Protection, risk and dieting: Intermittent fasting diets and disordered eating

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Thesis declaration form

I confirm that the work presented in this thesis is my own. Where information has been derived from other sources, I confirm that this has been indicated in the thesis.

Signature:

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Overview

Consideration of factors and behaviours which may increase the risk of disordered eating, or protect against these difficulties and promote resilience, can inform efforts to prevent and intervene.

Part One of this thesis is a systematic review of research into protective factors against eating disorders and disordered eating in proximal social systems. A range of potential protective factors in families, schools, peer groups and neighbourhoods are identified. Many of these factors may be non-specific to eating difficulties, promoting a range of positive outcomes, while others may be more specific to disordered eating. Methodological issues in the literature which limit the ability to draw firm conclusions are discussed.

Part Two presents empirical research into the impact of intermittent fasting (IF) diets on eating psychopathology, binge eating, food craving and mood. Contrary to expectation, starting a 5:2 IF diet did not result in increases in disordered eating or binge eating in healthy adult dieters, and in fact appeared to result in improvements in all outcomes. Higher scores on measures of risk factors for eating disorders at baseline were associated with greater reductions in disordered and binge-eating over the 28 day IF period. Limitations to interpretation of results are considered, along with potential clinical applications and suggestions for further research.

Part Three presents a critical appraisal of the literature review and empirical paper. Assumptions informing the research questions, aspects of the research process, and potential interpretations and implications of the findings are considered, with reference to the perspective of the scientist practitioner.

The empirical research in Part Two was completed as part of a joint research project. The details of the other part of this project can be found in Mahony, K. (2016). Nutrition and cognition: Exploring their relationship from two sides of the same coin. Clinical Psychology Doctorate Thesis.

Table of Contents

List of Tables

Part 1: Literature Review

Part 2: Empirical Paper

List of Figures

Part 1: Literature Review

Part 2: Empirical Paper

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Part 1: Literature Review

Systems of protection:
Protective factors against disordered eating
in the proximal social environment

Abstract

Aims: This review aims to identify and evaluate the literature investigating protective factors and eating disorders, to establish what is known about factors in proximal social systems (family, peer group, school and neighbourhood) that could be considered protective against the development of eating disorders/ eating pathology.

Methods: A systematic review of the literature was conducted on five databases, using search terms related to eating disorders/disordered eating and protective factors. Studies were systematically screened for inclusion, and all included studies were evaluated for study quality.

Results: Thirty-three studies met criteria for inclusion. The majority of studies looked at factors within families, including aspects of family relationships and family practices around food or eating. There was a particular research focus on the potential protective role of regular family meals. Potential protective factors also appear to exist at the level of schools, peer groups and neighbourhoods/communities. There were weaknesses in the methodology used in many studies, in particular an over-reliance on cross-sectional correlational methods.

Conclusions: Many of the potential protective factors identified, such as social support and family connectedness, may be non-specific to eating difficulties, promoting general adaptive development and a range of positive development outcomes. Factors in the family environment around food, eating and weight, such as frequent family meals and avoiding comments about weight, may be more specific to eating disorders and disordered eating. Issues with the methodologies used severely impact on the ability to draw conclusions about whether factors are 'protective'.

1.0 Introduction

Eating disorders, including anorexia nervosa (AN), bulimia nervosa (BN), binge eating disorder (BED), and those 'not otherwise specified' (ED-NOS), are serious mental health conditions associated with a range of negative physical, psychological and social outcomes, and with the highest mortality rates of any psychiatric disorder. Worryingly, the rate of new diagnoses of eating disorders appears to be growing over time, with the highest incidence found amongst girls and young women aged ten to nineteen years old (Micali, Hagberg, Petersen, & Treasure, 2013). Sub-clinical 'disordered eating' or 'eating pathology' affects a large proportion of the population, with some studies finding that up to 20% of young women report the use of disordered eating behaviours such as using diet pills, vomiting or laxatives to manage their weight (Austin, 2000) and as many as 25% meet the criteria for 'probable disordered eating' on various measures . This represents a major public health concern, and there is growing recognition of the need to understand the factors influencing the development of these difficulties in order to inform efforts to prevent them (Neumark-Sztainer, 2011). A large body of evidence now exists on risk factors for eating disorders at the biological, psychological, social, and cultural levels (for example see Steiner et al., 2003, Stice, 2002 for reviews). There has also been a move towards trying to identify 'protective factors', perhaps linked to a growing focus on 'resilience' and 'positive psychology' in clinical psychology as a whole (Masten, Best, & Garmezy, 1991; Seligman & Csikszentmihalyi, 2000; Steck, Abrams, & Phelps, 2004). It has been suggested that a focus on protective factors and building strengths, rather than on reducing not fully developed risks, may be particularly useful in designing universal prevention programmes for young people (Levine & Smolak, 2016).

In recent years a range of eating disorder prevention programmes have been developed and evaluated, with some success (Stice, 1999; Stice, Shaw & Marti, 2007). These interventions tend to intervene at the level of the individual to address risk, protective and mediating factors, for example by increasing self-esteem, body satisfaction or media literacy. Far fewer prevention

programmes have been developed to intervene at different ecological levels, such as the level of the family, peer group, school, or wider social and cultural systems, despite the known importance of considering these factors in the etiology of eating disorders and other difficulties, and evidence of the importance of involving families in treatment (Austin, 2000; Levine & Smolak, 2016). Consideration of protective factors at the proximal social level may present new opportunities and inform efforts to prevent the onset of disordered eating, by promoting processes and practices which protect against those outcomes (Neumark-Sztainer, 2011; Steck et al., 2004).

1.1 Protective factors

The term 'protective factor' has been defined in different ways dependent on the theoretical framework in use. The term refers to more than simply the absence of risk factors (Steck et al., 2004; Levine & Smolak, 2016). Within the developmental psychopathology framework, a protective factor is something which moderates the effect of a vulnerability or risk factor on development, promoting adaptive development and 'resilience' (the capacity for positive outcomes despite challenging circumstances) (Masten et al., 1991; Rutter, 1987). Theorists from the 'positive psychology' movement have argued that the dominant paradigm focus on risk factors and pathology has resulted in neglect of strengths and positive aspects of individuals and systems (Seligman & Csikszentmihalyi, 2000; Steck et al., 2004). They encourage the investigation of protective factors, those which promote health and wellbeing, and suggest that prevention programmes should aim to develop and foster these. These ideas have informed the concept of 'developmental assets', a range of contextual (family, peer, school, neighbourhood) and individual factors which form a set of 'building blocks' for successful development and positive outcomes. The presence of these developmental assets is thought to protect against the initiation of a range of health risk behaviours (French et al., 2001). Indeed, many protective factors are thought to be non-specific, reducing the probability of the onset of a

range of difficulties (Levine & Smolak, 2016). Despite this, protective factors are not necessarily universal, and what constitutes a protective factor will vary depending on gender, social class, ethnicity, age, and other variables.

Kraemer et al. (1997), approaching the issue from a psychiatric perspective, argue that there is a need for more precision in the definition and use of the terms 'risk factor' and 'protective factor'. They defined protective factors as "factors that either identify subjects at lower risk for the disorder or a higher probability of welcome outcomes". Key to their definition is the idea of establishing precedence, without which a factor can only be considered a 'correlate', and may well be a 'concomitant' or a 'consequence'. They suggest a five-point process in identifying a risk/protective factor: clearly define and validly and reliably measure the outcome; define and properly sample the population; define the risk factor, establish precedence and measure it properly; use analyses which allow the definition of high/low risk groups and show a statistically significant difference between the likelihood of the outcome in each; and finally use analyses that give an estimate of the potency of this association, to establish that the difference between high and low risk groups is clinically significant. Their 'typography of risk' also distinguishes between fixed factors (which cannot change), variable risk/protective factors (which vary over time spontaneously or can be modified) and causal risk/protective factors (which are manipulable, and change the likelihood of an outcome when modified). They suggest that 'causal factors' may be of interest to clinicians, but are clear to differentiate them from a 'cause', highlighting the probabilistic nature of outcomes, the likely complex interaction of many factors, and the possibility of multiple pathways to the same outcome. This definition has clear implications for study design, and they have pointed out that the preponderance of cross-sectional and retrospective designs in the psychiatric literature means that many factors named as 'risk' or 'protective' factors should in fact only be considered 'correlates', limiting their clinical or theoretical utility.

1.2 Proximal social influences and eating disorders

Bronfenbrenner's (1979) ecological model highlights the importance of different levels of social context in understanding a person's development and behaviour. Theorists in the eating disorder field have highlighted the core role of the socio-cultural environment in the aetiology of eating disorders, while clinicians working to treat these disorders have emphasised the importance of considering and working with the family system in formulation and intervention (see Austin, 2000). Large numbers of studies have aimed to elucidate the risk factors for the development of eating disorders at different levels of influence, and there is widespread recognition of the role of the immediate, proximal social environment, families, peers, schools and local communities, (termed the 'microsystem' and 'mesosystem' in Bronfenbrenner's model) in the aetiology of eating pathology (Neumark-Sztainer, 2011; Steiner et al., 2003). For example, parental and peer weight related teasing and parental encouragement to diet have been associated with increased risk of disordered eating (Rodgers & Chabrol, 2009; Sweetingham & Waller, 2008).

Protective factors also occur across multiple ecological levels, including at the level of the family system, community and culture (Levine and Smolak, 2016). However the vast majority of prevention programmes intervene at the level of the individual, for example by teaching media literacy, promoting body acceptance or inducing 'cognitive dissonance' (Stice et al., 2007; Shaw, Stice, & Becker, 2009). This may represent a missed opportunity to intervene at different levels of influence (Austin, 2000). Understanding more about protective factors at the proximal social level, which promote adaptive development and reduce the likelihood of developing eating pathology, may open up possibilities to develop prevention approaches which intervene at the level of the family, peer group, school or neighbourhood (Neumark-Sztainer, 2011; Steck et al., 2004). Studies have begun to identify potential protective factors in social systems, for example in relation to family relationships, family meal practices, or school environments (French et al., 2001; Lampis, Agus, & Cacciarru, 2014; Neumark-Sztainer, Wall, Story, & Fulkerson, 2004;

Perkins, Luster, & Jank, 2002). Increased awareness of these factors is likely to be useful not just to clinicians but also to families, schools and communities hoping to promote adaptive development and help to prevent eating disorders and disordered eating difficulties.

1.3 Aims

While a number of studies have identified potential protective factors for eating pathology in families, peer groups, schools and neighbourhoods, no cohesive picture of research into these factors has been developed currently. Reviewing this literature to create a 'roadmap' of key findings, areas of strength, and limitations, could be of relevance to prevention efforts at the social systems level, and would allow the identification of opportunities for further research. This review aims to systematically identify and evaluate studies with a view to answering the following questions:

1. Which factors in the proximal social environment (family, peer group, school and neighbourhood) can be considered protective against the development of eating disorders/ eating pathology?
2. What are the strengths and limitations of the literature in this field?

2.0 Methods

2.1 Inclusion criteria

Studies were included in the review if they met the following criteria:

1. Included an outcome measure which assessed eating disorder symptoms/ eating pathology/ disordered eating behaviours.
2. Identified and measured a potential protective factor in the proximal social environment, defined as the family, peer-group, school or neighbourhood.
3. Not an evaluation of a prevention programme/ intervention for disordered eating.
4. Only quantitative studies in peer-reviewed journals which could be accessed in the English language were included.

2.2 Systematic search protocol

The databases Psychinfo, Pubmed, Embase, CINAHL and Web of Science Core Collection were searched for the following terms: "eating disorder*", "disordered eating", "anorexi*", "bulimi*", "binge eating", "binge-eating", "EDNOS" or "ED-NOS"; and "protective factor", "protective" or "resilienc*". Subject heading searches ("Eating disorders" and "protective factors") were also used where available. Results were limited to journal articles and those in the English language. No limitations on the year of publication were applied.

Once duplicates had been removed, titles and abstracts were screened to exclude those obviously not relevant to the inclusion criteria. Remaining papers were accessed and read in full, with a two stage process used to identify papers to include in the final review. At Stage One, papers including an appropriate outcome measure and the measurement/identification of any potential protective factor were included. Reviewing reference lists of papers at this stage lead to the identification of two additional papers potentially meeting these inclusion criteria. The level of influence of protective factors measured/ identified in each paper was noted in a table, coded as individual/ family/ peer/ school/ neighbourhood/socio-cultural. At Stage Two, papers measuring/identifying a protective factor at the level of the proximal social environment (family, peer group, school, neighbourhood) were identified for inclusion in the final review.

2.3 Quality assessment and synthesis

Included papers were read to identify key findings and methodological issues. The Standard Quality Assessment Criteria (QualSyst) from the Alberta Heritage Foundation for Medical Research was used to appraise study quality (Kmet, Lee, & Cook, 2004; see Appendix A). This critical appraisal tool was designed to provide health researchers with a standardised means to assess the quality of studies with varying designs. It includes fourteen criteria, for which each study can score two points if the criterion is fully met, one point for partially met, zero points if not met, or 'not applicable' if the criterion cannot be applied appropriately to the study

design. A final 'score' for each study is given by dividing the total score by the total possible score for all applicable items. Due to the wide variation of study designs, outcomes and methods of analyses, it was decided that a narrative synthesis would be most appropriate to present the key findings and methodological issues indentified in the review.

3.0 Results

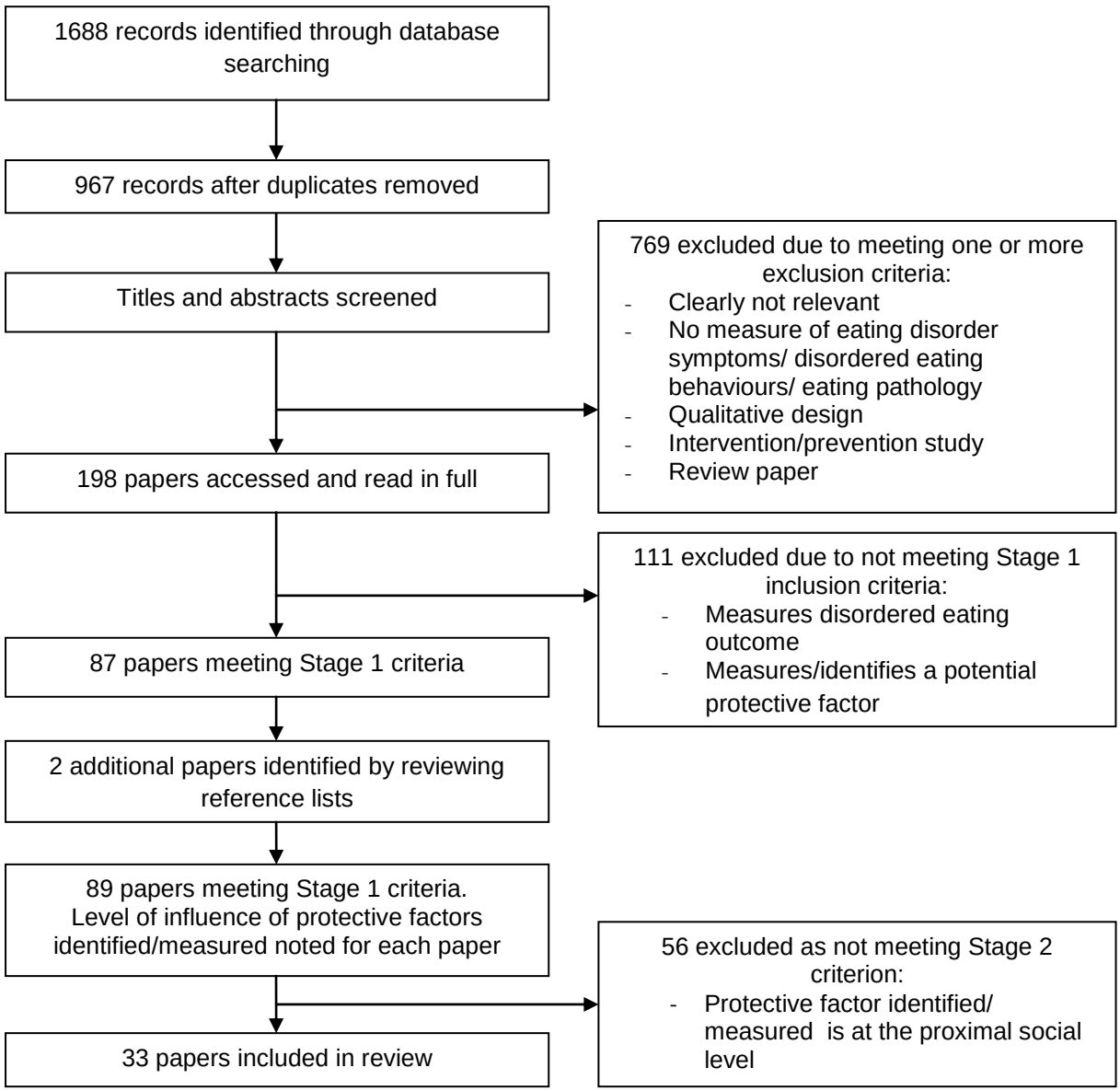
Database searching identified 967 papers for possible inclusion, once duplicates had been removed. Following review using the search protocol outlined above, 33 papers were identified as meeting the inclusion criteria and were included in the review. Full details of the number of papers included or excluded at each stage of the search protocol can be seen in Figure 1.

