	0.8
Cindy	02.07.2016	40	female	121	115	6.0	38.2	36.3	1.9
Claudia	02.07.2016	52	female	62	59	3.0	24.5	23.3	1.2
David	15.8.2016	41	male	72	69	3.0	24.1	23.1	1.0
Heather	28.07.2016	35	female	71	70	1.0	22.4	22.1	0.3
Ingrid	02.07.2016	74	female	64	64	0.0	21.4	21.4	0.0
Jill	02.07.2016	54	female	63	60	3.0	24.9	23.7	1.2
Justine	02.07.2016	37	female	52	53	-1.0	19.1	19.5	-0.4
Karl	28.07.2016	41	male	99	94	5.0	25.8	24.5	1.3
Lois	15.8.2016	37	female	67	67	0.0	23.2	23.2	0.0
Ltisch	30.6.2016	32	female	64	60	4.0	24.7	23.1	1.5
Mitch	30.6.2016	27	male	137	129	8.0	42.3	39.8	2.5
Nikki	02.07.2016	46	female	62	58	4.0	23.6	22.1	1.5
Pat	02.07.2016	60	female	77	72	5.0	29.0	27.1	1.9
Paul	02.07.2016	79	male	95	86	9.0	27.5	24.9	2.6
Rael	02.07.2016	50	male	95	85	10.0	28.4	25.4	3.0
Renate	02.07.2016	72	female	80	78	2.0	32.5	31.6	0.8
Renee	02.07.2016	75	female	92	88	4.0	32.6	31.2	1.4
Roland	18.08.2016	44	male	127	120	7.0	35.2	33.2	1.9
Sally Jane	02.07.2016	52	female	51	52	-1.0	18.5	18.9	-0.4
Sam	15.6.2016	39	female	92	86	6.0	29.0	27.1	1.9
Sheena	02.07.2016	30	female	72	69	3.0	27.8	26.6	1.2
Stevie	30.6.2016	27	female	72	69	3.0	32.4	31.1	1.4
Tarryn	02.07.2016	34	female	60	59	1.0	20.0	19.7	0.3
Ulrike	02.07.2016	46	female	70	67	3.0	22.9	21.9	1.0
Valerie	02.07.2016	68	female	89	85	4.0	34.8	33.2	1.6
	standard deviation weight l			21.68	19.47	2.2	5.7	5.1	**0.6**
				average weight loss		**3.5**			
				standard deviation weight		**2.9**			
					average BMI reduction				1.2
					standard deviation BMI		**5.7**	**5.1**	**0.6**

In the expectation of an improvement after the intervention, a one tailed t-test was applied. There was a statistically significant decrease in BMI from day 1 (M= 27.3, SD =5.71) to day 30 (M= 26.1, SD =5.12), t (29) = 6.8, $p<0.05$). The mean decrease in weight between the two periods was 1.2. The eta squared statistic (0.61) indicated a large effect size according to Cohen's (1988) guidelines.

4.3 IS THERE A MEASURABLE DIFFERENCE IN ADDICTION SEVERITY BEFORE AND AFTER THE INTERVENTION?

Figure 4.3.1.1 *YFAS food addiction classification: improvement (pre- and post-test)*



The diagnostic threshold for food addiction based on the YFAS (that is, three or more 'symptoms' and clinically significant impairment or distress) was met by 66.7% of participants before the intervention and by 33.3% of participants after the intervention (see table 4.3.1.1). In the overall sample, the mean number of food addiction 'symptoms' met on the YFAS was 3.33 (SD=1.67) before the intervention and 1.33 (SD=1.21) after the intervention. High FA (Food Addiction) was met by 16 participants (high FA plus impairment = 5 participants) before the intervention and by 8 participants AFTER (high FA plus impairment = 1 participant). The latter result is significant in that it shows a reduction in the addiction 'symptoms' in participants (less addiction) overall after the intervention by half of the participants, and across all eight subgroups (YFAS sub-criterion), the mean symptom count pre-intervention over all participants totalled 100, and post intervention this decreased to 46.

A dependent sample t-test showed that the decrease in addictive symptoms from the pre-test (M=3.33, SD=1.67) to the posttest (M=1.33, SD=1.21) was statistically significant with t(29) = -9.103, p<.05. The eta squared statistic (1.21) indicated that the effect size is large according to Cohen's (1988) guidelines. The data representing the change in addictive scores from the pre-treatment to the post-treatment are shown in table 4.3.1.2

Table 4.3.1.2 *Mean food addiction count pre- and post-treatment and standard deviation*

	before	after
	4	0
	6	3
	5	1
	2	1
	6	2
	7	3
	3	0
	1	1
	4	3
	1	0
	2	0
	2	2
	5	2
	3	2
	4	2
	5	2
	2	0
	2	0
	2	1
	6	3
	2	1
	2	0
	5	3
	2	0
	2	0
	3	2
	4	4
	2	0
	2	1
	4	1
Average	**3.33**	**1.33**
Std dev	**1.67**	**1.21**

The study comprised one (categorical) dependent variable with two categories (addiction/non-addiction) and one independent variable, the LCHF diet regime which is also a categorical variable (i.e. pre- and post-treatment). The two categories in the dependent variable are mutually exclusive (participants do not appear in more than one category). Given two paired variables where each variable has exactly two possible outcomes (criterion for addiction met or not met), the McNemar test was used to test for the presence (= 1) or absence (= 0) of addiction per criterion assessed. To establish whether there was a statistically significant improvement (less addiction) for each subpart of the scale, the McNemar calculation was done (see table 4.3.1.3), resulting in 8 additional analyses showing the following:

Table 4.3.1.3 *McNemar calculation p-values*

Subtest/ criterion	McNemar's	df	p-value
criterion1	7.111	1	0.0077
criterion2	8.1	1	0.004427
criterion3	4.9	1	0.02686
criterion4	4.1667	1	0.04123
criterion5	17.391	1	3.04E-05
criterion6	0.8	1	0.3711
criterion7	7.1111	1	0.007661
criterion8	4.1667	1	0.04123

While the criteria as set out by the YFAS appear to show a statistically significant improvement overall, criterion 6 could be seen as questionable according to the alpha size determined by the researcher. (<0.1) As discussed in Chapter 3 (see 3.6.1 concerns regarding the YFAS), the suitability and contextual questioning of the YFAS raised several concerns in its applicability to certain sectors, and should be reconsidered when recreating a study of similar sort. Due to the small sample size of the study the alpha was set high (<0.1). After Bonferroni correction for multiple testing the threshold for statistical significance was set at <0.0125.

4.3.1 Continuous scores and internal reliability before and after the intervention

To verify the reliability of the instrument used, the Cronbach alphas were calculated before and after the intervention using the *'R' package Psych* version 1.7.5 (Revelle, 2017). While Primer questions were excluded, it was found that the score for question 24 required to be calculated in reverse after the intervention to maintain internal consistency. Before the intervention, 24 and question 25 were reversed (both questions are part of symptom no.2). This supports the researcher's assumption in regard to the questions being 'two-fold' in answer (please refer to 3.5.2). Both questions resulted in a negative response (indicating high addiction severity) when scored according to the YFAS scoring principles, but displayed an inherently positive outcome in relation to the study aims (low addiction severity equalling exercising increased control in choice). Scoring them in the inverse therefore supports the hypothesis that question 24 and 25 should both display a high score if successful for the outcome for this study, which they did inversely (meaning that a high score or a 'yes' answer was not indicative of heightened addiction, but of an improvement in addictive behaviour). Before the intervention, the Cronbach alpha coefficient amounted to 0.9, and after the intervention it remained at 0.89, showing high internal consistency (see Appendix 16), whereas without inverse scoring on questions 24 and 25 the Cronbach alpha before would have been equal to 0.847 and after the intervention equal to 0,878 (see Appendix 16). Question 10 (before intervention, see Appendix 13) and 8 (after intervention, see Appendix 12) were omitted in the calculations due to a lack of variance (see Appendix16). This is in support of the argument that there were no overt or established eating pathologies present in any participants, whereby question 8 in particular pertained to addictive eating behaviour displayed in extreme eating pathologies. Question 10 indicated an avoidance of venues or events and social situations not supplying food according to the recommended eating guidelines after the intervention only. Because only after the change of eating habits the need

for or the avoidance of certain foods on account of reducing the overall carbohydrate intake became topical. As stipulated, the presence of a carbohydrate addiction features in 'normal' population samples is not topical except to obese people or people with eating pathologies, hence what is classified as being 'addictive behaviour' has to be adjusted according to the context it is used in and the parameters or study hypothesis set.

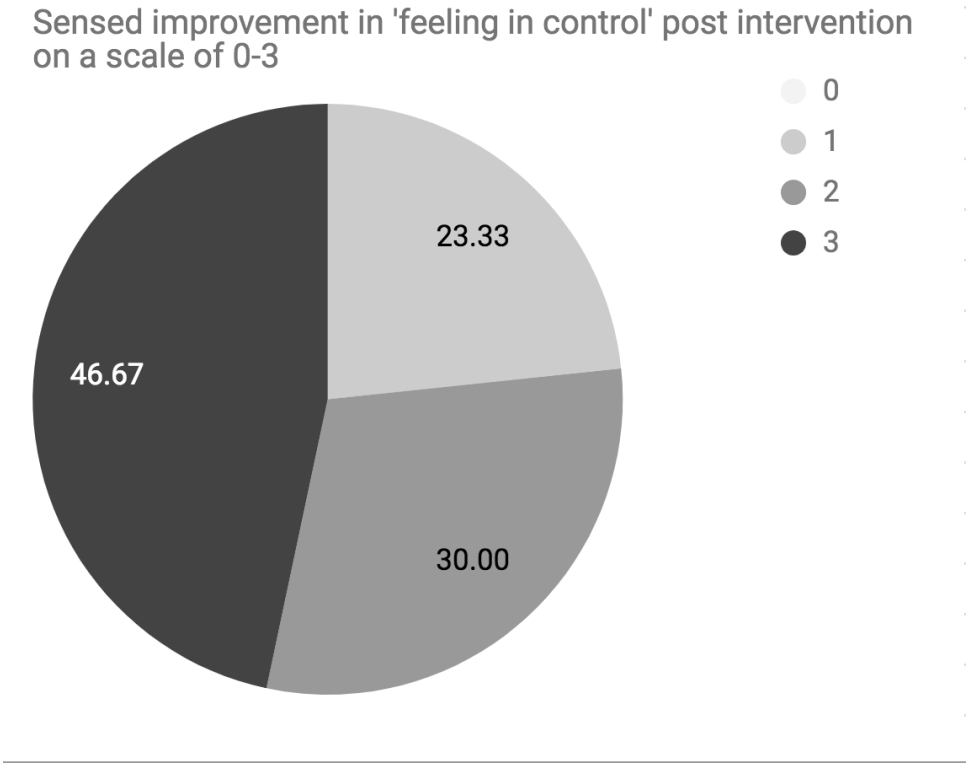
Overall, the Cronbach alphas from before and after the intervention displayed high internal reliability (all calculated at an 95% confidence interval) upon considering inverse scoring on 24 and 25, whereby the omission of symptom count 2 in a second calculation after the intervention (counting a total of 18 questions) showed no difference in alphas (before 0.88, after intervention 0.88). This implies that the exclusion of symptom 2 did not significantly affect the overall internal reliability and renders its calculation unnecessary in relation to the interpretation and the reliability of the results (see 16, 12, 13).

4.4 QUALITATIVE ANALYSIS OF 'FEELING OF CONTROL' IMPROVEMENT AFTER THE INTERVENTION

The mean perceived improvement of 'feeling in control' had an outcome score of 2.2 points, suggesting that a 'noticeable to significant' improvement was felt by participants overall (see figure 4.4.1 and appendix 11). An improved sense of control regarding eating behaviour is beneficial in that it leads to the conclusion that the nutritional intervention enabled an increased sense of control, which in turn allows for the curbing of addictive behaviour on a self-initiated level.

Figure 4.4.1

Increase of 'feeling in control' post intervention (scale of 0-3)



4.5 OTHER NOTICEABLE PSYCHOLOGICAL AND OTHER PHYSIOLOGICAL CHANGES OR IMPROVEMENTS BEFORE AND AFTER THE INTERVENTION?

Generally, participants noted overall positive physical and mental improvements after the intervention as reflected in statements such as the following:

"This is the first time I've slept through in years"

"My skin is clearing up"

"I am not hungry on a continuous basis anymore"

"The cravings have gone"

"I am much calmer than before"

"My joint aches have improved drastically"

"My brain fog is lifting, I can remember my phone number again"

"I don't struggle getting up in the mornings as much as I used to"

"I find that I am less lethargic and wanting to move more"

"I can cope with stress better at work"

"I feel less frustrated and angry"

"I am not disappointed with myself anymore"

"I don't get that emotional anymore"

These statements were part of additional records and notes participants made during the follow up of the intervention upon completing the second questionnaire.

More than two thirds (22) of 30 participants opted to continue with the regimen, acknowledging that its effectiveness relies upon its continued application. Participants were surprised as to how 'far-ranging' the effects were. They observed that they were positively affected by the re-sensitization of their own bodies, and were now capable of responding appropriately to essential physical and mental demands. Feeling in control again, being able

to influence and direct pain (as in the case of joint pain) was unexpected for most and the main determinant in opting to maintain this newly-found lifestyle.

It is worth the consideration, in any subsequent investigations, to include the issues addressed by the participants in a measurable format. Regarded in a context of motivation for any participants, these positive side-effects could act as extra leverage to ensure intervention adherence and increase the participant's determination. The lifestyle aspects not addressed within the context of this study specifically, that is an improvement of 'quality of life', such as better sleeping patterns, increased ability to concentrate, less feelings of anxiety and frustration, an improved ability to cope with stress and the alleviation of joint pains, could point to additional treatment options in varying fields of an experienced 'low quality of life' and the willingness to change or a wish for personal betterment within the addiction framework.

In future applications of the study, specific contingencies could be established in order to determine other relevant key improvement markers to be expected using the Chi Square test. Associations between the intervention and variables such as 'less joint pain', 'increased ability to concentrate', etc., over and above the increased 'feeling in control' could be indicated and established within the possible influences of the proposed nutritional intervention. Strategies advocating for the health benefits of the nutritional intervention, promoting the outcome of feeling better about oneself and improved self-confidence, could address factors possibly influencing treatment adherence such as a lack of motivation, time constraints and cost.

The successful application of a behavioural programme to curtail addictive eating behaviour depends on an individual's ability to sustain the adopted lifestyle changes involving the dietary modifications of the nutritional intervention. A focus on improving motivation, desire, self-efficacy, attitudes, knowledge, and goal setting may be particularly useful (West et al., 2017). Whereas this study concerned itself solely with devising a way to

lessen addiction by employing a LCHF nutritional intervention, further research could include the participants feedback concerning other positive 'side effects' and in turn establish their strength of association, thereby offering focal points in addressing motivation in regards to possible positive outcomes of the intervention to desire, aspire and look forward to over and above lessening the perceived addiction severity and the increased 'feeling of control'.

4.6 OTHER CONSIDERATIONS

Two participants (both female) could be classified as 'problematic' upon final assessments, as their mean body weight increased (by +-1kg) despite a full compliance to the nutritional guidelines set out. After further enquiry one participant was identified as having an allergic response on account of a substantial increase in the consumption of eggs (positive egg white allergy test). The second participant could be considered as having been borderline underweight before the intervention (height 1.65, before weight 52kg, afterwards 53kg). It should therefore be noted that future studies of this nature have to take allergic tendencies into consideration as a health criterion upon study entrance. This study has only considered the participants as a single group. It is suggested that any follow ups or a possible repetition takes variables such as gender, age, and level of obesity into account.

CHAPTER 5
DISCUSSION, FUTURE DIRECTIONS AND CONCLUSION

5.1 DISCUSSION

In this final chapter the quantitative and qualitative research findings are first discussed in relation to the three main research questions, after this some of the limitations of the current study are mentioned, and then a few recommendations are made for future research on this topic. The chapter concludes by discussing the general implications of this nutritional intervention in the context of a developing country such as South Africa.

5.1.1 Research findings

RQ1: Research Question 1

The first research question investigated was whether there was a measurable difference in body composition before and after the LCHF diet intervention, and entailed two sub-questions:

1) Is there a measurable difference in weight from before and after intervention?

2) Is there a measurable difference in BMI from before and after intervention?

The results supported the research expectations. There was a measurable difference with statistical significance in body composition from before and after the intervention. The average measurement of the participants' weights showed a decrease after the intervention and there was also a statistically significant decrease in their average BMI measurements after the intervention. In the case of the weight decrease as well as the BMI decrease, the effect sizes were large. The results were thus in accordance with some of the previous

research findings discussed in chapter 2, and suggest that a low carbohydrate, high fat diet regime has a positive effect on body composition.

RQ2: Research Question 2

The second research question explored whether there was a measurable difference in addiction severity before and after the diet intervention?

The results were again in line with the research expectations. There was a measurable difference in addiction severity at the posttest stage, with the participants' addiction severity showing a statistically significant decrease from the pre-test measurements, and this decrease may be attributable to the diet intervention. A paired-samples t-test showed that the decrease was statistically significant and the effect size (eta squared = 1.21) was also large. However, since this is only a quasi-experimental study the possibility cannot be excluded that other variables, that were not controlled for, could have played a role in the decrease.

RQ3: Research Question 3

The third question aimed to establish whether there was a noticeable self-perceived improvement in the participants 'feeling in control' over their eating behaviour after the intervention.

The qualitative analysis suggests that the participants did experience a noticeable self-perceived improvement in this 'feeling in control' after the intervention, and the results therefore supported the research expectation.

Overall, the study hypotheses were thus met, and the research served to indicate the positive impact that a low carbohydrate high fat (LCHF) nutritional intervention could have on weight control and eating behaviour, because the participants exhibited an improved eating addiction score on the Yale Food Addiction Questionnaire as well as a self-perceived

improvement in their 'feeling in control' of their own eating behaviour. Furthermore, their weights and BMI measurements improved significantly in only thirty days after adopting the LCHF eating regimen.

It would therefore appear that self-regulation in the realm of eating behaviour must be considered in context of the addiction potential of the substance(s) consumed. If the omission of simple carbohydrates (simple sugars), and the addition of healthy fats in the diet can lead to significant positive physical and mental manifestations in such a short time-span, this type of diet intervention is clearly a topic that governments and their healthcare systems should explore in view of the current rise in chronic diseases, because these are at least partly due to nutritional issues in developing countries. The role of carbohydrate consumption needs to be acknowledged as a risk factor in disease progression.

5.1.2 General discussion of findings

Although the dichotomous scores obtained with the YFAS clearly showed an improvement in the degree of addiction 'severity' from before and after the intervention, the researcher of the study chose to rely on the outcome of the continuous score as a basis for interpretation, and this decision was supported by the reliability analysis (i.e. inverse scoring of questions 24 and 25 in relation to the study's premises). This suggests that addiction behaviour symptom question scoring needs to be carefully reconsidered within the confines of the YFAS when applied in different regimes such as a LCHF nutritional intervention, and that dichotomous scoring alone will not allow for a complete and detailed interpretation of the results.

The need for the omission of questions 8 and 10 within the continuous scoring regimen (lacking variance) underlined that the presence of a food addiction despite a possibly low or standard body weight is a point to be stressed in this study: food addiction does not

translate into being overweight or is to be identified as being pathological alone. It is present in all shapes and forms, ages and colours. The continuous scoring outcomes support this in that they show that behaviour in particular pertaining to identified 'eating pathologies' have no weight in the internal reliability assessment of the final outcomes. Particular questions in relation to addictive behaviour have to be reconsidered in context, in turn leading to the conclusion that the YFAS can and should be readjusted if assessing addictive eating behaviour in a general population sample, while continuous scoring is incremental in assessing the internal reliability of the questions. Since the calculation of the alphas enabled an assessment of singular issues pertaining to certain questions, YFAS 'grey zones' could be identified and highlighted, which the dichotomous scores would not have enabled.

The study participants were deliberately chosen as being 'run of the mill' - your average human without evident disease markers in order to be able to underline the fact that 'food addiction' is present universally and not discernible by weight alone.

Independence and 'feeling in control', as well as interpreting your body's own pain and discomfort signs, all account for an autonomous state of being, that is not dictated by an addiction. This can be seen as indicative of the potential success in assisting in and treating carbohydrate addiction warranting further investigation. It should also be noted, that considering the rapid discoveries and change of baseline principles within nutritional fields, measures such as the YFSA or EAT-26, as much as the reliance upon a BMI measurement as a predictor, warrant consideration.

The reliance on the participant's full compliance could be judged as an incremental problem of the study. Conversely, only a diligent adherence to the nutritional regimen would have achieved the self- recorded results. The expected physical manifestations in improved well-being and sense of control or being 'in tune' with your body were remarked upon by the participants without prompting or formal initiation, while the overall recorded improvement

(mean improvement of 3) in 'feeling in control' serves in support of this. It was therefore accepted that the recorded results, albeit in different degree of severity when comparing the original YFAS scores and the adjusted scores, served as an indication of a successful study follow through by participants. The scores and implications of such are seen as warranting further investigation and could set the grounding for future directions to be outlined.

The 'problem foods' recorded on average either had a high sugar profile such as soda pop, apples, chips, candy or ice cream, or a high simple carbohydrate (high 'hidden' sugar) profile such as white bread, rolls, cookies and cheese burgers. Seeing that 'grain derived' simple carbohydrates dominated the degree of addiction difficulty perceived, is supporting the quint-essence of the study - namely that 'grain derived' food staples threaten the integrity of food choice and are potential causal factors in food addiction. A low carbohydrate nutritional intervention poses the real possibility of treating symptoms of food-addiction (lessening addiction), while improving the participant's overall well-being psychologically and physically. Since sugar addiction is likened to cocaine addiction etc., it is important to consider the implications of the outcome of this study - namely the possibility that a LCHF nutritional intervention might not only curb addictive behaviour and tendencies to food, but might also pave the way for an easier 'weaning off' of other drugs following similar addiction pathways.

5.1.3 Limitations

This study is a quasi-experimental study without randomization or a control group. The small sample size and the use of self-report measures only allowed for rather basic statistical analysis. The researcher acknowledges that the study's results are therefore tentative at best and that further research is needed to investigate the topic more systematically in future.

5.2 PROPOSED FUTURE DIRECTION

5.2.1 The Microbiome and nutrition.

Essentially, the KD/ LCHF diet changes the gut microbiota. David et al. (2014) have demonstrated that the gut microbiome can rapidly respond to an altered diet, potentially facilitating the diversity of human dietary lifestyles. The human gut microbiota comprises approximately 100 trillion microbial cells and has a significant effect on many aspects of human physiology including metabolism, nutrient absorption and immune function. As Bäckhed et al. (2012, p. 612) puts it: "An emerging paradigm is that diseases such as obesity and inflammatory bowel diseases (IBD) are associated with reduced diversity in the intestinal microbiome, which may represent evidence of a suboptimal microbiome".

A successful nutritional intervention such as this low carbohydrate diet might have concomitant effects on the microbiome. Future research should therefore try to establish if the change in the addictive behaviour resulting from the nutritional intervention used in this study, and measured with only a psychometric instrument, could also induce a change in the microbiome. Further research is warranted in the efficacy of employing a nutritional change regimen (or an additional method such as faecal transplants*), in order to attend to and improve addictive behaviour specifically in relation to obesity, thereby providing bio-markers to measure disease and disease progression.

Addictive behaviour such as compulsions, 'needs', cravings, self-debilitating and unhealthy traits related to carbohydrate addiction showed up differences from before and after the intervention: A) On the YFAS Questionnaire and B) physical measurements taken. Subjects felt 'more in control', experienced severely less 'cravings' and unwarranted wants, while noticing an improved and stabilised mood in general. Their sleeping patterns improved and subjects felt more rested. In some cases, dermatological issues resolved themselves and minor aches and pains subsided. The expected weight loss was more of a side effect and a

number of subjects opted to maintain the new 'lifestyle', as they felt considerably more in charge, able to cope with stress better and even getting as adventurous as exercising more out of their own free will. It seems that the low carb nutritional intervention has achieved what it promised in a timespan as short as one month. Not a single candidate did not experience a significant shift in physical and mental health.

Two participants stopped the intervention because of not feeling capable of adhering to protocol. Upon enquiry, both participants appointed their respective failure to experiencing difficulties from environmental factors such as family and social pressure, rather than problems with the actual intervention. Since a low-carb approach is to this day received with scepticism by most medical professionals, a rebuke by peers and family would not seem unusual. Both participants however, indicated the wish to reattempt 'when the timing is better', having seen and been informed of the success achieved in general by other participants. In acknowledgement of the importance of the 'right timing' and potential 'pressures' to be experienced to fully enable 100% compliance, the researcher suggests due consideration and counselling on such before any attempt in nutritional intervention is undertaken in future.

Following this, further fields of interest would be: Can the gut microbiome be changed selectively and specifically? Do certain behaviours such as addiction reflect in the microbiome and can therefore be targeted and identified? How is a change in the gut microbiome best achieved? Is LCHF the best method of changing the gut microbiome? Are options such as faecal transplants viable? How sustainable is a diet regimen like that without education?

Such findings could be highly relevant to other areas of addiction (any behavioural modification for that matter), allowing for biological markers and processes to be identified, which could possibly swerve the physiological course and therefore alter the psychological course of addictive behaviour. Instead of treating symptomology, this could offer a cost-

effective and practical physiological resolution pathway.

* The topic of faecal transplants as a viable additional treatment method rapidly changing the gut microbiota exceeds the scope of this study - but would be highly relevant in the preventative steps to take in the disease progression of obesity, that is addiction.

Referencing

Please refer to Fishman, & Thomson, 2015; Claes, Vargas García, & Lebeer, 2015) for further information on this topic.

5.3 CONCLUSION

In answer to all three research questions posed, the study indicates a significant finding of 'less addiction' overall. Two key questions regarding nutrition and addiction (and consequently the microbiome) in human healthcare are therefore of interest: (A) whether the microbiome should be manipulated therapeutically and (B) the appropriate means to do so. There are multiple proposed approaches for specific microbiota manipulation, including probiotics, prebiotics, diet-based therapies, antibiotics, immune modulation, and faecal transplantation (Kuczynski, 2012). If an alteration in gut microbiome by dietary intervention instigates behavioural modification (less addiction) leading to overall physical and psychological improvement, it could facilitate further research in accordance with the most suitable, practical or time efficient and financially viable approach that should or could be taken.

The study implied that a simple dietary intervention (as being the most cost-effective) enabled an increased ability to exercise self-control and regulation by physiologically altering addiction-pathways, thereby lessening addiction.

This in turn would allow for more efficient and long-term treatment, be highly cost-

effective and open vital implications in the educational and public health sector in general. South Africa is in dire need of nutritional education, which is the most practical and cost-effective tool to combat the healthcare crisis in the face of diabetes, autoimmune disease, obesity, cancer, dementia and pathological behaviour.

A further implication in this regard could be the procedural application of medical aid policies. If biomarkers and YFAS measures can be refined/ defined to such an extent that they can be employed as 'diagnostic' tools - the first step required would be to instruct the patient in a nutritional change regimen. If successfully applied the patient enjoys the benefit of a low cost medical aid whereas non-compliance could lead to adequate premium increases in foresight of the disease progression to follow. Not only would governmental structures benefit from a 'reward-system' such as this, but would be gifted with a joint force to educate the population.