3.1 Study characteristics

Participants and samples: Participants in the studies spanned an age range from 10 (Allen et al., 2014) to 81 years of age (Bertoli et al., 2015) at the time of outcome measurement. 24 papers reported mean age of participants, with means ranging from 12.76 (Allen et al., 2014) to 30 years of age (Nicholls & Viner, 2009) at baseline. Twenty-five of the papers used primarily or exclusively child and/or adolescent participants, taking data from sixteen participant samples. Seven of the papers were drawn from the same 'Project EAT' sample (Berge et al., 2013, 2014; Loth et al., 2015; Neumark-Sztainer et al., 2007; Neumark-Sztainer, Eisenberg, Fulkerson, Story, & Larson, 2008; Neumark-Sztainer et al., 2004; Neumark-Sztainer, Wall, Story, & Sherwood, 2009) while two papers were written about the Growing Up Today cohort (Haines, Gillman, Rifas-Shiman, Field, & Austin, 2009; Haines, Kleinman, Rifas-Shiman, Field, & Bryn Austin, 2010), two were written about the same USA-wide school-based sample (French et al., 2001; Fulkerson et al., 2006), and two used samples drawn from the same population of children whose parents misused substances (Chandy, Blum, & Resnick, 1995; Chandy, Harris, Blum, & Resnick, 1994). Eight of the papers used adult samples. Full details of the participant groups in

each study, along with study designs and outcome measures used, are included in Table 2 (all ages are given for baseline).

Figure 1: Diagram of systematic search protocol with studies excluded at each stage



Sample sizes ranged from 93 participants (Kunstman, Smith, & Maner, 2014).) to 249,894 participants (Ahrén et al., 2013). Fifteen of the papers used normative samples of school children (making use of seven different samples), six papers made use of birth cohorts (from five cohorts), five used university student or alumni samples (one of which only included students reporting symptoms of AN), three studied adolescents identified as 'at-risk' due to abuse or

substance abuse in the family home (from two different study populations), while one used a sample considered 'at risk' due to having experienced intimate partner violence. Two studied samples of 'elite aesthetic athletes', and one used a sample from a weight loss clinic.

Designs: Twenty-three studies made use of cross-sectional designs, with potential protective factors and disordered eating outcomes measured concurrently. Seven papers made use of longitudinal designs, exploring associations between potential protective and risk factor measures at baseline and eating pathology measures two- to five- years later. The majority of the studies used child report, or retrospective reports for studies with adult populations, to measure family and peer relationship variables, although three studies also included measures completed by parents (Allen et al., 2014; Berge et al., 2013; Loth et al., 2015). Two studies used prospective cohort designs, following a cohort of participants over time to identify cases of eating disorders and looking for predictors of positive or negative outcomes from earlier time points (Ahrén et al., 2013; Nicholls & Viner, 2009). One study made use of a randomised experimental design (Kunstman et al., 2014).

Outcome measures: A range of measures were used to assess disordered eating in the studies. The details of the outcome measures used in each study are included in Table 2. Two studies identified diagnosed cases of eating disorders. Ahren et al. (2013) looked at health records for relevant diagnostic codes or evidence of having received treatment for any eating disorder. Nicholls and Viner (2009) asked participants if they had ever received a diagnosis of AN. Kunstman et al. (2014) was the only study to use a behavioural measure, measuring the amount consumed on a test meal by participants reporting symptoms of anorexia nervosa, to compare those in the experimental condition to controls. The great majority of studies made use of self-report measures of eating pathology.

Table 1: Standardised measures of disordered eating used in studies

Measure	Abbreviation	No. of items	Factors assessed	No. of studies
Binge Eating Scale	BES	16	Binge eating behaviour Emotional and cognitive response to binge eating	1
Bulimia Test Revised	BULIT-R	36	Bulimic symptoms	2
Eating Attitudes Test /	EAT-26 /	26	Dieting	4
Child Eating Attitudes Test	ChEAT		Bulimia and food preoccupation Oral control	2
Eating Disorder Inventory	EDI-2	91 (11 subscales)	Drive for thinness Bulimia Body dissatisfaction Ineffectiveness Perfectionism Interpersonal distrust Interoceptive awareness Maturity fears Asceticism Impulse regulation Social insecurity	3
Eating Disorder Examination Questionnaire/	EDEQ	28	Restraint Eating Concern Weight Concern Shape Concern Frequency of binge eating /compensatory behaviours	1
Child Eating Disorder Examination	ChEDE	Clinical interview		1
Eating Disorder Screen for Primary Care	ESP	5	Satisfaction with eating pattern Eating in secret Impact of weight on self-concept Personal/family history of ED	1
Three Factor Eating Questionnaire	TFEQ	51	Cognitive restraint of eating Disinhibition Hunger	1

Thirteen of the studies used standardised measures of disordered eating (see Table 1 for details). Four studies used the Eating Attitudes Test (Garner, Olmsted, Bohr, & Garfinkel, 1982), with two studies using the child version of this test (Maloney, McGuire, Daniels, & Specker, 1988). Three studies made use of parts of the Eating Disorder Inventory (Garner, 1991). Other validated measures used included the Eating Disorder Examination Questionnaire (Fairburn & Beglin, 1994), the child's version of this interview (Bryant-Waugh, Cooper, Taylor, & Lask, 1996), the Bulimia Test Revised (Thelen, Farmer, Wonderlich, & Smith, 1991), the Eating Disorder Screen for Primary Care (Cotton, Ball, & Robinson, 2003), the Three Factor Eating

Questionnaire (Stunkard & Messick, 1985), and the Binge Eating Scale (Gormally, Black, Daston, & Rardin, 1982).

The rest of the studies included items asking about disordered eating behaviours in their surveys. The Project Eat survey used by seven of the studies included items asking about dieting, binge eating, 'unhealthy weight control behaviours' (WCBs; such as skipping meals, eating very little or smoking to control appetite) and 'extreme WCBs', (vomiting or use of laxatives, diuretics or diet pills to control weight), as well as asking about current body weight (Berge et al., 2013, 2014; Loth et al., 2015; Neumark-Sztainer et al., 2004, 2007, 2008, 2009). Generally, an indication of 'Yes' once or more in the past year to any of these items was taken as evidence of the presence of disordered eating. Note that such individuals may not have met criteria for a full eating disorder. A similar approach, asking for the presence of specific disordered weight control behaviours such as bingeing, purging, dieting or the use of diet pills at least once in the last year, was taken by Croll et al. (2002), Fonseca, Ireland, & Resnick (2002), French et al. (2001), Fulkerson et al. (2006) Haines et al. (2009, 2010), Perkins et al. (2002) and Wang et al. (2013). The studies by Chandy's group (Chandy et al., 1994; Chandy, Harris, Blum, & Resnick, 1995) asked about a list of seven 'disordered eating behaviours', with the presence of three or more taken to indicate disordered eating.

Table 2: Participant, sample, design, outcome measures, QualSyst score and key findings for all studies

Study	Country	Participant age (years)	Sample size	Participant gender	Participant ethnicity	Sample population	Design	Disordered eating outcome measures	Qual Syst quality score	Protective factors identified
Ackard & Neumark-Sztainer (2001)	USA	M= 20.6, SD= 3.1	560	Female	78.6% White 14.3% Black	University students	Cross sectional design	BULIT-R, EDI -2 (bulimia subscale)	.900	Family meals
Ahren, Chiesa, Koupil, Magnusson, Dalman & Goodman (2013)	Sweden	12 to 23	249894	Mixed (49% female)	Not described	Stockholm Youth Cohort	Prospective cohort study	Cases of ED	1.000	Having full siblings (for females only)
Allen, Gibson, McLean, Davis & Byrne (2014)	Australia	M=10.78 SD= 1.72	211	Mixed (54% female)	Not described	Childhood Growth and Development Cohort	Longitudinal design (2 years)	ChEDE, CARES (emotional eating)	1.000	Child satisfaction with family life
Berge, Maclehose, Loth, Eisenberg, Bucchianeri, Neumark-Sztainer (2013)*	USA	M= 14.4 SD= 2.0	2348	Mixed (53% female) (Separate analyses)	18.9% White, 29.0% Black, 19.9% Asian, 16.9%Hispanic, 3.7% Native American, 11.6% mixed/ other	School students in Project EAT (+parents)	Longitudinal study (2 years)	Dieting/ unhealthy or extreme WCBs/ binge eating in last year (Yes/No)	.900	Parent discussions around healthy eating, *Parent discussions about weight NOT protective*
Berge, Wall, Larson, Eisenberg, Loth, Neumark-Sztainer (2014)*	USA	M= 14.4 SD= 2.0	2793	Mixed (Separate analyses)	18.9% White, 29.0% Black, 19.9% Asian, 16.9%Hispanic, 3.7% Native American, 11.6% Mixed/ Other	School students in Project EAT	Cross sectional design	Dieting/ unhealthy or extreme WCBs/ binge eating in last year (Yes/No)	.950	Family functioning, Higher sense of connection with either parent, Mothers having knowledge of children's whereabouts, Father's knowledge of

										whereabouts (girls only). *Parental control NOT protective.*
Bertoli, Leone, Ponissi, Bedogni, Beggio, Strepparava & Battezzati (2015)	Italy	18- 81 Median =46	211	Mixed (72% female)	Not described	Patients at a weight loss clinic	Cross sectional design	Binge Eating Scale – Italian version	1.000	Being married
Brown & Keel (2012)	USA	M= 29.14 SD = 8.69	578	Male	76.5% Caucasian	University alumni	Cross sectional design	EDI-2 (bulimia and drive for thinness subscales)	.900	Being in a romantic relationship (homosexual men), NOT relationship satisfaction
Chandy, Harris, Blum, Resnick (1994) +	USA	M= 15.37	838	Mixed (64% female)	88% White, 8% Black, 4% Other	Children of parents who misuse substances	Cross sectional design	Disordered eating behaviours (3+)	.800	Perception of an alcohol free school environment
Chandy, Harris, Blum, Resnick (1995) +	USA	M= 15.4	532	Female	89.5% White	Children of parents who misuse alcohol	Cross sectional design	Disordered eating behaviours (3+)	.800	Perception that school staff care
Cordero & Israel (2009)	USA	Mode=19	212	Female	55.9% White, 19.0% Asian/ Pacific Islander, 10.9% Latino/ Hispanic, 2.8% Black, 1.4% Middle-Eastern, 9.5% Other/Mixed	University students	Cross sectional design	EAT-26	.950	Low negative parental comments about shape and weight
Croll, Neumark-Sztainer,	USA	9^{th} and 12^{th} grade students	81247	Mixed (49% female)	87% White, 3.5% Asian, 2% Black,	School students completing	Cross sectional design	Extreme WCBs/ binge eating in last	.850	Two parent household Family

Story & Ireland (2002)				(Separate analyses)	1.5% Hispanic, 1% American Indian.	Minnesota Student Survey		year (Yes/No)		connectedness School achievement
Ferreiro, Seoana & Senra (2012)	Spain	M= 10.84 SD= .78	942	Mixed (49% female) (Separate analyses)	98.5% White	School students	Longitudinal study (4 years)	ChEAT – Spanish Version	.900	Social support (boys only)
Fonseca, Ireland & Resnick (2002)	USA	M=14.4	9042	Mixed (51% female) (Separate analyses)	Not described	School students completing Voice of Connecticut Survey	Cross sectional design	Disordered WCBs (Yes/No)	.950	Family connectedness, Maternal presence in the home, Strong family communication (girls only), High parental expectations (boys only), High supervision and monitoring (girls only- risk factor for boys)
Francisco, Narcisco & Alarcao (2013)	Portugal	M= 15.34 SD= 2.12	724	Mixed (62% female) (Separate analyses)	Not described	245 aesthetic athletes and same age controls	Cross sectional design	EDEQ – Portuguese version (global score)	.900	None - *Social support NOT protective*
French, Leffert, Story, Neumark-Sztainer, Hannan & Benson (2001) [#]	USA	6^{th} -12^{th} grade	95395	Mixed (50% female) (Separate analyses)	86% White, 5% multiracial, 4% Hispanic, 2% each African-American, American Indian and Asian.	School students	Cross sectional design	Binge/purge behaviour, weight loss to make others worry ever (Yes/No)	.900	Developmental assets: Family support, Positive family communication, Clear family boundaries, School engagement, Positive peer influence

										Community that values youth, Youth programme in the neighbourhood
Fulkerson, Story, Mellin, Leffert, Neumark-Sztainer & French (2006) #	USA	6th -12th grade	99462	Mixed (50% female) (Separate analyses)	86% White, 5% multiracial, 4% Hispanic, 2% each African-American, American Indian and Asian.	School students	Cross sectional design	Binge/purge behaviour, weight loss to make others worry ever (Yes/No)	.900	Family meals
Haines, Gilman, Rifas-Shiman, Field & Austin (2009) ~	USA	M= 11.9 SD= 1.6	13448	Mixed (56% female) (Separate analyses)	93% White	Growing Up Today (GUTS) cohort	Longitudinal design (4 years)	Vomiting/use of laxatives/ binge eating monthly, dieting weekly (Yes/No)	.900	Family meals
Haines, Kleinman, Rifas-Shiman, Field & Byrn Austin (2010) ~	USA	11 – 18	10540	Mixed (57% female) (Separate analyses)	Not described	Growing Up Today (GUTS) cohort	Longitudinal design (4 years)	Vomiting /use of laxatives/ binge eating in past year. Overweight.	.850	Family meals
Kunstman, Smith & Maner (2014)	USA	M= 18.5 SD= 1.31	93	Female	100% White	University students reporting symptoms of AN	Experimental design	Amount consumed in a test meal	.887	Experience of social power (for those high in self-oriented perfectionism)
Lampis, Agus & Cacciarru (2014)	Italy	M= 15.9 SD= 1.4	1083	Mixed (55% female)	Not described	School students	Cross sectional design	EDI – Italian version	.900	Family functioning, Mother and father caring style, Family cohesiveness.

Loth, Wall, Choi, Bucchianeri, Quick, Larson, Neumark-Sztainer (2015) *	USA	M= 14.5 SD= 1.98	2793	Mixed (53.3% female) (Separate analyses)	18.9% White, 29.0% Black, 19.9% Asian, 16.9%Hispanic, 3.7% Native American, 11.6% mixed/ other	School students in Project EAT (+parents)	Cross sectional design	Dieting/ unhealthy or extreme WCBs/ binge eating in the last year (Yes/No)	.900	Family meals only where: High levels of parent dieting + High enjoyment (boys), Little teasing + Good family functioning + Low levels of weight talk (girls).
Mazur, Dzielska & Malkowska-Szkutnik (2011)	Poland	13	605	Mixed (50.4%)	Not described	1995 Polish birth cohort	Cross sectional design	TFEQ – Polish Version	.900	Positive attitude to school, Social acceptance
McVey, Pepler, Davis, Flett & Abdolell (2002)	Canada	M= 12.9 SD= .62	363	Female	74% Caucasian	School students	Cross sectional design	ChEAT	.950	Paternal involvement, Unconditional parental support
Neumark-Sztainer, Eisenberg, Fulkerson, Story & Larson (2008) *	USA	1/3 M= 12.8± 0.8, 2/3 M= 15.8±0.8	2516	Mixed (55% Female) (Separate analyses)	48.5% White, 19.2% Asian, 19.0% African American, 5.8% Hispanic, 3.5% Native American, 3.9% Mixed/Other	School students in Project EAT	Longitudinal design (5 years)	Chronic dieting/ unhealthy or extreme WCBs/ binge eating in the last year (Yes/No)	.950	Family meals

Neumark-Sztainer, Wall, Story & Falkerson (2004) *	USA	M=14.9 SD=1.7	4746	Mixed (Separate analyses)	'Ethnically diverse'	School students in Project EAT	Cross sectional design	Chronic dieting/ unhealthy or extreme WCBs/ binge eating in the last year (Yes/No)	.950	Family meals
Neumark-Sztainer, Wall, Story & Sherwood, (2009) *	USA	M= 12.7 ± 0.8	412	Mixed (56% female) (Separate analyses)	45% Caucasian, 24% African-American, 16% Hispanic, 6% Asian, 5% Native American, 4% mixed/other	School students in Project EAT identified as overweight	Longitudinal design (5 years)	Extreme WCBs/ binge eating in the last year (Yes/No)	.950	Family meals, Family connectedness
Neumark-Sztainer, Wall, Haines, Story, Sherwood & Van der Berg (2007) *	USA	1/3 M= 12.8± 0.8, 2/3 M= 15.8±0.8	2380	Mixed (55% Female) (Separate analyses)	48.5% White, 19.2% Asian, 19.0% African American, 5.8% Hispanic, 3.5% Native American, 3.9% Mixed/Other	School students in Project EAT	Longitudinal design (5 years)	Extreme WCBs, binge eating in the last year (Yes/No). Overweight.	.850	Family meals
Nicholls & Viner (2009)	UK	30	11211	Mixed	Not described	1970 British Cohort Study	Prospective cohort study	Cases of AN	.950	High maternal BMI, NOT parenting style
Perkins, Luster & Yank (2002)	USA	M=14.9 SD= 1.75	18592	Female	83% European American, 8% African American, 3% Native American, 3% Hispanic, 1%	Adolescents who have experienced physical abuse	Cross sectional design	Vomiting after eating to control weight two or more times per week.	.950	Family support, Positive school climate, NOT relationships with adults outside of family

					Asian or Pacific Islander					
Schirk, Lehman, Perry, Ornstein, & McCall-Hosenfeld (2015)	USA	18 - 64	302	Female	89% non-Hispanic White	Women who have experienced intimate partner violence	Cross sectional design	Eating disorder screen for primary care	1.000	Social support
Scoffier, Maiano, & D'Arripe-Longueville (2010)	France	M= 15.75 SD= 3.00	227	Female	Not described	Elite aesthetic athletes (dancers/ gymnasts/ synchronised swimmers)	Cross sectional design	EAT-26 – French Version	.950	Quality of relationship with parents, Peer acceptance, NOT coach relantionship
Twamley & Davis (1999)	USA	M= 20 SD= 2.4	249	Female	77% Caucasian	University students	Cross sectional design	EAT-26, BULIT-R combined into composite score	.900	Low family influence to control weight in childhood
Wang, Peterson, Richmond, Spadano-Gasbarro, Greaney, Mezgebu McCormick, & Byrn Austin (2013)	USA	6^{th} to 8^{th} graders	15461	Mixed (49% female) (separate analyses)	82.3% White, 6.7% Hispanic, 4.3% Black, 4.2% Asian	School students in Massachusetts Healthy Choices Study	Cross sectional design	Disordered WCBs (Yes/No)	.950	Family meals, Parents providing lifts to physical activity (girls only)

All mean ages for longitudinal designs given for baseline

*(* / + / # / ~ = same participant pool)*

3.2 Methodological quality

Methodological quality was assessed using the QualSyst tool. Total scores for each paper are included in Table 2 (maximum score is 1.000; raw scores available in Appendix A). In general study quality was high, with scores ranging from .800 (Chandy et al., 1994, 1995) to 1.000 (Ahrén et al., 2013; Allen et al., 2014; Bertoli et al., 2015; Schirk et al., 2015).

All papers described their research questions and objectives sufficiently in their introductory sections. Several papers did not explicitly state an intention to identify protective factors in addition to risk factors in their introduction, meaning that protective factors were often identified 'post-hoc' through the identification of an inverse association with disordered eating outcomes (Ahrén et al., 2013; Bertoli et al., 2015; Nicholls & Viner, 2009). This means that there were few null findings in the literature. All papers were deemed to have designs which were appropriate to answer the study question. Cross sectional designs, including those asking questions retrospectively (Cordero & Israel, 2009; Twamley & Davis, 1999), were thought to be appropriate to identify associations between potential protective and risk factors and disordered eating. According to Kraemer et al.'s (1997) criteria these papers could only identify 'correlates', not 'risk' or 'protective' factors, as temporal precedence cannot be established. Only one paper (Kunstman et al., 2014) used an experimental design, suitable for the identification of a 'causal protective factor'. This was a well designed experimental study, with an appropriate control condition, random allocation to treatment group and blinding of participants to intervention.

Most studies used a method of subject selection designed to obtain an unbiased sample of the relevant target population. Two studies recruited female volunteers studying psychology, women's studies or continuing education at college, (Ackard & Neumark-Sztainer, 2001; Kunstman et al., 2014), while another study used participants who were all alumni of the same university (Brown & Keel, 2012), both groups unlikely to be representative of the whole population. Cordero & Israel (2009) were specifically interested in female college students as an 'at risk' group, but their reliance on self-selected volunteer student participants could have

resulted in a sample that was less representative of the wider student population. French et al. (2001) and Fulkerson et al. (2006) used the same sample of students from self-selected schools, and comment that their participants were 'significantly more likely to be white and have college educated parents than the USA population as a whole'. All the participants used in McVey et al. (2002) attended the same two suburban schools. All of the children in the Growing Up Today cohort used in Haines et al. (2009, 2010) were the children of nurses, who were themselves participants in the Nurses' Health Study II. Three studies did not fully describe the method of subject selection used in their studies (Francisco et al, 2010). The majority of papers included sufficient description of participant characteristics, although a number of papers included less information (Francisco et al., 2013; Haines et al., 2010; Mazur et al., 2011; Nicholls & Viner, 2009). All studies used samples of sufficient size to allow adequate statistical power. French et al. (2001) note that their very large sample size lends their study so much statistical power that they are able to detect even very weak associations, although many of the associations found were notably large. Several other studies had equivalently large sample sizes.

All papers used well defined reproducible outcome measures. Two studies assessed prevalence of eating disorders, whereas the rest used self-report measures. Studies using un-validated lists of symptoms rather than standardised measures scored lower, and none of these papers included information about the reliability and validity of measures used. Kunstman et al. (2014) used a behavioural measure, amount eaten in a test meal, with more eaten taken to represent less pathological restriction, however it is unclear how this measure relates to AN symptomatology and no mention is made of how it might relate to EDs where binge eating is a problem. Almost all of the studies used self-report measures to assess potential protective factors such as family relationships. Two papers used retrospective measures of childhood family environments assessed in adulthood, leaving these measures at risk of recall bias (Cordero & Israel, 2009; Twamley & Davis, 1999). Most papers assessed interpersonal factors such as peer acceptance, supportiveness of school environment, or family connectedness using self-report

measures completed by the individual, meaning that conclusions can only be drawn about the relation of the subjective perception of the relationship in question to disordered eating, rather than 'objective' characteristics of these relationships (Kraemer et al., 1997).

All papers used appropriate analytic methods, primarily logistic regression analyses. Chandy et al. (1994, 1995) used chi-squared analyses inappropriately to compare data for a subset of young people (those with parents misusing substances) with the whole sample, but this did not affect the analyses investigating protective or risk factors. Many papers made multiple comparisons and drew post-hoc conclusions, suggesting a possible risk of Type 1 errors. Several of the papers did not include any estimate of variance for their main results (Chandy et al., 1994, 1995; Croll et al., 2002; Ferreiro et al., 2012; Lampis et al., 2014; Neumark-Sztainer et al., 2007). The conclusions of almost all of the studies were well supported by the results. However, Mazur et al. (2011) refer to protective and risk factors for emotional eating in their conclusions, when in fact the predictive power of the model tested is non-significant. Berge et al. (2013) draw conclusions about a factor being protective for all adolescents, when in fact this only seems to be the case for overweight adolescents, whose data was analysed separately.

3.3 Results of studies

The papers included in the review identified a number of potential protective factors against the development of eating disorders, along with a range of risk factors. Findings related to the proximal social environment of family, peers, schools and neighbourhoods are summarised here.

Social support: Several studies looked at the impact of 'social support' in general. In a longitudinal study, Ferreiro et al. (2012) explored the impact of social support (a composite of feeling loved/supported by family and loved by friends) on the risk of adolescents developing 'disordered eating' (scoring above 15 on the ChEAT) over a 5 year period. For both boys and girls, greater depressive symptoms at baseline was predictive of disordered eating at five years

later, but for boys only, social support appeared to moderate this effect. Social support was also directly predictive of reduced disordered eating at the end of the study for boys only. They concluded that social support may be protective against the development of disordered eating in boys. Mazur et al (2011) studied adolescents in Poland using the Three Factor Eating Questionnaire. They found that social support was negatively associated cross-sectionally with uncontrolled eating. They report that a model incorporating stress, emotional social support and attitude to school predicted uncontrolled eating, explaining 7% of the variance, while instrumental social support, stress, social acceptance from peers and coping style predicted emotional eating, explaining 7.8% of the variance. Based on this model, they concluded that social support may be protective against uncontrolled and emotional eating. However given the very small proportion of the variance explained by their models and the absence of an independent association between social support and emotional eating, their conclusions regarding emotional eating are not supported by the evidence.

Schirk et al. (2015) looked at the impact of social support on women who could be considered 'at risk' due to having experienced intimate partner violence. They found that 12% of women reporting current high levels of social support were at current high risk of ED (scoring 3 or 4 on the Eating Disorder Screen for Primary Care), compared to 24% of women reporting low social support. There was a significant association of social support and ED risk. A 5-unit increase in social support was associated with reduced odds of being at high risk for ED, controlling for other variables. They concluded that social support may be protective against disordered eating for women who have experienced intimate partner violence. Francisco et al. (2013) were also interested in 'at risk' groups and studied the role of social support on disordered eating in adolescent aesthetic athletes (dancers or gymnasts) and non-athlete controls. In line with previous research, elite aesthetic athletes reported higher levels of disordered eating on the EDEQ. In contrast to the other studies, they found that social support was not cross-sectionally

associated with disordered eating for athletes or controls, and concluded that social support did not appear to be protective for this 'at risk' group.

Key relationships: A number of the studies identified that the existence of particular relationships appeared to be protective. Ahren et al.'s (2013) prospective analysis of health and other public records for the very large Stockholm Youth Cohort found that for females only, having a greater number of full siblings was associated with a lower rate of ED by the end of the study period (age 12-27), while the reverse was true for number of half-siblings. In their discussion, they reflected on how full siblings might exert a protective effect by 'diluting' parental expectations and pressure, which might be otherwise, when experienced in the milieu of a high cultural value of thinness for females, contribute to the development of ED in females. They suggested that the finding of the opposite pattern for half-siblings may reflect the fact that these families were more likely to have experienced adverse events such as loss of a parent or divorce. In their large cross-sectional study of adolescents in the USA, Croll et al (2002) found a small protective effect of living in a two parent household, with this being associated with reduced risk of disordered weight control behaviours (Disordered WCBs).

Other researchers have explored the influence of romantic relationships. Bertoli et al. (2015) studied a mixed gender group of patients in a weight loss clinic, and found that being married, along with older age and increased physical activity, was cross-sectionally associated with a 12% lower chance of scoring more than 18 on the Binge Eating Scale, and suggested that being married might be protective against binge eating in overweight individuals. In contrast to this, Schirk et al. (2015) found that in their sample of women who had experienced intimate partner violence, those partnered but not married were less likely to be at current high risk of having an eating disorder. Brown and Keel (2012) hypothesised that being in a romantic relationship might be protective for gay and bisexual (GB) men, due to reduced pressure to find a male partner which is thought to drive increased ED rates in this group. Their cross-sectional

study of male alumni from one university found that GB men scored significantly higher on the EDI-2, and that for men in their 'middle' (30s) and 'older' (40s) age groups there was an interaction with relationship status, with GB men in relationships reporting fewer ED symptoms than those who were single. There was no association between relationship satisfaction and ED symptoms.

Quality of family relationships: Many of the studies looked for associations between the quality of relationships with family and ED outcomes. Allen et al. (2014) conducted a longitudinal study over two years following the children of mothers with or without eating disorder history. They found that increases in the child's reported satisfaction with their family between the ages of 10 and 12 predicted decreases in scores on the ChEDE and loss of control over eating over the two-year study period. The association with control over eating remained even when controlling for child depression and self esteem. They concluded that being satisfied with family life might be protective against the development of disordered eating difficulties in early adolescence, although they did not explore what aspects of family life contributed to a sense of greater satisfaction.

Looking at slightly older children (aged 11 to 19) in the USA, Berge et al. (2014) found that children's reports of better 'family functioning' were cross-sectionally associated with lower odds of engaging in dieting, binge eating, and a range of unusual and extreme WCBs, as was a reported higher sense of connection with either parent. Mothers generally having knowledge of their children's whereabouts was associated with fewer disordered eating behaviours for both boys and girls, whereas father's knowledge of whereabouts was associated with lower odds of Disordered WCBs for girls only. Conversely, high reported 'parental control' was associated with higher rates of disordered eating, and weakened the protective effect of high family functioning. Lampis et al. (2014) also found a protective effect of family functioning, in a study of children of the same age in Italy. Increased mother and father caring style on the Parental Bonding

Instrument was cross-sectionally associated with reduced likelihood of scoring above clinical cut offs on the EDI, as was increased 'family cohesiveness'. Conversely, increased scores for 'family adaptability' was associated with increased odds of scoring highly. They concluded that parental caring and family cohesion may be protective against EDs.