As Ramphele states in his foreword to 'Substance use and addiction in South Africa' (Bhana, 2012): "Increasingly we are understanding that substance use disorders are a major public health problem; there is also value to considering these conditions as brain disorders, which require appropriate professional help. Effective and cost-effective prevention is of course a crucial goal to strive for. At the same time, once addiction is present, then evidence-based treatments are required. The current gap in South Africa is non-acceptable; there are too few services for too many. There is a great need to improve health and mental health literacy in the community, and to train addiction professionals."

While it seems that a great awareness of addiction and its destructive forces within society is present, food ironically does not seem to be considered as part of it. And while a good deal of attention is given to the 'brain', our 'second brain' is left out of account. This study is indicative of the possible success of a LCHF nutritional intervention in treating addiction, and could propose a treatment at low cost, creating much warranted awareness of how important nutrition actually is in the progression of addiction and disease.

Validation of food addiction at the neurobiological level is absolutely critical, and while the debate of the link obesity and food addiction rages on (Ziauddeen & Fletcher, 2013), this study serves as preliminary 'indication' of such. While caution in the definition of this construct is of the utmost importance, thought processes such as Ziauddeen and Fletcher (2013) embody, seem to disregard the obvious need for recognition and subsequent improvement possibilities within our system: "Despite continuing uncertainty about the concept and relative lack of support, it has remarkable, and, in our view, unjustified, influence in developing neurobiological models of obesity and in framing debates about the formulation of public health policy." (Ziauddeen & Fletcher, 2013, p. 19). If not establish the fact that there is such a concept of food addiction and/or obesity - are we to turn a blind eye to the epidemic of our century?

The issue is rather one of defining the addictive and disease potential of certain food groups such as refined carbohydrates, and in turn create the necessary awareness. Since the terminology of food-addiction is misleading, the debate at hand could possibly be alleviated by enabling the term 'food' to be more closely defined to allow for progression and education - rather than opting for disbelief, non-acceptance or pure ignorance.

REFERENCES

Abu-Shanab A., Quigley E.M. (2010) The role of the gut microbiota in nonalcoholic fatty liver disease. *Nat Rev Gastroenterol Hepatol, 7*(12), 691-701 [PMID:21045794].

Alcock, J., Maley, C. C., & Aktipis, C. A. (2014). Is eating behavior manipulated by the gastrointestinal microbiota? Evolutionary pressures and potential mechanisms. *BioEssays, 36*(10), 940–949. doi:10.1002/bies.201400071

Almond, M. (2013). Depression and Inflammation: Examining the Link: Inflammatory Conditions May Precipitate or Perpetuate Depression, but the Precise Relationship Is Unclear. *Current Psychiatry, 6*(12), 24-32.

Appleton, K. M., Rogers, P. J., & Ness, A. R. (2010). Updated systematic review and meta-analysis of the effects of n-3 long-chain polyunsaturated fatty acids on depressed mood. *American Journal of Clinical Nutrition, 91*(3), 757–770. doi:10.3945/ajcn.2009.28313

Avena, N. M., Rada, P., & Hoebel, B. G. (2008). Evidence for sugar addiction: Behavioral and neurochemical effects of intermittent, excessive sugar intake. *Neuroscience & Biobehavioral Reviews, 32*(1), 20–39. doi:10.1016/j.neubiorev.2007.04.019

Bäckhed, F., Fraser, C. M., Ringel, Y., Sanders, M. E., Sartor, R. B., Sherman, P. M., & Finlay, B. B. (2012). Defining a healthy human gut microbiome: current concepts, future directions, and clinical applications. *Cell Host & Microbe, 12*(5), 611-622. doi:10.1016/j.chom.2012.10.012

Bäckhed, F., Ding, H., Wang, T., Hooper, L. V., Koh, G. Y., Nagy, A., … Gordon, J. I. (2004). The gut microbiota as an environmental factor that regulates fat storage. *Proceedings of the National Academy of Sciences of the United States of America, 101*(44), 15718–15723.

Bandura, A., Freeman, W. H., & Lightsey, R. (1999). Self-efficacy: The exercise of control. *Journal of Cognitive Psychotherapy, 13*(2), 158-166.

Bazan, N. G. (2016). Omega-3 fatty acids and neuroinflammation in Alzheimer's disease: the unraveling of neurorestorative cell signaling. *Future Neurology, 11*(2), 99–103. doi:10.2217/fnl-2016-0008

Bazzano, L. A., Hu, T., Reynolds, K., Yao, L., Bunol, C., Liu, Y., … He, J. (2014). Effects of Low-Carbohydrate and Low-Fat Diets: A Randomized Trial. *Annals of Internal Medicine*, *161*(5), 309. doi:10.7326/M14-0180

Bercik, P., Denou, E., Collins, J., Jackson, W., Lu, J., Jury, J., & ... Collins, S. M. (2011). The intestinal microbiota affect central levels of brain-derived neurotropic factor and behavior in mice. *Gastroenterology, 141*(2), 599. doi:10.1053/j.gastro.2011.04.052

Bested, A. C., Logan, A. C., & Selhub, E. M. (2013). Intestinal microbiota, probiotics and mental health: from Metchnikoff to modern advances: Part II-contemporary contextual research. *Gut Pathog, 5*(3), 1–14.

Bhana, A. (2012). Substance Use and Abuse in South Africa: Insights from Brain and Behavioural Sciences, George FR Ellis, Dan J. Stein, Kevin GF Thomas, Ernesta M. Meintjies (Eds.): book review-izindaba. *South African Medical Journal*, *102*(8), 651.

Blumenthal, D. M., & Gold, M. S. (2010). Neurobiology of food addiction. *Current Opinion In Clinical Nutrition And Metabolic Care, 13*(4), 359-365. doi:10.1097/MCO.0b013e32833ad4d4

Bosch-Bouju, C., & Layé, S. (2016). Dietary Omega-6/Omega-3 and Endocannabinoids: Implications for Brain Health and Diseases. In *Cannabinoids in Health and Disease*. InTech.

Cani, P. D. (2013). Gut microbiota and obesity: lessons from the microbiome. *Briefings In Functional Genomics, 12*(4), 381-387. doi:10.1093/bfgp/elt014

Cassim, S. B. (2010). Food and beverage marketing to children in South Africa: mapping the

terrain. *South African Journal of Clinical Nutrition, 23(*4), 181–185.

Centres for Disease Control and Prevention (2014) National Diabetes Statistics Report. Retrieved 2015 from http://www.cdc.gov/diabetes/pubs/statsreport14/national-diabetes- report-web.pdf.

Chiurchiù, V., Battistini, L., & Maccarrone, M. (2015). Endocannabinoid signalling in innate and adaptive immunity. *Immunology*, *144*(3), 352–364. doi:10.1111/imm.12441

Claes, I. J., Vargas García, C. E., & Lebeer, S. (2015). Novel opportunities for the exploitation of host-microbiome interactions in the intestine. *Current Opinion In Biotechnology*, 3228-34. doi:10.1016/j.copbio.2014.10.008

Clark, S. M., & Saules, K. K. (2013). Validation of the Yale Food Addiction Scale among a weight-loss surgery population. *Eating Behaviors, 14*(2), 216–219. doi:10.1016/j.eatbeh.2013.01.002

Coca-colaproductfacts. 2017. Retrieved from http://www.coca-colaproductfacts.com/en/products/ coca-cola/

Cohen, J. (1983). The Cost of Dichotomization. *Applied Psychological Measurement, 7*(3), 249–253. doi:10.1177/014662168300700301

Cohen, J.W. (1988). *Statistical power analysis for the behavioral sciences* (2nd edn). Hillsdale, NJ: Lawrence Erlbaum Associates.

Corsica, J. A., & Pelchat, M. L. (2010). Food addiction: true or false? *Current opinion in gastroenterology, 26*(2), 165-169. doi:10.1097/MOG.0b013e328336528d

Crane, P. K., Walker, R., Hubbard, R. A., Li, G., Nathan, D. M., Zheng, H., & ... Larson, E. B. (2013). Glucose levels and risk of dementia. *The New England Journal Of Medicine, 369*(6), 540-548. doi:10.1056/NEJMoa1215740

Daulatzai, M. A. (2015). Non-celiac gluten sensitivity triggers gut dysbiosis, neuroinflammation, gut-brain axis dysfunction, and vulnerability for dementia. *CNS & Neurological Disorders Drug Targets, 14*(1), 110-131.

David, L. A., Maurice, C. F., Carmody, R. N., Gootenberg, D. B., Button, J. E., Wolfe, B. E., … Turnbaugh, P. J. (2014). Diet rapidly and reproducibly alters the human gut microbiome. *Nature*, *505*(7484), 559–563. DOI:10.1038/nature12820

Debette, S., Beiser, A., Hoffmann, U., DeCarli, C., O'Donnell, C. J., Massaro, J. M., ... & Seshadri, S. (2010). Visceral fat is associated with lower brain volume in healthy middle-aged adults. Annals of neurology, 68(2), 136-144.

Debette, S., Beiser, A., Hoffmann, U., DeCarli, C., O'Donnell, C. J., Massaro, J. M., … Fox, C. S. (2010). Visceral fat is associated with lower brain volume in healthy middle-aged adults. *Annals of Neurology*, *68*(2), 136–144.

De Filippo, C., Cavalieri, D., Di Paola, M., Ramazzotti, M., Poullet, J. B., Massart, S., ... & Lionetti, P. (2010). Impact of diet in shaping gut microbiota revealed by a comparative study in children from Europe and rural Africa. *Proceedings of the National Academy of Sciences*, *107*(33), 14691-14696. DOI:10.1073/pnas.1005963107

Dehghan, M., Mente, A., Zhang, X., Swaminathan, S., Li, W., Mohan, V., … Mapanga, R. (2017). Associations of fats and carbohydrate intake with cardiovascular disease and mortality in 18 countries from five continents (PURE): a prospective cohort study. *The Lancet*. doi:10.1016/S0140-6736(17)32252-3

Desbonnet, L., Garrett, L., Clarke, G., Bienenstock, J., & Dinan, T. G. (2008). The probiotic Bifidobacteria infantis: An assessment of potential antidepressant properties in the rat. *Journal Of Psychiatric Research, 43*(2), 164-174. doi:10.1016/j.jpsychires.2008.03.009

Duseja, A., Chawla Y.K. (2013). Obesity and NAFLD. *Clinics in Liver Disease, 18*(1), 59-71. doi:10.1016/j.cld.2013.09.002.

Elliott, S. S., Keim, N. L., Stern, J. S., Teff, K., & Havel, P. J. (2002). Fructose, weight gain, and the insulin resistance syndrome. *The American Journal of Clinical Nutrition,*

76(5), 911–922.

El-Mallakh, R. S., & Paskitti, M. E. (2001). The ketogenic diet may have mood-stabilizing properties. *Medical hypotheses, 57*(6), 724-726. doi:10.1054/mehy.2001.1446

Fasano, A. (2011). Zonulin and its regulation of intestinal barrier function: the biological door to inflammation, autoimmunity, and cancer. *Physiological Reviews, 91*(1), 151-175. doi:10.1152/physrev.00003.2008org/10.1016/j.fshw.2013.09.002

Finegold, S. M. (2011). State of the art; microbiology in health and disease. Intestinal bacterial flora in autism. *Anaerobe, 17*(6), 367-368. doi:10.1016/j.anaerobe.2011.03.007

Fishman, J. A., & Thomson, A. W. (2015). Clinical Implications of Basic Science Discoveries: Immune Homeostasis and the Microbiome—Dietary and Therapeutic Modulation and Implications for Transplantation. *American Journal of Transplantation*, *15*(7), 1755-1758. doi:10.1111/ajt.13236

Flint, A.J., Gearhardt, A.N., Corbin, W.R., Brownell, K.D., Field, A.E., Rimm, E.B., 2014. Food-addiction scale measurement in 2 cohorts of middle-aged and older women. *The American Journal of Clinical Nutrition, 99*(3), 578–586. doi:10.3945/ajcn.113.068965

Forbush, K. T., Wildes, J. E., Pollack, L. O., Dunbar, D., Luo, J., Patterson, K., … Watson, D. (2013). Development and validation of the Eating Pathology Symptoms Inventory (EPSI). *Psychological Assessment*, *25*(3), 859–878. doi:10.1037/a0032639

Forbush, K. T. (2015). Eating Pathology Symptoms Inventory (EPSI). In T. Wade (Ed.), *Encyclopedia of Feeding and Eating Disorders* (pp. 1–3). Singapore: Springer Singapore. Retrieved from http://link.springer.com/10.1007/978-981-287-087-2_50-1

Fortuna, J. L. (2010). Sweet preference, sugar addiction and the familial history of alcohol dependence: shared neural pathways and genes. *Journal of Psychoactive Drugs, 42*(2), 147–151.

Foster, J. A., & McVey Neufeld, K. (2013). Gut–brain axis: how the microbiome influences anxiety and depression. *Trends In Neurosciences, 36*(5), 305-312. doi:10.1016/j.tins.2013.01.005

Freedman, M. R., King, J., & Kennedy, E. (2001). Popular diets: a scientific review. *Obesity Research*, 9 Suppl 1, 1S–40S. doi:10.1038/oby.2001.113

Friese, M., Hofmann, W., & Wänke, M. (2008). When impulses take over: Moderated predictive validity of explicit and implicit attitude measures in predicting food choice and consumption behaviour. *British Journal of Social Psychology, 47*(3), 397-419. doi:10.1348/014466607X241540

Garner, D. M., Olmsted, M. P., Bohr, Y., & Garfinkel, P. E. (1982). The eating attitudes test: psychometric features and clinical correlates. *Psychological medicine*, *12*(4), 871-878. doi:10.1017/S0033291700049163

Gearhardt, A. N., Corbin, W. R., & Brownell, K. D. (2009). Food addiction: an examination of the diagnostic criteria for dependence. *Journal of Addiction Medicine, 3*(1), 1–7. doi:10.1097/ADM.0b013e318193c993

Gearhardt, A. N., Corbin, W. R., & Brownell, K. D. (2009). Preliminary validation of the Yale Food Addiction Scale. *Appetite, 52*(2), 430-436. doi:10.1016/j.appet.2008.12.003

Gearhardt A. N., Yokum, S., Orr, P.T., Stice, E., Corbin, W.R., & Brownell, K.D. (2011). Neural correlates of food addiction. *Archives of General Psychiatry, 68*(8), 808–816. doi:10.1001/archgenpsychiatry.2011.32

N Gearhardt, A., Davis, C., Kuschner, R., & D Brownell, K. (2011). The addiction potential of hyperpalatable foods. *Current drug abuse reviews*, *4*(3), 140-145. doi:10.2174/1874473711104030140

Gearhardt, A. N., White, M. A., Masheb, R. M., Morgan, P. T., Crosby, R. D., & Grilo, C. M. (2012). An examination of the food addiction construct in obese patients with binge

eating disorder. *International Journal of Eating Disorders*, *45*(5), 657-663. doi: 10.1002/eat.20957

Gearhardt, A., Roberts, M., & Ashe, M. (2013). If Sugar Is Addictive...What Does It Mean for the Law?. *Journal Of Law, Medicine & Ethics*, 4146-49. doi:10.1111/jlme.12038

Gerritsen, J., Smidt, H., Rijkers, G. T., & de Vos, W. M. (2011). Intestinal microbiota in human health and disease: the impact of probiotics. *Genes & Nutrition, 6*(3), 209-240. doi:10.1007/s12263-011-0229-7

Gibney, M., & Vorster, H. (2001). South African food-based dietary guidelines. *S Afr J Clin Nutr*, *14*(3), S2.

Gold, M. S., Frost-Pineda, K., & Jacobs, W. S. (2003). Overeating, binge eating, and eating disorders as addictions. *Psychiatric Annals, 33*(2), 117.

Harcombe Z., Noakes T. (2016) The universities of Stellenbosch/Cape Town low-carbohydrate diet review: Mistake or mischief? *South African Med J., 106*(11), 79–82.

Harding, K. L., Judah, R. D., & Gant, C. E. (2003). Outcome-based comparison of Ritalin® versus food-supplement treated children with AD/HD. *Alternative Medicine Review, 8*(3), 319–330.

Hartman, A. L., Gasior, M., Vining, E. P. G., & Rogawski, M. A. (2007). The neuropharmacology of the ketogenic diet. *Pediatric Neurology, 36*(5), 281–292. doi:10.1016/j. pediatrneurol.2007.02.008

Hession, M., Rolland, C., Kulkarni, U., Wise, A., & Broom, J. (2009). Systematic review of randomized controlled trials of low-carbohydrate vs. low-fat/low-calorie diets in the management of obesity and its comorbidities. *Obesity Reviews, 10*(1), 36–50. doi:10.1111/j.1467-789X.2008.00518.x

Higginson, A. D., McNamara, J. M., & Houston, A. I. (2016). Fatness and fitness: exposing the logic of evolutionary explanations for obesity. *Proc. R. Soc. B*, *283*(1822), 20152443. doi:10.1098/rspb.2015.2443

Hill, J. M., Bhattacharjee, S., Pogue, A. I., & Lukiw, W. J. (2014). The gastrointestinal tract microbiome and potential link to Alzheimer's disease. *Frontiers In Neurology, 543*. doi:10.3389/fneur.2014.00043

Hill, M.N., Miller, G.E., Carrier ,E.J., Gorzalka B.B., Hillard C.J. (2009). Circulating endocannabnoids and N-acyl ethanolamines are differentially regulated in major depression and following exposure to social stress. *Psychoneuroendocrinology, 34*: 1257–1262. doi:10.1016/j.psyneuen.2009.03.013

Hoebel, B. G., Rada, P. V., Mark, G. P., & Pothos, E. N. (1999). Neural systems for reinforcement and inhibition of behavior: Relevance to eating, addiction, and depression. In D. Kahneman, E. Diener, & N. Schwarz (Eds.), *Well-being: The foundations of hedonic psychology* (pp. 558-572). New York: Russell Sage Foundation.

Hoffman, N. E., Chandramoorthy, H. C., Shanmughapriya, S., Zhang, X. Q., Vallem, S., Doonan, P. J., ... & Koch, W. J. (2014). SLC25A23 augments mitochondrial Ca2+ uptake, interacts with MCU, and induces oxidative stress–mediated cell death. *Molecular biology of the cell*, *25*(6), 936-947.

Holmes, E., Li, J. V., Marchesi, J. R., & Nicholson, J. K. (2012). Gut Microbiota Composition and Activity in Relation to Host Metabolic Phenotype and Disease Risk. *Cell Metabolism, 16*(5), 559–564. doi:10.1016/j.cmet.2012.10.007

Hotamisligil, G. S. (2006). Inflammation and metabolic disorders. *Nature 444*, 860–867.

Hrncir, T., Stepankova, R., Kozakova, H., Hudcovic, T., & Tlaskalova-Hogenova, H. (2008). Gut microbiota and lipopolysaccharide content of the diet influence development of regulatory T cells: studies in germ-free mice. *BMC Immunology, 9*(1), 65. doi:10.1186/1471-2172-9-65

Hsiao, E. Y., McBride, S. W., Hsien, S., Sharon, G., Hyde, E. R., McCue, T., … Mazmanian, S. K. (2013). Microbiota Modulate Behavioral and Physiological Abnormalities

Associated with Neurodevelopmental Disorders. *Cell, 155*(7), 1451–1463. doi:10.1016/j. cell.2013.11.024

Jenkins, T., Nguyen, J., Polglaze, K., & Bertrand, P. (2016). Influence of Tryptophan and Serotonin on Mood and Cognition with a Possible Role of the Gut-Brain Axis. *Nutrients, 8*(1), 56. doi:10.3390/nu8010056

Kahn, M. S. (2011). A potential role for LPS-induced inflammation in the induction of Alzheimer's disease-related pathology and cognitive deficits [electronic resource]. *UMIA Thesis*. Retrieved from https://repository.tcu.edu/handle/116099117/4320

Kalliomäki, M., Collado, M. C., Salminen, S., & Isolauri, E. (2008). Early differences in fecal microbiota composition in children may predict overweight. *The American journal of clinical nutrition*, *87*(3), 534-538.

Kimani-Murage, E. W. (2013). Exploring the paradox: double burden of malnutrition in rural South Africa. *Global health action, 6*.

Kossoff, E. H., & Hartman, A. L. (2012). Ketogenic diets: new advances for metabolism-based therapies. *Current Opinion in Neurology, 25*(2), 173–178. doi:10.1097/WCO.0b013e3283515e4a

Krebs, H. A. (1966). The regulation of the release of ketone bodies by the liver. *Advances in Enzyme Regulation, 4*, 339–354.

Kruger, F.C., Daniels, C., Kidd, M., Swart, G., Brundyn, K., Van Rensburg, C., Kotze, M.J. (2010). Non-alcoholic fatty liver disease (NAFLD) in the Western Cape: a descriptive analysis. *S Afr Med J., 100*(3):168-71.

Kuczynski, J., Lauber, C. L., Walters, W. A., Parfrey, L. W., Clemente, J. C., Gevers, D., & Knight, R. (2012). Experimental and analytical tools for studying the human microbiome. *Nature Reviews Genetics, 13*(1), 47-58. doi:10.1038/nrg3129

Lamb, J.J. (2012). Homeostasis: A dynamic Balance, In Jones, D.S., Quinn, S. *Textbook of Functional Medicine,* pp.93-96. Gig Harbour: The Institute Of Functional Medicine

Lambert, E. V., & Kolbe-Alexander, T. L. (2013). Innovative strategies targeting obesity and non-communicable diseases in South Africa: what can we learn from the private healthcare sector?. *Obesity reviews, 14*(S2), 141-149. doi:10.1111/obr.12094

Landete, J. M., Arqués, J., Medina, M., Gaya, P., de Las Rivas, B., & Muñoz, R. (2016). Bioactivation of phytoestrogens: intestinal bacteria and health. *Critical reviews in food science and nutrition*, *56*(11), 1826-1843. doi:10.1080/10408398.2013.789823

Lehninger, A. L., Nelson, D. L., & Cox, M. M. (2008). *Lehninger principles of biochemistry* (5th ed). New York: W.H. Freeman.

Lesch, E., & Adams, A. R. (2016). Couples living with and around alcohol abuse: A study of a farmworker community in the Cape Winelands, South Africa. *Social Science & Medicine*, *156*, 167-174.

Leung, M. Y. M., Carlsson, N. P., Colditz, G. A., & Chang, S.-H. (2017). The Burden of Obesity on Diabetes in the United States: Medical Expenditure Panel Survey, 2008 to 2012. *Value in Health, 20*(1), 77–84. doi:10.1016/j.jval.2016.08.735

Ley, R. E., Bäckhed, F., Turnbaugh, P., Lozupone, C. A., Knight, R. D., & Gordon, J. I. (2005). Obesity alters gut microbial ecology. *Proceedings of the National Academy of Sciences of the United States of America, 102*(31), 11070–11075.

Lin, Z., Owen, A. B., & Altman, R. B. (2004). Genomic research and human subject privacy. *Science*, *305*(5681), 183-183.

Liska, D.,Lukaczer, D. (2010). Web-like interconnections of physiological Symptoms. In Jones, D.S., Quinn, S., *Textbook of Functional Medicine*, pp.97-99. Gig Harbour: The Institute Of Functional Medicine

Lumeng, C. N., & Saltiel, A. R. (2011). Inflammatory links between obesity and metabolic disease. *The Journal Of Clinical Investigation, 121*(6), 2111-2117. doi:10.1172/JCI57132

Luppino, F. S., de Wit, L. M., Bouvy, P. F., Stijnen, T., Cuijpers, P., Penninx, B. H., &

Zitman, F. G. (2010). Overweight, obesity, and depression: a systematic review and meta-analysis of longitudinal studies. *Archives Of General Psychiatry, 67*(3), 220-229. doi:10.1001.archgenpsychiatry.2010.2

Ma, J., Fox, C. S., Jacques, P. F., Speliotes, E. K., Hoffmann, U., Smith, C. E., … McKeown, N. M. (2015). Sugar-sweetened beverage, diet soda, and fatty liver disease in the Framingham Heart Study cohorts. *Journal of Hepatology, 63*(2), 462–469. doi:10.1016/j.jhep.2015.03.032

Manninen, A. H. (2004). Metabolic Effects of the Very-Low-Carbohydrate Diets: Misunderstood "Villains" of Human Metabolism. *Journal of the International Society of Sports Nutrition, 1*(2), 7–11. doi:10.1186/1550-2783-1-2-7

Mattes, R. D., & Popkin, B. M. (2009). Nonnutritive sweetener consumption in humans: effects on appetite and food intake and their putative mechanisms–. *The American Journal of Clinical Nutrition, 89*(1), 1–14. doi:10.3945/ajcn.2008.26792

Melnik, B. C. (2009). Milk–the promoter of chronic Western diseases. *Medical hypotheses*, *72*(6), 631-639.

Meule, A., Heckel, D., & Kübler, A. (2012). Factor structure and item analysis of the Yale Food Addiction Scale in obese candidates for bariatric surgery. *European Eating Disorders Review, 20*(5), 419-422. doi:10.1002/erv.2189

Miura, K. (2014). Role of gut microbiota and Toll-like receptors in nonalcoholic fatty liver disease. *World Journal of Gastroenterology, 20*(23), 7381. doi:10.3748/wjg.v20 i23.7381

Mokdad, A. H., Marks, J. S., Stroup, M. F., & Gerberding, J. L. (2004). Actual causes of death in the United States. *Journal of the American Medical Association, 291*(10), 1238–1245.

Monteiro, R., & Azevedo, I. (2010). Chronic Inflammation in Obesity and the Metabolic Syndrome. *Mediators of Inflammation*, 1–10. doi:10.1155/2010/289645

Murphy, P., Likhodii, S., Nylen, K., & Burnham, W. M. (2004). The antidepressant properties of the ketogenic diet. *Biological psychiatry, 56*(12), 981-983. doi:10.1016/j.biopsych.2004.09.019

Nakamura, Y., Ueshima, H., Okuda, N., Miura, K., Kita, Y., Miyagawa, N., … Stamler, J. (2016). Relationship of three different types of low-carbohydrate diet to cardiometabolic risk factors in a Japanese population: the Intermap/interlipid Study. *European Journal of Nutrition, 55*(4), 1515–1524. doi:10.1007/s00394-015-0969-z

Nestle, M. (2013). *Food politics: How the food industry influences nutrition and health (Vol. 3)*. Univ of California Press.

Neufeld, K. M., Kang, N., Bienenstock, J., & Foster, J. A. (2011). Reduced anxiety-like behavior and central neurochemical change in germ-free mice. *Neurogastroenterology & Motility, 23*(3), 255-e119. doi:10.1111/j.1365-2982.2010.01620.x

Nhlapo, N., Lues, R. F., Kativu, E., & Groenewald, W. H. (2015). Assessing the quality of food served under a South African school feeding scheme: A nutritional analysis. *South African Journal Of Science, 111*(1/2), 1-9. doi:10.17159/sajs.2015/20140044

Nieto, M. M., Wilson, J., Cupo, A., Roques, B. P., & Noble, F. (2002). Chronic morphine treatment modulates the extracellular levels of endogenous enkephalins in rat brain structures involved in opiate dependence: a microdialysis study. *The Journal of Neuroscience, 22*(3), 1034–1041.