Several studies looked at 'family connectedness'. Croll et al (2002) found that adolescents reports of higher family connectedness were cross-sectionally associated with reduced odds of engaging in Disordered WCBs, and they concluded that family connectedness was a significant protective factor against the development of disordered eating. A similar cross-sectional study by Fonseca et al. (2002) also found that greater adolescent reported family connectedness appeared to be protective. They also found that maternal presence in the home appeared to be protective for both boys and girls, while strong family communication appeared to be protective for girls only, and high parental expectations were protective for boys only. There was a different pattern of associations with parental monitoring and supervision for boys and girls, with high supervision and monitoring appearing protective for girls, while being a risk factor for boys. These findings are interesting considered in relation to the findings of Berge et al (2014) regarding parental control and knowledge of adolescents' whereabouts. Neumark-Sztainer et al. (2009) looked at the impact of family connectedness in overweight children using data from Project EAT. They found that for both boys and girls, greater reported family connectedness at baseline (around age 12) was again associated with reduced likelihood of engaging in binge eating or extreme WCBs five years later.

French et al. (2001) looked for associations of a wide range of 'developmental assets' and disordered eating outcomes in adolescents (aged 12 to 18). All of the developmental assets, assessed in this very large study using the Profiles of Student Life: Attitudes and Behaviour Survey, were cross-sectionally associated with lower rates of Disordered WCBs. Family support, positive family communication and clear family boundaries were amongst the strongest 'discriminating assets' between those reporting/not-reporting disordered eating. Several other

studies looked at reports of support from families. McVey et al. (2002) study of early adolescent females looked at unconditional support vs conditional support, as assessed using the Conditional Support Scale for Parents and the Children's Perceptions of Parents Scale, and found that unconditional paternal support was cross-sectionally inversely associated with disordered eating assessed using the ChEAT. They found an interaction with negative life events, such that a history of negative life events were less strongly associated with current disordered eating where current unconditional paternal support was high.

A number of studies looked at the possible protective role of family factors in adolescents that might be considered 'at risk' of developing disordered eating difficulties. Perkins et al. (2002) identified female adolescents who had experienced physical abuse from a large sample of Search Institute surveys. Females reporting physical abuse were more likely to engage in purging behaviour. Current family support appeared to be protective against purging in this group. There was no association between positive family communication and purging. Scoffier et al. (2010) studied female adolescents practicing aesthetic sport (dance, gymnastics and synchronised swimming) at an elite level. They found that the quality of parent relationships currently reported on the Self Description Questionnaire was inversely associated with disturbed eating attitudes on the EAT. They suggested that high quality relationships with parents might be protective against the development of disturbed eating attitudes in elite aesthetic athletes.

Family environment around eating and weight: A very large prospective cohort study, by Nicholls and Viner (2009), looked for associations between a wide range of childhood variables and prevalence of anorexia nervosa by age 30. Higher maternal BMI at age 10 was found to be associated with reduced risk of AN, as was high self-esteem in childhood. Interestingly, parenting style and the experience of separations from mothers showed no predictive association with disordered eating. Maternal depression in early childhood, female sex, and a history of under-eating in late childhood were associated with increased risk.

Berge et al. (2013) looked at the cross-sectional relationships between the discussions parents were having with their children about healthy eating and weight and the disordered WCBs reported by their children. Many parents were having discussions about healthy eating and/or weight with their children. Overall, parental conversations about healthy eating were associated with the lowest prevalence of disordered WCBs, and parent conversations about weight were associated with higher prevalence. There were slightly different patterns of results for type of behaviour, mothers/fathers and overweight/normal weight children, suggesting healthy eating conversations may only be protective against some behaviours, and more so for overweight than normal weight children.

Cordero & Israel (2009) asked female university students to report retrospectively about potential protective factors in earlier life while reporting current disordered eating on the EAT-26. Lower levels of parental negative comments about weight and shape reported during childhood predicted lower scores on the EAT-26. The strongest predictor of ED symptoms was acceptance of socio-cultural attitudes about appearance. The impact of parental comments was fully mediated by the internalisation of socio-cultural attitudes. They suggested that internalisation of socio-cultural attitudes may act as a pathway for the impact of parental comments on eating pathology. There was no association of parental acceptance or availability with ED symptoms. Twamley and Davis (1999) also looked at associations between parents comments and influence to control weight and internalisation of thinness norms. In their sample of female undergraduates, awareness of thinness norms was associated with eating pathology, and this effect was mediated by internalisation of thinness norms and body dissatisfaction. Retrospectively reported family influence to control weight moderated the relationship between awareness and internalisation of thinness norms, such that low family influence to control weight in childhood was associated with reduced internalisation of norms in early adulthood, but only where awareness of norms was low. They concluded that low family influence to lose weight may

protect against the later development of eating pathology, by buffering against the internalisation of thin-ideals.

Ten of the studies investigated the potential protective effect of family meals. Ackard and Neumark-Sztainer (2001) first explored the association between frequency of family dinners in childhood and bulimia symptoms on the BULIT-R and EDI-2 in a sample of female college students. They found that frequency of family dinners while growing up (reported retrospectively) was inversely associated with current bulimic symptoms. The association between family meals and bulimic behaviours remained statistically significant after adjusting for other familial factors including family cohesion, independence and achievement orientation. This finding was further supported by four studies by Neumark-Sztainer et al. (2004, 2007, 2008, 2009) using data from the Project EAT survey. Increased frequency of family meals, along with prioritising these meals and having a positive meal atmosphere, was shown to be associated cross-sectionally with reduced risk of unhealthy and extreme weight control behaviours for both girls and boys, and with reduced risk of chronic dieting for girls only (Neumark-Sztainer et al., 2004). This protective relation of family meal frequency and disordered eating was replicated longitudinally. Neumark-Sztainer et al. (2007) report that family meal frequency and a positive atmosphere at meals at baseline was inversely associated with 'weight related problems' (extreme WCBs, bingeing and overweight) 5 years later for girls only. Parental weight concern, weight teasing, dieting, weight concern and body dissatisfaction were risk factors for girls developing weight related problems. Neumark-Sztainer et al. (2008) also found that for girls, frequency of family meals at baseline was associated with lower likelihood of engaging in extreme WCBs (but not chronic dieting or binge eating) 5 years later. Interestingly, for boys, regular family meals at baseline were associated with greater likelihood of unhealthy WCBs (non-extreme), in particular skipping meals and eating very little food in meals, 5 years later, but not with other disordered eating outcomes. Family meals may be protective against extreme weight control behaviours for girls. Finally, Neumark-Sztainer et al. (2009) looked at data for a subset of the Project EAT

sample who were overweight, and found (in addition to their findings related to family connectedness mentioned earlier) that for girls only family meals with a positive atmosphere at baseline appeared protective against disordered eating 5 years later, as were higher levels of self esteem body satisfaction, and eating regular lunch or dinner.

Similar findings are reported by Haines et al. (2009, 2010) following their analysis of data from the Growing Up Today study. Haines et al. (2009) found that more frequent family meals in the previous year were associated with significantly lower incidence of purging (for females only), binge eating, and frequent dieting (for females only). These effects were not modified by age, importance of thinness to parents, frequency of parental comments to child about weight, or maternal dieting behaviours. They concluded that eating family meals frequently may be protective against the development of disordered eating behaviours, and this effect is not moderated by negative family interactions around weight and food. Haines et al. (2010) looked at 'weight related problems' (bingeing, purging and overweight) in the same dataset and report that family meal frequency was inversely associated with purging and binge eating for females, both cross-sectionally and longitudinally.

Fulkerson et al (2006) used the same dataset as used by French et al. (2001) to explore the association of family meal frequency, disordered eating (bingeing, purging and excessive weight loss) and other high risk behaviours and 'developmental assets'. Family meal frequency was cross-sectionally associated with other developmental assets including family support, boundaries and expectations, positive identity, commitment to learning, social competencies and positive values. All high risk behaviours, including disordered eating, were inversely associated with family meal frequency. These associations remained significant even when controlling for family support and communication. They concluded that family meals could be considered a 'developmental asset' promoting a range of positive outcomes.

Wang et al. (2013) looked at family meal practices and also behaviours around exercise. Adolescents who had family meals most days or every day had decreased odds of disordered

WCB relative to those who never did. Parents' provision of rides to/from physical activity events was cross-sectionally inversely associated with disordered WCBs for girls. Parental participation in physical activity with children was associated with increased odds of disordered WCBs. There was no association of parental encouragement of physical activity or parents stating to watch sports with disordered WCBs. They concluded that frequent family meals were protective against disordered WCBs, and this effect appears consistent across race/ethnicity and weight status. Parental provision of lifts to physical activities may be protective for girls.

Most recently, Loth et al. (2015) have looked again at data from Project EAT. They found that greater frequency of meals was cross-sectionally associated with lower levels of dieting (girls only), unhealthy weight control behaviours (boys and girls), and extreme weight control behaviours (girls only). There was no association with binge eating. These effects were fairly robust, but a number of interactions were found. For boys, low enjoyment of family meals reversed this protective effect, while high levels of parent dieting were associated with greater protective effect. For girls, family meals were protective where there was little teasing, good family functioning, or low levels of weight talk, but were a risk factor where teasing or weight talk were high, or functioning low. They concluded that family meals appear to exert a robust protective effect, but elements of negative mealtime and family weight- related environment may diminish or even reverse this protective effect.

School: A number of papers looked for protective factors in the school environment. In addition to their findings about family relationships, Croll et al. (2002) found that self reported current school achievement was 'protective' against current disordered weight control behaviours for both males and females, while 'school connectedness' was a protective factor for male adolescents only. French et al. (2001) found that high levels of self reported school engagement, which they described as one 'developmental asset', was cross-sectionally associated with reduced likelihood of engaging in binge/purge behaviour. Mazur et al. (2011) studied adolescents in

Poland and found that having a positive attitude to school (along with stress and emotional social support) predicted uncontrolled eating cross-sectionally on the Three Factor Eating Questionnaire, and concluded that having a positive attitude to school may protect against uncontrolled eating. None of these studies asked questions about what factors in the school contributed to the adolescent's subjective sense of engagement, connectedness or positive attitude.

Three studies of 'at risk' adolescents found a protective role for schools. Chandy et al. (1994, 1995) wrote two papers on children who had parents misusing substances, using data from the same Adolescent Health Survey. In the first paper, the children of parents who misused substances reported significantly higher rates of disordered eating behaviours than the general population. The perception that school was an alcohol free environment was cross-sectionally associated with a higher probability of reporting no symptoms. The second paper looked at female adolescents who indicated that their parents misused alcohol. As above, these adolescents reported higher rates of disordered eating than the general population. The perception that school staff cared about them was associated with reporting fewer disordered eating behaviours. They concluded that a supportive school environment may be protective against disordered eating for at-risk adolescents. In addition to their findings about family support, Perkins et al. (2002) found that a current reports of a 'positive school climate' was 'protective' against current purging behaviour in for female adolescents who had experienced physical abuse.

Peers: Two studies looked at the role of self-reported social acceptance by peers. Mazur et al. (2011) found that social acceptance by peers was a cross-sectional predictor of reduced emotional eating, and concluded it was protective. Scoffier et al. (2010) found that in their sample of elite aesthetic athletes, current perceived peer acceptance was inversely associated with current disturbed eating attitudes on the Eating Attitudes Test, and concluded that peer acceptance may be protective against the disordered eating in this group. They also looked at

'sports friendships', but found that rather than being protective, sport friendship quality was directly correlated with perceived physical ability, which was in turn directly correlated with increased disordered eating. French et al (2001) asked about positive peer influence (having friends who abstain from alcohol, drugs and sexual intercourse) in their study of developmental assets, and found that this was one of the strongest 'discriminating assets', cross-sectionally associated with lower rates of disordered eating.

Other interpersonal factors and relationships: A number of studies looked at protective factors in the proximal social environment outside of family, school and peers. French et al. (2001) looked at neighbourhood factors in their study of developmental assets and found that having a community that values youth, and having a youth programme in the neighbourhood, were both amongst the strongest discriminating assets associated with lower risk of disordered eating. Perkins et al. (2002) hypothesised that for adolescents that had experienced physical abuse, having relationships with adults outside of their family would be protective against disordered eating. Contrary to this expectation, they found that reported support from adults outside of the family was cross-sectionally associated with increased risk of engaging in purging behaviour. They suggested that this may be because seeking support from adults outside of the family may be an indicator of a lack of support within the family. Scoffier et al. (2010) also failed to find an expected protective influence, in their study of aesthetic athletes. They found that a higher quality of relationship with sporting coaches was not protective, and instead was directly correlated with perceived physical ability, which was correlated with disordered eating attitudes.

In the only study with an experimental design, Kunstman et al. (2014) studied the impact of experiencing social 'power', which they defined as 'asymmetrical interpersonal resource and outcome control', having the capacity to control outcomes for others, having more access to resources and being less subject to the judgements of others. Having recruited female students

who reported one or more symptoms of AN, they randomly allocated participants to a 'situational power' condition, where they completed a computer task where they had power over another participant's outcomes, and a control condition where they had no control over others' outcomes. Participants were unaware that the researchers were studying power or eating. Following the task, participants were asked to 'taste test' a milkshake, and the amount they consumed was recorded. They also assessed 'self-oriented perfectionism' (SOP). They found that there was no main effect of power, but there was an interaction with SOP. In the control condition, SOP was inversely associated with consumption. This relationship was attenuated in the power condition. At high levels of SOP, those in the power condition consumed more than controls. They suggested that situational power may be protective against pathological dietary restraint for those high in SOP, and may increase consumption for at risk individuals. They suggested that experiencing power over others may 'over power restriction' by creating a sense of control, counteracting the perceived loss of control experienced by those high in SOP when failing to meet unrealistic goals, which might otherwise contribute to self-criticism and restraint.

4.0 Discussion

4.1 Key findings

The aim of this review was to identify studies which looked for or identified protective factors against the development of eating disorders and disordered eating existing in the proximal social environment. The papers included in the review make use of a range of designs to explore these associations. Their results highlight a wide range of potential protective factors in social systems such as families, schools, peer groups and neighbourhoods.

Social support in general was identified by a number of studies as a protective factor for various groups. Ferreiro et al. (2012) found social support was protective against disordered WCBs for the adolescent boys in their study, while Mazur et al. (2011) identified support as protective against uncontrolled and emotional eating in their adolescent sample. Schirk et al.

(2015) found that social support protected against ED risk in women who had experienced intimate partner violence. Social support is well established as a protective factor promoting general psychological wellbeing. It is thought to play a direct beneficial role over the course of development, while also exerting an indirect effect by 'buffering' individuals against the impact of stressful life events (Cohen & Wills, 1985). It is therefore perhaps surprising that not all of the studies found a consistent protective effect of social support. There was no association between support and disordered eating outcomes for the girls in Ferreiro et al.'s (2012) study, while Francisco et al. (2013) found no association in their sample of aesthetic athletes. The reasons for this difference are unclear, but it is notable that both females and aesthetic athletes are more 'at risk' of developing difficulties with disordered eating. It may be that the protective influence of social support is insufficient to overcome the wider pressures in social systems promoting eating pathology in these groups.

The existence of a number of key relationships appeared to be protective. For children and adolescents, having full siblings, and living in a two parent household were identified as protective (Ahrén et al., 2013; Croll et al., 2002). In adulthood, one study identified being married as protective against binge eating for overweight individuals, while in another study of women who had experienced intimate partner violence, being partnered but not married was protective (Bertoli et al., 2015; Schirk et al., 2015). It seems plausible that the apparently contradictory findings of these two studies may reflect the violent nature of some of the current relationships in Schirk et al.'s study. Being in a romantic relationship of any kind was protective for bisexual and gay men in their 30s and 40s (Brown & Keel, 2012). Their suggestion that this is due to reduced pressure to find a male partner is also of relevance to the studies of heterosexual women, and highlights links to wider socio-cultural levels of influence on eating pathology, such as discourses around what is, and is not, a desirable body appearance.

Many of the studies looked at the qualities of relationships, and in particular the qualities of relationships within families. Children's satisfaction with family life appeared to be protective

as was a reported sense of 'family connectedness' and reporting high quality relationships with parents (Allen et al., 2014; Croll et al., 2002; Fonseca et al., 2002; Neumark-Sztainer et al., 2007; Scoffier et al., 2010). Unconditional family support was identified as protective in a number of studies (French et al., 2001; McVey et al., 2002; Perkins et al., 2002). Positive family communication was identified as a protective factor against purging in one sample of adolescents, while another study found this association for girls only , and a study of children who had experienced physical abuse found no such association (Fonseca et al., 2002; French et al., 2001; Perkins et al., 2002). Having clear family boundaries was indentified in one study as protective, while two studies identified having good 'family functioning' as protective against disordered weight control behaviours (Berge et al., 2014; French et al., 2001; Lampis et al., 2014). Many of these factors can be identified as 'developmental assets', in that they are factors which have been shown to be related to a wide range of positive developmental outcomes, not just to the absence of eating pathology (French et al., 2001; Levine and Smolak, 2016).

In relation to parenting practices, parents having a caring style was identified by one study as protective, while high levels of 'parental control' appeared to increase risk (Lampis et al., 2014). One study found that parents having a good knowledge of their children's whereabouts to be protective against disordered WCBs, while another study found that high levels of parental monitoring and supervision was protective only for girls, while in fact being a risk factor for boys (Berge et al., 2014; Fonseca et al., 2002). This difference highlights the importance of careful consideration of factors such as gender rather than taking a 'one-size-fits-all' approach when studying protective factors, or when using findings such as these to inform prevention efforts (Levine and Smolak, 2016).

The only study looking at parenting practices in adulthood, and looking at cases of AN rather than disordered WCBs, found no association between parenting style and AN (Nicholls & Viner, 2009). They highlight that putative risk and protective factors identified cross-sectionally in childhood do not always show consistent effects when studied longitudinally. This could also

reflect the different outcomes, with many disordered weight control behaviours reflecting a 'binge/purge' type presentation, while AN involves more restriction. It may be that different kinds of eating pathology are influenced by different protective and risk factors (Waller,1994)

Family practices around eating and weight have been studied in some detail. Ten studies, incorporating data from five major samples, have highlighted a protective role of eating regular family meals against developing disordered weight control behaviours. This association has been demonstrated cross sectionally and longitudinally in a range of samples of school children from the USA (Ackard & Neumark-Sztainer, 2001; Fulkerson et al., 2006; Haines et al., 2009, 2010; Loth et al., 2015; Neumark-Sztainer et al., 2004, 2007, 2008, 2009; Wang, Peterson, et al., 2013). More recently Loth et al. (2015) have looked in more detail at meal frequency in relation to the family environment around food and weight and suggest that while meals are generally protective, in cases where families report high levels of weight talk and teasing and poor family functioning (for girls) or low enjoyment of family meals (for boys) their effect is reversed, becoming a risk factor for disordered WCBs. Other aspects of the family weight and eating environment identified as protective include high maternal BMI (protective against AN), avoiding negative comments about weight or parents exerting influence to lose weight (against disordered eating symptoms in adults), having discussions about healthy eating but not about weight (against disordered WCBs) and provision of lifts to physical activities (against disordered WCBs in females) (Berge et al., 2013; Cordero & Israel, 2009; Nicholls & Viner, 2009; Twamley & Davis, 1999; Wang, Peterson, et al., 2013). These results fit well with the findings of qualitative research into the strategies used by parents to promote positive body image and resilence against disordered eating, such as sensitively filtering communication around body image issues, and promoting positivity around food by shifting the focus of conversations away from body size and weight loss towards healthy choices and pleasure in food (Maor & Cwikel, 2015).

Outside of the family, school achievement and a sense of engagement with and connection to school were identified as factors protecting against disordered WCBs, bingeing and uncontrolled eating (Croll et al., 2002; French et al., 2001; Mazur et al., 2011). Having a supportive school environment was identified as an important protective factor for adolescents who might be considered at risk due to parental substance misuse or experiences of physical abuse (Chandy et al., 1994, 1995; Perkins et al., 2002). Feeling accepted by peers was identified in two studies as a protective factor (Mazur et al., 2011; Scoffier et al., 2010). As with many of the other relational factors discussed in these papers, it is important to note that these studies assessed the subjective experience of feeling accepted, rather an objective metric, and as such may be assessing an aspect of self concept rather than the 'real' experience of relations with peers. One study identified having relationships with peers who abstained from risk behaviours as protective against bingeing and purging (French et al., 2001). Looking at the wider social environment, having communities that value youth and the provision of youth activities in the neighbourhood was identified as protective against bingeing and purging (French et al., 2001). Having experiences of social power over others was identified as protective against restriction in individuals high in self-oriented perfectionism. It is interesting to consider whether social systems provide many opportunities for adolescents and young people to have experiences of power.

4.2 Limitations of the literature

The studies included in the review made use of a range of designs, some of which were more or less suitable for the identification of risk and protective factors. The most commonly used design was a cross-sectional survey, allowing the identification of factors correlated with current disordered weight control behaviours. According to the guidance provided by Kraemer et al. (1997), these designs are appropriate to identify 'correlates', but these correlates cannot be considered 'protective' or 'risk' factors unless precedence can be established. Following this

guidance would mean that only those studies making use of longitudinal designs could actually be considered to have identified a protective factor. They also highlight the importance of consideration of time of testing, suggesting that different factors have an influencing role at different times. All of the studies included participants from a range of ages, ignoring the role of time. Nicholls and Viner (2009) highlight this in their paper when commenting that risk factors from childhood often do not show consistent associations when measured longitudinally. Going further than this, Kraemer et al. (1997) suggest that having identified a protective or risk factor, in order to establish it as causal it is necessary to modify the risk factor and then observe an effect on the outcome of interest. Only one study made use of an experimental design which would allow this. This failure by many studies to make use of appropriate designs for the identification of protective or risk factors severely limits the strength of the conclusions that can be drawn from the literature.

There are a number of sources of potential bias in the literature. Many of the papers identified protective factors retrospectively, having set out to measure risk factors and then having found a factor with an inverse association with disordered eating. This post-hoc method of drawing conclusions leaves the literature as a whole at risk of publication bias, as 'protective' associations only get mentioned if they have been found. This problem is conpounded by the fact that many of the papers assessed high numbers of variables, and had large samples, increasing the chance of 'Type 1' false positive errors. It is possible therefore that some of the protective relationships identified may be spurious. Support for the existence of a publication bias in this field is evident in the fact that very few papers reported null findings. Where researchers have set out with a specific intention to explore protective factors, the literature is further skewed by the factors that the researchers have chosen to measure. Ten of the papers assessed the same construct, family meal frequency, making this effect seem particularly robust. However seven of these studies are based on the same Project EAT dataset, so this finding may be less robust than the number of studies suggests. These papers have also been written

exclusively about students in middle or high school in the USA, compromising the generalisability of these findings to other groups.

Regarding the clinical utility of the literature, some of the papers have assessed factors which could be targeted by interventions, such as family discussions about weight, while other have assessed factors which could be considered more 'fixed', such as having full vs half siblings. Many of the factors assessed, such as 'good family functioning' or 'family connectedness', are poorly defined and may be highly subjective. Without clearer definition of these factors, and some level of operationalisation of the behaviours which contribute to, for example, 'family connectedness', it is hard to know what guidance could be offered to families and other social systems, if indeed it was practicable to do so. Neither have many of the papers have any made attempt to consider the mechanisms by which these factors may exert influence on outcomes, or to explore factors which may mediate or moderate these associations. Consideration of mediating and moderating variables may go some way to elucidating situations where there appear to be different patterns of results, for example where there are differences according to gender or according to type of pathology. Failure to fully understand these mechanisms reduces the potential of findings to inform prevention efforts.

4.3 Clinical applications

The identification of protective factors against disordered eating outcomes can inform the development of prevention strategies and interventions (Levine & Smolak, 2016; Neumark-Sztainer, 2011; Steck et al., 2004). Currently, although the role of the social systems in eating pathology is widely accepted, the vast majority of prevention interventions work at the level of the individual (Austin, 2000; Shaw et al., 2009). The findings of these studies could be used to inform the development of prevention approaches at the level of the family, the school, the peer group, or even wider social levels. Interventions to address the family environment around eating and weight present one such opportunity, for example supporting families to introduce more frequent, regular family meals and encouraging them to avoid weight related discussions and

comments. Even these fairly straightforward seeming interventions would require careful implementation and evaluation, to reduce the risk of iatrogenic effects. For example, it is possible that advising parents to the frequency of family meals without offering guidance and support to reduce weight talk and teasing might be counterproductive and even harmful, increasing risk for girls.

Some findings, those related to 'fixed' factors such as the number of siblings, have less obvious clinical utility as they cannot realistically be changed (although they can be used to identify those who are less or more at risk). Elucidation of the mechanisms by which these factors exert influence might open up more potential opportunities for prevention efforts. Making full use of the findings of these studies also requires a shift in focus from individual pathology to wider social systems. Creative thinking is required to develop ways to intervene at the level of families, schools, communities and policies, with a focus on promoting positive eating and weight related wellbeing outcomes rather than just preventing pathology (Bronfenbrenner, 1979; Seligman & Csikszentmihalyi, 2000). Many of the protective factors identified, such as unconditional family support, are also protective factors for a range of positive developmental outcomes, meaning effective interventions to promote these factors would be likely to have wide ranging positive effects (Levine and Smolak, 2016). There is a need for a wider scientific and political debate around ways to support families, schools, and communities to promote adaptive development, resilience and wellbeing.

4.4 Suggestions for further research

To address the limitations identified above, there is a need for more studies with an explicit objective to investigate protective factors in social systems. Areas which have been relatively neglected so far in the field, such as factors in peer groups, school and neighbourhoods, could be promising avenues for further research. Identification of protective factors outside of families might be of particular value to those young people developing in

family environments which could otherwise place them at higher risk. Where potential protective factors have been identified, such as high quality relationships with parents, or conversations about healthy eating, future studies should aim to replicate these findings and explore their applicability across different genders, cultures and other groups.

Further research is also needed to explore the mechanisms and mediating and moderating variables underlying the protective effects identified. This will contribute to explanatory models for adaptive outcomes and disordered eating, help to clarify cases where different patterns of results have been found in different groups, and provide further information and potential targets for prevention efforts. The studies of Twamley and Davis (1999), which looks at the interaction of family influence to lose weight with internalisation of thinness norms, and Loth et al. (2015) which studies the moderating influence of various qualities of the family meal environment, provide a useful model of how such studies could be conducted. Future studies could also benefit from incorporating data from multiple perspectives and potentially from direct observation, rather than relying on an individual's perception of their relationships with others. This would allow researchers to draw firmer conclusions about objective characteristics of relationships (rather than just an individual's subjective experience of them) and could potentially indentify specific behaviours or patterns of interaction which contribute to a sense of, for example, family connectedness or family meal enjoyment. This information could then be incorporated into guidance for families, schools and communities, and intervention efforts.

In many cases 'correlates' have been identified through cross sectional designs, presenting potential opportunities for carefully designed prospective studies to fully test hypotheses about protective factors. Intervention studies to modify protective factors would allow the exploration of 'causality', and strengthen the clinical utility of these findings. Certain more easily modifiable factors, such as the frequency of family meals, present clearer opportunities for studies of this kind. However at this point in many cases the potential

protective factors that have been identified are fixed, or poorly defined, and one can question whether experimentally manipulating factors such as 'family connectedness' would be ethical, or indeed possible. Work of this kind could be more useful following more thorough and co-ordinated exploratory research efforts.

4.5 Limitations of this review

This review represents an attempt to systematically search and review the literature. Several databases were searched using broad search terms in an attempt to identify all papers relevant to the topic of the review. However, it is possible that some papers meeting inclusion criteria may not have been identified. In particular, while reference lists for studies meeting Stage One inclusion criteria were scanned for further relevant papers, scanning the reference lists of papers which were excluded was not feasible within the scope of the review. The low number of additional papers identified in this way (two papers from the reference lists of eighty-seven papers), does provide some evidence in support of the potential of the search strategy used to provide a complete picture of the literature.

Given the wide range of study designs, outcome measures and methods of analysis used, it was decided to present a narrative synthesis of key findings and methodological issues in the literature. The lack of a quantitative synthesis limits the potential to draw conclusions about the strength and reliability of particular associations. The broad focus of the review and wide range of factors studied also means that conclusions are not drawn about the applicability of findings to different groups, or to different forms of disordered eating.

Extraction of relevant data and assessment of study quality was undertaken by a single researcher. Every attempt was made to complete this task in a systematic fashion. The involvement of an independent quality checker would have allowed an assessment of the reliability of this process, however this was not feasible within the scope of the review.

4.6 Conclusions

The aim of this review was to identify and evaluate the current literature regarding protective factors against eating disorders and eating pathology that exist in the proximal social environment. The papers discussed in this review investigate a range of potential protective factors including the role of social support, high quality family relationships, a healthy family environment around eating and weight, and aspects of schools, peer groups and wider social networks. Informed by the positive psychology and developmental psychopathology approaches, there is a growing interest understanding the factors which promote positive psychosocial development and resilience. The interpersonal environment has a key role to play in protecting young people against the development of eating disorders and eating pathology and supporting adaptive outcomes. Identifying protective factors at this level presents opportunities to understand and the potential to prevent the development of these difficulties. Further research into protective factors at this level is essential to inform efforts by clinicians, families, schools and communities to prevent disordered eating and to promote positive development.

5.0 References

Ackard, D. M., & Neumark-Sztainer, D. (2001). Family mealtime while growing up: associations with symptoms of bulimia nervosa. *Eating Disorders*, *9*, 239–49. doi:10.1080/10640260127551

Ahrén, J. C., Chiesa, F., Koupil, I., Magnusson, C., Dalman, C., & Goodman, A. (2013). We are family - Parents, siblings, and eating disorders in a prospective total-population study of 250,000 Swedish males and females. *International Journal of Eating Disorders*, *46*, 693–700. doi:10.1002/eat.22146

Allen, K. L., Gibson, L. Y., McLean, N. J., Davis, E. A., Byrne, S. M. (2014). Maternal and family factors and child eating pathology: Risk and protective relationships. *Journal of Eating Disorders*, *2*, 11. doi:10.1186/2050-2974-2-11

Austin, S. (2000). Prevention research in eating disorders: theory and new directions. *Psychological Medicine*, *30*, 1249–1262.

Berge, J. M., Maclehose, R., Loth, K. A., Eisenberg, M., Bucchianeri, M. M., & Neumark-Sztainer, D. (2013). Parent conversations about healthful eating and weight associations with adolescent disordered eating behaviors. *JAMA Pediatrics*, *167,* 746–753. doi:10.1001/jamapediatrics.2013.78

Berge, J. M., Wall, M., Larson, N., Eisenberg, M. E., Loth, K. A., & Neumark-Sztainer, D. (2014). The unique and additive associations of family functioning and parenting practices with disordered eating behaviors in diverse adolescents. *Journal of Behavioral Medicine*. 37, 205-217

Bertoli, S., Leone, A., Ponissi, V., Bedogni, G., Beggio, V., Strepparava, M. G., & Battezzati, A. (2015). Prevalence of and risk factors for binge eating behaviour in 6930 adults starting a weight loss or maintenance programme. *Public Health Nutrition*, 1–7. doi:10.1017/S1368980015001068

Bronfenbrenner, U. (1979). *The Ecology of Human Development: Experiments by Nature and Design*. Cambridge, MA: Harvard University Press.