Nutrition Society of South Africa [NSSA] (2013). *The new South African food-based dietary guidelines in perspective*. Retrieved October 7, 2015, from http://www.nutritionsociety. co.za/index.php/11-useful-information/26-the-new-south-african-food-based-dietary- guidelines-in-perspective

Oriach, C. S., Robertson, R. C., Stanton, C., Cryan, J. F., & Dinan, T. G. (2016). Food for thought: The role of nutrition in the microbiota-gut–brain axis. *Clinical Nutrition*

Experimental, 6, 25–38. doi:10.1016/j.yclnex.2016.01.003

O'Malley, M. A., & Stotz, K. (2011). Intervention, integration and translation in obesity research: Genetic, developmental and metaorganismal approaches. *Philosophy, Ethics, And Humanities In Medicine: PEHM*, *62*. doi:10.1186/1747-5341-6-2

Paoli, A. (2014). Ketogenic Diet for Obesity: Friend or Foe? *International Journal of Environmental Research and Public Health, 11*(2), 2092–2107. doi:10.3390/ijerph110202092

Paoli, A., Bianco, A., Grimaldi, K. A., Lodi, A., & Bosco, G. (2013). Long term successful weight loss with a combination biphasic ketogenic Mediterranean diet and Mediterranean diet maintenance protocol. *Nutrients, 5*(12), 5205-5217. doi:10.3390/nu5125205

Parry, C. D. H., (2005) South Africa: alcohol today. *Addiction, 100*(4), 426–429. doi:10.1111/j.1360-0443.2005.01015.x.

Paruk, I., Pirie, F., Motala, A., & Kolawole, B. (2011). High prevalence of abnormal liver enzymes in South African patients with type 2 diabetes mellitus attending a diabetes clinic. Journal of Endocrinology, *Metabolism and Diabetes of South Africa, 16*(1), 43–47. doi:10.1080/22201009.2011.10872250

Perlmutter, D., & Loberg, K. (2013). *Grain Brain: The Surprising Truth about Wheat, Carbs, and Sugar--Your Brain's Silent Killers (1 edition)*. New York, NY: Little, Brown and Company.

Phy Ali M, J. L. (2015). Low Starch/Low Dairy Diet Results in Successful Treatment of Obesity and Co- Morbidities Linked to Polycystic Ovary Syndrome (PCOS). *Journal of Obesity & Weight Loss Therapy, 05*(02). doi:10.4172/2165-7904.1000259

Pivovarova, O., Jürchott, K., Rudovich, N., Hornemann, S., Lu, Y., Möckel, S., ... & Pfeiffer, A. F. (2015). Changes of dietary fat and carbohydrate content alter central and peripheral clock in humans. *The Journal of Clinical Endocrinology & Metabolism,*

122(03). doi:10.1055/s-0035-1547612

Poobalan, A., & Aucott, L. (2016). Obesity Among Young Adults in Developing Countries: A Systematic Overview. *Current Obesity Reports, 5*(1), 2–13. doi:10.1007/ s13679-016-0187-x

Putnam, J. J., & Allshouse, J. E. (1999). Food Consumption, Prices and Expenditure, 1970–1997. USDA Food & Rural Economic Division. *Economic Research Service Bulletin, 34*(965).

Qin, J., Li, Y., Cai, Z., Li, S., Zhu, J., Zhang, F., & ... Wang, J. (2012). A metagenome-wide association study of gut microbiota in type 2 diabetes. *Nature, 490*(7418), 55-60. doi:10.1038/nature11450

Rajilić-Stojanović, M., Jonkers, D. M., Salonen, A., Hanevik, K., Raes, J., Jalanka, J., ... & Penders, J. (2015). Intestinal Microbiota And Diet in IBS: Causes, Consequences, or Epiphenomena&quest. *The American journal of gastroenterology*. doi:10.1038/ajg.2014.427

Revelle, W. (2017, May 3). *R-package "psych"*, Procedures for psychological, psychometric, and personality research. Retrieved from http://personality-project.org/r/psych

Riccio, P. (2011). The molecular basis of nutritional intervention in multiple sclerosis: A narrative review. *Complementary Therapies in Medicine, 19*(4), 228–237. doi:10.1016/j.ctim.2011.06.006

Ridaura, V. K., Faith, J. J., Rey, F. E., Cheng, J., Duncan, A. E., Kau, A. L., ... & Muehlbauer, M. J. (2013). Gut microbiota from twins discordant for obesity modulate metabolism in mice. *Science*, *341*(6150), 1241214. doi:10.1126/science.1241214

Romero-Corral, A., Somers, V. K., Sierra-Johnson, J., Thomas, R. J., Collazo-Clavell, M. L., Korinek, J., … Lopez-Jimenez, F. (2008). Accuracy of body mass index in diagnosing obesity in the adult general population. *International Journal of Obesity, 32*(6), 959–966.

Runow, K.D. (2011). *Der Darm denkt mit (The GI thinks*). Munich: Südwest Verlag, Random House

Sánchez-Villegas, A., Delgado-Rodríguez, M., Alonso, A., Schlatter, J., Lahortiga, F., Serra Majem, L., & Martínez-González, M. A. (2009). Association of the Mediterranean dietary pattern with the incidence of depression: the Seguimiento Universidad de Navarra/ University of Navarra follow-up (SUN) cohort. *Archives Of General Psychiatry, 66*(10), 1090-1098. doi:10.1001/archgenpsychiatry.2009.129

Schaubeck, M., Clavel, T., Calasan, J., Lagkouvardos, I., Haange, S. B., Jehmlich, N., … Haller, D. (2015). Dysbiotic gut microbiota causes transmissible Crohn's disease-like ileitis independent of failure in antimicrobial defence. *Gut*. doi:10.1136/ gutjnl-2015-309333

Schmidt, R., Schmidt, H., Curb, J. D., Masaki, K., White, L. R., & Launer, L. J. (2002). Early inflammation and dementia: a 25-year follow-up of the Honolulu-Asia Aging Study. *Annals Of Neurology, 5*2(2), 168-174. doi:10.1002/ana.10265

Schönfeldt, H. C., Hall, N., & Bester, M. (2013). Relevance of food-based dietary guidelines to food and nutrition security: A South African perspective. *Nutrition Bulletin, 38*(2), 226- 235. doi:10.1111/nbu.12027

Seves, S., Verkaik-Kloosterman, J., Biesbroek, S., & Temme, E. (2017). Are more environmentally sustainable diets with less meat and dairy nutritionally adequate? *Public Health Nutrition, 20*(11), 2050-2062. doi:10.1017/S1368980017000763

Sonnenburg, E. D., & Sonnenburg, J. L. (2014). Starving our microbial self: the deleterious consequences of a diet deficient in microbiota-accessible carbohydrates. *Cell Metabolism, 20*(5), 779-786. doi:10.1016/j.cmet.2014.07.003

Spyridaki, E. C., Avgoustinaki, P. D., & Margioris, A. N. (2016). Obesity, inflammation and cognition. *Current Opinion in Behavioral Sciences, 9*, 169–175. doi:10.1016/j.cobeha.2016.05.004

Stafstrom, C. E., & Rho, J. M. (2012). The ketogenic diet as a treatment paradigm for diverse neurological disorders. *Frontiers In Pharmacology, 359*. doi:10.3389/fphar.2012.00059

Stipanuk, M. H. (2006). *Biochemical, physiological, & molecular aspects of human nutrition*. St. Louis: Saunders Elsevier.

Suez, J., Korem, T., Zeevi, D., Zilberman-Schapira, G., Thaiss, C. A., Maza, O., ... & Elinav, E. (2014). Artificial sweeteners induce glucose intolerance by altering the gut microbiota. *Nature, 514*(7521), 181-186. doi:10.1038/nature13793

Tatum, J. (2010). Psychosocial influences. In Jones, D.S., Quinn, S., *Textbook of Functional Medicine*, pp.137-140. Gig Harbour: The Institute Of Functional Medicine

Thornley, S., Russell, B., & Kydd, R. (2011). Carbohydrate reward and psychosis: an explanation for neuroleptic induced weight gain and path to improved mental health? *Current Neuropharmacology, 9*(2), 370.

Tillisch, K., Labus, J., Kilpatrick, L., Jiang, Z., Stains, J., Ebrat, B., & ... Mayer, E. A. (2013). Consumption of fermented milk product with probiotic modulates brain activity. *Gastroenterology, 144*(7), 1394. doi:10.1053/j.gastro.2013.02.043

Tucker, D. M., Penland, J. G., Sandstead, H. H., Milne, D. B., Heck, D. G., & Klevay, L. M. (1990). Nutrition status and brain function in aging. *The American Journal Of Clinical Nutrition, 52*(1), 93-102.

Turnbaugh, P. J., Ley, R. E., Mahowald, M. A., Magrini, V., Mardis, E. R., & Gordon, J. I. (2006). An obesity-associated gut microbiome with increased capacity for energy harvest. *Nature, 444*(7122), 1027-1031. doi:10.1038/nature05414

Turnbaugh, P. J., Bäckhed, F., Fulton, L., & Gordon, J. I. (2008). Diet-Induced Obesity Is Linked to Marked but Reversible Alterations in the Mouse Distal Gut Microbiome. *Cell Host & Microbe, 3*(4), 213–223. doi:10.1016/j.chom.2008.02.015

Turnbaugh, P. J., Hamady, M., Yatsunenko, T., Cantarel, B. L., Duncan, A., Ley, R. E., ... &

Egholm, M. (2009). A core gut microbiome in obese and lean twins. *nature*, *457*(7228), 480-484.

Turnbaugh, P. J., Ridaura, V. K., Faith, J. J., Rey, F. E., Knight, R., & Gordon, J. I. (2009). The effect of diet on the human gut microbiome: a metagenomic analysis in humanized gnotobiotic mice. *Science translational medicine*, *1*(6), 6ra14-6ra14.

Turner, H., & Bryant-Waugh, R. (2004). Eating disorder not otherwise specified (EDNOS): profiles of clients presenting at a community eating disorder service. *European Eating Disorders Review*, *12*(1), 18-26. doi:10.1002/erv.552

Vasquez, A., (2010). Organ System Function and the Underlying Mechanisms: The Interconnected Web. In Jones, D.S., Quinn, S., *Textbook of Functional Medicine*, 99-103. Gig Harbour: The Institute Of Functional Medicine

Veech, R. L. (2004). The therapeutic implications of ketone bodies: the effects of ketone bodies in pathological conditions: ketosis, ketogenic diet, redox states, insulin resistance, and mitochondrial metabolism. *Prostaglandins, Leukotrienes, and Essential Fatty Acids, 70*(3), 309–319. doi:10.1016/j.plefa.2003.09.007

Volek, J. S., Noakes, T., & Phinney, S. D. (2015). Rethinking fat as a fuel for endurance exercise. *European Journal of Sport Science, 15*(1), 13–20. doi:10.1080/17461391.2014.959564

Volkow, N. D., Fowler, J. S., Wang, G., & Goldstein, R. Z. (2002). Role of dopamine, the frontal cortex and memory circuits in drug addiction: insight from imaging studies. *Neurobiology of Learning and Memory,78*(3), 610-624. doi:10.1006/nlme.2002.4009

Vojdani, A., O'Bryan, T., & Kellermann, G. H. (2008). The immunology of immediate and delayed hypersensitivity reaction to gluten. *European Journal of Inflammation, 6*(1), 1–10.

Vrieze, A., Van Nood, E., Holleman, F., Salojärvi, J., Kootte, R. S., Bartelsman, J. F. W. M., … Nieuwdorp, M. (2012). Transfer of Intestinal Microbiota From Lean Donors

Increases Insulin Sensitivity in Individuals With Metabolic Syndrome. *Gastroenterology, 143*(4), 913–916.e7. doi:10.1053/j.gastro.2012.06.031

Vučić, V., Arsić, A., Petrović, S., Milanović, S., Gurinović, M., & Glibetić, M. (2015). Trans fatty acid content in Serbian margarines: Urgent need for legislative changes and consumer information. *Food chemistry, 185*, 437-440. doi:10.1016/j.foodchem.2015.04.018

Wang, G., Volkow, N. D., Pappas, N. R., Netusil, N., Wong, C. T., Logan, Fowler, J. S. & Zhu, W. (2001). Brain dopamine and obesity. *Lancet, 357*(9253), 354. doi:10.1016/S0140-6736(00)03643-6

Werbach, M. (1992). Nutritional influences on aggressive behavior. *J Orthomol Med, 7*, 45-51.

West, J. H., Belvedere, L. M., Andreasen, R., Frandsen, C., Hall, P. C., & Crookston, B. T. (2017). Controlling Your "App"etite: How Diet and Nutrition-Related Mobile Apps Lead to Behavior Change. *JMIR MHealth and UHealth, 5*(7). doi:10.2196/mhealth.7410

Westerterp-Plantenga, M. S., Nieuwenhuizen, A., Tomé, D., Soenen, S., & Westerterp, K. R. (2009). Dietary protein, weight loss, and weight maintenance. *Annual Review of Nutrition, 29*, 21–41. doi:10.1146/annurev-nutr-080508-141056

World health Organization [WHO] *Obesity*. Retrieved from http://www.who.int/topics/obesity/en/, 2015

Wilsgaard, T., & Jacobsen, B. K. (2007). Lifestyle factors and incident metabolic syndrome. *Diabetes Research and Clinical Practice, 78*(2), 217–224. doi:10.1016/j.diabres.2007.03.006

World health Organization [WHO] *Obesity and Overweight*. Retrieved from http://www.who.int/ mediacentre/factsheets/fs311/en/, 2017

Yang, H., Youm, Y. H., Vandanmagsar, B., Ravussin, A., Gimble, J. M., Greenway, F., ... &

Dixit, V. D. (2010). Obesity increases the production of proinflammatory mediators from adipose tissue T cells and compromises TCR repertoire diversity: implications for systemic inflammation and insulin resistance. *The Journal of Immunology, 185*(3), 1836- 1845. doi:10.4049/jimmunol.1000021

Younossi, Z.M., Koenig, A.B., Abdelatif, D., Fazel, Y., Henry, L., Wymer, M. (2016) Global epidemiology of nonalcoholic fatty liver disease-Meta-analytic assessment of prevalence, incidence, and outcomes. *Hepatology, 64*(1):73-84. doi: 10.1002/hep.28431

Zhang, R., Miller, R. G., Gascon, R., Champion, S., Katz, J., Lancero, M., & ... McGrath, M. S. (2009). Circulating endotoxin and systemic immune activation in sporadic amyotrophic lateral sclerosis (sALS). *Journal Of Neuroimmunology, 206*(1-2), 121-124. doi:10.1016/j.jneuroim.2008.09.017

Zhang, Y., & Zhang, H. (2013). Microbiota associated with type 2 diabetes and its related complications. *Food Science and Human Wellness, 2*(3-4), 167–172. doi:10.1016/j.fshw.2013.09.002

Ziauddeen, H., & Fletcher, P. C. (2013). Is food addiction a valid and useful concept?: Food addiction: valid and useful? *Obesity Reviews, 14*(1), 19–28. doi:10.1111/j.1467-789X.2012.01046.x

Zilberter, T., (2012). Food addiction and obesity: do macronutrients matter? *Frontiers in neuroenergetics, 4* . doi:10.3389/fnene.2012.00007AA

Appendix 1: DSM-V

Table 1
Fifth edition of the Diagnostic Statistical Manual's proposed criteria for substance-use disorder

Substance-use disorder

A maladaptive pattern of substance use leading to clinically significant impairment or distress, as manifested by two (or more) of the following, occurring within a 12-month period

1. Recurrent substance use resulting in a failure to fulfill major role obligations at work, school, or home (e.g., repeated absences or poor work performance related to substance use; substance-related absences, suspensions, or expulsions from school; neglect of children or household)

2. Recurrent substance use in situations in which it is physically hazardous (e.g., driving an automobile or operating a machine when impaired by substance use)

3. Continued substance use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of the substance (e.g., arguments with spouse about consequences of intoxication and physical fights)

4. Tolerance, as defined by either of the following:
(a) Need for markedly increased amounts of the substance to achieve intoxication or desired effect
(b) Markedly diminished effect with continued use of the same amount of the substance (Note: tolerance is not counted for those taking medications under medical supervision such as analgesics, antidepressants, ant-anxiety medications, or beta-blockers)

5. Withdrawal, as manifested by either of the following:
(a) The characteristic withdrawal syndrome for the substance (refer to Criteria A and B of the criteria sets for withdrawal from the specific substances)
(b) The same (or a closely related) substance is taken to relieve or avoid withdrawal symptoms (Note: withdrawal is not counted for those taking medications under medical supervision such as analgesics, antidepressants, antianxiety medications, or beta-blockers)

6. The substance is often taken in larger amounts or over a longer period than was intended

7. There is a persistent desire or unsuccessful efforts to cut down or control substance use

8. A great deal of time is spent on activities necessary to obtain the substance, use the substance, or recover from its effects

9. Important social, occupational, or recreational activities are given up or reduced because of substance use

10. The substance use is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by the substance

11. Craving or a strong desire or urge to use a specific substance

Appendix 2: Consent form

Low Carbohydrate Nutritional Intervention
Informed CONSENT FORM

This Informed Consent Form is for men and women who would like to participate in a one month long nutritional intervention aiming at establishing the addictive potential of carbohydrates.

Ann Kistenmacher is the Principal Investigator. The study is part of her Masters Dissertation at UNISA. The Study will be supported and monitored by a nutritional expert.

You will be given a copy of the full Informed Consent Form

Introduction

I am Ann Kistenmacher, a masters Student at UNISA. I would like to determine the effect of a low carbohydrate nutritional intervention on cravings and needs commonly associated with carbohydrate consumption. I am going to give you information and invite you to be part of this research. You do not have to decide today whether or not you will participate in the research. Before you decide, you can talk to anyone you feel comfortable with about the research. There may be some words that you do not understand. Please ask me to stop as we go through the information and I will take time to explain. If you have questions later, you can ask them of me or the study nutritionist.

Purpose of the Research/Type of Intervention/What to expect

Principally, your food intake will be altered from a high carbohydrate intake to a low one. This does not mean that you will go hungry - on the contrary - you might find your mind quite at ease without snacking inbetween meals. The way to cook, shop and what to look out for will be explained to you by our nutritionist. It will not incur extra cost and might even save on meal preparation time. Ideally you will follow your new eating plan for 1 month. We will assess your physical state before and after the intervention (body weight and waist to hip ratio). Additionally you will be asked to fill out a questionnaire to establish your relationship to food before and after the intervention. We will be available upon request to all participants to share problems and thoughts. The intervention will be of a duration of 1 month. If, at any stage, you feel unable to follow the protocol of the intervention, we will have to ask you to stop participating. You will be asked to fill out a questionnaire on why you felt you could not continue.

Benefits of Participating

Discoveries made as a result of this research could be used to understand the basic causes of addictive behaviour. The Low Carb High fat dietary intervention could potentially improve your overall state of well-being.
This study does not seek to and does not treat or cure any medical condition, and participation should not be used as a substitute for any medical treatment.

Risks of Participating

• Some survey questions may make you uncomfortable.

Information that participants choose to share with their physician or other health care provider may become part of their medical record.

None of the surveys or other procedures used by the investigators in the Research study are invasive or experimental. The procedures involved do not involve significant risks, and no compensation or treatment is available if injury occurs as a result of participation.

Voluntary Participation

At any time, you may choose to withdraw all or some of your information from our research by sending a request to the Administrator at annkiste@gmail.com.

Any research on your data that has been performed or published prior to this date will not be reversed, undone, or withdrawn. Your information may still be used.

Confidentiality

The information that we collect from this research project will be kept confidential. Information about you that will be collected during the research will be put away and no-one but the researchers will be able to see it. Any information about you will have a number on it instead of your name. Only the researchers will know what your number is. It will not be shared with or given to anyone.

The knowledge that we get from doing this research will be shared with you before it is made widely available to the public. Confidential information will not be shared. We will publish the results in order that other interested people may learn from our research.

Contact Information

If you have any questions or concerns about the study, if you suffer a esearch related injury, or if you have a question about participants' rights, please contact the following: annkiste@gmail.com

This proposal has been reviewed and approved by the ethics committe of UNISA, which is a committee whose task it is to make sure that research participants are protected from harm.

I have read the foregoing information, or it has been read to me. I have had the opportunity to ask questions about it and any questions that I have asked have been answered to my satisfaction. I consent voluntarily to participate as a participant in this research.

☐ I AGREE TO PARTICIPATE

☐ I DO NOT WISH TO PARTICIPATE AT THIS TIME

☐ You may contact me in the future to invite my participation in additional research

_____ _____

Signature of participant Date

Appendix 3: YFAS participation questionnaire

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, ice cream
- Starches like white bread, rolls, pasta, and rice
- Salty snacks like chips, pretzels, 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 food similar to those listed in the food group or ANY OTHER foods you have had a problem with in the past year

Name .....

Gender .....

Age .....

Weight .....

Height .....

Waist to hip ratio .....

YFAS

in the past 12 months:

		never	once a month	2-3 times a month	2-3 times a week	4 or more daily
1	I find that when I start eating certain foods, I end up eating much more than planned	0	1	2	3	4
2	I find myself continuing to consume certain foods even though I am no longer hungry	0	1	2	3	4
3	I eat to the point where I feel physically ill	0	1	2	3	4
4	Not eating certain types of food or cutting down on certain types of food is something I worry about	0	1	2	3	4
5	I spend a lot of time feeling sluggish or fatigued from overeating	0	1	2	3	4
6	I find myself constantly eating certain foods throughout the day	0	1	2	3	4
7	I find that when certain foods are not available, I will go out of my way to obtain them. For example, I will drive to the store to purchase certain foods even though I have other options available to me at home.	0	1	2	3	4
8	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.	0	1	2	3	4
9	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.	0	1	2	3	4
10	There have been times when I avoided professional or social situations where certain foods were available, because I was afraid I would overeat.	0	1	2	3	4
11	There have been times when I avoided professional or social situations because I was not able to consume certain foods there.	0	1	2	3	4
12	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.)	0	1	2	3	4
13	I have consumed certain foods to prevent feelings of anxiety, agitation, or other physical symptoms that were developing. (Please do NOT include consumption of caffeinated beverages such as soda pop, coffee, tea, energy drinks, etc.)	0	1	2	3	4
14	I have found that I have elevated desire for or urges to consume certain foods when I cut down or stop eating them.	0	1	2	3	4
15	My behavior with respect to food and eating causes significant distress.	0	1	2	3	4
16	I experience significant problems in my ability to function effectively (daily routine, job/school, social activities, family activities, health difficulties) because of food and eating.	0	1	2	3	4
17	My food consumption has caused significant psychological problems such as depression, anxiety, self-loathing, or guilt.	0	1	2	3	4
18	My food consumption has caused significant physical problems or made a physical problem worse.	0	1	2	3	4
19	I kept consuming the same types of food or the same amount of food even though I was having emotional and/or physical problems.	0	1	2	3	4
20	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.	0	1	2	3	4
21	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.	0	1	2	3	4
22	I want to cut down or stop eating certain kinds of food.	0	1	2	3	4
23	I have tried to cut down or stop eating certain kinds of food.	0	1	2	3	4
24	I have been successful at cutting down or not eating these kinds of food	0	1	2	3	4

		1 time	2 time	3 time	4 time	5 or more times
25	How many times in the past year did you try to cut down or stop eating certain foods altogether?					

Please circle ALL of the following foods you have problems with:

Ice Cream	Chocolate	Apples	Doughnuts	Broccoli	Cookies
White Bread	Rolls	Lettuce	Pasta	Stawberries	Rice
Cake	Candy	Crackers	Chips	Pretzels	French Fries
Carrots	Steak	Bananas	Bacon	Hamburgers	Cheese Burgers
	Pizza	Soda Pop	None of the above		

Please list any other foods that you have problems with that were not previously listed:

Appendix 4: Eating Attitudes Test (EAT-26)

Eating Attitudes Test (EAT-26)©

Instructions: This is a screening measure to help you determine whether you might have an eating disorder that needs professional attention. This screening measure is not designed to make a diagnosis of an eating disorder or take the place of a professional consultation. Please fill out the below form as accurately, honestly and completely as possible. There are no right or wrong answers. All of your responses are confidential.

Part A: Complete the following questions:

1) Birth Date Month:	Day:	Year:	2) Gender:	Male	Female
3) Height Feet :	Inches:			□	□
4) Current Weight (lbs.):	5) Highest Weight (excluding pregnancy):				
6) Lowest Adult Weight:	7: Ideal Weight:				

Part B: Check a response for each of the following statements:	Always	Usually	Often	Some times	Rarely	Never
1. Am terrified about being overweight.	□	□	□	□	□	□
2. Avoid eating when I am hungry.	□	□	□	□	□	□
3. Find myself preoccupied with food.	□	□	□	□	□	□
4. Have gone on eating binges where I feel that I may not be able to stop.	□	□	□	□	□	□
5. Cut my food into small pieces.	□	□	□	□	□	□
6. Aware of the calorie content of foods that I eat.	□	□	□	□	□	□
7. Particularly avoid food with a high carbohydrate content (i.e. bread, rice, potatoes, etc.)	□	□	□	□	□	□
8. Feel that others would prefer if I ate more.	□	□	□	□	□	□
9. Vomit after I have eaten.	□	□	□	□	□	□
10. Feel extremely guilty after eating.	□	□	□	□	□	□
11. Am preoccupied with a desire to be thinner.	□	□	□	□	□	□
12. Think about burning up calories when I exercise.	□	□	□	□	□	□
13. Other people think that I am too thin.	□	□	□	□	□	□
14. Am preoccupied with the thought of having fat on my body.	□	□	□	□	□	□
15. Take longer than others to eat my meals.	□	□	□	□	□	□
16. Avoid foods with sugar in them.	□	□	□	□	□	□
17. Eat diet foods.	□	□	□	□	□	□
18. Feel that food controls my life.	□	□	□	□	□	□
19. Display self-control around food.	□	□	□	□	□	□
20. Feel that others pressure me to eat.	□	□	□	□	□	□
21. Give too much time and thought to food.	□	□	□	□	□	□
22. Feel uncomfortable after eating sweets.	□	□	□	□	□	□
23. Engage in dieting behavior.	□	□	□	□	□	□
24. Like my stomach to be empty.	□	□	□	□	□	□
25. Have the impulse to vomit after meals.	□	□	□	□	□	□
26. Enjoy trying new rich foods.	□	□	□	□	□	□

Part C: Behavioral Questions: In the past 6 months have you:		Never	Once a month or less	2-3 times a month	Once a week	2-6 times a week	Once a day or more
A	Gone on eating binges where you feel that you may not be able to stop? *	□	□	□	□	□	□
B	Ever made yourself sick (vomited) to control your weight or shape?	□	□	□	□	□	□
C	Ever used laxatives, diet pills or diuretics (water pills) to control your weight or shape?	□	□	□	□	□	□
D	Exercised more than 60 minutes a day to lose or to control your weight?	□	□	□	□	□	□
E	Lost 20 pounds or more in the past 6 months	Yes □		No □			

* Defined as eating much more than most people would under the same circumstances and feeling that eating is out of control

Appendix 5: YFAS Instruction sheet

Instruction Sheet for the Yale Food Addiction Scale

(Gearhardt, Corbin, & Brownell, 2009)

Contact Information: ashle gearhardt ale edu

The ale ood ddiction Scale is a measure that has been de eloped to identif those who are most li el to be e hibiting mar ers of substance dependence with the consumption of high fat high sugar foods

e elopment

The scale questions fall under specific criteria that resemble the s mptoms for substance dependence as stated in the iagnostic and Statistical anual of ental isorders I R and operationali ed in the Structured Clinical Inter iew for S I is I isorders

1) Substance taken in larger amount and for longer period than intended
Questions #1, #2, #3

2) Persistent desire or repeated unsuccessful attempts to quit
Questions #4, #22, # 24, #25

3) Much time/activity to obtain, use, recover
Questions #5, #6, #7

4) Important social, occupational, or recreational activities given up or reduced
Questions #8, #9, #10, #11

5) Use continues despite knowledge of adverse consequences (e.g., failure to fulfill role obligation, use when physically hazardous)
Question #19

6) Tolerance (marked increase in amount; marked decrease in effect)
Questions #20, #21

7) Characteristic withdrawal symptoms; substance taken to relieve withdrawal
Questions #12, #13, #14

8) Use causes clinically significant impairment or distress
Questions #15, #16

Cut offs

The following cut offs were de eloped for the continuous questions

0 criterion not met, 1 criterion is met

The following questions are scored 0 (0), 1 (1): #19, #20, #21, #22
The following question is scored 0 (1), 1 (0): #2
The following questions are scored 0 (0 thru 1), 1 (2 thru): #8, #10, #11
The following questions are scored 0 (0 thru 2), 1 (3 &): #3, # , #7, #9, #12, #13, #1 , #1 , #1
The following questions are scored 0 (0 thru 3), 1 ( ): #1, #2, # , #
The following questions are scored 0 (0 thru), 1 ( ): #2

The following questions are NOT scored, but are primers for other questions: #17, #18, #23

SCORING

fter computing cut offs, sum up the questions under each substance dependence criterion (e g Tolerance, ithdrawal, Clinical Significance, etc) If the score for the criterion is _ 1, then the criterion has been met and is scored as 1 If the score 0, then the criteria has not been met

ample:
Tolerance: (#20 1) (#21 0) 1, Criterion et
ithdrawal (#12 0) (#13 0) (#1 0) 0, Criterion Not et
Gi en up (#8 1) (#9 0) (#10 1) (#11 1) 3, Criterion et and scored as 1

To score the continuous ersion of the scale, which resembles a s mptom count without diagnosis, add up all of the scores for each of the criterion (e g Tolerance, ithdrawal, se espite Negati e Consequence) o NOT add clinical significance to the score This score should range from 0 to 7 (0 s mptoms to 7 s mptoms)

To score the dichotomous ersion, which resembles a diagnosis of substance dependence, compute a iable in which clinical significance must 1 (items 1 or 1 1), and the s mptom count must be _ 3 This should be either a 0 or 1 score (no diagnosis or diagnosis met)

Norms (undergraduates)

iagnosis of ood ependence – 11
edian S mptom Count Score – 1 0
ithdrawal – 1 3
Tolerance – 13
Continued se espite roblems – 28 3
Important cti ities Gi en p – 10 3
arge mounts of Time Spent – 2 0
oss of Control – 21 7
a e Tried nsuccessfull to Cut own or orried bout Cutting own – 71 3
Clinicall Significant Impairment 1

Appendix 6: Eat This_30 day reset_Carn Addiction Study AK

eat this.

hello sweetie

Congratulations, you have just bought yourself a new lifestyle starting with your individual nutritional plan. Major health benefits are about to surprise you within the first days, weeks and month already, and you will feel increasingly better as you continue. **Don't think of it as giving something up - you're shedding an old skin** (or old pants..:)

All our nutritional plans follow Paleo principles, the foremost being eating in accordance with human nature: Eat food without antibiotics, hormones, preservatives, 'natural flavours', food which is untreated and without any toxic manipulation during production. ThinkFood **eat this.** reflects your individual situation: the food items chosen are specifically based on you, your frequency & lab values provided, questionnaires and information from your anamnesis.