Brown, T. A., & Keel, P. K. (2012). The impact of relationships on the association between sexual orientation and disordered eating in men. *International Journal of Eating Disorders*, *45*(6), 792–799. doi:10.1002/eat.22013

Bryant-Waugh, R. J., Cooper, P. J., Taylor, C. L., & Lask, B. D. (1996). The use of the eating disorder examination with children: A pilot study. *The International Journal of Eating Disorders*, *19*, 391–397. doi:10.1002/(SICI)1098-108X(199605)19:4<391::AID-EAT6>3.0.CO;2-G

Chandy, J. M., Harris, L., Blum, R. W., & Resnick, M. D. (1994). Disordered eating among adolescents whose parents misuse alcohol: Protective and risk factors. *International Journal of the Addictions*. 29, 505-516.

Chandy, J. M., Harris, L., Blum, R. W., & Resnick, M. D. (1995). Female adolescents of alcohol misusers: Disordered eating features. *International Journal of Eating Disorders*, 17, 281-289

Cohen, S., & Wills, T. (1985). Stress, social support and the buffering hypothesis. *Psychological Bulletin*, *98*, 310-357.

Cordero, E. D., & Israel, T. (2009). Parents as protective factors in eating problems of college women, *Eating Disorders: The Journal of Treatment & Prevention*, 17, 146-161.

Cotton, M. A., Ball, C., & Robinson, P. (2003). Four simple questions can help screen for eating disorders. *Journal of General Internal Medicine*, *18*, 53–56.

Croll, J. K., Neumark-Sztainer, D., Story, M., Ireland, M. (2002). Prevalence and risk and protective factors related to disordered eating behaviors among adolescents: Relationship to gender and ethnicity. *Journal of Adolescent Health*. 31, 166-175

Fairburn, C. G., & Beglin, S. J. (1994). Assessment of eating disorders: Interview or self report questionnaire? *International Journal of Eating Disorders*, *16*, 363–370.

Ferreiro, F., Seoane, G., & Senra, C. (2012). Gender-related risk and protective factors for depressive symptoms and disordered eating in adolescence: A 4-year longitudinal study. *Journal of Youth and Adolescence*. 41, 607-622

Fonseca, H., Ireland, M., & Resnick, M. D. (2002). Familial correlates of extreme weight control behaviors among adolescents. *International Journal of Eating Disorders*. 32, 441-448

Francisco, R., Narciso, I., & Alarcao, M. (2013). Individual and relational risk factors for the development of eating disorders in adolescent aesthetic athletes and general adolescents. *Eating and Weight Disorders*. 18, 403-411

French, S. A., Leffert, N., Story, M., Neumark-Sztainer, D., Hannan, P., & Benson, P. L. (2001). Adolescent binge/purge and weight loss behaviors: Associations with developmental assets. *Journal of Adolescent Health*. 28, 211-221.

Fulkerson, J. A., Story, M., Mellin, A., Leffert, N., Neumark-Sztainer, D., & French, S. A. (2006). Family Dinner Meal Frequency and Adolescent Development: Relationships with Developmental Assets and High-Risk Behaviors. *Journal of Adolescent Health*. 39, 337-345.

Garner, D. M. (1991). *Eating Disorder Inventory—2: Professional manual*. Odessa: Psychological Assessment Resources.

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

Gormally, J., Black, S., Daston, S., & Rardin, D. (1982). The assessment of binge eating severity among obese persons. *Addiction Behaviour*, *7*, 47–55.

Haines, J., Gillman, M. W., Rifas-Shiman, S., Field, A. E., & Austin, S. B. (2009). Family Dinner and Disordered Eating Behaviors in a Large Cohort of Adolescents. *Eating Disorders*, *18*, 10–24. doi:10.1080/10640260903439516

Haines, J., Kleinman, K. P., Rifas-Shiman, S. L., Field, A. E., & Bryn Austin, S. (2010). Examination of shared risk and protective factors for overweight and disordered eating among adolescents. *Archives of Pediatrics and Adolescent Medicine*, *164*(4), 336–343. doi:10.1001/archpediatrics.2010.19

Kmet, L., Lee, R., & Cook, L. (2004). *Standard Quality Assessment Criteria for Evaluating Primary Research Papers from a Variety of Fields*. Alberta: Alberta Heritage Foundation for Medical Research.

Kraemer, H., Kazdin, A., Offord, D., Kessler, R., Jensen, P., & Kupfer, D. (1997). Coming to Terms with the Terms of Risk. *Archives of General Psychiatry*, *54*, 337–343.

Kunstman, J. W., Smith, A. R., & Maner, J. K. (2014). Overpowering restriction: Power reduces restriction among self-critical perfectionists. *Journal of Social and Clinical Psychology*. 33, 630-652.

Lampis, J., Agus, M., & Cacciarru, B. (2014). Quality of family relationships as protective factors of eating disorders: An investigation amongst Italian teenagers. *Applied Research in Quality of Life*. 9, 309-324.

Levine, M. P. & Smolak, L. (2016) The role of protective factors in the prevention of negative body image and disordered eating. *Eating disorders*. 24, 39-46.

Loth, K., Wall, M., Choi, C.-W., Bucchianeri, M., Quick, V., Larson, N., & Neumark-Sztainer, D. (2015). Family meals and disordered eating in adolescents: Are the benefits the same for everyone? *International Journal of Eating Disorders*, *48*(1), 100–110.

Maloney, M., McGuire, J., Daniels, S., & Specker, B. (1988). Reliability testing of a children's version of the Eating Attitudes Test. *Journal of the American Academy of Child and Adolescent Psychiatry*, *27*, 541–543.

Maor, M. & Cwikel, J. (2015) Mother's strategies to strengthen their duaghters' body image. *Feminism Psychology. 26,* 11-29.

Masten, A. N. N. S., Best, K. M., & Garmezy, N. (1991). Resilience and development : Contributions from the study of children who overcome adversity. *Development and Psychopathology*, *2,* 425–444.

Mazur, J., Dzielska, A., Malkowska-Szkutnik, A. (2011). Psychological determinants of selected eating behaviours in adolescents. *Medycyna Wieku Rozwojowego*, *15*(3), 240–249.

McVey, G. L., Pepler, D., Davis, R., Flett, G. L., & Abdolell, M. (2002). Risk and protective factors associated with disordered eating during early adolescence. *The Journal of Early Adolescence*. 22, 75-95.

Micali, N., Hagberg, K. W., Petersen, I., & Treasure, J. L. (2013). The incidence of eating disorders in the UK in 2000-2009: findings from the General Practice Research Database. *BMJ Open*, *3*.

Neumark-Sztainer, D. (2011). Prevention of Eating Disorders in Children and Adolescents. In D. Le Grange & J. Lock (Eds.), *Eating Disorders in Children and Adolescents: A Clinical Handbook* . Guildford: Guilford Press.

Neumark-Sztainer, D., Eisenberg, M. E., Fulkerson, J. A., Story, M., & Larson, N. I. (2008). Family meals and disordered eating in adolescents: Longitudinal findings from Project EAT. *Archives of Pediatrics and Adolescent Medicine*, *162*, 17–22. doi:10.1001/archpediatrics.2007.9

Neumark-Sztainer, D., Wall, M. M., Haines, J. I., Story, M. T., Sherwood, N. E., & van den Berg, P. A. (2007). Shared Risk and Protective Factors for Overweight and Disordered Eating in Adolescents. *American Journal of Preventive Medicine*, *33,* 359-369

Neumark-Sztainer, D., Wall, M., Story, M., & Fulkerson, J. A. (2004). Are family meal patterns associated with disordered eating behaviors among adolescents? *Journal of Adolescent Health*, *35*, 350–359. doi:10.1016/j.jadohealth.2004.01.004

Neumark-Sztainer, D., Wall, M., Story, M., & Sherwood, N. E. (2009). Five-year longitudinal predictive factors for disordered eating in a population-based sample of overweight adolescents: Implications for prevention and treatment. *International Journal of Eating Disorders*. 42, 664-672.

Nicholls, D. E., & Viner, R. M. (2009). Childhood risk factors for lifetime anorexia nervosa by age 30 in a national birth cohort. *Journal of the American Academy of Child and Adolescent Psychiatry*. 48.791-799.

Perkins, D. F., Luster, T., & Jank, W. (2002). Protective factors, physical abuse, and purging from community-wide surveys of female adolescents. *Journal of Adolescent Research*. 17, 377-400.

Rodgers, R., & Chabrol, H. (2009). Parental attitudes, body image disturbance and disordered eating amongst adolescents and young adults: A review. *European Eating Disorders Review*. 17, 137-151.

Rutter, M. (1987). Psychosocial resilience and protective mechanisms. *The American Journal of Orthopsychiatry*, *57*, 316–331. doi:10.1111/j.1939-0025.1987.tb03541.x

Schirk, D., Lehman, E., Perry, A., Ornstein, R., & McCall-Hosenfeld, J. (2015). The impact of social support on the risk of eating disorders in women exposed to intimate partner violence. *International Journal of Women's Health*, 919-931. doi:10.2147/IJWH.S85359

Scoffier, S., Maiano, C., & d'Arripe-Longueville, F. (2010). The effects of social relationships and acceptance on disturbed eating attitudes in elite adolescent female athletes: The mediating role of physical self-perceptions. *International Journal of Eating Disorders*. 43, 65-71.

Seligman, M. E. P., & Csikszentmihalyi, M. (2000). Positive psychology: An introduction. *American Psychologist*, *55*, 5–14. doi:10.1037//0003-066X.55.1.5

Shaw, H., Stice, E., & Becker, C. B. (2009). Preventing Eating Disorders. *Child and Adolescent Psychiatric Clinics of North America*, *18*, 199–207. doi:10.1016/j.chc.2008.07.012

Steck, E. L., Abrams, L. M., & Phelps, L. (2004). Positive Psychology in the Prevention of Eating Disorders. *Psychology in the Schools. 41,* 111-117.

Steiner, H., Kwan, W., Shaffer, T. G., Walker, S., Miller, S., Sagar, A., & Lock, J. (2003). Risk and protective factors for juvenile eating disorders. *European Child & Adolescent Psychiatry*, *12 Suppl 1*, I38–6. doi:10.1007/s00787-003-1106-8

Stice, E. (1999). Clinical implications of psychosocial research on bulimia nervosa and binge-eating disorder. *Journal of Clinical Psychology*. 55, 675-683.

Stice, E. (2002). Risk and maintenance factors for eating pathology: A meta-analytic review. *Psychological Bulletin*, *128*, 825–848. doi:10.1037//0033-2909.128.5.825

Stice, E., Shaw, H., Marti, N.C. (2007). A meta-analytic review of eating disorder prevention programmes: encouraging findings. *Annual Review Clinical Psychology*. 3, 207-31

Stunkard, A. J., & Messick, S. (1985). The three-factor eating questionnaire to measure dietary restraint, disinhibition and hunger. *Journal of Psychosomatic Research*, *29*, 71–83. doi:10.1016/0022-3999(85)90010-8

Sweetingham, R., & Waller, G. (2008). Childhood Experiences of Being Bullied and Teased in the Eating Disorders. *European Eating Disorders Review : The Journal of the Eating Disorders Association*, *16*, 401–407. doi:10.1002/erv

Thelen, M. H., Farmer, J., Wonderlich, D. A., & Smith, M. (1991). A revision of the bulimia test: The BULIT-R. *Psychological Assessment*, *3*, 119–124.

Twamley, E. W., & Davis, M. C. (1999). The sociocultural model of eating disturbance in young women: The effects of personal attributes and family environment. *Journal of Social and Clinical Psychology*. 18, 467-489.

Waller, G. (1994). Bulimic women's perceptions of interaction within their families. *Psychological Reports*, *74*(1), 27–32. doi:10.2466/pr0.1994.74.1.27

Wang, M. L., Peterson, K. E., Richmond, T. K., Spadano-Gasbarro, J., Greaney, M. L., Mezgebu, S., Byrn Austin, S. (2013). Family Physical Activity and Meal Practices Associated With Disordered Weight Control Behaviors in a Multiethnic Sample of Middle-School Youth. *Academic Pediatrics*, *13*(4), 379–385.

Wang, M. L., Walls, C. E., Peterson, K. E., Richmond, T. K., Spadano-Gasbarro, J., Greaney, M. L., … Bryn Austin, S. (2013). Dietary and physical activity factors related to eating disorder symptoms among middle school youth. *The Journal of School Health*, *83*(1), 14–20. doi:10.1111/j.1746-1561.2012.00742.x

Part 2: Empirical Paper

Believe the hype? An exploration of the impact of intermittent fasting (5:2) diets on disordered eating, binge eating and mood in healthy participants

Abstract

Background: Intermittent fasting (IF) diets, in which food intake is restricted on some days of the week and unrestricted on others, have been popularised in the media as an easy and effective way to lose weight and improve health. The eating disorder (ED) field has been sceptical, suggesting that calorie restriction and fasting may lead to rises in disordered eating, preoccupation with food and bingeing. Few studies have yet investigated the psychological and behavioural impact of eating in this way on dieters.

Aims: This prospective study explored the impact of starting the '5:2 diet', a popular version of IF, on symptoms of ED, binge eating, food craving and mood, and looked for interactions with risk factors for the development of disordered eating behaviours such as dieting history, low self-esteem, dichotomous thinking, weight suppression and weight dissatisfaction.

Method: Using online media, English speaking healthy individuals planning to start 5:2 IF were recruited globally. Participants completed food diaries and online self-report measures prior to starting IF (N=144), and again after four weeks of IF (N=81).

Results: After four weeks of IF, participants reported reduced ED symptoms, binge frequency, binge eating disorder symptoms, food craving and mood symptoms. Exploration of risk factors showed an interaction with dieting history, such that only those who had dieted in the past showed significant reductions in ED symptoms, binge frequency and food craving. Reductions in ED symptoms on some measures were positively correlated with weight dissatisfaction and dichotomous thinking, and negatively correlated with self-esteem. Participants did not increase their calorie intake on non-fasting days after starting the diet.

Conclusions: Contrary to expectation, commencing IF did not lead to increases in disordered eating symptoms or binge eating in healthy people, and in fact appeared to be associated with reductions in these behaviours in those completing the one-month study period. These findings should be interpreted with care due to the high drop-out rate and lack of other diet control.

Introduction

1.1 Background

In recent years, intermittent fasting (IF) diets, involving fasting or restricting calorie intake for short periods of time, interspersed with periods of eating normally, have emerged as a popular dieting approach. A popular version of IF is the '5:2' or 'Fast Diet' (Mosley & Spencer, 2013), in which participants restrict calorie intake on two days of the week and eat normally for the other five. IF has been presented in the media as an easy and evidence-based means to improve health, and lose weight. The rationale for such diets comes from the field of 'calorie restriction' (CR) research. CR (to around 60% to 70% of normal intake, while maintaining 'optimal nutrition' through careful consumption of adequate micronutrients) has been posited as a means to extend longevity, by placing the body into a 'restore and repair' state (see Vitousek, Gray, & Grubbs, (2004) for a review). Although there is some evidence (from animal studies) of the potential of long-term CR to extend life spans, even the strongest proponents of the approach admit that for humans eating restricting food intake in this way over months, years and decades is incredibly difficult, although some people are attempting it (see CR Society International, 2002). A number of researchers therefore began to take interest in the idea of briefer 'intermittent' periods of fasting as a means to get the benefits of restriction and fasting in a more realistically achievable and maintainable way.