Do. Or do not. There is no try. (Yoda)

At the beginning only permitted food items are to be consumed. Your immune system (IS) has a memory like an elephant and will remain alert long after the triggering food has passed your digestive tract. Therefore you really need to ban these items for quite a while before you cautiously re-introduce them. **From our experience, only 20% are successful with a 80/20 version of Paleo,** where the 'odd flat white' or 'just one cookie' is permitted during the weekends... even if that cake is 'homemade' and 'you always used to like it'. Detrimental can be the hidden things, like soya and gluten even in organic soya sauce or milk derived digestive enzymes in your expensive supplement. For some people this (a cookie crumble) may mean back to square one.

You should only really start to **eat this.** if your life will allow for 30 days where you, your personal well-being and food are of the utmost importance. Our *Beginners Guide* will give you practical tips how to get started and organise your household. Choose a time when you have less stress and don't have too many work or social commitments.

eat this.

eat this & be nice to people

Despite multiple attempts to tighten your belt, the waistline has stayed the same. Your skin just doesn't reflect the glow that the price of the face cream promised. Another restless night hasn't made you feel or look any better. Even making breakfast in the morning is too much of an effort. The coffee gets you up and running but does not elevate your mood enough to deal with the onslaught of communication during the day. You are tired. And grumpy. Your back is sore and your knee is acting up despite there not 'being any apparent cause'. Perhaps you are taking pain medication or mood elevators to help you through the day. Wouldn't you change this if you could? Certain food groups not only have a negative impact on your health and fitness, but also on your state of mind - on your work and on your family, your partners, your kids, your dog....

Particular foods have addictive properties, such as sugar. Other foods cause allergic reactions or are a causal factor in symptomatology such as a 'leaky gut', auto-immune reactions, skin eruptions or impaired wound healing. Yet other food groups deplete vital vitamins and minerals necessary for chemical reactions in your brain, stabilizing your mood and allowing you to remember Auntie Claire's aversion to Old Spice.

The only way to figure out which foods are good and not so good for your beaten body is to cut them out for a while. Completely. Not even one little crumb or drop. This allows your body a chance to heal and re-sensitize itself so that it can pull the line when the ground is getting close.

back to **basics**

When your computer starts running slowly, applications are crashing left and right and you can't even move the cursor anymore, what do you do? **Control-alt-delete**. Or if you're a Mac user, you hold down the power button to restart.

Sometimes we need to do the same thing with our bodies. They're under constant assault in the modern world. Refined, processed food, environmental toxins, stress, sleep deprivation and chronic infections can all wreak havoc on our health. We're simply not adapted to live this way.

Life might look a lot different today, with smartphones, electric cars and space travel, but our genes haven't changed all that much. This means - with a few exceptions that we'll cover later - we're still hard-wired to eat the foods our hunter-gatherer ancestors ate. When we follow that genetic template, as we did for thousands of generations, we're naturally healthy and vital. But when we stray from it, as we have in the recent past, we suffer. So when things start to go awry, the best thing to do is to get back to basics. To return to that 2.5 million year-old genetic template that humans are meant to follow. **In other words:**

hit the reset button

How do you do this? You commit to a 30-day period where you eliminate the modern foods that cause disease as well as the foods people are most often allergic to or intolerant of, and focus on the safe, nourishing foods our ancestors have thrived on for 77,000 generations. Then, after you've "hit the reset button" and returned to that basic template, you can customize it to find the approach that works best for you over the long term.

But first things first. Let's look at the 30-day Reset in more detail.[1]

1 adapted from 30-day Paleo Reset, Kresser Institue, 2017

eat this. 30-day reset

how does it work

The **eat this.** Reset phase is designed to reduce inflammation, improve digestion, burn fat, identify food sensitivities, reduce allergic reactions, boost energy, regulate blood sugar and stabilize mood. It almost seems too good to be true, but it works. **No other therapy - natural or otherwise - can come even remotely close to accomplishing all of these goals in such a short period of time.**

How long does this phase last? There's no hard and fast answer to that question, but it's absolutely essential that you commit to making these changes for at least 30 days - without cheating.

After completing the **eat this.** Reset you'll have a bit more leeway to go off the rails every now and then. (After all, there's more to life than food!) But the Reset phase is not one of those times. This is where you gather your strength and buckle down. You can do it.

By removing the foods that most commonly cause problems, you allow your body to rest and recover from whatever symptoms those foods have been provoking. Just one cheat could trigger a whole new cascade of reactions. Don't do it. It's not worth it.

Remember, 30 days is just a minimum. Some people may need 45, 60 or even 90 days to get the full benefits of the Reset phase.

what foods **can you eat**

There are **three categories** to make it as easy as possible.

1. eat this. liberally: You can enjoy as much of these foods as you like. No counting calories or calculating ratios of protein, fat or carbohydrate. This isn't a "cleanse" or a fast. If a food is one this list, you're free to eat it.

2. eat this. in moderation: You can eat these foods, but don't go hog wild with them. I've indicated how often or how much of them I think is safe, but in general you want to limit consumption of these foods compared to those in the "eat liberally" category.

3. Avoid completely: Yep, completely. This is where the rubber hits the road. The success (or failure) of the program hinges on your ability to steer clear of these foods during the **eat this.** 30-day Reset.

eat this. **liberally**

+ Meat and poultry. Emphasize beef and lamb, but also pork, chicken, turkey, duck and wild game like gemsbok, kudu, ostrich, etc. Organic and free-range is always preferable, but is especially so during this program.

+ Organ meats (especially liver). Liver is the most nutrient-dense food on the planet. If you don't like the taste of liver, one good trick is to put one chicken liver in each cube of an ice cube tray and freeze them. Then, when you're making any meat dish, dice up one chicken liver and add it to the meat.

+ Bone broth soups. It's essential to balance your intake of muscle meats and organ meats with homemade bone broths. Bone broths are rich in glycine, and amino acid found in collagen, which is a protein important in maintaining a healthy gut lining.

+ Fish. Especially fatty fish like salmon, mackerel and herring. Wild is preferable. You need to eat three 150-200g servings of fatty fish per week to balance your omega-6 to omega-3 ratio.

+ Eggs. Preferably free-range and organic.

+ Starchy tubers. Yams, sweet potatoes, yucca/manioc, taro, lotus root, etc.

+ Non-starchy vegetables. Cooked and raw.

+ Fermented vegetables and fruits. Sauerkraut, kim chi, beet kvaas, coconut kefir, etc. These are excellent for gut health.

+ Traditional fats. Coconut oil, palm oil, lard, duck fat, beef tallow and olive oil.

+ Olives, avocados and coconuts (including coconut milk).

+ Sea salt and spices. Avoid sugar or artificial flavorings.

eat this. **in moderation**

+ Processed meat. Sausage, bacon and biltong. Make sure they are gluten, sugar and soy free and organic/freerange meat is preferable.

+ Whole fruit. Approximately 1-3 servings per day, depending on your blood sugar balance. Favour low sugar fruits like berries and peaches over tropical fruits, apples & pears.

+ Nuts and seeds. A maximum of a handful per day, preferably soaked overnight and dehydrated or roasted at low temperature (60-80°C) to improve digestibility. Favor nuts lower in omega-6, like hazelnuts and macadamias, and minimize nuts high in omega-6, like brazil nuts and almonds.

+ Green beans, sugar peas and snap peas. Though technically legumes, they are usually well tolerated.

+ Coffee and black tea. Black, or with coconut milk. *Only if you don't suffer from fatigue, insomnia or hypoglycemia, and only before 12:00 PM.* Limit to one cup (not one triple expresso - one cup).

+ Dark chocolate. 70% (Tip: L**dt 90% 'mild') or higher in small amounts (i.e. about the size of a 5 Rand coin per serving) is permitted.

+ Vinegar. Apple cider vinegar is especially well tolerated.

+ Restaurant food. The main problem with eating out is that restaurants cook with industrial seed oils, which wreak havoc on the body and cause serious inflammation. You don't need to become a cave dweller, but it's best to limit eating out as much as possible during this initial period.

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! don't eat that

AVOID COMPLETELY:

+ Dairy. Including butter, cheese, yogurt, milk, cream & any dairy product that comes from a cow, goat or sheep.

+ Grains. Including bread, rice, cereal, oats, or any gluten-free pseudo grains like sorghum, teff, quinoa, amaranth, buckwheat, etc.

+ Legumes. Including beans of all kinds (soy, black, kidney, pinto, etc.), peas, lentils and peanuts.

+ Concentrated sweeteners, real or artificial. Including sugar, high fructose corn syrup, maple syrup, honey, agave, brown rice syrup, Splenda, Equal, Nutrasweet, xylitol, stevia, etc.

+ Processed or refined foods. As a general rule, if it comes in a bag or a box, don't eat it. This also includes highly processed "health foods" like protein powder, energy bars, dairyfree creamers, etc.

+ Industrial seed oils. Soybean, corn, safflower, sunflower, cottonseed, canola, etc. Read labels - seed oils are in almost all processed, packaged and refined foods (which you should be mostly avoiding anyway).

+ Sodas and diet sodas. All forms.

+ Alcohol. In any form. (Don't freak out. It's just 30 days.)

+ Processed sauces and seasonings. Soy sauce, tamari, and other processed seasonings and sauces (which often have sugar, soy, gluten, or all of the above).

eat this. 30-day reset

caveats & **tweaks**

With certain health conditions the basic program above needs further modification:

1. Those with arthritis, joint pain, autoimmune disease and severe gut issues should also eliminate nightshades and eggs. Nightshades include potatoes, tomatoes, sweet and hot peppers, eggplant, tomatillos, pepinos, pimentos, paprika and cayenne pepper. Nightshades have compounds called alkaloids that can cause inflammation and worsen joint pain in susceptible people. Eggs contain proteins that are common allergens, particularly in susceptible people.

2. Those with insulin resistance, hypoglycemia or reactive hypoglycemia, and those wishing to lose weight, should limit fruit and starchy vegetables. The total amount eaten each day should equal roughly 50 grams per day of carbohydrate, which is the amount contained in 2 servings of low-glycemic fruit (berries) and 1-2 servings of starch (i.e. sweet potato, taro, yucca, etc.).

3. Those with fatigue, insomnia, anxiety, mood swings or depression should eliminate coffee, tea and all caffeine entirely. Caffeine stimulates the adrenals and can worsen all of these conditions. Once your adrenal issues have been addressed, you may be able to add them back in moderation.

4. Those who are athletes or have high levels of physical activity may want to increase their carbohydrate intake, especially after training. As a general idea, a minimum of 600 calories (150g) per day of carbohydrate, and as much as 800 calories (200g) or more may be required to meet energy needs, depending on the intensity of training and individual tolerance.

Okay, there it is. If you're completely new to this whole Paleo thing you might be feeling pretty overwhelmed right about now. "I thought saturated fats were bad", you say. "Aren't whole grains healthy?"

If you have questions about why the program includes some foods commonly thought to be dangerous and excludes other foods commonly thought to be healthy, you may want to read the book *The Paleo Cure*. In it, you'll learn the "what and why" in great detail, and all of your questions will be answered.

This handout, however, is much more about "how" than what or why. And if you're like most people, that's what you're most concerned about.

So dive in and give this a shot. Then, once you're feeling better than you have in years, you might be a lot more motivated to find out why. At that point you can go back and read *The Paleo Cure* and other resources to learn the theory behind what we're doing here.

eat this. **FAQ's**

HOW DO I DO IT?

I recognize this will be a dramatic change for many of you. The best way to do it is to just dive right in. Begin right now. If you procrastinate or delay, it just gets harder.

WHEN WILL I GET RESULTS?

The first few days can be hard. Your body will be going through withdrawal. Sugar and wheat in particular are addictive and you may notice symptoms like mood swings, strong cravings, irritability and fatigue as your body adjusts to life without them.

But at some point you will recover and start feeling better than you did before you began the program. Your energy will improve, your skin will clear up, your digestion will smooth out, your sleep will get deeper, your moods will stabilize and you'll start shedding some pounds (only if you need to, usually). Aches, pains and mysterious symptoms you've had for ages will - seemingly miraculously - begin to improve. This program has the potential to change your life. Though it may be difficult, the results are worth the effort.

I THOUGHT FAT WAS BAD FOR ME. SHOULDN'T I LIMIT IT?

The biggest mistake people make on this program is not eating enough fat. You're eliminating a lot of foods from your diet (bread, grains, beans, etc.), and you have to replace those calories with something. Healthy fat is that something. Healthy fat doesn't make you fat. Food toxins like wheat, fructose and seed oils - along with other aspects of the modern lifestyle - make you fat. Fat is the preferred fuel source of the body, and should constitute about 60-70% of calories.

A LITTLE CHEAT HERE AND THERE CAN'T HURT, RIGHT?

In general, once you've figured out your ideal diet, this is true. But this isn't the time to cheat. Don't do it. It's not worth it. One piece of bread or one glass of milk could re-start the inflammatory process and throw your body back into the chaos that led you to this in the first place. If you can stick this initial period out, it will get easier. At some point you won't even miss those foods you think you can't live without.

SHOULDN'T I BE COUNTING CALORIES AND CALCULATING MACRONUTRIENT RATIOS?

Try to relax into this as much as possible. Don't overanalyze what you're eating. Enjoy your food. Make cooking fun and leave time to savor your creations.

eat this. 30-day reset

THIS IS TOO HARD. HOW CAN I MAKE IT EASIER?

No man (or woman) is an island. Making big changes is hard, and the more support you have in doing this, the easier it will go. See if you can enlist your spouse, significant other or a good friend to do this with you. (They may not be eager to join, but they'll thank you later.) Have a "paleo pot luck". Invite friends over to cook with you.

I'VE GOT A TRIP PLANNED OR I'M GOING OUT ON A DATE. WHAT DO I DO?

First, check out the On the Go guide for ideas on easy-to-pack Paleo snacks. If you know you're going out to dinner with some friends this weekend, choose a place that can accommodate your needs. Call ahead and ask if they have gluten-free items on the menu. Pick a place that has meat and vegetable dishes, and order a salad on the side. Don't put yourself in a situation where you're starving because you haven't planned in advance, and then eat a bagel with cream cheese because that's all that's available. If you're going on a road trip, stock up on paleo-friendly snacks. This is all possible, but it does require some planning and foresight.

I'M TAKING A BOATLOAD OF SUPPLEMENTS. SHOULD I CONTINUE TAKING THEM DURING THE 30-DAY RESET?

This one's a little harder to answer. If you know the supplement helps you, or you're taking it for a specific goal or purpose (i.e. iodine for thyroid function), by all means continue. But if it's something you started taking a while ago and you can't even remember why, and it doesn't seem to be helping you, then go ahead and stop taking it. You can always start again later if you need to.

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eat this.

core food plan

eat this.

eat this.

- Meat
- Sea Food
- Eggs
- Vegetables
- Some Fruit
- Good Fats
- Nuts & Seeds

don't eat that.

- no added sugar of any kind
  no maple syrup, honey, argave nectar, coconut sugar Nutrasweet, Xylitol, Stevia - read your label!
- no alcohol
  not even for cooking - and no tobacco products of any sort either!
- no grains
  wheat, rye, barley, oats, corn, rice, millet, bulgur, sorghum, amaranth, buckwheat, sprouted grains, quinoa
- no legumes
  beans of all kinds, peas , chickpeas, lentils and peanuts, soy, miso, tofu, tempeh, edamame, lecithin...
- no dairy
  this includes cow, goat & sheep's milk products such as cream, cheese, kefir, yogurt and sour cream
- **MSG** no carrageenan, MSG,
  please check your labels, if any of these are listed, do not eat or drink
- sulfite's
  no experiments with 'paleo approved' ingredients, don't try to recreate the 'have beens'
- don't recreate baked goods

Eat real foods with pronounceable ingredients. Aim for foods that have no ingredients listed at all because they are totally unprocessed and natural. Shop on Farmers Markets and look out for Free Range Produce. Rather buy a good piece of meat once in a while than go for the affordable option on a daily basis. Even food without an obvious ingredients label tends to contain some form of preservative or additive, if not sugar to encourage your continued and excessive consumption of such (meat, biltong, chicken, fish and nuts especially tend to be marinated or treated with additives and preservatives). Omitting these foods will help you regain a healthy metabolism and reduce systemic inflammation.

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eat this.

eat this. the fine print

no scale. no measurements. no excessive exercise.

Focusing on body composition will make you miss out on the real benefits of **eat this**. This is about your whole body, inside and out - it is a time to heal, rethink and re-sensitize, during which your body needs time and rest. Don't stress about missing out on the treadmill or what you weigh. Listen to what your body is telling you to do.

exceptions

~~Clarified butter or ghee.~~ ~~Plain butter is a no-go, as the milk proteins could impact the results of **eat this.**~~

Fruit juice as a sweetener. Fine in moderation, please do not make it a habit and think half an apple rather than two added to your smoothie

Certain legumes. Green beans, sugar snap peas and snow peas are fine, technically being far more 'pod' than 'bean'

Vinegar. Apple cider, naturally cloudy is your best bet as it contains neither sugar nor sulfite's. Balsamic, white, red or rice vinegar are good choices too.

Salt. All iodized table salt contains sugar. Generally though all pre-packaged and most restaurant foods contain salt, so we'll bend the rules on this one.

eat this. protein

Eggs	Chicken (Sausage)	Whitefish (hake)	Pork (Chops)
Beef (Ground)	Chicken (Whole)	Shrimp	Pork (Sausage)
Beef (Steak)	Turkey (Whole)	Scallops	Pork (Bacon)
Beef (Other)	Turkey (Other)	Seafood (Other)	Biltong
Chicken (Breast/Thigh)	Salmon	Pork (Ground)	Deli Meat

Best choice: *look for words like 100% grass-fed, pastured, wild-caught, and organic on the label*
Avoid: *processed meats (pre-made sausage, burgers, bacon, deli meat, etc.) with added sugar, 'cereal', carrageenan, MSG, or sulfite's*

eat this. vegetables

Acorn Squash	Celery	Lettuce (all)	Spaghetti Squash
Artichoke	Collard Greens	Mushrooms (all)	Spinach
Arugula (rocket)	Cucumber	Okra	Sprouts
Asparagus	Delicata Squash	Onion	Summer Squash
Beets	Eggplant	Parsnips	Sweet Potato/Yams
Bell Peppers	Endive	Potatoes (all)	Swiss Chard
Bok Choy	Fennel (Anise)	Pumpkin	Tomato
Broccoli/Broccolini	Frisee (Curly Endive)	Radish	Turnip
Broccoli Rabe	Garlic	Rutabaga	Zucchini
Brussels Sprouts	Green Beans	Rhubarb	
Butternut Squash	Greens (beet, mustard, turnip)	Romaine	
Cabbage	Jalapeño/Hot Peppers (all)	Shallots	
Carrots	Kale	Snow Peas	
Cauliflower	Leeks	Sugar Snap Peas	

This is a comprehensive but not exhaustive list. All vegetables but corn, peas, and Lima beans are allowed.

eat this. fruit

Apples (all)	Figs	Melon (cantaloupe)	Pineapple
Apricots	Grapefruit	Melon (honeydew)	Plantains
Bananas (ripe)	Grapes (all)	Naartjie	Plum
Bananas (green)	Kiwi	Nectarines	Pomegranate
Blackberries	Lemon	Oranges (all)	Raspberries
Blueberries	Lime	Paw paw (papaya)	Strawberries
Cherries	Lychee	Peaches	Tangerines
Dates	Mango	Pears (all)	Watermelon

This is a comprehensive but not exhaustive list. All fruit are allowed in moderation.

eat this. fats

COOKING FATS	EATING/DRESSING	NUTS AND SEEDS	
Clarified Butter	Avocado	Almonds	Flax Seeds
Duck Fat	Avocado Oil	Nut Butters (except peanut!)	Pine Nuts
Ghee	Coconut Butter	Brazil Nuts	Pumpkin Seeds/Pepitas
Coconut Oil	Coconut (Flakes, Shredded)	Cashews	Sesame Seeds
Extra-Virgin Olive Oil (cool!)	Coconut Milk (Canned)	Hazelnuts/Filberts	Sunflower Seeds
Lard (Pork Fat)	Light Olive Oil	Macadamia Nuts & Butter	Sunflower Seed Butter
Palm Oil	Olives (all)	Pecans	Walnuts
Tallow (Beef Fat)	Sesame Oil	Pistachio	

eat this. herbs and spices

Allspice	Cloves (Ground)	Onion Powder	Thyme (Fresh/Dried)
Basil (Fresh/Dried)	Cumin	Oregano {Fresh/Dried)	Wasabi Powder
Bay Leaves	Curry Powder (Red/Yellow)	Paprika	
Black Pepper	Dill (Fresh/Dried)	Parsley (Fresh/Dried)	
Black Peppercorns	Garlic Powder	Red Pepper Flakes	
Cayenne	Ginger (Fresh/Dried)	Rosemary (Fresh/Dried)	
Chili Powder	Ground Cloves	Sage (Fresh/Dried)	
Chipotle Powder D Chives	Lemongrass	Salt (Iodized/Sea Salt)	
Cilantro (Fresh/Dried)	Mustard Powder	Salt (Himalayan)	
Cinnamon	Nutmeg	Salt (Karoo, desert)	

eat this. pantry

Almond Flour	Canned Salmon	Dried Fruit	Roasted Red Peppers
Apple Cider Vinegar	Canned Tuna	Fish Sauce	Sardines
Arrowroot Powder	Capers	Hot Sauce	Tapioca Starch
Balsamic Vinegar	Chicken Broth	Mustard (all)	Tomato Paste
Beef Broth	Cocoa (100% Cacao)	Pickles (all)	Tomatoes
Canned Butternut Squash	Coconut Ami nos	Raisins	Tomatoes (Sun-Dried)
Canned Pumpkin	Coconut Flour	Red Wine Vinegar	Vegetable Broth
Canned Sweet Potato	Dried Cranberries	Rice Vinegar	White Vinegar

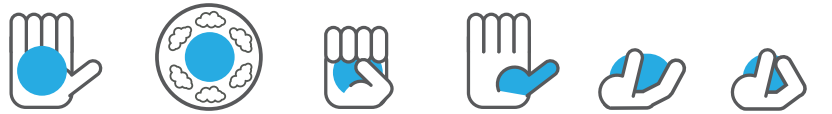
eat this. drinks

Apple Cider (diluted)	Coffee	Naturally Flavored Water	Vegetables Juice
Cacao (100%) Drinks	Fruit Juice (all)	Seltzer Water	Water Kefir
Club Soda	Kombucha	Sparkling Water	
Coconut Water	Mineral Water	Tea (all)	

eat this. shopper

eat this. meal planner

Make each meal large enough to satisfy you until the next meal - **don't snack**. Stop eating a few hours in advance of your bedtime.



Practice good mealtime habits. Have your meals at the table while relaxing. Don't distract yourself by watching TV, checking your phone or incoming mail while you are eating. Chew slowly and thoroughly. Take the time to enjoy the food you have prepared! Eat three meals a day. Start with a good breakfast. Base each meal on 1-2 palm-sized protein sources. The rest of your plate are vegetables. Occasionally add a serving of fruit. Add fat in the following recommended amounts **per meal**:

- All oils and cooking fats (olive oil, animal fats, etc.): 1-2 thumb-sized portions
- All butters (ghee, coconut butter, nut butters, etc.): 1-2 thumb-sized portions
- Coconut (shredded or flaked): 1-2 open (heaping) handfuls
- Olives: 1-2 open (heaping) handfuls
- Nuts and seeds: Up to one closed handful
- Avocado: ½ - 1 avocado
- Coconut milk: Between 1/4 and 1/2 of one (14 oz.) can

Eat 15 - 75 minutes **pre-workout**, as a signal to prepare your body for activity. If you train first thing in the morning, something is better than nothing. Choose foods that are easily digestible and palatable.
Include a small amount of protein {½ a meal size or smaller), and (optionally) a small amount of fat (½ a meal size or smaller). Do not add fruit or carb-dense vegetables to your pre-workout snack.