While a growing number of studies have investigated the impact of IF on a range of physical health biomarkers, there has been a marked neglect of psychological and behavioural outcomes. Researchers in the eating disorders field, citing classical starvation studies as well as research in clinical populations, have argued that extreme dietary restriction and restraint are associated with a range of adverse outcomes, including disordered eating symptoms, binge eating, preoccupation with food, and symptoms of depression, stress and anxiety (Vitousek, Manke, Gray, & Vitousek, 2004). It is unclear to what extent, if at all, these risks may apply to IF diets, yet thousands of dieters are undertaking such programmes with little or no supervision.

While some may already be reaping the benefits of improved health, it is possible that certain at-risk individuals might already experiencing harmful effects.

One of the few published studies directly investigating these factors looked at the impact of a clinic controlled 'alternate day fasting' (eating 25% of baseline energy needs, with meals provided by the clinic, every other day, and eating 'ad libitum' on other days) in a group of 59 obese women (Hoddy et al., 2015). They found that after 8 weeks of this diet, participants reported reduced depression and binge-eating, and increased restrictive eating on the brief Multidimensional Assessment of Eating-Disorder Symptoms screening measure. Another study looking at weight loss and biomarkers of diseases in obese women, compared those doing a clinic-controlled '5:2' type diet IF to a control group following a more 'traditional' low calorie diet (Harvie et al., 2011). More women in the IF group than the low calorie group reported minor adverse psychological effects of dieting, such as difficulties concentrating, a bad temper and preoccupation with food. Fewer women in the IF group reported improvements in mood and confidence, while both groups experienced equivalent weight loss. As with much of the research onto the biological effects of IF, both of these studies included solely obese participants, and participants were provided with direct instruction about what they should eat on fasting days, rather than making their own food choices.

Other studies have looked at the effects of IF in young women, a group long recognised as at higher risk for developing problems with disordered eating (American Psychiatric Association, 1994). One study looking at the impact of IF on mood asked a group of 19 female students to fast (restrict to 500 calories) for two consecutive days, comparing self-reported mood, hunger, and distraction to non-fasting days (Appleton & Baker, 2015). They found that on fasting days, participants reported lower positive mood and higher negative mood. The degree of mood disturbance reported was associated with the level of distraction reported by the participant, although not with the level of hunger reported. One older study made use of IF (instructing participants to restrict to 500 calories every other day) as an analogue for bulimic symptoms in

young women who had not dieted (Laessle, Platte, Schweiger, & Pirke, 1996). They found that participants steadily increased the calories they ate and experienced increased impulse to overeat on 'non-fast' days over the four weeks of the study, and reported significantly more symptoms of depression, irritability, preoccupation with food, and drive to eat more than allowed on fasting days. The study did not, however, observe any actual bingeing behaviour during the study.

More is known about the biological impacts of IF, although here too there is a paucity of studies and findings are inconclusive. Studies, again often including participants who are obese or who have conditions such as diabetes, have shown some improvements on biomarkers of breast cancer, diabetes and cardiovascular diseases (Harvie et al., 2011; Johnson, Laub, & John, 2006; Varady & Hellerstein, 2007). However, a number of recent reviews have suggested that despite the media excitement, IF may be no more effective than other diets for promoting weight loss, and may have no more impact on biological markers than equivalent weight loss from continuous restriction (Seimon et al., 2015; Tinsley & La Bounty, 2015).

Moving beyond IF to look at restriction and dieting more generally, dieting to lose weight has long been recognised through correlational and prospective studies as a risk factor or precipitating factor in the development of a whole range of eating disorders (Polivy & Herman, 2002; Stice, Marti, & Durant, 2011). Restriction of caloric intake has also been implicated in the development of a wide variety of 'disordered' eating behaviours and experiences in the general population, such as preoccupation with food, weight and shape, and binge eating (Herman & Polivy, 1975; Keys, Brozek, Henschel, Mickelson, & Taylor, 1950; Ogden, 2010; Stice, 2001). A variety of mechanisms have been posited to explain these links. Cognitive theorists have attempted to explain why attempts at dietary restraint can lead to over- and binge- eating by referring to Abstinence Violation or the 'What the hell' effect, the idea that once a cognitively set 'diet' boundary or strict 'diet rule' has been crossed or broken, an 'all or nothing' view and negative emotional impact makes the individual vulnerable to an episode of overeating (Herman & Polivy, 1984; Ogden, 2010; Wilson, Fairburn, & Agras, 1997). Where this overeating is

accompanied by a sense of loss of control, it can be defined as a 'binge' (American Psychiatric Association, 1994). Others have suggested physiological or motivational mechanisms with an adaptive evolutionary function to protect against starvation, which mean that humans respond to a reduction in calorie intake with increases in hunger, physical changes, preoccupation with food and food craving, attributing higher reward value to food, and other emotional and behavioural changes (Stice et al., 2008; Tapper & Pothos, 2010).

Here too though, there are inconsistencies in the literature. More recently, the consensus that attempts to diet result in negative psychological consequences, bingeing and other ED symptoms has been challenged by experimental studies in which participants following diets have experienced no negative, and even positive, impacts on their psychological wellbeing and have not developed bingeing and other ED symptoms (Stice, 2002; Williamson et al., 2008). It is clear, both from longitudinal studies and also from the experience of everyday dieters, that not all dieters go on to experience highly adverse outcomes. It has been suggested that some forms of dieting behaviour might be particularly likely to lead to difficulties. For example, in a longitudinal study of female adolescents, Stice et al. (2008) found that fasting behaviour, defined here as going without eating for 24 hours for the purposes of weight control, was a strong predictor of future onset of bingeing and bulimic symptoms, over and above any association with dietary restraint. It is also likely that some dieters are more likely to experience adverse consequences than others. Researchers have attempted to identify characteristics which might make a dieters particularly 'at risk' of developing disordered eating symptoms, bingeing, or other negative psychological consequences. Epidemiological research has shown clearly that EDs are more common in young females, although other groups can also be affected (American Psychiatric Association, 1994). A wide variety of other potential risk factors have been identified, such as low pre-existing body weight, 'suppressed' weight, body dissatisfaction, low self-esteem, self-esteem which is highly dependent on shape and weight, and a 'black and white' thinking style (Button, Sonuga-Barke, Davies, & Thompson, 1996; Fairburn, Cooper, Doll, &

Davies, 2005; Geller, Johnston, & Madsen, 1997; Ogden, 2010; Polivy & Herman, 2002; Stice et al., 2011; Stice et al., 2008; Vitousek & Hollon, 1990; Wilson et al., 1997).

1.2 Aims

Research into the psychological and behavioural impact of IF is necessary to allow dieters to make informed choices about choosing to eat in this way. At present, the studies conducted into these factors have made use of restricted populations (female only, either obese women or young women), which are anecdotally not representative of the mainstream population following these diets. The forms of IF used in these studies (clinic-controlled, alternative day fasting, or only very brief fasting) differ from the form of IF currently in most popular use (generally so called '5:2' fasting applied flexibly with little supervision). The results of studies appear somewhat contradictory, and the applicability of their findings to everyday IF dieters is unclear. This study therefore aims to follow healthy adult dieters as they begin self-directed IF in an attempt to answer the following questions:

- Does commencing a 5:2 IF diet result in change on measures of disordered eating and other related experiences such as binge-eating and preoccupation with food/food craving?
- What is the impact of starting IF on mood factors such as depression, stress and anxiety?
- Does commencing IF lead to an increase in eating disorder symptoms, binge eating and/or preoccupation with food/food craving in individuals who could be considered more 'at risk' of developing these kinds of difficulties?

Based on these research questions, and the discourse within the eating disorder research community outlined above, a number of hypotheses were developed:

- Participants commencing IF may report increases in eating psychopathology, binge eating and food craving.

- Participants following IF may experience an increase in adverse mood experiences (depression, stress or anxiety).
- Participants scoring higher on risk factors for disordered eating will experience a greater adverse impact on eating related outcomes after commencing IF.

2.0 Methods

2.1 Participant characteristics

Participants were males and females who had decided that they wanted to undertake a 5:2 intermittent fasting diet, but had not yet commenced the diet. All participants met the following inclusion criteria:

a) Individuals intending to commence a 5:2 intermittent fasting diet within the next few weeks.
b) Aged 18 and above
c) Sufficient level of English language and computer literacy to complete the study
d) No current or history of eating disorders
e) No current diagnosed mental health problem.
f) Not currently pregnant, or with health conditions such as diabetes which would make fasting medically unadvisable.

Participants were recruited though online advertising, social media and word of mouth (see Appendix B for advertising material). All parts of the study were completed online or over the telephone, allowing recruitment internationally. 73.84% of participants completing the study were currently residing in the UK, 12.35% were resident in the USA, 8.64% were resident in Australia or New Zealand, with the remaining participants resident in other European countries, Asia or Central America. The mean age of participants completing the study was 45.22 years (SD=10.71). 85.19% were female and 14.82% were male. 83.95% reported their ethnicity as

White or Caucasian (6.17% Asian, 3.70% Afro-Carribbean, 3.70% Mediterranean/Hispanic, 2.47% Mixed ethnicity). Mean body mass index at the start of the study was 27.23 (SD=5.07).

The power calculation for this study was informed by the work of Telch and Agras (1993) who explored the impact of following a very low calorie diet on the frequency of bingeing behaviour. Like most studies in the field, this study used overweight participants. They found an increase in bingeing behaviour following completion of a three month diet in those that had no history of bingeing, with an effect size of d=.66 at the end of the diet period. A power calculation was carried out using G Power (Faul, Erdfelber, Lang, & Buchner, 2007), giving an estimated sample size of 31 to provide 80% power with an alpha level of 0.01 for a dependent means design. Given the differences in design and scope between this study and our own, and the wide differences in effect sizes found in related fields, the convention from Cohen (1992) for the size of a medium effect size (d=.05), was used to calculate a more conservative minimum sample size of 51 to identify a main effect of commencing the diet on a core outcome measures.

2.2 Design

The study made use of a within subjects quasi-experimental design. Participants completed all measures twice, once at baseline prior to starting the diet, and once after 28 days of following the diet.

2.3 Measures

Key outcomes:

Eating disorder symptomology and binge eating: Eating disorder symptomology was assessed using the Eating Disorder Examination Questionnaire (EDEQ), a 28-item self- report instrument adapted from the clinical Eating Disorders Examination interview (Fairburn & Beglin, 1994). This 28-item questionnaire measure has been shown to have high reliability (.72 to .93) and validity (sensitivity=0.83, specificity=0.96 in a community sample) (Mond, Hay, Rogers & Beumont, 2004; Rose, Vaewsorn, Rosselli-Navarra, Wilson & Weissman, 2013). The global

EDE-Q score is a measure of global eating disorder symptomology. The EDE-Q also asks participants to list how often they have engaged in binge eating (described as eating a large amount of food, accompanied by a sense of loss of control over eating), providing an estimate of the frequency of binge eating episodes. Binge eating symptoms were also assessed using the Binge Eating Disorder Test (BEDT), a 26 item scale adapted from the BULIT-R bulimia scale, which assesses binge eating, loss of control, and body dissatisfaction. This scale shows high sensitivity (100% for a cut off value of 75) and specificity (100% for a cut off value of 75) to binge eating disorder, and high internal consistency reliability (α=.96) (Thelen, Farmer, Wonderlich, & Smith, 1991; Vander Wal, Stein, & Blashill, 2011).

Other disordered-eating-related outcomes: Preoccupation with food and eating and food craving were assessed using the State Food Craving Questionnaire (FCQ-S) (Cepeda-Benito, Gleaves, Williams, & Erath, 2000). This 15-item scale demonstrates high validity ($F(1,102) > 11.40$, $p<.001$) and internal reliability (α=.96). Test re-test reliability for this scale is lower (r=.56) as it is designed to be a 'state' measure Individuals with bulimic disorders score significantly higher on this scale than controls (Van den Eynde et al., 2012).

Food intake: Participants completed a food diary for one week at baseline and for the last week of the 28 day diet period (see Appendix B). The food diary asked participants to record everything that they ate or drank, specifying the amount, along with information about the time, situation, hunger level and sense of control/loss of control, and any other thoughts/emotions associated with the eating episode. Under-reporting of intake on food diaries is common. . To increase validity and adherence participants were asked to complete the diary as soon as possible after consumption. They were encouraged to be as honest and accurate as possible and reassured that their data was confidential. Keeping a food diary may lead to changes in food intake or have other unintended consequences (Anderson, Lundgren, Shapiro, & Paulosky, 2004). However the 'Fast Diet' book and online guidance suggest that individuals following the diet keep a food diary

as a means of promoting adherence, meaning that recording intake in this way was not inconsistent with naturalistic intermittent fasting (Mosley & Spencer, 2013).

Mood: Mood was assessed using the DASS-21, a standardised self-report mood scale which assesses depression and low mood, anxiety, and stress and irritability (Lovibond & Lovibond, 1995). This commonly used 21-item measure has been shown to be highly reliable (.87 to .94) and shows good concurrent validity (r=.68 to .85) (Anthony, Bieling, Cox, Enns, Swinson & Haynes, 1998).

Risk factors:

Demographics and descriptive variables: All participants completed a form asking them to give their age, gender, ethnicity, height and weight (allowing calculation of BMI), and history of dieting.

Body dissatisfaction and weight suppression: Participants were asked to state their ideal weight and highest ever weight. This was combined with information about their current weight to calculate their current level of weight suppression (highest weight minus current weight) (according to the method of Lowe, 1993) and body dissatisfaction (ideal weight minus current weight) (as in Mizes, Heffner, Madison, & Varnado-Sullivan, 2004).

Self-esteem: Participants completed the Shape and Weight Based Self Esteem Inventory (SAWBS) as a measure of the degree to which their self-esteem is dependent on their body shape and weight (Geller et al., 1997). This scale has been demonstrated to be reliable (.81) and to have high validity (r=.83). To measure more global self esteem, they also completed the Rosenberg Self Esteem Scale (SES) (Rosenberg, 1965). This very commonly used 11-item scale has consistently been shown to have high convergent validity (r=.57 to .79) , internal reliability (.91) and test-retest reliability (Sinclair, Blais, Gansler, Sandberg, Bistis & Locicero, 2010).

Dichotomous thinking: The Dichotomous Thinking in Eating Disorders Scale is a self report measure of 'black and white' thinking in eating-specific and general domains, and was used to

assess the degree to which participants engaged in dichotomous thinking (Byrne, Allen, Dove, Watt, & Nathan, 2008). This 11-item scale shows high internal reliability (α=.88) and has been validated with eating disordered, obese and control populations (r=.41 to .62, p<.01).

2.4 Procedure

Individuals responding to the advertisements were emailed a copy of the Information Sheet for Participants and a flow chart summarising the study protocol (see Appendix D). Those who replied to this email to say that they were interested in taking part were contacted by a researcher to arrange an initial telephone screening call (for UK participants) or Skype call (for those based outside of the UK). The purpose of this call was to explain the study process and to ensure that participants met all inclusion criteria for the study. Following this call, participants were emailed a copy of the consent form to sign and return, and were assigned a participant number. As an incentive for participation, all participants were informed that they would be entered into two prize draws to win online shopping vouchers (valued between £20 and £100), with one draw held for all participants completing baseline measures, and a further draw for those completing all parts of the study. They were also informed that a donation of £1 per participant would be made to charity, and were given the opportunity to vote for the charity this donation would be made to.

Participants who consented to take part in the study were then asked to name the date that they planned to commence 5:2 intermittent fasting. Prior to starting the diet, participants were emailed a food diary to complete for one week while eating normally. At the end of this week, participants received an email prompting them to complete the baseline outcome measures (eating disorder symptomology, binge eating, food craving, mood, and body weight) and measures of risk factor variables (age, gender, history of dieting, weight dissatisfaction and suppression, self-esteem, proportion of self-esteem dependent on weight and shape, and dichotomous thinking). All measures were completed using Qualtrics online survey software (Qualtrics, 2005).