Eat immediately **post-workout** (15-30 minutes). Eat a meal-sized easily digestible protein, plus the appropriate amount of carb-dense vegetables, e.g. sweet potatoes/yams, turnip, parsnip, butternut squash, acorn squash, pumpkin or beets.
Do not use fruit as your primary post-workout carb, and add little to no fat. Your PWO meal is additional to breakfast, lunch or dinner. It's designed to help you recover faster and more efficiently from high intensity exercise.

Source: whole30.com

Appendix 7: Whole 30_sugar

WHOLE30®
http://whole30.com

WHOLE30 RESOURCES: SNEAKY SUGARS
practice your label reading

Companies sneak sugar into their products under the guise of a label that sounds vaguely plant-like and harmless, or in plain sight under its scientific name, easy to overlook because you just don't know what it is. Don't be fooled. These are all of the sneaky ways sugar may try to hide in the foods you eat. Educate yourself, read your labels, and avoid regular consumption of products with added sugar in any form.

just plain sugar

- _____ Sugar (brown sugar, cane sugar, raw sugar, beet sugar, confectioner's sugar, etc.)
- _____ Syrup (high fructose corn syrup, malt syrup, refiner's syrup, rice syrup, etc.)

science-y names for sugar

- Dextrose
- Disaccharide
- Fructose
- Glucose
- Galactose
- Lactose
- Maltose
- Monosaccharide
- Polysaccharide
- Ribose
- Saccharose
- Sucrose

"natural" sugars

- Agave Nectar
- Coconut Nectar
- Coconut Sugar
- Date Sugar
- (Evaporated) Cane Juice
- Fruit Juice*
- Honey
- Maple Syrup
- Molasses
- Rice Malt (Extract)
- (Sweet) Sorghum
- Treacle

artificial sweeteners

- Aspartame
- Acesulfame-K
- Equal
- Nutra-Sweet
- Saccharin
- Splenda
- Stevia
- Sucralose
- Sweetleaf
- Sweet-n-Low
- Truvia

sugar alcohols

- Arabitol
- Dulcitol
- Erythritol
- Glycol
- Glycerin (Glycerol)
- HSH
- Iditol
- Isomalt
- Lactitol
- Maltitol
- Mannitol
- Polyglycitol
- Ribitol
- Sorbitol
- Threitol
- Xylitol

**Admittedly, it can be hard to know where to stop in your quest to remove added sugar from your diet. Fruit juice is basically just sugar, which is why we're including it here - but this is the one "added sweetener" that is not excluded from our Whole30® program.*

Appendix 8: Whole 30_pantry

WHOLE30®
http://whole30.com

WHOLE30 RESOURCES: PANTRY STOCKING
good food on your shelves

Your healthy eating pantry (and fridge) are not complete without these staple items, easily found in most health food stores. Read your labels here too! On the Whole30, no added sugar, soy, carrageenan, MSG, sulfites, or other off-plan ingredients.

pantry item	helpful hints
Almond flour	Use almond flour/meal to thicken a sauce or to coat meat or fish before baking.
Applesauce	Unsweetened brands, like Santa Cruz Organics.
Beef or chicken broth	Some Imagine broths are Whole30-friendly, but it's best to make your own.
Butter (clarified) or ghee	Pastured and organic - like Whole30 Approved Pure Indian Foods or OMGhee.
Canned meats (salmon, tuna, chicken)	Read your labels—no soy, sugar or other less healthy ingredients.
Canned vegetables (sweet potato, squash, pumpkin)	The only ingredient should be the vegetable itself (and maybe water).
Cocoa (or 100% cacao)	100% cocoa or cacao adds flavor to meals and sauces. Treat it like a spice.
Coconut aminos	From Coconut Secret: Whole30-friendly, found in the soy sauce aisle.
Coconut butter	Also called "creamed coconut" or "coconut manna." Try Artisana brand.
Coconut (flaked or shredded)	Great for snacking or in recipes . Buy organic to avoid added sulfites.
Coconut milk	Get the full fat version, no sulfites. Try Thai Kitchen or Whole Foods 365.
Coconut oil	Look for the unrefined kind—organic isn't important here.
Curry paste	Red, green, or yellow will spice up any curry. Try Thai Kitchen brand.
Fish sauce	Watch for added sugar here! We like Whole30 Approved Red Boat Fish Sauce.
Hot sauce	Try Whole30 Approved Tessemae's or Horsetooth Hot Sauce.
Jerky	Remember, no added sugar! Try Primal Pacs, Chomps, and Gourmet Grassfed.
Mustard	Read your labels—no added sugar, corn starch, maltodextrin, etc.
Nuts and seeds	Raw or dry-roasted, salt is optional.
Nut butters	Look for no added sugar, like Whole30-friendly Organic Sunbutter.
Olives (black, green, etc.)	Any variety, canned or fresh - as long as there are no added sulfites.
Olive oil	Extra-virgin for dressings, sauces, and cooking; light for homemade mayo.
Pickles, relish, diced green chiles, capers, etc.	Add spice and flavor to meals and sauces—but as always, read your labels.
Raisins, currants, dried figs, etc.	A little goes a long way to flavor a dish or add some sweetness. Use sparingly.
Sesame oil	On our "limit" list, but a small amount can add lots of flavor.
Tomatoes (crushed, paste)	The best ingredient list would read just "tomatoes," like Pomi brand.
Vinegar (balsamic, cider, red wine, white, etc.)	All vinegar varieties (except for malt) are Whole30 permitted, even rice or wine.

Appendix 9: Data Set 25 Questions 30 Before & After
('r' calculation data input)

Q1	Q2	Q3	Q4	Q5	Q6	Q7	Q8	Q9	Q10	Q11	Q12	Q13	Q14	Q15	Q16	Q17	Q18	Q19	Q20	Q21	Q22	Q23	Q24	Q25
1	1	0	0	0	0	0	0	0	0	2	0	0	0	0	0	0	0	0	0	0	0	4	4	2
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2	4	1	2	2	3	0	0	0	0	0	3	2	3	2	2	2	2	1	0	0	3	3	1	2

Appendix 9: 22 (25-PRIMER) Questions BEFORE
('r' calculation data input)

Q1	Q2	Q3	Q4	Q5	Q6	Q7	Q8	Q9	Q10	Q11	Q12	Q13	Q14	Q15	Q16	Q19	Q20	Q21	Q22	Q24	Q25
2	4	1	1	2	0	2	0	0	0	0	0	0	1	2	0	1	0	1	2	0	2
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2	3	1	1	2	0	0	0	0	0	0	0	0	1	0	0	1	0	0	1	3	2
2	4	1	1	2	3	4	1	0	0	0	2	2	3	3	3	2	2	2	4	0	2
2	3	1	1	2	0	0	0	0	0	0	0	0	1	0	0	1	0	0	2	2	2
2	3	1	2	2	0	0	0	0	0	0	0	0	1	0	0	1	0	0	2	2	2
4	3	0	0	0	3	2	0	0	0	0	0	0	3	1	0	3	1	0	4	4	3
2	3	1	2	2	0	0	0	0	0	0	0	0	1	0	0	1	0	0	1	3	2
3	3	1	1	2	0	0	0	0	0	0	0	0	1	0	0	1	0	0	2	3	2
2	3	1	1	2	3	2	0	0	0	0	0	2	2	0	3	1	1	1	3	1	2
3	3	1	4	1	3	3	0	0	0	0	4	1	4	4	0	3	0	0	4	1	2
2	3	1	1	2	0	1	0	0	0	0	0	0	1	0	0	1	0	0	2	2	2
2	2	1	1	2	0	0	0	0	0	0	0	0	1	0	0	1	0	0	2	3	2
2	4	1	2	2	3	0	0	0	0	0	3	2	3	2	2	1	0	0	3	1	2

Appendix 9: 22 (25-PRIMER) Questions AFTER
('r' calculation data input)

Q1	Q2	Q3	Q4	Q5	Q6	Q7	Q8	Q9	Q10	Q11	Q12	Q13	Q14	Q15	Q16	Q19	Q20	Q21	Q22	Q24	Q25
1	1	0	0	0	0	0	0	0	0	2	0	0	0	0	0	0	0	0	0	4	2
2	0	0	0	1	0	0	0	0	0	0	1	0	0	0	0	1	0	1	1	4	2
1	1	0	0	2	0	0	0	0	0	3	1	0	2	0	0	0	0	0	1	4	2
1	0	0	0	0	0	0	0	0	0	0	0	0	0	2	0	0	0	0	2	4	2
1	2	0	0	2	0	2	0	0	0	4	1	1	2	2	2	0	0	1	2	4	2
1	1	0	2	0	0	1	0	0	0	3	2	1	1	2	2	1	0	1	2	4	2
1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	4	2
0	2	0	2	1	1	1	0	0	0	0	0	0	1	0	0	1	0	0	0	4	1
2	3	1	2	2	1	1	0	1	0	0	1	1	2	2	0	1	1	1	3	2	3
1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	4	2
1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	4	2
1	0	0	1	0	0	0	0	0	0	0	0	0	0	2	0	1	0	0	2	4	2
2	3	1	3	2	0	2	0	0	0	0	1	1	2	1	0	1	0	0	3	3	1
1	0	0	2	0	0	1	0	0	0	0	1	0	0	0	0	0	0	2	1	4	1
0	0	0	2	0	0	**3**	0	0	0	0	1	0	0	0	0	0	0	0	1	**4**	1
1	2	0	0	0	0	0	0	0	0	3	1	1	4	0	0	0	0	0	2	4	3
1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	4	2
1	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	4	2
1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	4	2
1	1	0	0	0	0	1	0	0	0	0	0	0	2	1	1	1	0	1	2	4	2
1	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	4	2
1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	4	2
2	0	0	1	0	0	0	0	0	0	0	0	0	0	2	0	3	1	0	1	4	3
1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	4	2
1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	4	2
1	0	0	0	0	0	0	0	0	0	0	0	0	1	0	1	0	0	1	3	4	2
1	1	1	1	3	4	1	0	0	4	4	2	0	4	4	4	0	0	0	4	4	2
1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	4	2
1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	4	2
1	2	0	1	2	0	0	0	0	0	2	1	1	0	0	0	0	0	0	1	4	2

Appendix 9: 18 (25-SYM2, PRIMER) Questions ('r' calculation data input)

Q1	Q2	Q3	Q5	Q6	Q7	Q8	Q9	Q10	Q11	Q12	Q13	Q14	Q15	Q16	Q19	Q20	Q21
1	1	0	0	0	0	0	0	0	2	0	0	0	0	0	0	0	0
2	4	1	2	0	2	0	0	0	0	0	0	1	2	0	1	0	1
2	0	0	1	0	0	0	0	0	0	1	0	0	0	0	1	0	1
4	3	1	3	3	0	1	1	0	0	3	0	1	0	0	3	2	2
3	3	1	3	4	2	0	0	0	0	3	1	3	1	0	1	1	3
1	1	0	2	0	0	0	0	0	3	1	0	2	0	0	0	0	0
1	0	0	0	0	0	0	0	0	0	0	0	0	2	0	0	0	0
2	3	1	1	0	1	0	0	0	0	0	0	1	3	0	1	0	0
1	2	0	2	0	2	0	0	0	4	1	1	2	2	2	0	0	1
4	4	1	3	4	3	0	2	0	0	3	3	3	3	3	1	2	2
1	1	0	0	0	1	0	0	0	3	2	1	1	2	2	1	0	1
4	4	2	2	0	3	2	2	0	0	4	3	3	3	3	2	1	2
1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
2	3	1	2	0	0	0	0	0	0	0	0	1	0	0	1	0	1
2	2	2	2	3	1	0	0	0	0	2	1	2	0	0	0	0	0
0	2	0	1	1	1	0	0	0	0	0	0	1	0	0	1	0	0
2	3	1	2	1	1	0	1	0	0	1	1	2	2	0	1	1	1
3	3	2	2	2	3	0	2	0	0	1	1	2	2	0	2	1	1
1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
2	3	1	2	0	0	0	0	0	0	0	0	1	0	0	1	0	0
1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
2	3	1	2	0	0	0	0	0	0	0	0	1	0	0	1	0	0
1	0	0	0	0	0	0	0	0	0	0	0	0	2	0	1	0	0
2	3	1	2	2	0	0	0	0	0	2	2	1	3	0	3	0	0
4	3	2	2	0	3	0	0	0	1	1	1	3	2	0	2	0	0
2	3	1	2	0	2	0	0	0	0	1	1	2	1	0	1	0	0
1	0	0	0	0	1	0	0	0	0	1	0	0	0	0	0	0	2
2	2	0	1	2	3	0	0	0	0	0	1	2	0	1	1	0	0
0	0	0	0	0	3	0	0	0	0	1	0	0	0	0	0	0	0
2	2	0	0	1	0	0	1	0	0	1	2	3	2	1	1	0	3
3	4	1	3	3	3	0	0	0	1	3	3	1	1	0	1	0	0
1	2	0	0	0	0	0	0	0	3	1	1	4	0	0	0	0	0
1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
2	3	1	2	0	0	0	0	0	0	0	0	1	1	0	1	0	0
1	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0
2	3	1	2	0	2	0	0	0	0	0	0	1	2	0	1	0	0
2	3	1	2	0	0	0	0	0	0	0	0	1	0	0	1	0	0
1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
2	4	1	2	3	4	1	0	0	0	2	2	3	3	3	2	2	2
1	1	0	0	0	1	0	0	0	0	0	0	2	1	1	1	0	1
1	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0
2	3	1	2	0	0	0	0	0	0	0	0	1	0	0	1	0	0
1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
2	3	1	2	0	0	0	0	0	0	0	0	1	0	0	1	0	0
4	3	0	0	3	2	0	0	0	0	0	0	3	1	0	3	1	0
2	0	0	0	0	0	0	0	0	0	0	0	0	2	0	3	1	0
1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
2	3	1	2	0	0	0	0	0	0	0	0	1	0	0	1	0	0
1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
3	3	1	2	0	0	0	0	0	0	0	0	1	0	0	1	0	0
1	0	0	0	0	0	0	0	0	0	0	0	1	0	1	0	0	1
2	3	1	2	3	2	0	0	0	0	0	2	2	0	3	1	1	1
1	1	1	3	4	1	0	0	4	4	2	0	4	4	4	0	0	0
3	3	1	1	3	3	0	0	0	0	4	1	4	4	0	3	0	0
1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
2	3	1	2	0	1	0	0	0	0	0	0	1	0	0	1	0	0
1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
2	2	1	2	0	0	0	0	0	0	0	0	1	0	0	1	0	0
1	2	0	2	0	0	0	0	0	2	1	1	0	0	0	0	0	0
2	4	1	2	3	0	0	0	0	0	3	2	3	2	2	1	0	0

Appendix 10: Att3_YFAS_ExcelFormula_AKiste.xlsx (dichotomous analysis)

Study ID	date	before / after	age	gender	weight	height	waist	hip	BMI	WTH	1. I find that when I start eating certain foods, I end up eating much more than planned.	2. I find myself continuing to consume certain foods even though I am no longer hungry.	3. I eat to the point where I feel physically ill.	4. Not eating certain types of food or cutting down on certain types of food is something I worry about.	5. I spend a lot of time feeling sluggish or fatigued from overeating.
Angie	03.08.2016	after	40	female	72	167	84	103	25,8	0,8	1	1	0	0	0
Angie	02.07.2016	before	40	female	72	167	92	105	25,8	0,9	2	4	1	1	2
Anja	03.08.2016	after	40	female	83	170	90	104	28,7	0,9	2	0	0	0	1
Anja	02.07.2016	before	40	female	87	170	97	106	30,1	0,9	4	3	1	1	3
Bill	02.07.2016	before	48	male	105	183	115	112	31,4	1,0	3	3	1	1	3
Bill	03.08.2016	after	48	male	97	183	109	109	29,0	1,0	1	1	0	0	2
Candice	03.08.2016	after	33	female	64	167	78	98	22,9	0,8	1	0	0	0	0
Candice	02.07.2016	before	33	female	64	167	77	96	22,9	0,8	2	3	1	1	1
Catherine	03.08.2016	after	43	female	61	162	77	99	23,2	0,8	1	2	0	0	2
Catherine	02.07.2016	before	43	female	63	162	80	102	24,0	0,8	4	4	1	1	3
Cindy	03.08.2016	after	40	female	115	178	102	129	36,3	0,8	1	1	0	2	0
Cindy	02.07.2016	before	40	female	121	178	105	137	38,2	0,8	4	4	2	3	2
Claudia	03.08.2016	after	52	female	59	159	82	102	23,3	0,8	1	1	0	0	0
Claudia	02.07.2016	before	52	female	62	159	85	103	24,5	0,8	2	3	1	1	2
David	15.8.2016	before	41	male	72	173	83	80	24,1	1,0	2	2	2	1	2
David	12.11.2016	after	41	male	69	173	79	80	23,1	1,0	0	2	0	2	1
Heather	31.08.2016	after	35	female	70	178	81	91	22,1	0,9	2	3	1	2	2
Heather	28.07.2016	before	35	female	71	178	76	89	22,4	0,9	3	3	2	3	2
Ingrid	03.08.2016	after	74	female	64	173	81	94	21,4	0,9	1	0	0	0	0
Ingrid	02.07.2016	before	74	female	64	173	82	94	21,4	0,9	2	3	1	1	2
Jill	03.08.2016	after	54	female	60	159	72	104	23,7	0,7	1	0	0	0	0
Jill	02.07.2016	before	54	female	63	159	76	107	24,9	0,7	2	3	1	1	2
Justine	03.08.2016	after	37	female	53	165	72	92	19,5	0,8	1	0	0	1	0
Justine	02.07.2016	before	37	female	52	165	70	91	19,1	0,8	2	3	1	3	2
Karl	28.07.2016	before	41	male	99	196	106	100	25,8	1,1	4	3	2	4	2
Karl	01.09.2016	after	41	male	94	196	101	99	24,5	1,0	2	3	1	3	2
Lois	12.11.2016	after	37	female	67	170	77	93	23,2	0,8	1	0	0	2	0
Lois	15.8.2016	before	37	female	67	170	78	93	23,2	0,8	2	2	0	3	1
Ltisch	3.8.2016	after	32	female	60	161	70	89	23,1	0,8	0	0	0	2	0
Ltisch	30.6.2016	before	32	female	64	161	76	90	24,7	0,8	2	2	0	2	0
Mitch	30.6.2016	before	27	male	137	180	129	125	42,3	1,0	3	4	1	3	3
Mitch	3.8.2016	after	27	male	129	180	120	118	39,8	1,0	1	2	0	0	0
Nikki	03.08.2016	after	46	female	58	162	76	100	22,1	0,8	1	0	0	0	0
Nikki	02.07.2016	before	46	female	62	162	80	101	23,6	0,8	2	3	1	1	2
Pat	03.08.2016	after	60	female	72	163	88	114	27,1	0,8	1	0	0	0	0
Pat	02.07.2016	before	60	female	77	163	91	116	29,0	0,8	2	3	1	1	2
Paul	02.07.2016	before	79	male	95	186	103	109	27,5	0,9	2	3	1	1	2
Paul	03.08.2016	after	79	male	86	186	99	106	24,9	0,9	1	0	0	0	0
Rael	02.07.2016	before	50	male	95	183	100	109	28,4	0,9	2	4	1	1	2
Rael	03.08.2016	after	50	male	85	183	95	104	25,4	0,9	1	1	0	0	0
Renate	03.08.2016	after	72	female	78	157	106	115	31,6	0,9	1	0	0	0	1
Renate	02.07.2016	before	72	female	80	157	108	118	32,5	0,9	2	3	1	1	2
Renee	03.08.2016	after	75	female	88	168	102	117	31,2	0,9	1	0	0	0	0
Renee	02.07.2016	before	75	female	92	168	105	119	32,6	0,9	2	3	1	2	2
Roland	18.08.2016	before	44	male	127	190	115	112	35,2	1,0	4	3	0	0	0
Roland	18.08.2016	after	44	male	120	190	109	109	33,2	1,0	2	0	0	1	0
Sally Jane	03.08.2016	after	52	female	52	166	71	86	18,9	0,8	1	0	0	0	0
Sally Jane	02.07.2016	before	52	female	51	166	71	86	18,5	0,8	2	3	1	2	2
Sam	15.7.2016	after	39	female	86	178	104	112	27,1	0,9	1	0	0	0	0
Sam	15.6.2016	before	39	female	92	178	110	114	29,0	1,0	3	3	1	1	2
Sheena	03.08.2016	after	30	female	69	161	78	113	26,6	0,7	1	0	0	0	0
Sheena	02.07.2016	before	30	female	72	161	80	114	27,8	0,7	2	3	1	1	2
Stevie	3.8.2016	after	27	female	69	149	72	98	31,1	0,7	1	1	1	1	3
Stevie	30.6.2016	before	27	female	72	149	77	101	32,4	0,8	3	3	1	4	1
Tarryn	03.08.2016	after	34	female	59	173	75	93	19,7	0,8	1	0	0	0	0
Tarryn	02.07.2016	before	34	female	60	173	78	93	20,0	0,8	2	3	1	1	2
Ulrike	03.08.2016	after	46	female	67	175	75	110	21,9	0,7	1	0	0	0	0
Ulrike	02.07.2016	before	46	female	70	175	77	112	22,9	0,7	2	2	1	1	2
Valerie	03.08.2016	after	68	female	85	160	104	111	33,2	0,9	1	2	0	1	2
Valerie	02.07.2016	before	68	female	89	160	108	113	34,8	1,0	2	4	1	2	2

Appendix 10: Att3_YFAS_ExcelFormula_AKiste.xlsx (dichotomous analysis)

Study ID	date	before / after	6. I find myself constantly eating certain foods throughout the day.	7. I find that when certain foods are not available, I will go out of my way to obtain them. For example, I will drive to the store to purchase certain foods even though I have other options available	8. 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	9. 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	10. There have been times when I avoided professional or social situations where certain foods were available because I was afraid I would overeat.
Angie	03.08.2016	after	0	0	0	0	0
Angie	02.07.2016	before	0	2	0	0	0
Anja	03.08.2016	after	0	0	0	0	0
Anja	02.07.2016	before	3	0	1	1	0
Bill	02.07.2016	before	4	2	0	0	0
Bill	03.08.2016	after	0	0	0	0	0
Candice	03.08.2016	after	0	0	0	0	0
Candice	02.07.2016	before	0	1	0	0	0
Catherine	03.08.2016	after	0	2	0	0	0
Catherine	02.07.2016	before	4	3	0	2	0
Cindy	03.08.2016	after	0	1	0	0	0
Cindy	02.07.2016	before	0	3	2	2	0
Claudia	03.08.2016	after	0	0	0	0	0
Claudia	02.07.2016	before	0	0	0	0	0
David	15.8.2016	before	3	1	0	0	0
David	12.11.2016	after	1	1	0	0	0
Heather	31.08.2016	after	1	1	0	1	0
Heather	28.07.2016	before	2	3	0	2	0
Ingrid	03.08.2016	after	0	0	0	0	0
Ingrid	02.07.2016	before	0	0	0	0	0
Jill	03.08.2016	after	0	0	0	0	0
Jill	02.07.2016	before	0	0	0	0	0
Justine	03.08.2016	after	0	0	0	0	0
Justine	02.07.2016	before	2	0	0	0	0
Karl	28.07.2016	before	0	3	0	0	0
Karl	01.09.2016	after	0	2	0	0	0
Lois	12.11.2016	after	0	1	0	0	0
Lois	15.8.2016	before	2	3	0	0	0
Ltisch	3.8.2016	after	0	**3**	0	0	0
Ltisch	30.6.2016	before	1	0	0	1	0
Mitch	30.6.2016	before	3	3	0	0	0
Mitch	3.8.2016	after	0	0	0	0	0
Nikki	03.08.2016	after	0	0	0	0	0
Nikki	02.07.2016	before	0	0	0	0	0
Pat	03.08.2016	after	0	1	0	0	0
Pat	02.07.2016	before	0	2	0	0	0
Paul	02.07.2016	before	0	0	0	0	0
Paul	03.08.2016	after	0	0	0	0	0
Rael	02.07.2016	before	3	4	1	0	0
Rael	03.08.2016	after	0	1	0	0	0
Renate	03.08.2016	after	0	0	0	0	0
Renate	02.07.2016	before	0	0	0	0	0
Renee	03.08.2016	after	0	0	0	0	0
Renee	02.07.2016	before	0	0	0	0	0
Roland	18.08.2016	before	3	2	0	0	0
Roland	18.08.2016	after	0	0	0	0	0
Sally Jane	03.08.2016	after	0	0	0	0	0
Sally Jane	02.07.2016	before	0	0	0	0	0
Sam	15.7.2016	after	0	0	0	0	0
Sam	15.6.2016	before	0	0	0	0	0
Sheena	03.08.2016	after	0	0	0	0	0
Sheena	02.07.2016	before	3	2	0	0	0
Stevie	3.8.2016	after	4	1	0	0	4
Stevie	30.6.2016	before	3	3	0	0	0
Tarryn	03.08.2016	after	0	0	0	0	0
Tarryn	02.07.2016	before	0	1	0	0	0
Ulrike	03.08.2016	after	0	0	0	0	0
Ulrike	02.07.2016	before	0	0	0	0	0
Valerie	03.08.2016	after	0	0	0	0	0
Valerie	02.07.2016	before	3	0	0	0	0

Appendix 10: Att3_YFAS_ExcelFormula_AKiste.xlsx (dichotomous analysis)

Study ID	date	before / after	11. There have been times when I avoided professional or social situations because I was not able to consume certain foods there.	12. 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	13. I have consumed certain foods to prevent feelings of anxiety, agitation, or other physical symptoms that were developing. (Please do NOT include consumption of	14. I have found that I have elevated desire for or urges to consume certain foods when I cut down or stop eating them.	15. My behavior with respect to food and eating causes significant distress.
Angie	03.08.2016	after	2	0	0	0	0
Angie	02.07.2016	before	0	0	0	1	2
Anja	03.08.2016	after	0	1	0	0	0
Anja	02.07.2016	before	0	3	0	1	0
Bill	02.07.2016	before	0	3	1	3	1
Bill	03.08.2016	after	3	1	0	2	0
Candice	03.08.2016	after	0	0	0	0	2
Candice	02.07.2016	before	0	0	0	1	3
Catherine	03.08.2016	after	4	1	1	2	2
Catherine	02.07.2016	before	0	3	3	3	3
Cindy	03.08.2016	after	3	2	1	1	2
Cindy	02.07.2016	before	0	4	3	3	3
Claudia	03.08.2016	after	0	0	0	0	0
Claudia	02.07.2016	before	0	0	0	1	0
David	15.8.2016	before	0	2	1	2	0
David	12.11.2016	after	0	0	0	1	0
Heather	31.08.2016	after	0	1	1	2	2
Heather	28.07.2016	before	0	1	1	2	2
Ingrid	03.08.2016	after	0	0	0	0	0
Ingrid	02.07.2016	before	0	0	0	1	0
Jill	03.08.2016	after	0	0	0	0	0
Jill	02.07.2016	before	0	0	0	1	0
Justine	03.08.2016	after	0	0	0	0	2
Justine	02.07.2016	before	0	2	2	1	3
Karl	28.07.2016	before	1	1	1	3	2
Karl	01.09.2016	after	0	1	1	2	1
Lois	12.11.2016	after	0	1	0	0	0
Lois	15.8.2016	before	0	0	1	2	0
Ltisch	3.8.2016	after	0	1	0	0	0
Ltisch	30.6.2016	before	0	1	2	3	2
Mitch	30.6.2016	before	1	3	3	1	1
Mitch	3.8.2016	after	3	1	1	4	0
Nikki	03.08.2016	after	0	0	0	0	0
Nikki	02.07.2016	before	0	0	0	1	1
Pat	03.08.2016	after	0	0	0	0	0
Pat	02.07.2016	before	0	0	0	1	2
Paul	02.07.2016	before	0	0	0	1	0
Paul	03.08.2016	after	0	0	0	0	0
Rael	02.07.2016	before	0	2	2	3	3
Rael	03.08.2016	after	0	0	0	2	1
Renate	03.08.2016	after	0	0	0	0	0
Renate	02.07.2016	before	0	0	0	1	0
Renee	03.08.2016	after	0	0	0	0	0
Renee	02.07.2016	before	0	0	0	1	0
Roland	18.08.2016	before	0	0	0	3	1
Roland	18.08.2016	after	0	0	0	0	2
Sally Jane	03.08.2016	after	0	0	0	0	0
Sally Jane	02.07.2016	before	0	0	0	1	0
Sam	15.7.2016	after	0	0	0	0	0
Sam	15.6.2016	before	0	0	0	1	0
Sheena	03.08.2016	after	0	0	0	1	0
Sheena	02.07.2016	before	0	0	2	2	0
Stevie	3.8.2016	after	4	2	0	4	4
Stevie	30.6.2016	before	0	4	1	4	4
Tarryn	03.08.2016	after	0	0	0	0	0
Tarryn	02.07.2016	before	0	0	0	1	0
Ulrike	03.08.2016	after	0	0	0	0	0
Ulrike	02.07.2016	before	0	0	0	1	0
Valerie	03.08.2016	after	2	1	1	0	0
Valerie	02.07.2016	before	0	3	2	3	2

Appendix 10: Att3_YFAS_ExcelFormula_AKiste.xlsx (dichotomous analysis)

Study ID	date	before / after	16. I experience significant problems in my ability to function effectively (daily routine, job/school, social activities, family activities, health difficulties) because of food and eating.	17. My food consumption has caused significant psychological problems such as depression, anxiety, self-loathing, or guilt.	18. My food consumption has caused significant physical problems or made a physical problem worse.	19. I kept consuming the same types of food or the same amount of food even though I was having emotional and/or physical problems.	20. 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.
Angie	03.08.2016	after	0	0	0	0	0
Angie	02.07.2016	before	0	0	0	1	0
Anja	03.08.2016	after	0	0	0	1	0
Anja	02.07.2016	before	0	0	2	3	2
Bill	02.07.2016	before	0	0	2	1	1
Bill	03.08.2016	after	0	0	0	0	0
Candice	03.08.2016	after	0	0	0	0	0
Candice	02.07.2016	before	0	1	1	1	0
Catherine	03.08.2016	after	2	0	0	0	0
Catherine	02.07.2016	before	3	1	1	1	2
Cindy	03.08.2016	after	2	2	1	1	0
Cindy	02.07.2016	before	3	2	2	2	1
Claudia	03.08.2016	after	0	0	0	0	0
Claudia	02.07.2016	before	0	0	0	1	0
David	15.8.2016	before	0	0	0	0	0
David	12.11.2016	after	0	0	0	1	0
Heather	31.08.2016	after	0	2	1	1	1
Heather	28.07.2016	before	0	2	1	2	1
Ingrid	03.08.2016	after	0	0	0	0	0
Ingrid	02.07.2016	before	0	0	0	1	0
Jill	03.08.2016	after	0	0	0	0	0
Jill	02.07.2016	before	0	0	0	1	0
Justine	03.08.2016	after	0	0	1	1	0
Justine	02.07.2016	before	0	1	2	3	0
Karl	28.07.2016	before	0	1	1	2	0
Karl	01.09.2016	after	0	1	1	1	0
Lois	12.11.2016	after	0	0	0	0	0
Lois	15.8.2016	before	1	0	1	1	0
Ltisch	3.8.2016	after	0	0	0	0	0
Ltisch	30.6.2016	before	1	1	1	1	0
Mitch	30.6.2016	before	0	1	1	1	0
Mitch	3.8.2016	after	0	0	0	0	0
Nikki	03.08.2016	after	0	0	0	0	0
Nikki	02.07.2016	before	0	0	0	1	0
Pat	03.08.2016	after	0	0	0	0	0
Pat	02.07.2016	before	0	0	0	1	0
Paul	02.07.2016	before	0	0	0	1	0
Paul	03.08.2016	after	0	0	0	0	0
Rael	02.07.2016	before	3	2	1	2	2
Rael	03.08.2016	after	1	0	0	1	0
Renate	03.08.2016	after	0	0	0	0	0
Renate	02.07.2016	before	0	0	0	1	0
Renee	03.08.2016	after	0	0	0	0	0
Renee	02.07.2016	before	0	0	0	1	0
Roland	18.08.2016	before	0	2	2	3	1
Roland	18.08.2016	after	0	1	0	3	1
Sally Jane	03.08.2016	after	0	0	0	0	0
Sally Jane	02.07.2016	before	0	0	0	1	0
Sam	15.7.2016	after	0	0	0	0	0
Sam	15.6.2016	before	0	0	0	1	0
Sheena	03.08.2016	after	1	0	0	0	0
Sheena	02.07.2016	before	3	2	0	1	1
Stevie	3.8.2016	after	4	3	0	0	0
Stevie	30.6.2016	before	0	2	0	3	0
Tarryn	03.08.2016	after	0	0	0	0	0
Tarryn	02.07.2016	before	0	0	0	1	0
Ulrike	03.08.2016	after	0	0	0	0	0
Ulrike	02.07.2016	before	0	1	0	1	0
Valerie	03.08.2016	after	0	1	0	0	0
Valerie	02.07.2016	before	2	2	2	1	0

Appendix 10: Att3_YFAS_ExcelFormula_AKiste.xlsx (dichotomous analysis)

Study ID	date	before / after	21. 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.	22. I want to cut down or stop eating certain kinds of food.	23. I have tried to cut down or stop eating certain kinds of food.	24. I have been successful at cutting down or not eating these kinds of food.
Angie	03.08.2016	after	0	0	4	4
Angie	02.07.2016	before	1	2	3	0
Anja	03.08.2016	after	1	1	4	4
Anja	02.07.2016	before	2	3	3	1
Bill	02.07.2016	before	3	2	3	1
Bill	03.08.2016	after	0	1	4	4
Candice	03.08.2016	after	0	2	4	4
Candice	02.07.2016	before	0	4	3	2
Catherine	03.08.2016	after	1	2	4	4
Catherine	02.07.2016	before	2	3	3	0
Cindy	03.08.2016	after	1	2	4	4
Cindy	02.07.2016	before	2	4	3	0
Claudia	03.08.2016	after	0	0	4	4
Claudia	02.07.2016	before	1	2	3	3
David	15.8.2016	before	0	2	2	1
David	12.11.2016	after	0	0	4	4
Heather	31.08.2016	after	1	3	2	2
Heather	28.07.2016	before	1	3	1	2
Ingrid	03.08.2016	after	0	0	4	4
Ingrid	02.07.2016	before	0	0	3	3
Jill	03.08.2016	after	0	0	4	4
Jill	02.07.2016	before	0	2	3	1
Justine	03.08.2016	after	0	2	4	4
Justine	02.07.2016	before	0	3	3	2
Karl	28.07.2016	before	0	3	2	2
Karl	01.09.2016	after	0	3	1	3
Lois	12.11.2016	after	2	1	4	4
Lois	15.8.2016	before	0	4	1	1
Ltisch	3.8.2016	after	0	1	4	**4**
Ltisch	30.6.2016	before	3	3	3	2
Mitch	30.6.2016	before	0	3	2	0
Mitch	3.8.2016	after	0	2	4	4
Nikki	03.08.2016	after	0	0	4	4
Nikki	02.07.2016	before	0	2	3	2
Pat	03.08.2016	after	0	0	4	4
Pat	02.07.2016	before	0	2	3	1
Paul	02.07.2016	before	0	1	3	3
Paul	03.08.2016	after	0	1	4	4
Rael	02.07.2016	before	2	4	3	0
Rael	03.08.2016	after	1	2	4	4
Renate	03.08.2016	after	0	1	4	4
Renate	02.07.2016	before	0	2	3	2
Renee	03.08.2016	after	0	0	4	4
Renee	02.07.2016	before	0	2	3	2
Roland	18.08.2016	before	0	4	1	4
Roland	18.08.2016	after	0	1	0	4
Sally Jane	03.08.2016	after	0	0	4	4
Sally Jane	02.07.2016	before	0	1	3	3
Sam	15.7.2016	after	0	0	4	4
Sam	15.6.2016	before	0	2	3	3
Sheena	03.08.2016	after	1	3	4	4
Sheena	02.07.2016	before	1	3	3	1
Stevie	3.8.2016	after	0	4	4	4
Stevie	30.6.2016	before	0	4	4	1
Tarryn	03.08.2016	after	0	0	4	4
Tarryn	02.07.2016	before	0	2	3	2
Ulrike	03.08.2016	after	0	1	4	4
Ulrike	02.07.2016	before	0	2	3	3
Valerie	03.08.2016	after	0	1	4	4
Valerie	02.07.2016	before	0	3	3	1

Appendix 10: Att3_YFAS_ExcelFormula_AKiste.xlsx (dichotomous analysis)

Study ID	date	before / after	25. How many times in the past year did you try to cut down or stop eating certain foods altogether?	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
Angie	03.08.2016	after		2	0	0	0	0	0	0	0	0	0	1	0	0	0	0
Angie	02.07.2016	before		2	0	1	0	0	0	0	0	0	0	0	0	0	0	0
Anja	03.08.2016	after		2	0	0	0	0	0	0	0	0	0	1	0	0	0	0
Anja	02.07.2016	before		2	1	0	0	0	1	0	0	0	0	0	1	0	0	0
Bill	02.07.2016	before		2	0	0	0	0	1	1	0	0	0	0	1	0	1	0
Bill	03.08.2016	after		2	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Candice	03.08.2016	after		2	0	0	0	0	0	0	0	0	0	1	0	0	0	0
Candice	02.07.2016	before		2	0	0	0	0	0	0	0	0	0	0	0	0	0	1
Catherine	03.08.2016	after		2	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Catherine	02.07.2016	before		2	1	1	0	0	1	1	1	0	0	0	1	1	1	1
Cindy	03.08.2016	after		2	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Cindy	02.07.2016	before		2	1	1	0	0	0	0	1	1	0	0	1	1	1	1
Claudia	03.08.2016	after		2	0	0	0	0	0	0	0	0	0	1	0	0	0	0
Claudia	02.07.2016	before		2	0	0	0	0	0	0	0	0	0	0	0	0	0	0
David	15.8.2016	before		2	0	0	0	0	0	0	0	0	0	0	0	0	0	0
David	12.11.2016	after		1	0	0	0	0	0	0	0	0	0	1	0	0	0	0
Heather	31.08.2016	after		3	0	0	0	0	0	0	0	0	0	1	0	0	0	0
Heather	28.07.2016	before		2	0	0	0	0	0	0	1	0	0	0	0	0	0	0
Ingrid	03.08.2016	after		2	0	0	0	0	0	0	0	0	0	1	0	0	0	0
Ingrid	02.07.2016	before		2	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Jill	03.08.2016	after		2	0	0	0	0	0	0	0	0	0	1	0	0	0	0
Jill	02.07.2016	before		2	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Justine	03.08.2016	after		2	0	0	0	0	0	0	0	0	0	1	0	0	0	0
Justine	02.07.2016	before		2	0	0	0	0	0	0	0	0	0	0	0	0	0	1
Karl	28.07.2016	before		1	1	0	0	1	0	0	1	0	0	0	0	0	1	0
Karl	01.09.2016	after		1	0	0	0	0	0	0	0	0	0	1	0	0	0	0
Lois	12.11.2016	after		1	0	0	0	0	0	0	0	0	0	1	0	0	0	0
Lois	15.8.2016	before		2	0	0	0	0	0	0	1	0	0	0	0	0	0	0
Ltisch	3.8.2016	after		1	0	0	0	0	0	0	1	0	0	1	0	0	0	0
Ltisch	30.6.2016	before		5	0	0	0	0	0	0	0	0	0	0	0	0	1	0
Mitch	30.6.2016	before		2	0	1	0	0	1	0	1	0	0	0	0	1	1	0
Mitch	3.8.2016	after		3	0	0	0	0	0	0	0	0	0	0	0	0	1	0
Nikki	03.08.2016	after		2	0	0	0	0	0	0	0	0	0	1	0	0	0	0
Nikki	02.07.2016	before		2	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Pat	03.08.2016	after		2	0	0	0	0	0	0	0	0	0	1	0	0	0	0
Pat	02.07.2016	before		2	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Paul	02.07.2016	before		2	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Paul	03.08.2016	after		2	0	0	0	0	0	0	0	0	0	1	0	0	0	0
Rael	02.07.2016	before		2	0	1	0	0	0	0	1	0	0	0	0	0	1	1
Rael	03.08.2016	after		2	0	0	0	0	0	0	0	0	0	1	0	0	0	0
Renate	03.08.2016	after		2	0	0	0	0	0	0	0	0	0	1	0	0	0	0
Renate	02.07.2016	before		2	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Renee	03.08.2016	after		2	0	0	0	0	0	0	0	0	0	1	0	0	0	0
Renee	02.07.2016	before		2	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Roland	18.08.2016	before		3	1	0	0	0	0	0	0	0	0	0	0	0	1	0
Roland	18.08.2016	after		3	0	0	0	0	0	0	0	0	0	1	0	0	0	0
Sally Jane	03.08.2016	after		2	0	0	0	0	0	0	0	0	0	1	0	0	0	0
Sally Jane	02.07.2016	before		2	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Sam	15.7.2016	after		2	0	0	0	0	0	0	0	0	0	1	0	0	0	0
Sam	15.6.2016	before		2	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Sheena	03.08.2016	after		2	0	0	0	0	0	0	0	0	0	1	0	0	0	0
Sheena	02.07.2016	before		2	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Stevie	3.8.2016	after		2	0	0	0	0	1	1	0	0	0	1	0	0	1	1
Stevie	30.6.2016	before		2	0	0	0	1	0	0	1	0	0	0	0	1	0	1	1
Tarryn	03.08.2016	after		2	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0
Tarryn	02.07.2016	before		2	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Ulrike	03.08.2016	after		2	0	0	0	0	0	0	0	0	0	1	0	0	0	0
Ulrike	02.07.2016	before		2	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Valerie	03.08.2016	after		2	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Valerie	02.07.2016	before		2	0	1	0	0	0	0	0	0	0	0	1	0	1	0

Appendix 10: Att3_YFAS_ExcelFormula_AKiste.xlsx (dichotomous analysis)

Study ID	date	before / after	16	17	18	19	20	21	22	23	24
Angie	03.08.2016	after	0	not sc	not sc	0	0	0	0	not sc	0
Angie	02.07.2016	before	0	not sc	not sc	1	0	1	2	not sc	1
Anja	03.08.2016	after	0	not sc	not sc	1	0	1	1	not sc	0
Anja	02.07.2016	before	0	not sc	not sc	3	2	2	3	not sc	0
Bill	02.07.2016	before	0	not sc	not sc	1	1	3	2	not sc	0
Bill	03.08.2016	after	0	not sc	not sc	0	0	0	1	not sc	0
Candice	03.08.2016	after	0	not sc	not sc	0	0	0	2	not sc	0
Candice	02.07.2016	before	0	not sc	not sc	1	0	0	4	not sc	0
Catherine	03.08.2016	after	0	not sc	not sc	0	0	1	2	not sc	0
Catherine	02.07.2016	before	1	not sc	not sc	1	2	2	3	not sc	1
Cindy	03.08.2016	after	0	not sc	not sc	1	0	1	2	not sc	0
Cindy	02.07.2016	before	1	not sc	not sc	2	1	2	4	not sc	1
Claudia	03.08.2016	after	0	not sc	not sc	0	0	0	0	not sc	0
Claudia	02.07.2016	before	0	not sc	not sc	1	0	1	2	not sc	0
David	15.8.2016	before	0	not sc	not sc	0	0	0	2	not sc	0
David	12.11.2016	after	0	not sc	not sc	1	0	0	0	not sc	0
Heather	31.08.2016	after	0	not sc	not sc	1	1	1	3	not sc	0
Heather	28.07.2016	before	0	not sc	not sc	2	1	1	3	not sc	0
Ingrid	03.08.2016	after	0	not sc	not sc	0	0	0	0	not sc	0
Ingrid	02.07.2016	before	0	not sc	not sc	1	0	0	0	not sc	0
Jill	03.08.2016	after	0	not sc	not sc	0	0	0	0	not sc	0
Jill	02.07.2016	before	0	not sc	not sc	1	0	0	2	not sc	0
Justine	03.08.2016	after	0	not sc	not sc	1	0	0	2	not sc	0
Justine	02.07.2016	before	0	not sc	not sc	3	0	0	3	not sc	0
Karl	28.07.2016	before	0			2	0	0	3		0
Karl	01.09.2016	after	0			1	0	0	3		0
Lois	12.11.2016	after	0	not sc	not sc	0	0	2	1	not sc	0
Lois	15.8.2016	before	0	not sc	not sc	1	0	0	4	not sc	0
Ltisch	3.8.2016	after	0	not sc	not sc	0	0	0	1	not sc	0
Ltisch	30.6.2016	before	0	not sc	not sc	1	1	0	3	not sc	0
Mitch	30.6.2016	before	0	not sc	not sc	1	0	0	3	not sc	1
Mitch	3.8.2016	after	0	not sc	not sc	0	0	0	2	not sc	0
Nikki	03.08.2016	after	0	not sc	not sc	0	0	0	0	not sc	0
Nikki	02.07.2016	before	0	not sc	not sc	1	0	0	2	not sc	0
Pat	03.08.2016	after	0	not sc	not sc	0	0	0	0	not sc	0
Pat	02.07.2016	before	0	not sc	not sc	1	0	0	2	not sc	0
Paul	02.07.2016	before	0	not sc	not sc	1	0	0	1	not sc	0
Paul	03.08.2016	after	0	not sc	not sc	0	0	0	1	not sc	0
Rael	02.07.2016	before	1	not sc	not sc	2	2	2	4	not sc	1
Rael	03.08.2016	after	0	not sc	not sc	1	0	1	2	not sc	0
Renate	03.08.2016	after	0	not sc	not sc	0	0	0	1	not sc	0
Renate	02.07.2016	before	0	not sc	not sc	1	0	0	2	not sc	0
Renee	03.08.2016	after	0	not sc	not sc	0	0	0	0	not sc	0
Renee	02.07.2016	before	0	not sc	not sc	1	0	0	2	not sc	0
Roland	18.08.2016	before	0	not sc	not sc	3	1	0	4	not sc	0
Roland	18.08.2016	after	0	not sc	not sc	3	1	0	1	not sc	0
Sally Jane	03.08.2016	after	0	not sc	not sc	0	0	0	0	not sc	0
Sally Jane	02.07.2016	before	0	not sc	not sc	1	0	0	1	not sc	0
Sam	15.7.2016	after	0	not sc	not sc	0	0	0	0	not sc	0
Sam	15.6.2016	before	0	not sc	not sc	1	0	0	2	not sc	0
Sheena	03.08.2016	after	0	not sc	not sc	0	0	1	3	not sc	0
Sheena	02.07.2016	before	1	not sc	not sc	1	1	1	3	not sc	0
Stevie	3.8.2016	after	1	not sc	not sc	0	0	0	4	not sc	0
Stevie	30.6.2016	before	0	not sc	not sc	3	0	0	4	not sc	0
Tarryn	03.08.2016	after	0	not sc	not sc	0	0	0	0	not sc	0
Tarryn	02.07.2016	before	0	not sc	not sc	1	0	0	2	not sc	0
Ulrike	03.08.2016	after	0	not sc	not sc	0	0	0	1	not sc	0
Ulrike	02.07.2016	before	0	not sc	not sc	1	0	0	2	not sc	0
Valerie	03.08.2016	after	0	not sc	not sc	0	0	0	1	not sc	0
Valerie	02.07.2016	before	0	not sc	not sc	1	0	0	3	not sc	0

Appendix 10: Att3_YFAS_ExcelFormula_AKiste.xlsx (dichotomous analysis)

Study ID	date	before / after	25	1. Substance taken in larger amount and for longer period than intended	Met?	Criterion Score	2. Persistent desire or repeated unsuccessful attempts at quitting	Met?	Criterion Score	3. Much time/activity to obtain, use, recover	Met?	Criterion Score	4. Important social, occupational, or recreational activities given up or reduced	Met?	Criterion Score
Angie	03.08.2016	after	0	0	No	0	0	No	0	0	No	0	0	No	0
Angie	02.07.2016	before	0	1	Yes	1	3	Yes	1	0	No	0	0	No	0
Anja	03.08.2016	after	0	0	No	0	1	Yes	1	0	No	0	0	No	0
Anja	02.07.2016	before	0	1	Yes	1	3	Yes	1	1	Yes	1	0	No	0
Bill	02.07.2016	before	0	0	No	0	2	Yes	1	2	Yes	1	0	No	0
Bill	03.08.2016	after	0	0	No	0	1	Yes	1	0	No	0	0	No	0
Candice	03.08.2016	after	0	0	No	0	2	Yes	1	0	No	0	0	No	0
Candice	02.07.2016	before	0	0	No	0	4	Yes	1	0	No	0	0	No	0
Catherine	03.08.2016	after	0	0	No	0	2	Yes	1	0	No	0	0	No	0
Catherine	02.07.2016	before	0	2	Yes	1	4	Yes	1	3	Yes	1	0	No	0
Cindy	03.08.2016	after	0	0	No	0	2	Yes	1	0	No	0	0	No	0
Cindy	02.07.2016	before	0	2	Yes	1	5	Yes	1	1	Yes	1	1	Yes	1
Claudia	03.08.2016	after	0	0	No	0	0	No	0	0	No	0	0	No	0
Claudia	02.07.2016	before	0	0	No	0	2	Yes	1	0	No	0	0	No	0
David	15.8.2016	before	0	0	No	0	2	Yes	1	0	No	0	0	No	0
David	12.11.2016	after	0	0	No	0	0	No	0	0	No	0	0	No	0
Heather	31.08.2016	after	0	0	No	0	3	Yes	1	0	No	0	0	No	0
Heather	28.07.2016	before	0	0	No	0	3	Yes	1	1	Yes	1	0	No	0
Ingrid	03.08.2016	after	0	0	No	0	0	No	0	0	No	0	0	No	0
Ingrid	02.07.2016	before	0	0	No	0	0	No	0	0	No	0	0	No	0
Jill	03.08.2016	after	0	0	No	0	0	No	0	0	No	0	0	No	0
Jill	02.07.2016	before	0	0	No	0	2	Yes	1	0	No	0	0	No	0
Justine	03.08.2016	after	0	0	No	0	2	Yes	1	0	No	0	0	No	0
Justine	02.07.2016	before	0	0	No	0	3	Yes	1	0	No	0	0	No	0
Karl	28.07.2016	before	0	1	Yes	1	4	Yes	1	1	Yes	1	0	No	0
Karl	01.09.2016	after	0	0	No	0	3	Yes	1	0	No	0	0	No	0
Lois	12.11.2016	after	0	0	No	0	1	Yes	1	0	No	0	0	No	0
Lois	15.8.2016	before	0	0	No	0	4	Yes	1	1	Yes	1	0	No	0
Ltisch	3.8.2016	after	0	0	No	0	1	Yes	1	1	Yes	1	0	No	0
Ltisch	30.6.2016	before	0	0	No	0	3	Yes	1	0	No	0	0	No	0
Mitch	30.6.2016	before	0	1	Yes	1	4	Yes	1	2	Yes	1	0	No	0
Mitch	3.8.2016	after	0	0	No	0	2	Yes	1	0	No	0	0	No	0
Nikki	03.08.2016	after	0	0	No	0	0	No	0	0	No	0	0	No	0
Nikki	02.07.2016	before	0	0	No	0	2	Yes	1	0	No	0	0	No	0
Pat	03.08.2016	after	0	0	No	0	0	No	0	0	No	0	0	No	0
Pat	02.07.2016	before	0	0	No	0	2	Yes	1	0	No	0	0	No	0
Paul	02.07.2016	before	0	0	No	0	1	Yes	1	0	No	0	0	No	0
Paul	03.08.2016	after	0	0	No	0	1	Yes	1	0	No	0	0	No	0
Rael	02.07.2016	before	0	1	Yes	1	5	Yes	1	1	Yes	1	0	No	0
Rael	03.08.2016	after	0	0	No	0	2	Yes	1	0	No	0	0	No	0
Renate	03.08.2016	after	0	0	No	0	1	Yes	1	0	No	0	0	No	0
Renate	02.07.2016	before	0	0	No	0	2	Yes	1	0	No	0	0	No	0
Renee	03.08.2016	after	0	0	No	0	0	No	0	0	No	0	0	No	0
Renee	02.07.2016	before	0	0	No	0	2	Yes	1	0	No	0	0	No	0
Roland	18.08.2016	before	0	1	Yes	1	4	Yes	1	0	No	0	0	No	0
Roland	18.08.2016	after	0	0	No	0	1	Yes	1	0	No	0	0	No	0
Sally Jane	03.08.2016	after	0	0	No	0	0	No	0	0	No	0	0	No	0
Sally Jane	02.07.2016	before	0	0	No	0	1	Yes	1	0	No	0	0	No	0
Sam	15.7.2016	after	0	0	No	0	0	No	0	0	No	0	0	No	0
Sam	15.6.2016	before	0	0	No	0	2	Yes	1	0	No	0	0	No	0
Sheena	03.08.2016	after	0	0	No	0	3	Yes	1	0	No	0	0	No	0
Sheena	02.07.2016	before	0	0	No	0	3	Yes	1	0	No	0	0	No	0
Stevie	3.8.2016	after	0	0	No	0	4	Yes	1	2	Yes	1	1	Yes	1
Stevie	30.6.2016	before	0	0	No	0	5	Yes	1	1	Yes	1	0	No	0
Tarryn	03.08.2016	after	0	0	No	0	0	No	0	0	No	0	0	No	0
Tarryn	02.07.2016	before	0	0	No	0	2	Yes	1	0	No	0	0	No	0
Ulrike	03.08.2016	after	0	0	No	0	1	Yes	1	0	No	0	0	No	0
Ulrike	02.07.2016	before	0	0	No	0	2	Yes	1	0	No	0	0	No	0
Valerie	03.08.2016	after	0	0	No	0	1	Yes	1	0	No	0	0	No	0
Valerie	02.07.2016	before	0	1	Yes	1	3	Yes	1	0	No	0	0	No	0

Appendix 10: Att3_YFAS_ExcelFormula_AKiste.xlsx (dichotomous analysis)

Study ID	date	before / after	5. Use continues despite knowledge of adverse consequences (e.g., failure to fulfill role obligation, use when physically hazardous	Met?	Criterion Score	6. Tolerance (marked increase in amount; marked decrease in effect)	Met?	Criterion Score	7. Characteristic withdrawal symptoms; substance taken to relieve withdrawal	Met?	Criterion Score	8. Use causes clinically significant impairment	Met?	Criterion Score
Angie	03.08.2016	after		0	No	0	0	No	0	0	No	0	0	No	0
Angie	02.07.2016	before		1	Yes	1	1	Yes	1	0	No	0	0	No	0
Anja	03.08.2016	after		1	Yes	1	1	Yes	1	0	No	0	0	No	0
Anja	02.07.2016	before		3	Yes	1	4	Yes	1	1	Yes	1	0	No	0
Bill	02.07.2016	before		1	Yes	1	4	Yes	1	2	Yes	1	0	No	0
Bill	03.08.2016	after		0	No	0	0	No	0	0	No	0	0	No	0
Candice	03.08.2016	after		0	No	0	0	No	0	0	No	0	0	No	0
Candice	02.07.2016	before		1	Yes	1	0	No	0	0	No	0	1	Yes	1
Catherine	03.08.2016	after		0	No	0	1	Yes	1	0	No	0	0	No	0
Catherine	02.07.2016	before		1	Yes	1	4	Yes	1	3	Yes	1	2	Yes	1
Cindy	03.08.2016	after		1	Yes	1	1	Yes	1	0	No	0	0	No	0
Cindy	02.07.2016	before		2	Yes	1	3	Yes	1	3	Yes	1	2	Yes	1
Claudia	03.08.2016	after		0	No	0	0	No	0	0	No	0	0	No	0
Claudia	02.07.2016	before		1	Yes	1	1	Yes	1	0	No	0	0	No	0
David	15.8.2016	before		0	No	0	0	No	0	0	No	0	0	No	0
David	12.11.2016	after		1	Yes	1	0	No	0	0	No	0	0	No	0
Heather	31.08.2016	after		1	Yes	1	2	Yes	1	0	No	0	0	No	0
Heather	28.07.2016	before		2	Yes	1	2	Yes	1	0	No	0	0	No	0
Ingrid	03.08.2016	after		0	No	0	0	No	0	0	No	0	0	No	0
Ingrid	02.07.2016	before		1	Yes	1	0	No	0	0	No	0	0	No	0
Jill	03.08.2016	after		0	No	0	0	No	0	0	No	0	0	No	0
Jill	02.07.2016	before		1	Yes	1	0	No	0	0	No	0	0	No	0
Justine	03.08.2016	after		1	Yes	1	0	No	0	0	No	0	0	No	0
Justine	02.07.2016	before		3	Yes	1	0	No	0	0	No	0	1	Yes	1
Karl	28.07.2016	before		2	Yes	1	0	No	0	1	Yes	1	0	No	0
Karl	01.09.2016	after		1	Yes	1	0	No	0	0	No	0	0	No	0
Lois	12.11.2016	after		0	No	0	2	Yes	1	0	No	0	0	No	0
Lois	15.8.2016	before		1	Yes	1	0	No	0	0	No	0	0	No	0
Ltisch	3.8.2016	after		0	No	0	0	No	0	0	No	0	0	No	0
Ltisch	30.6.2016	before		1	Yes	1	1	Yes	1	1	Yes	1	0	No	0
Mitch	30.6.2016	before		1	Yes	1	0	No	0	2	Yes	1	0	No	0
Mitch	3.8.2016	after		0	No	0	0	No	0	1	Yes	1	0	No	0
Nikki	03.08.2016	after		0	No	0	0	No	0	0	No	0	0	No	0
Nikki	02.07.2016	before		1	Yes	1	0	No	0	0	No	0	0	No	0
Pat	03.08.2016	after		0	No	0	0	No	0	0	No	0	0	No	0
Pat	02.07.2016	before		1	Yes	1	0	No	0	0	No	0	0	No	0
Paul	02.07.2016	before		1	Yes	1	0	No	0	0	No	0	0	No	0
Paul	03.08.2016	after		0	No	0	0	No	0	0	No	0	0	No	0
Rael	02.07.2016	before		2	Yes	1	4	Yes	1	1	Yes	1	2	Yes	1
Rael	03.08.2016	after		1	Yes	1	1	Yes	1	0	No	0	0	No	0
Renate	03.08.2016	after		0	No	0	0	No	0	0	No	0	0	No	0
Renate	02.07.2016	before		1	Yes	1	0	No	0	0	No	0	0	No	0
Renee	03.08.2016	after		0	No	0	0	No	0	0	No	0	0	No	0
Renee	02.07.2016	before		1	Yes	1	0	No	0	0	No	0	0	No	0
Roland	18.08.2016	before		3	Yes	1	1	Yes	1	1	Yes	1	0	No	0
Roland	18.08.2016	after		3	Yes	1	1	Yes	1	0	No	0	0	No	0
Sally Jane	03.08.2016	after		0	No	0	0	No	0	0	No	0	0	No	0
Sally Jane	02.07.2016	before		1	Yes	1	0	No	0	0	No	0	0	No	0
Sam	15.7.2016	after		0	No	0	0	No	0	0	No	0	0	No	0
Sam	15.6.2016	before		1	Yes	1	0	No	0	0	No	0	0	No	0
Sheena	03.08.2016	after		0	No	0	1	Yes	1	0	No	0	0	No	0
Sheena	02.07.2016	before		1	Yes	1	2	Yes	1	0	No	0	1	Yes	1
Stevie	3.8.2016	after		0	No	0	0	No	0	1	Yes	1	2	Yes	1
Stevie	30.6.2016	before		3	Yes	1	0	No	0	2	Yes	1	1	Yes	1
Tarryn	03.08.2016	after		0	No	0	0	No	0	0	No	0	0	No	0
Tarryn	02.07.2016	before		1	Yes	1	0	No	0	0	No	0	0	No	0
Ulrike	03.08.2016	after		0	No	0	0	No	0	0	No	0	0	No	0
Ulrike	02.07.2016	before		1	Yes	1	0	No	0	0	No	0	0	No	0
Valerie	03.08.2016	after		0	No	0	0	No	0	0	No	0	0	No	0
Valerie	02.07.2016	before		1	Yes	1	0	No	0	2	Yes	1	0	No	0

Appendix 10: Att3_YFAS_ExcelFormula_AKiste.xlsx (dichotomous analysis)

Study ID	date	before / after	Symptom Count	High FA Met	High FA + Impairment
Angie	03.08.2016	after	0	0	0
Angie	02.07.2016	before	4	1	0
Anja	03.08.2016	after	3	1	0
Anja	02.07.2016	before	6	1	0
Bill	02.07.2016	before	5	1	0
Bill	03.08.2016	after	1	0	0
Candice	03.08.2016	after	1	0	0
Candice	02.07.2016	before	2	0	0
Catherine	03.08.2016	after	2	0	0
Catherine	02.07.2016	before	6	1	1
Cindy	03.08.2016	after	3	1	0
Cindy	02.07.2016	before	7	1	1
Claudia	03.08.2016	after	0	0	0
Claudia	02.07.2016	before	3	1	0
David	15.8.2016	before	1	0	0
David	12.11.2016	after	1	0	0
Heather	31.08.2016	after	3	1	0
Heather	28.07.2016	before	4	1	0
Ingrid	03.08.2016	after	0	0	0
Ingrid	02.07.2016	before	1	0	0
Jill	03.08.2016	after	0	0	0
Jill	02.07.2016	before	2	0	0
Justine	03.08.2016	after	2	0	0
Justine	02.07.2016	before	2	0	0
Karl	28.07.2016	before	5	1	0
Karl	01.09.2016	after	2	0	0
Lois	12.11.2016	after	2	0	0
Lois	15.8.2016	before	3	1	0
Ltisch	3.8.2016	after	2	0	0
Ltisch	30.6.2016	before	4	1	0
Mitch	30.6.2016	before	5	1	0
Mitch	3.8.2016	after	2	0	0
Nikki	03.08.2016	after	0	0	0
Nikki	02.07.2016	before	2	0	0
Pat	03.08.2016	after	0	0	0
Pat	02.07.2016	before	2	0	0
Paul	02.07.2016	before	2	0	0
Paul	03.08.2016	after	1	0	0
Rael	02.07.2016	before	6	1	1
Rael	03.08.2016	after	3	1	0
Renate	03.08.2016	after	1	0	0
Renate	02.07.2016	before	2	0	0
Renee	03.08.2016	after	0	0	0
Renee	02.07.2016	before	2	0	0
Roland	18.08.2016	before	5	1	0
Roland	18.08.2016	after	3	1	0
Sally Jane	03.08.2016	after	0	0	0
Sally Jane	02.07.2016	before	2	0	0
Sam	15.7.2016	after	0	0	0
Sam	15.6.2016	before	2	0	0
Sheena	03.08.2016	after	2	0	0
Sheena	02.07.2016	before	3	1	1
Stevie	3.8.2016	after	4	1	1
Stevie	30.6.2016	before	4	1	1
Tarryn	03.08.2016	after	0	0	0
Tarryn	02.07.2016	before	2	0	0
Ulrike	03.08.2016	after	1	0	0
Ulrike	02.07.2016	before	2	0	0
Valerie	03.08.2016	after	1	0	0
Valerie	02.07.2016	before	4	1	0

Appendix 10: Att3_YFAS_ExcelFormula_AKiste.xlsx (Cronbach alpha)

Anova: Two-Factor Without Replication

SUMMARY	*Count*	*Sum*	*Average*	*Variance*
Row 23	22	4	0,181818182	0,251082251
Row 23	22	19	0,863636364	1,170995671
Row 23	22	7	0,318181818	0,322510823
Row 23	22	33	1,5	1,785714286
Row 23	22	34	1,545454545	1,688311688
Row 23	22	11	0,5	0,738095238
Row 23	22	5	0,227272727	0,374458874
Row 23	22	20	0,909090909	1,324675325
Row 23	22	22	1	1,238095238
Row 23	22	47	2,136363636	1,742424242
Row 23	22	23	1,045454545	0,807359307
Row 23	22	51	2,318181818	1,560606061
Row 23	22	2	0,090909091	0,086580087
Row 23	22	14	0,636363636	0,813852814
Row 23	22	20	0,909090909	1,038961039
Row 23	22	9	0,409090909	0,443722944
Row 23	22	28	1,272727273	0,779220779
Row 23	22	36	1,636363636	1,0995671
Row 23	22	1	0,045454545	0,045454545
Row 23	22	11	0,5	0,738095238
Row 23	22	1	0,045454545	0,045454545
Row 23	22	13	0,590909091	0,824675325
Row 23	22	8	0,363636364	0,432900433
Row 23	22	30	1,363636364	1,480519481
Row 23	22	33	1,5	1,880952381
Row 23	22	24	1,090909091	1,134199134
Row 23	22	8	0,363636364	0,432900433
Row 23	22	23	1,045454545	1,474025974
Row 23	22	7	0,318181818	0,608225108
Row 23	22	26	1,181818182	1,108225108
Row 23	22	35	1,590909091	1,872294372
Row 23	22	14	0,636363636	1,29004329
Row 23	22	1	0,045454545	0,045454545
Row 23	22	14	0,636363636	0,813852814
Row 23	22	2	0,090909091	0,086580087
Row 23	22	17	0,772727273	0,945887446
Row 23	22	12	0,545454545	0,735930736
Row 23	22	2	0,090909091	0,086580087
Row 23	22	44	2	1,523809524
Row 23	22	11	0,5	0,452380952
Row 23	22	3	0,136363636	0,123376623
Row 23	22	13	0,590909091	0,824675325
Row 23	22	1	0,045454545	0,045454545
Row 23	22	14	0,636363636	0,909090909
Row 23	22	28	1,272727273	2,207792208
Row 23	22	11	0,5	0,738095238
Row 23	22	1	0,045454545	0,045454545
Row 23	22	13	0,590909091	0,824675325
Row 23	22	1	0,045454545	0,045454545
Row 23	22	14	0,636363636	1,004329004
Row 23	22	7	0,318181818	0,512987013
Row 23	22	29	1,318181818	1,274891775
Row 23	22	41	1,863636364	2,980519481
Row 23	22	40	1,818181818	2,822510823
Row 23	22	1	0,045454545	0,045454545
Row 23	22	14	0,636363636	0,813852814
Row 23	22	2	0,090909091	0,086580087
Row 23	22	13	0,590909091	0,634199134
Row 23	22	12	0,545454545	0,545454545
Row 23	22	34	1,545454545	1,593073593
Column 1	60	107	1,783333333	0,918361582
Column 2	60	112	1,866666667	2,083615819
Column 3	60	34	0,566666667	0,385310734
Column 4	60	66	1,1	1,210169492
Column 5	60	73	1,216666667	1,121751412
Column 6	60	45	0,75	1,716101695
Column 7	60	52	0,866666667	1,371751412
Column 8	60	4	0,066666667	0,097175141
Column 9	60	9	0,15	0,231355932
Column 10	60	4	0,066666667	0,266666667
Column 11	60	23	0,383333333	1,020056497
Column 12	60	45	0,75	1,275423729
Column 13	60	31	0,516666667	0,728531073
Column 14	60	74	1,233333333	1,436158192
Column 15	60	53	0,883333333	1,426836158
Column 16	60	26	0,433333333	0,961581921
Column 17	60	31	0,516666667	0,660734463
Column 18	60	24	0,4	0,447457627
Column 19	60	51	0,85	0,773728814
Column 20	60	13	0,216666667	0,274293785
Column 21	60	26	0,433333333	0,62259887

Appendix 10: Att3_YFAS_ExcelFormula_AKiste.xlsx (Cronbach alpha)

Column 22	60	111	1,85	1,655084746

ANOVA

Source of Variation	*SS*	*df*	*MS*	*F*	*P-value*	*F crit*
Rows	477,6090909	59	8,095069337	13,50284553	0	1,331957353
Columns	378,6636364	21	18,03160173	30,07731284	0	1,564326245
Error	742,7909091	1239	0,599508401			
Total	1599,063636	1319				

Cronbach's alpha	**0,925941536**
no. of items	25

Appendix 11: ATT5_QA Table-1
(qualitative analysis 'feeling in control)

stuyID	DOB	age	gender	in control' on a scale of 0-3 (whereby 0 implied no
Angie	02.07.2016	40	female	3
Anja	02.07.2016	40	female	2
Bill	02.07.2016	48	male	2
Candice	02.07.2016	33	female	1
Catherine	02.07.2016	43	female	1
Cindy	02.07.2016	40	female	1
Claudia	02.07.2016	52	female	3
David	15.8.2016	41	male	3
Heather	28.07.2016	35	female	2
Ingrid	02.07.2016	74	female	3
Jill	02.07.2016	54	female	3
Justine	02.07.2016	37	female	2
Karl	28.07.2016	41	male	1
Lois	15.8.2016	37	female	3
Ltisch	30.6.2016	32	female	3
Mitch	30.6.2016	27	male	2
Nikki	02.07.2016	46	female	3
Pat	02.07.2016	60	female	3
Paul	02.07.2016	79	male	2
Rael	02.07.2016	50	male	1
Renate	02.07.2016	72	female	2
Renee	02.07.2016	75	female	3
Roland	18.08.2016	44	male	2
Sally Jane	02.07.2016	52	female	3
Sam	15.6.2016	39	female	3
Sheena	02.07.2016	30	female	1
Stevie	30.6.2016	27	female	1
Tarryn	02.07.2016	34	female	3
Ulrike	02.07.2016	46	female	3
Valerie	02.07.2016	68	female	2
			AVERAGE	2,23

Appendix 12: Results 18 25-SYM2 PRIMER Questions AFTER ('r' calculations on Cronbach)

```
Sourcing: C:\Shtats\BBQBreakfast\BBQBreakfast\BBQBreakfast\script.R
Rows Read: 30, Total Rows Processed: 30, Total Chunk Time: 0.009 seconds
'data.frame':   30 obs. of  18 variables:
 $ Q1 : num  1 2 1 1 1 1 1 0 2 1 ...
 $ Q2 : num  1 0 1 0 2 1 1 2 3 0 ...
 $ Q3 : num  0 0 0 0 0 0 0 0 1 0 ...
 $ Q5 : num  0 1 2 0 2 0 0 1 2 0 ...
 $ Q6 : num  0 0 0 0 0 0 0 0 1 1 0 ...
 $ Q7 : num  0 0 0 0 2 1 0 1 1 0 ...
 $ Q8 : num  0 0 0 0 0 0 0 0 0 0 0 ...
 $ Q9 : num  0 0 0 0 0 0 0 0 1 0 0 ...
 $ Q10: num  0 0 0 0 0 0 0 0 0 0 0 ...
 $ Q11: num  2 0 3 0 4 3 0 0 0 0 0 ...
 $ Q12: num  0 1 1 0 1 2 0 0 1 0 0 ...
 $ Q13: num  0 0 0 0 1 1 0 0 1 0 0 ...
 $ Q14: num  0 0 2 0 2 1 0 1 2 0 0 ...
 $ Q15: num  0 0 0 2 2 2 0 0 2 0 0 ...
 $ Q16: num  0 0 0 0 2 2 0 0 0 0 0 ...
 $ Q19: num  0 1 0 0 0 1 0 1 1 0 0 ...
 $ Q20: num  0 0 0 0 0 0 0 0 1 0 0 ...
 $ Q21: num  0 1 0 0 1 1 0 0 1 0 0 ...

Reliability analysis
Call: psych::alpha(x = d, check.keys = TRUE)

 raw_alpha std.alpha G6(smc) average_r S/N   ase mean   sd
      0.88      0.88    0.98      0.31 7.6 0.028  0.4 0.45

 lower alpha upper     95% confidence boundaries
0.82 0.88 0.93

Reliability if an item is dropped:
    raw_alpha std.alpha G6(smc) average_r S/N alpha se
Q1       0.88      0.88    0.98      0.32 7.7    0.027
Q2       0.87      0.87    0.98      0.30 6.9    0.030
Q3       0.87      0.87    0.98      0.29 6.6    0.029
Q5       0.86      0.87    0.98      0.29 6.7    0.032
Q6       0.86      0.87    0.98      0.30 7.0    0.030
Q7       0.87      0.88    0.98      0.32 7.5    0.028
Q9       0.88      0.88    0.98      0.32 7.4    0.028
Q10      0.87      0.88    0.98      0.31 7.2    0.030
Q11      0.87      0.88    0.98      0.31 7.2    0.030
Q12      0.86      0.87    0.97      0.29 6.6    0.031
Q13      0.87      0.88    0.98      0.31 7.1    0.029
Q14      0.86      0.87    0.98      0.29 6.7    0.033
Q15      0.86      0.87    0.98      0.29 6.7    0.031
Q16      0.86      0.87    0.98      0.30 7.0    0.032
Q19      0.88      0.89    0.98      0.33 7.9    0.026
Q20      0.88      0.88    0.98      0.32 7.6    0.028
Q21      0.88      0.89    0.98      0.33 7.9    0.027

Item statistics
     n raw.r std.r r.cor r.drop  mean   sd
Q1  30  0.26  0.40  0.40   0.20 1.067 0.45
Q2  30  0.67  0.69  0.69   0.59 0.667 0.96
Q3  30  0.72  0.80  0.80   0.70 0.100 0.31
Q5  30  0.78  0.76  0.75   0.73 0.533 0.90
Q6  30  0.70  0.65  0.66   0.65 0.200 0.76
Q7  30  0.47  0.46  0.45   0.38 0.467 0.78
Q9  30  0.33  0.50  0.50   0.31 0.033 0.18
Q10 30  0.65  0.57  0.57   0.59 0.133 0.73
Q11 30  0.70  0.57  0.57   0.60 0.700 1.34
```

Appendix 12: Results 18 25-SYM2 PRIMER Questions AFTER ('r' calculations on Cronbach)

```
Item = Q8 had no variance and was deleted

Q12 30  0.81  0.77  0.77   0.77 0.433 0.63
Q13 30  0.58  0.61  0.61   0.54 0.200 0.41
Q14 30  0.82  0.76  0.74   0.76 0.700 1.18
Q15 30  0.77  0.76  0.76   0.70 0.600 1.04
Q16 30  0.76  0.66  0.66   0.71 0.333 0.88
Q19 30  0.23  0.33  0.33   0.15 0.333 0.66
Q20 30  0.26  0.43  0.42   0.22 0.067 0.25
Q21 30  0.26  0.33  0.31   0.20 0.267 0.52

Non missing response frequency for each item
      0    1    2    3    4 miss
Q1  0.07 0.80 0.13 0.00 0.00    0
Q2  0.60 0.20 0.13 0.07 0.00    0
Q3  0.90 0.10 0.00 0.00 0.00    0
Q5  0.70 0.10 0.17 0.03 0.00    0
Q6  0.90 0.07 0.00 0.00 0.03    0
Q7  0.67 0.23 0.07 0.03 0.00    0
Q9  0.97 0.03 0.00 0.00 0.00    0
Q10 0.97 0.00 0.00 0.00 0.03    0
Q11 0.77 0.00 0.07 0.10 0.07    0
Q12 0.63 0.30 0.07 0.00 0.00    0
Q13 0.80 0.20 0.00 0.00 0.00    0
Q14 0.67 0.10 0.17 0.00 0.07    0
Q15 0.70 0.07 0.20 0.00 0.03    0
Q16 0.83 0.07 0.07 0.00 0.03    0
Q19 0.73 0.23 0.00 0.03 0.00    0
Q20 0.93 0.07 0.00 0.00 0.00    0
Q21 0.77 0.20 0.03 0.00 0.00    0
Warning message:
In psych::alpha(d, check.keys = TRUE) :
```

Appendix 13: Results 18 25-SYM2 PRIMER Questions BEFORE ('r' calculations on Cronbach)

```
Sourcing: C:\Shtats\BBQBreakfast\BBQBreakfast\BBQBreakfast\script.R
Rows Read: 30, Total Rows Processed: 30, Total Chunk Time: 0.038 seconds
'data.frame':   30 obs. of  18 variables:
 $ Q1 : num  2 4 3 2 4 4 2 2 3 2 ...
 $ Q2 : num  4 3 3 3 4 4 3 2 3 3 ...
 $ Q3 : num  1 1 1 1 1 2 1 2 2 1 ...
 $ Q5 : num  2 3 3 1 3 2 2 2 2 2 ...
 $ Q6 : num  0 3 4 0 4 0 0 3 2 0 ...
 $ Q7 : num  2 0 2 1 3 3 0 1 3 0 ...
 $ Q8 : num  0 1 0 0 0 2 0 0 0 0 ...
 $ Q9 : num  0 1 0 0 2 2 0 0 2 0 ...
 $ Q10: num  0 0 0 0 0 0 0 0 0 0 ...
 $ Q11: num  0 0 0 0 0 0 0 0 0 0 ...
 $ Q12: num  0 3 3 0 3 4 0 2 1 0 ...
 $ Q13: num  0 0 1 0 3 3 0 1 1 0 ...
 $ Q14: num  1 1 3 1 3 3 1 2 2 1 ...
 $ Q15: num  2 0 1 3 3 3 0 0 2 0 ...
 $ Q16: num  0 0 0 0 3 3 0 0 0 0 ...
 $ Q19: num  1 3 1 1 1 2 1 0 2 1 ...
 $ Q20: num  0 2 1 0 2 1 0 0 1 0 ...
 $ Q21: num  1 2 3 0 2 2 1 0 1 0 ...

Reliability analysis
Call: psych::alpha(x = d, check.keys = TRUE)

  raw_alpha std.alpha G6(smc) average_r S/N   ase mean   sd
       0.88      0.88    0.98      0.31 7.6 0.026  1.1 0.56

 lower alpha upper     95% confidence boundaries
0.83 0.88 0.94

Reliability if an item is dropped:
    raw_alpha std.alpha G6(smc) average_r S/N alpha se
Q1       0.88      0.87    0.98      0.30 6.9    0.028
Q2       0.88      0.88    0.98      0.31 7.2    0.027
Q3       0.89      0.89    0.98      0.33 7.9    0.026
Q5       0.89      0.89    0.97      0.33 8.0    0.025
Q6       0.88      0.88    0.97      0.31 7.1    0.028
Q7       0.87      0.87    0.98      0.30 6.9    0.029
Q8       0.88      0.88    0.97      0.31 7.1    0.027
Q9       0.88      0.88    0.97      0.30 7.0    0.028
Q11      0.89      0.89    0.98      0.34 8.2    0.027
Q12      0.86      0.87    0.97      0.29 6.5    0.032
Q13      0.87      0.87    0.97      0.29 6.7    0.030
Q14      0.87      0.87    0.97      0.30 7.0    0.030
Q15      0.88      0.88    0.97      0.31 7.1    0.028
Q16      0.87      0.88    0.97      0.31 7.0    0.028
Q19      0.88      0.88    0.98      0.32 7.6    0.027
Q20      0.87      0.87    0.97      0.30 6.8    0.028
Q21      0.88      0.88    0.98      0.31 7.2    0.028

 Item statistics
       n raw.r std.r r.cor r.drop  mean   sd
Q1    30  0.65  0.70  0.69   0.60 2.500 0.78
Q2    30  0.54  0.59  0.56   0.49 3.067 0.58
Q3    30  0.26  0.35  0.33   0.21 1.033 0.49
Q5    30  0.23  0.31  0.31   0.16 1.900 0.71
Q6    30  0.69  0.61  0.61   0.60 1.300 1.51
Q7    30  0.72  0.69  0.68   0.64 1.267 1.36
Q8    30  0.55  0.62  0.62   0.52 0.133 0.43
Q9    30  0.61  0.65  0.64   0.57 0.267 0.64
Q11 30  0.18  0.24  0.23   0.15 0.067 0.25
```

Appendix 13: Results 18 25-SYM2 PRIMER Questions BEFORE ('r' calculations on Cronbach)

```
Q12 30  0.84  0.82  0.83   0.79 1.067 1.41
Q13 30  0.80  0.77  0.77   0.75 0.833 1.05
Q14 30  0.74  0.66  0.66   0.68 1.767 0.97
Q15 30  0.66  0.62  0.62   0.58 1.167 1.29
Q16 30  0.66  0.64  0.64   0.59 0.533 1.07
Q19 30  0.45  0.45  0.43   0.38 1.367 0.76
Q20 30  0.73  0.74  0.75   0.70 0.367 0.67
Q21 30  0.61  0.59  0.58   0.54 0.600 0.97

Item = Q10 had no variance and was deleted

Non missing response frequency for each item
      0    1    2    3    4 miss
Q1  0.00 0.00 0.67 0.17 0.17    0
Q2  0.00 0.00 0.13 0.67 0.20    0
Q3  0.10 0.77 0.13 0.00 0.00    0
Q5  0.07 0.10 0.70 0.13 0.00    0
Q6  0.53 0.03 0.10 0.27 0.07    0
Q7  0.47 0.10 0.17 0.23 0.03    0
Q8  0.90 0.07 0.03 0.00 0.00    0
Q9  0.83 0.07 0.10 0.00 0.00    0
Q11 0.93 0.07 0.00 0.00 0.00    0
Q12 0.57 0.10 0.10 0.17 0.07    0
Q13 0.53 0.20 0.17 0.10 0.00    0
Q14 0.00 0.57 0.13 0.27 0.03    0
Q15 0.47 0.13 0.20 0.17 0.03    0
Q16 0.77 0.07 0.03 0.13 0.00    0
Q19 0.03 0.70 0.13 0.13 0.00    0
Q20 0.73 0.17 0.10 0.00 0.00    0
Q21 0.67 0.13 0.13 0.07 0.00    0
Warning message:
In psych::alpha(d, check.keys = TRUE) :
```

Appendix 14: Results 22 25-PRIMER Questions AFTER ('r' calculations on Cronbach)

```
Sourcing: C:\Shtats\BBQBreakfast\BBQBreakfast\BBQBreakfast\script.R
Rows Read: 30, Total Rows Processed: 30, Total Chunk Time: 0.008 seconds
'data.frame':    30 obs. of  22 variables:
 $ Q1 : num  1 2 1 1 1 1 1 0 2 1 ...
 $ Q2 : num  1 0 1 0 2 1 1 2 3 0 ...
 $ Q3 : num  0 0 0 0 0 0 0 0 1 0 ...
 $ Q4 : num  0 0 0 0 0 2 0 2 2 0 ...
 $ Q5 : num  0 1 2 0 2 0 0 1 2 0 ...
 $ Q6 : num  0 0 0 0 0 0 0 1 1 0 ...
 $ Q7 : num  0 0 0 0 2 1 0 1 1 0 ...
 $ Q8 : num  0 0 0 0 0 0 0 0 0 0 ...
 $ Q9 : num  0 0 0 0 0 0 0 0 1 0 ...
 $ Q10: num  0 0 0 0 0 0 0 0 0 0 ...
 $ Q11: num  2 0 3 0 4 3 0 0 0 0 ...
 $ Q12: num  0 1 1 0 1 2 0 0 1 0 ...
 $ Q13: num  0 0 0 0 1 1 0 0 1 0 ...
 $ Q14: num  0 0 2 0 2 1 0 1 2 0 ...
 $ Q15: num  0 0 0 2 2 2 0 0 2 0 ...
 $ Q16: num  0 0 0 0 2 2 0 0 0 0 ...
 $ Q19: num  0 1 0 0 0 1 0 1 1 0 ...
 $ Q20: num  0 0 0 0 0 0 0 0 1 0 ...
 $ Q21: num  0 1 0 0 1 1 0 0 1 0 ...
 $ Q22: num  0 1 1 2 2 2 0 0 3 0 ...
 $ Q24: num  4 4 4 4 4 4 4 4 2 4 ...
 $ Q25: num  2 2 2 2 2 2 2 1 3 2 ...

Reliability analysis
Call: psych::alpha(x = d, check.keys = TRUE)

  raw_alpha std.alpha G6(smc) average_r S/N   ase mean   sd
      0.89       0.9       1      0.31 9.2 0.024  0.5 0.44

 lower alpha upper     95% confidence boundaries
0.85 0.89 0.94

 Reliability if an item is dropped:
     raw_alpha std.alpha G6(smc) average_r S/N alpha se
Q1        0.89      0.90    0.99      0.31 9.1    0.024
Q2        0.88      0.89    1.00      0.30 8.5    0.026
Q3        0.89      0.89    1.00      0.29 8.2    0.025
Q4        0.89      0.90    1.00      0.31 8.9    0.024
Q5        0.88      0.89    1.00      0.30 8.4    0.027
Q6        0.89      0.90    1.00      0.30 8.7    0.026
Q7        0.89      0.90    1.00      0.31 9.1    0.024
Q9        0.89      0.90    1.00      0.31 8.8    0.024
Q10       0.89      0.90    1.00      0.31 9.0    0.026
Q11       0.89      0.90    1.00      0.31 9.0    0.024
Q12       0.88      0.89    1.00      0.29 8.4    0.027
Q13       0.89      0.90    1.00      0.30 8.6    0.025
Q14       0.88      0.89    0.99      0.30 8.4    0.028
Q15       0.88      0.89    1.00      0.29 8.3    0.027
Q16       0.88      0.90    1.00      0.30 8.7    0.026
Q19       0.90      0.90    1.00      0.32 9.4    0.023
Q20       0.89      0.90    1.00      0.31 9.0    0.024
Q21       0.89      0.90    0.99      0.32 9.5    0.024
Q22       0.88      0.89    1.00      0.29 8.2    0.028
Q24-      0.89      0.90    1.00      0.30 8.6    0.025
Q25       0.90      0.91    0.99      0.33 9.7    0.024

 Item statistics
     n raw.r std.r r.cor r.drop  mean    sd
Q1  30  0.31  0.44  0.44  0.261 1.067 0.45
```

Appendix 14: Results 22 25-PRIMER Questions AFTER ('r' calculations on Cronbach)

```
Q2   30  0.69  0.70  0.70  0.627 0.667 0.96
Q3   30  0.76  0.82  0.82  0.750 0.100 0.31
Q4   30  0.52  0.55  0.55  0.444 0.567 0.90
Q5   30  0.76  0.73  0.73  0.714 0.533 0.90
Q6   30  0.67  0.61  0.61  0.622 0.200 0.76
Q7   30  0.48  0.45  0.44  0.412 0.467 0.78
Q9   30  0.40  0.56  0.57  0.381 0.033 0.18
Q10  30  0.60  0.51  0.51  0.549 0.133 0.73
Q11  30  0.63  0.50  0.50  0.534 0.700 1.34
Q12  30  0.80  0.74  0.74  0.770 0.433 0.63
Q13  30  0.61  0.64  0.63  0.583 0.200 0.41
Q14  30  0.81  0.74  0.74  0.757 0.700 1.18
Q15  30  0.78  0.76  0.75  0.731 0.600 1.04
Q16  30  0.71  0.59  0.59  0.660 0.333 0.88
Q19  30  0.28  0.37  0.37  0.216 0.333 0.66
Q20  30  0.32  0.49  0.49  0.297 0.067 0.25
Q21  30  0.29  0.33  0.33  0.236 0.267 0.52
Q22  30  0.83  0.80  0.79  0.780 1.133 1.14
Q24- 30  0.49  0.64  0.64  0.451 0.100 0.40
Q25  30  0.15  0.24  0.24  0.097 1.967 0.49

Non missing response frequency for each item
       0    1    2    3    4 miss
Q1  0.07 0.80 0.13 0.00 0.00    0
Q2  0.60 0.20 0.13 0.07 0.00    0
Q3  0.90 0.10 0.00 0.00 0.00    0
Q4  0.67 0.13 0.17 0.03 0.00    0
Q5  0.70 0.10 0.17 0.03 0.00    0
Q6  0.90 0.07 0.00 0.00 0.03    0
Q7  0.67 0.23 0.07 0.03 0.00    0
Q9  0.97 0.03 0.00 0.00 0.00    0
Q10 0.97 0.00 0.00 0.00 0.03    0
Q11 0.77 0.00 0.07 0.10 0.07    0
Q12 0.63 0.30 0.07 0.00 0.00    0
Q13 0.80 0.20 0.00 0.00 0.00    0
Q14 0.67 0.10 0.17 0.00 0.07    0
Q15 0.70 0.07 0.20 0.00 0.03    0
Q16 0.83 0.07 0.07 0.00 0.03    0
Q19 0.73 0.23 0.00 0.03 0.00    0
Q20 0.93 0.07 0.00 0.00 0.00    0
Q21 0.77 0.20 0.03 0.00 0.00    0
Q22 0.37 0.30 0.20 0.10 0.03    0
Q24 0.00 0.00 0.03 0.03 0.93    0
Q25 0.00 0.13 0.77 0.10 0.00    0
There were 22 warnings (use warnings() to see them)

Warning messages:
1: In psych::alpha(d, check.keys = TRUE) :
  Item = Q8 had no variance and was deleted
2: In cor.smooth(r) : Matrix was not positive definite, smoothing was done
3: In psych::alpha(d, check.keys = TRUE) :
  Some items were negatively correlated with total scale and were automatically reversed.
 This is indicated by a negative sign for the variable name.
4: In cor.smooth(R) : Matrix was not positive definite, smoothing was done
5: In cor.smooth(R) : Matrix was not positive definite, smoothing was done
6: In cor.smooth(R) : Matrix was not positive definite, smoothing was done
7: In cor.smooth(R) : Matrix was not positive definite, smoothing was done
8: In cor.smooth(R) : Matrix was not positive definite, smoothing was done
9: In cor.smooth(R) : Matrix was not positive definite, smoothing was done
10: In cor.smooth(R) : Matrix was not positive definite, smoothing was done
11: In cor.smooth(R) : Matrix was not positive definite, smoothing was done
12: In cor.smooth(R) : Matrix was not positive definite, smoothing was done
```

Appendix 14: Results 22 25-PRIMER Questions AFTER ('r' calculations on Cronbach)

13: In cor.smooth(R) : Matrix was not positive definite, smoothing was done
14: In cor.smooth(R) : Matrix was not positive definite, smoothing was done
15: In cor.smooth(R) : Matrix was not positive definite, smoothing was done
16: In cor.smooth(R) : Matrix was not positive definite, smoothing was done
17: In cor.smooth(R) : Matrix was not positive definite, smoothing was done
18: In cor.smooth(R) : Matrix was not positive definite, smoothing was done
19: In cor.smooth(R) : Matrix was not positive definite, smoothing was done
20: In cor.smooth(R) : Matrix was not positive definite, smoothing was done
21: In cor.smooth(R) : Matrix was not positive definite, smoothing was done
22: In cor.smooth(R) : Matrix was not positive definite, smoothing was done

Appendix 15: Results 22 25-PRIMER Questions BEFORE ('r' calculations on Cronbach)

```
Sourcing: C:\Shtats\BBQBreakfast\BBQBreakfast\BBQBreakfast\script.R
Rows Read: 30, Total Rows Processed: 30, Total Chunk Time: 0.010 seconds
'data.frame':    30 obs. of  22 variables:
 $ Q1 : num  2 4 3 2 4 4 2 2 3 2 ...
 $ Q2 : num  4 3 3 3 4 4 3 2 3 3 ...
 $ Q3 : num  1 1 1 1 1 2 1 2 2 1 ...
 $ Q4 : num  1 1 1 1 1 3 1 1 3 1 ...
 $ Q5 : num  2 3 3 1 3 2 2 2 2 2 ...
 $ Q6 : num  0 3 4 0 4 0 0 3 2 0 ...
 $ Q7 : num  2 0 2 1 3 3 0 1 3 0 ...
 $ Q8 : num  0 1 0 0 0 2 0 0 0 0 ...
 $ Q9 : num  0 1 0 0 2 2 0 0 2 0 ...
 $ Q10: num  0 0 0 0 0 0 0 0 0 0 ...
 $ Q11: num  0 0 0 0 0 0 0 0 0 0 ...
 $ Q12: num  0 3 3 0 3 4 0 2 1 0 ...
 $ Q13: num  0 0 1 0 3 3 0 1 1 0 ...
 $ Q14: num  1 1 3 1 3 3 1 2 2 1 ...
 $ Q15: num  2 0 1 3 3 3 0 0 2 0 ...
 $ Q16: num  0 0 0 0 3 3 0 0 0 0 ...
 $ Q19: num  1 3 1 1 1 2 1 0 2 1 ...
 $ Q20: num  0 2 1 0 2 1 0 0 1 0 ...
 $ Q21: num  1 2 3 0 2 2 1 0 1 0 ...
 $ Q22: num  2 3 2 4 3 4 2 2 3 0 ...
 $ Q24: num  0 1 1 2 0 0 3 1 2 3 ...
 $ Q25: num  2 2 2 2 2 2 2 2 2 2 ...

Reliability analysis
Call: psych::alpha(x = d, check.keys = TRUE)

 raw_alpha std.alpha G6(smc) average_r S/N   ase mean   sd
       0.9      0.89    0.99      0.29 8.4 0.024  1.4 0.54

 lower alpha upper     95% confidence boundaries
0.85 0.9 0.94

 Reliability if an item is dropped:
     raw_alpha std.alpha G6(smc) average_r S/N alpha se
Q1        0.89      0.89    0.99      0.28 7.8    0.025
Q2        0.89      0.89    0.99      0.28 8.0    0.024
Q3        0.90      0.89    0.99      0.30 8.5    0.024
Q4        0.90      0.89    0.99      0.29 8.3    0.023
Q5        0.90      0.90    0.99      0.30 8.6    0.023
Q6        0.89      0.89    0.98      0.28 8.0    0.025
Q7        0.89      0.88    0.99      0.28 7.6    0.026
Q8        0.89      0.89    0.98      0.28 7.9    0.024
Q9        0.89      0.89    0.98      0.28 7.9    0.025
Q11       0.90      0.90    0.99      0.30 8.7    0.024
Q12       0.88      0.88    0.98      0.27 7.4    0.028
Q13       0.88      0.88    0.98      0.27 7.5    0.027
Q14       0.89      0.89    0.99      0.28 7.8    0.026
Q15       0.89      0.89    0.99      0.28 7.8    0.025
Q16       0.89      0.89    0.98      0.28 7.9    0.025
Q19       0.89      0.89    0.99      0.29 8.3    0.024
Q20       0.89      0.89    0.98      0.28 7.7    0.025
Q21       0.89      0.89    0.99      0.29 8.1    0.024
Q22       0.89      0.89    0.99      0.28 7.9    0.025
Q24-      0.89      0.89    0.99      0.28 7.7    0.026
Q25-      0.90      0.90    0.99      0.31 9.0    0.023

 Item statistics
     n raw.r std.r r.cor r.drop  mean   sd
Q1  30 0.621  0.67  0.67  0.576 2.500 0.78
```

Appendix 15: Results 22 25-PRIMER Questions BEFORE ('r' calculations on Cronbach)

```
Q2   30 0.532  0.58  0.56  0.494 3.067 0.58
Q3   30 0.286  0.38  0.37  0.245 1.033 0.49
Q4   30 0.450  0.45  0.45  0.373 1.633 1.03
Q5   30 0.230  0.32  0.32  0.169 1.900 0.71
Q6   30 0.665  0.59  0.59  0.581 1.300 1.51
Q7   30 0.755  0.74  0.73  0.696 1.267 1.36
Q8   30 0.545  0.60  0.60  0.517 0.133 0.43
Q9   30 0.574  0.60  0.60  0.534 0.267 0.64
Q11  30 0.234  0.29  0.29  0.212 0.067 0.25
Q12  30 0.847  0.83  0.83  0.805 1.067 1.41
Q13  30 0.808  0.77  0.78  0.772 0.833 1.05
Q14  30 0.719  0.65  0.65  0.673 1.767 0.97
Q15  30 0.689  0.65  0.65  0.622 1.167 1.29
Q16  30 0.641  0.62  0.62  0.580 0.533 1.07
Q19  30 0.448  0.45  0.45  0.392 1.367 0.76
Q20  30 0.666  0.68  0.68  0.631 0.367 0.67
Q21  30 0.538  0.51  0.51  0.474 0.600 0.97
Q22  30 0.679  0.63  0.62  0.627 2.567 1.01
Q24- 30 0.706  0.69  0.68  0.652 3.367 1.10
Q25- 30 0.095  0.19  0.18  0.042 2.900 0.61

Non missing response frequency for each item
       0    1    2    3    4    5 miss
Q1  0.00 0.00 0.67 0.17 0.17 0.00    0
Q2  0.00 0.00 0.13 0.67 0.20 0.00    0
Q3  0.10 0.77 0.13 0.00 0.00 0.00    0
Q4  0.03 0.60 0.13 0.17 0.07 0.00    0
Q5  0.07 0.10 0.70 0.13 0.00 0.00    0
Q6  0.53 0.03 0.10 0.27 0.07 0.00    0
Q7  0.47 0.10 0.17 0.23 0.03 0.00    0
Q8  0.90 0.07 0.03 0.00 0.00 0.00    0
Q9  0.83 0.07 0.10 0.00 0.00 0.00    0
Q11 0.93 0.07 0.00 0.00 0.00 0.00    0
Q12 0.57 0.10 0.10 0.17 0.07 0.00    0
Q13 0.53 0.20 0.17 0.10 0.00 0.00    0
Q14 0.00 0.57 0.13 0.27 0.03 0.00    0
Q15 0.47 0.13 0.20 0.17 0.03 0.00    0
Q16 0.77 0.07 0.03 0.13 0.00 0.00    0
Q19 0.03 0.70 0.13 0.13 0.00 0.00    0
Q20 0.73 0.17 0.10 0.00 0.00 0.00    0
Q21 0.67 0.13 0.13 0.07 0.00 0.00    0
Q22 0.03 0.07 0.40 0.30 0.20 0.00    0
Q24 0.17 0.30 0.30 0.20 0.03 0.00    0
Q25 0.00 0.03 0.90 0.03 0.00 0.03    0
Warning messages:
1:
In psych::alpha(d, check.keys = TRUE) :

 Item = Q10 had no variance and was deleted
2:
In psych::alpha(d, check.keys = TRUE) :

Some items were negatively correlated with total scale and were automatically reversed.
This is indicated by a negative sign for the variable name.
```

Appendix 16: Results ('r' script with automated adjustments, Cronbach's Alpha)

Results

R. script with AUTOMATED adjustments

Some items were negatively correlated with total scale and were automatically reversed.
This is indicated by a negative sign for the variable name.
(i.e. Reverse Scoring and Deletion if Variance =0:
https://drive.google.com/open?id=1DV5fJH6aNo5nV74YuYhe8HrXzr70lBXvGs3u6bX_AAY

Results 22 (25-PRIMER Q17,18,23) Questions BEFORE

Cronbach's α: **0.9**

Q24- 0.89 0.89 0.99 0.28 7.7 0.026
Q25- 0.90 0.90 0.99 0.31 9.0 0.023
Item = Q10 had no variance and was deleted

Results 22 (25-PRIMER Q17,18,23)) Questions AFTER

Cronbach's α: **0.89**

Q24- 30 0.49 0.64 0.64 0.451 0.100 0.40
Item = Q8 had no variance and was deleted

Cronbach's alpha calculated in Excel

Results 22 (25-PRIMER Q17,18,23) Questions BEFORE

Cronbach's alpha	**0.847219212**

Results 22 (25-PRIMER Q17,18,23)) Questions AFTER

Cronbach's alpha	**0.8779182048**

Appendix 17: R Script to Get Data from CSV and run Cronbach's Alpha

```
#Set path to .csv file
dataFile <- file.path("C:\Stats", "25Questions30Before30After.csv")
#dataFile <- file.path("C:\Stats", "25Questions30After.csv")
#dataFile <- file.path("C:\Stats", "25Questions30Before.csv")

#Map fields to DTS object
inTextData <- RxTextData(file = dataFile, colClasses = c(
        "Q1" = "numeric",
        "Q2" = "numeric",
        "Q3" = "numeric",
        "Q4" = "numeric",
        "Q5" = "numeric",
        "Q6" = "numeric",
        "Q7" = "numeric",
        "Q8" = "numeric",
        "Q9" = "numeric",
        "Q10" = "numeric",
        "Q11" = "numeric",
        "Q12" = "numeric",
        "Q13" = "numeric",
        "Q14" = "numeric",
        "Q15" = "numeric",
        "Q16" = "numeric",
        "Q17" = "numeric",
        "Q18" = "numeric",
        "Q19" = "numeric",
        "Q20" = "numeric",
        "Q21" = "numeric",
        "Q22" = "numeric",
        "Q23" = "numeric",
        "Q24" = "numeric",
        "Q25" = "numeric"

    ))

#Import data from file
d <- rxDataStep(inData = inTextData, outFile = NULL)

#Echo data
str(d)

#Run Cronbach Alpha on data, remove keys automatic
psych::alpha(d, check.keys = TRUE)
```

Appendix 18: T-TEST_YFAS_ExcelFormula_AKiste_301

Study ID	date	before/ after	age	gender	weight	height	waist	hip	BMI	WTH	1. I find that when I start eating certain foods, I end up eating much more than planned.	2. I find myself continuing to consume certain foods even though I am no longer hungry.	3. I eat to the point where I feel physically ill.	4. Not eating certain types of food or cutting down on certain types of food is something I worry about.	5. I spend a lot of time feeling sluggish or fatigued from overeating.	6. I find myself constantly eating certain foods throughout the day.	7. I find that when certain foods are not available, I will go out of my way to obtain them. For example, I will drive to the store to purchase certain foods even though I have other options available to me at home.	8. 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	9. 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
		TTEST1 tail									1,54409E-13	8,16902E-15	1,67437E-12	4,57987E-08	2,8119E-08	0,000182541	0,000712914	0,051649855	0,016147084
		TTEST2 tail									7,5837E-13	7,26973E-14	1,23121E-11	1,99817E-07	1,54697E-07	0,000347415	0,002745077	0,103323298	0,032195315
		Cohen's D																	

10. There have been times when I avoided professional or social situations where certain foods were available because I was afraid I would overeat.	11. There have been times when I avoided professional or social situations because I was not able to consume certain foods there.	12. 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	13. I have consumed certain foods to prevent feelings of anxiety, agitation, or other physical symptoms that were developing. (Please do NOT include consumption of caffeinated beverages such as soda pop,	14. I have found that I have elevated desire for or urges to consume certain foods when I cut down or stop eating them.	15. My behavior with respect to food and eating causes significant distress.	16. I experience significant problems in my ability to function effectively (daily routine, job/school, social activities, family activities, health difficulties) because of food and eating.	17. My food consumption has caused significant psychological problems such as depression, anxiety, self-loathing, or guilt.	18. My food consumption has caused significant physical problems or made a physical problem worse.	19. I kept consuming the same types of food or the same amount of food even though I was having emotional and/or physical problems.	20. 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.	21. 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.
0,162790994	0,00697784	0,000822724	0,000161042	2,58153E-06	0,000345146	0,148287361	0,002726188	0,000200032	2,09417E-10	0,008686178	0,028653643
0,325874707	0,024062241	0,00160144	0,000305815	9,39332E-06	0,001385179	0,296848425	0,005374233	0,000381309	1,17523E-09	0,017266773	0,083060605

22. I want to cut down or stop eating certain kinds of food.	23. I have tried to cut down or stop eating certain kinds of food.	24. I have been successful at cutting down or not eating these kinds of food.	25. How many times in the past year did you try to cut down or stop eating certain foods altogether?	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	1. Substance taken in larger amount and for longer period than intended	Met?
6,29043E-10	2,02155E-08	1,56241E-11	0,190036457	0,011	0,006	#####	0,08	0,092	0,286	0,008	0,163	#####	0,163	0,003	0,003	0,042	0,008	0,011	0,09	#####	#####	0	0	0,05	0	#####	0,01	#####	0,001401576	#####
3,94516E-09	9,73302E-08	7,70175E-11	0,38039017	0,023	0,023	#####	0,161	0,184	0,573	0,017	0,326	#####	0,326	0,012	0,006	0,083	0,017	0,023	0,18	#####	#####	0	0,01	0,16	0	#####	0,04	#####	0,005280641	#####

Criterion Score	2. Persistent desire or repeated unsuccessful attempts at quitting	Met?	Criterion Score	3. Much time/activity to obtain, use, recover	Met?	Criterion Score	4. Important social, occupational, or recreational activities given up or reduced	Met?	Criterion Score	5. Use continues despite knowledge of adverse consequences (e.g., failure to fulfill role obligation, use when physically hazardous	Met?	Criterion Score	6. Tolerance (marked increase in amount; marked decrease in effect)	Met?	Criterion Score	7. Characteristic withdrawal symptoms; substance taken to relieve withdrawal	Met?	Criterion Score	8. Use causes clinically significant impairment	Met?	Criterion Score	Symptom Count	High FA Met
0,000713	4,1607E-11	#####	0	0,012554123	#####	0,00445	0,00843458	#####	0,005827	2,09417E-10	#####	3,4E-08	0,00956157	#####	0,09207	0,00085166	#DIV/0!	0,00071	0,02169871	#####	0,0058274	1,8552E-11	0,00146965
0,00288	2,0263E-10	#####	0	0,025003454	#####	0,00881	0,03125186	#####	0,022503	1,17523E-09	#####	1,9E-07	0,02668902	#####	0,32587	0,00165848	#DIV/0!	0,00139	0,04331307	#####	0,01155545	0,05490624	0,00583231

Appendix 19: McNemar Calculation

Study ID	before/after % von 30	Criterion1 #REF!	Criterion2 #REF!	Criterion3 #REF!	Criterion4 #REF!	Criterion5 #REF!	Criterion6 #REF!	Criterion7 #REF!	Criterion8 #REF!	Study ID	before/ after	Criterion1	Criterion2	Criterion3	Criterion4	Criterion5	Criterion6	Criterion7	Criterion8
1 after		0	0	0	1	0	0	0	0		countifafter	0	0	0	0	0	0	0	0
2 after		0	1	0	0	1	1	0	0										
3 after		0	1	0	1	0	0	0	0			0	0	0	0	0	0	0	0
4 after		0	1	0	0	0	0	0	0										
5 after		0	1	0	1	0	1	0	0										
6 after		0	1	0	0	1	0	0	0										
7 after		0	0	0	0	0	0	0	0										
8 after		0	0	0	0	0	1	0	0										
9 after		0	1	0	0	1	0	0	0										
10 after		0	0	0	0	0	0	0	0										
11 after		0	0	0	0	0	0	0	0										
12 after		0	1	0	0	1	0	0	0										
13 after		0	1	0	0	1	0	0	0										
14 after		0	1	0	0	0	1	0	0										
15 after		0	1	1	0	0	0	0	0										
16 after		0	1	0	1	0	0	1	0										
17 after		0	0	0	0	0	0	0	0										
18 after		0	0	0	0	0	0	0	0										
19 after		0	1	0	0	0	0	0	0										
20 after		0	1	0	0	1	0	0	0										
21 after		0	0	0	0	0	0	0	0										
22 after		0	0	0	0	0	0	0	0										
23 after		0	1	0	0	1	0	0	0										
24 after		0	0	0	0	0	0	0	0										
25 after		0	0	0	0	0	0	0	0										
26 after		0	1	0	0	0	0	0	0										
27 after		0	1	0	0	0	0	0	0										
28 after		0	1	0	0	0	0	0	0										
29 after		0	1	0	0	0	0	0	0										
30 after		0	1	0	0	1	0	0	0										
1 before		1	1	0	0	1	1	0	0										
2 before		1	1	1	0	0	1	1	0										
3 before		0	1	1	0	1	1	1	0										
4 before		0	1	0	0	1	0	0	1										
5 before		1	1	1	0	1	1	1	1										
6 before		1	1	1	1	1	1	1	1										
7 before		0	1	0	0	1	1	0	0										
8 before		0	1	0	0	0	0	0	0										
9 before		0	1	1	0	1	0	0	0										
10 before		0	0	0	0	1	0	0	0										
11 before		0	1	0	0	1	0	0	0										
12 before		0	1	0	0	1	0	0	1										
13 before		1	1	1	0	1	0	1	0										
14 before		0	1	1	0	1	0	0	0										
15 before		0	1	0	0	1	0	1	0										
16 before		1	1	1	0	1	0	1	0										
17 before		0	1	0	0	1	0	0	0										
18 before		0	1	0	0	1	0	0	0										
19 before		0	1	0	0	1	0	0	0										
20 before		1	1	1	0	1	1	1	1										
21 before		0	1	0	0	1	0	0	0										
22 before		0	1	0	0	1	0	0	0										
23 before		1	1	0	0	1	1	1	0										
24 before		0	1	0	0	1	0	0	0										
25 before		0	1	0	0	1	0	0	0										
26 before		0	1	0	0	1	1	0	1										
27 before		0	1	1	0	1	0	1	1										
28 before		0	1	0	0	1	0	0	0										
29 before		0	1	0	0	1	0	0	0										
30 before		1	1	0	0	1	0	1	0										
countifbefore		9	29	10	1	29	11	11	7										
			19	2															

Appendix 19: McNemar Calculation Results

Subtest/ criterion	McNemar's chi-squared	df	p-value
criterion1	7,111	1	0,0077
criterion2	8,1	1	0,004427
criterion3	4,9	1	0,02686
criterion4	4,1667	1	0,04123
criterion5	17,391	1	3,04E-05
criterion6	0,8	1	0,3711
criterion7	7,1111	1	0,007661
criterion8	4,1667	1	0,04123