Participants then began following a '5:2' intermittent fasting diet, as described by Mosley and Spencer (2013), requiring them to restrict their calorie intake to 500 (women) or 650 (men) calories on two 'fast' days per week and eat normally with no restrictions on the other five days. Participants were free to choose on which days they 'fasted' (Mosley & Spencer, 2013), and were encouraged to follow the diet as strictly or loosely as they would have if they had not been involved in the study, to provide a 'real world' picture of the sustainability and impact of attempting to eat in this way.

After three weeks of following the diet, participants received another email, asking them to complete a food diary for the fourth week of following the diet. At the end of the four week period, participants received a final email prompt, asking them to complete the outcome measures (eating disorder symptomology, binge eating, food craving, mood, and body weight) for the second time, and to submit their completed food diaries. Participants were instructed to complete these measures on a non-fasting day. After this, participants were advised that they were free to continue following the diet or to stop intermittent fasting if they preferred to do so.

2.5 Ethics

Ethical approval for the study was obtained from the University College London (UCL) Research Ethics Committee (Project ID Number: 3529/001, see Appendix E). All participants were provided with an Information Sheet about the study, and had the opportunity to ask questions, before being asked to sign a form to give informed consent. Participants were also asked to indicate continuing consent prior to completing each testing session by ticking a box on the online survey. Data from the study were anonymised and kept confidential. As the potential risks/ benefits of intermittent fasting diets are unknown, participants were recruited who had already independently decided to follow the diet. Participants disclosing a currently diagnosed mental health problems or eating disorder, those with a history of eating disorders, and those who were pregnant or had health conditions such as diabetes which would make fasting

medically unadvisable were not included in the study, and were advised of the potential risks of intermittent fasting (in line with NHS advice). Participants were advised that their participation in the study did not mean that they needed to adhere more strictly to the diet, and that if they began to feel unwell they should stop restricting. Participants were also made aware that they had the right to withdraw from the study at any time without giving a reason.

2.6 Data processing and statistical analysis

Data processing:

Baseline and final outcome data were matched for each participant using participant numbers. Questionnaires were scored according to guidance provided for each measure. To allow comparison of baseline, fasting and non-fasting days while minimising data processing burden for the researcher, food diary data was studied for each participant for a random selection of two days for baseline diaries, and on one fasting and one non-fasting day for final diaries. Data was processed using the MyFitnessPal website (MyFitnessPal, 2011) to provide an estimate of calories consumed and the percentage of intake that was carbohydrate or fat.

Change over time and interactions:

A series of paired samples t-tests or non-parametric alternatives were used to assess change over time on outcome variables (EDEQ global score, binge eating frequency on the EDE-Q, BEDT total score, State Food Craving Scale score and DASS-21 total score, calories consumed). To account for potential inflation of the family-wise Type 1 error rate brought about by multiple comparisons, the Bonferroni corrected criterion threshold for these tests was calculated as $p<.01$ ($=.05/5$). . To explore interactions between risk factor variables and outcome variables, for categorical risk factor variables (gender and dieting history), mixed ANOVAs were run with time as a within-subjects variable and the risk factor (gender or diet history) as a between-subjects variable. For continuous risk factor variables, a series of repeated measures ANCOVAs with risk factor variables as covariates. The Bonferroni corrected criterion

threshold for these tests was calculated as $p<.001$ (=.05/20). For food diary data repeated measures ANOVAs were run with four time points (pre-diet day 1, pre-diet day 2, non-fasting day and fasting day) to look for differences in dietary intake at different points in the study.

Missing data:

Only data for participants completing both baseline and final outcome measures were included in analyses. A series of t-tests and chi-squared analyses were used to establish whether there were any differences at baseline between those completing the study and those that did not complete final measures.

Assumptions of normality:

Data for change in outcome variables was subjected to tests of normality to assess adherence to assumptions for parametric testing. Skewness, kurtosis and Kolmogorov-Smirnov values, as well as inspection of histograms, indicated that data for change in binge-eating frequency on the EDE-Q, BEDT scores, State Food Craving scores and DASS-21 scores were not normally distributed and did not meet assumptions for parametric testing. Non-parametric analyses (Wilcoxon signed-rank tests) were run to assess main effects. There were no differences between the results of parametric and non-parametric analyses. There are no non-parametric alternatives to mixed ANOVAs or ANCOVAs. It has been suggested that the ANOVA analyses are robust to violations of normality, and that the use of non-parametric alternatives is associated with a loss of statistical power (Harwell, Rubinsteing, Hayes, & Olds, 1992). Studies using simulated data have demonstrated that non-normally distributed data does not bias ANOVA results (Schmider, Ziegler, Danay, Beyer, & Bühner, 2010). Furthermore, effects in this study identified as significant using parametric t-tests remained significant when non-parametric alternatives were used. The results of the ANOVA and ANCOVA analyses are therefore included in this paper, but should be interpreted with caution.

2.7 Collaboration

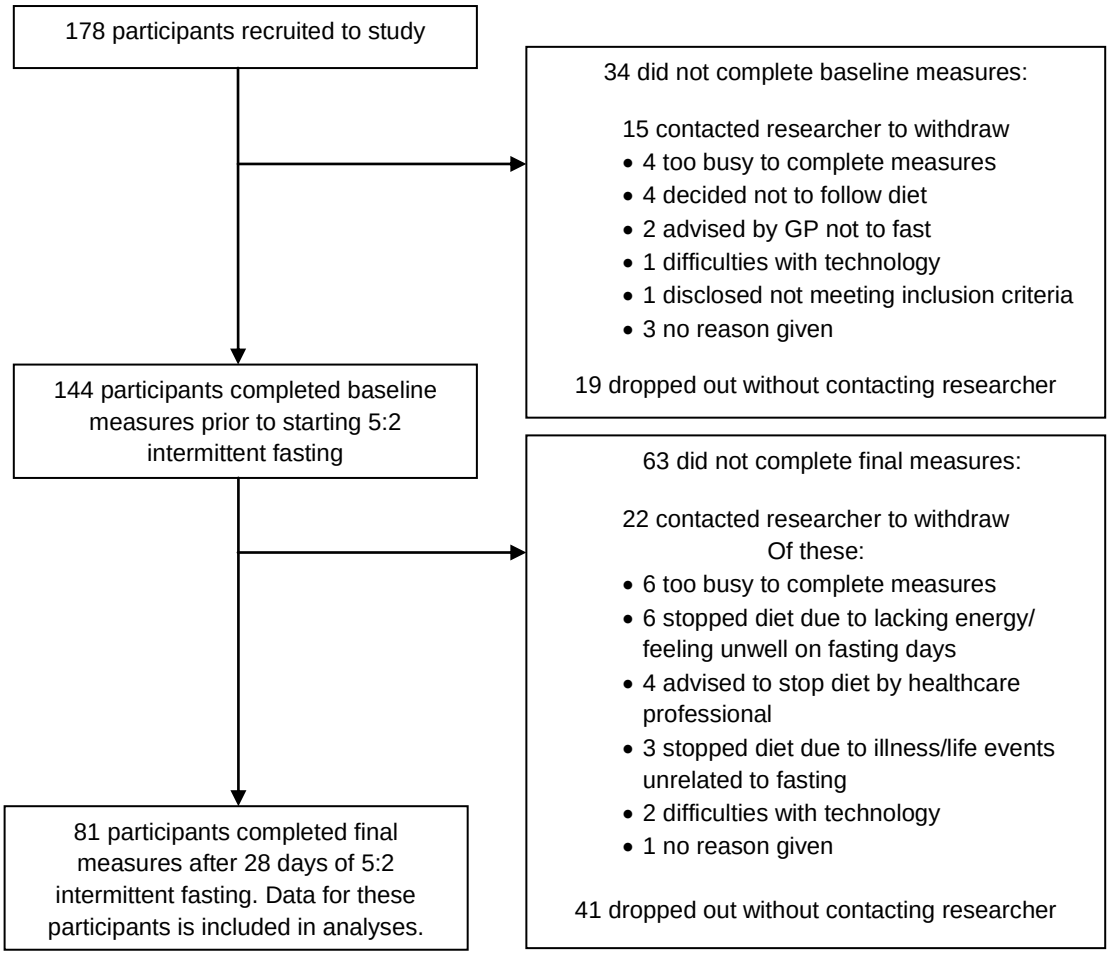
Recruitment and data collection for this study was undertaken as part of a joint project. Details of the other part of this study are reported in Mahony (2016): Nutrition and cognition: Exploring their relationship from two sides of the same coin (Clinical Psychology Doctorate Thesis). See Appendix F for further details of this collaboration.

3.0 Results

3.1 Participant baseline characteristics and attrition

There were 176 participants recruited for the study. 144 completed baseline measures, and of these 81 (56.25%) completed the measures at the end of the study period and were included in analyses. Figure 1 provides an overview of sample attrition at each stage (along with reasons given for withdrawing from the study if available).

Figure 1: Participant numbers and attrition at each stage



Mean baseline scores and standard deviations (or frequencies for categorical variables) for each of the outcome variables, age, gender, BMI and risk factor variables are reported in Table 1, for all participants completing baseline measures. A series of t-tests and chi-squared analyses were used to establish whether there were any significant differences between participants who completed the study (completers) and those who withdrew or dropped out before completing the final measures (non-completers). There were no significant differences between completers and non-completers on any of the outcome measures at baseline. When looking at risk factors, the group completing the study reported higher weight suppression ($t(142)= -2.345$, $p=.02$), and lower weight dissatisfaction ($t(141.965)= 2.257$, $p=.026$) than the group that did not complete final measures. There were no other significant differences between completers and non-completers on age, gender, BMI or any other risk factor.

Table 1: Baseline scores on outcome and risk factor variables for study completers and non-completers

Measure	Completers (n=81)		Non-completers (n=63)	
	Mean	**SD**	**Mean**	**SD**
EDEQ Global score	2.11	1.11	2.05	1.09
EDEQ Binge frequency	3.73	5.38	5.75	7.48
BEDT score	47.24	18.31	49.99	18.71
State Food Craving score	29.80	12.65	28.08	11.59
DASS-21 score	31.72	6.62	31.49	8.07
Age	45.37	10.7685	44.619	10.8738
Gender	85% Female, 15% Male		87% Female, 13% Male	
Body mass index	27.23	5.07	28.02	27.23
Weight dissatisfaction*	10.34	9.15	14.72	10.34
Weight suppression*	6.81	7.80	4.11	6.81
Dieting history (Yes/No)	18% No, 82% Yes		22% No, 78 % Yes	
Self-esteem scale score	21.03	5.67	21.73	5.59
SAWBS %	22.31	16.98	18.45	18.13
Dichotomous thinking score	23.35	7.28	23.21	7.23

** = significant difference between completers and non-completers (p<.05)*

Associations of baseline outcome variables and risk factors

At baseline, there were a number of associations between outcome variables and risk factor variables.

Categorical variables

At baseline (in participants who then went on to complete the study), females scored significantly higher than males for global eating disorder symptomology, as measured on the EDEQ ($t(79)=-2.187$), $p=.032$). There were no other differences between males and females on outcome variables at baseline. Compared with those who had not dieted previously, participants who had dieted in the past scored significantly higher at baseline on global disordered eating on the EDEQ ($t(79)=-3.823$), $p=.001$) and on binge eating disorder symptoms on the BEDT ($t(79)=-2.509$), $p=.014$).

Continuous variables

Full details of correlations between risk factor variables and outcome variables at baseline can be seen in Table 2. Age at baseline was negatively correlated with binge eating disorder symptoms reported on the BEDT and with mood symptoms reported on the DASS-21. Weight suppression was not correlated with any of the outcome variables at baseline. Weight dissatisfaction at baseline was correlated with global disordered eating and frequency of binge eating on the EDEQ, and with binge eating disorder symptoms on the BEDT. Self-esteem score on the SES was negatively correlated with all outcome measures (global EDEQ, binge frequency, BEDT score, State Food Craving scale score and DASS-21 score). The proportion of self-esteem reported as dependent on weight and shape on the SAWBS was correlated with global EDEQ score, binge eating frequency, BEDT score and state food craving at baseline. Dichotomous thinking about food and eating, as assessed using the DTED, was correlated with global EDEQ score, binge eating frequency, BEDT score and state food craving at baseline.

Summary:

There was high attrition over the course of the study. Apart from higher reported weight suppression, and lower weight dissatisfaction, there were no significant differences between study 'completers' and 'non-completers'. Many of the risk factor variables, including self-esteem, shape and weight based self-esteem, weight dissatisfaction and dichotomous thinking about food, were associated with higher scores on disordered and binge eating outcome measures at baseline.

Table 2: Correlations between outcome and risk factor variables at baseline, for participants completing the study

Outcome measure	Risk factor measure					
	Age	Weight suppression	Weight dissatisfaction	SES score	SAWBS score	DTED Score
EDEQ Global score	.004	.106	.287*	-.440**	.421**	.658**
EDEQ Binge frequency	-.215	-.095	.507**	-.404**	.280*	.521**
BEDT score	-305**	.152	.480**	-.560**	.464**	.673**
State Food Craving score	-.165	.129	.077	-.321**	.261*	.395**
DASS-21 Score	-.270*	.157	.044	-370**	.180	.186

**= significant at p<.05, **=significant at p<.01*

3.2 Change after 28 days of 5:2 intermittent fasting

Weight loss and calories consumed

Mean weight loss over the 28 day IF period was 1.94 kilos (SD=3.22), giving a mean BMI change of 0.70 (SD=1.17). Food diary data was available for 65 participants. Mean reported calorie intake, and the percentage of calorie intake coming from carbohydrates and fat, for two random pre-diet days and on one fasting and one non-fasting day after starting the diet are reported in Table 2. The results of a repeated measures ANOVA showed that there was a significant effect of time point on total reported calorie intake ($F(3, 192)=138.609$, $p<.001$). Exploration of pairwise comparisons showed that participants reported consuming significantly fewer calories on fasting days compared to both non-fasting days (mean difference = 1084.5 cal,

standard error= 68.857, p<.001) and to days before starting the diet (mean difference = 1176.8cal/1143.4cal, standard error= 59.097/67.192, p<.001). There were no significant differences between total calories consumed on days prior to the diet and non-fasting days after starting the diet, however, significantly fewer calories were consumed on fasting than on non-fasting days and days before the diet. There was no significant effect of time point on the proportion of calorie intake coming from carbohydrates or fat.

Table 3: Mean calorie intake and proportion of energy intake from carbohydrates and protein for two days pre-diet, non-fasting days, and fasting days

Time Point	Calorie Intake		Proportion carbohydrates (%)		Proportion fat (%)	
	Mean	SD	Mean	SD	Mean	SD
Pre Diet Day 1	1723.12	423.95	43.30	12.10	32.16	10.38
Pre Diet Day 2	1689.73	546.43	45.35	11.13	31.52	10.06
Non-Fasting Day	1630.83	512.27	42.93	12.07	31.59	9.95
Fasting Day	546.32	228.98	42.60	15.23	28.08	13.08

Global eating disorder symptomology:

For those who completed the study, there was a significant reduction in global EDEQ scores indicating a reduction in eating pathology over the 28 day IF period (see Table 4).

Binge-eating:

EDEQ binge-eating frequency: Those completing the 28 day IF period reported a reduction in the frequency of binge-eating episodes on the EDEQ.

BEDT score: After 28 days of IF, participants scored lower on the BEDT, indicating reduced binge eating.

Preoccupation with food and food craving:

Participants completing the study scored lower on the State Food Craving scale after 28 days of 5:2 IF, indicating a reduced experience of food craving and preoccupation with food.

Mood:

Those completing the study scored significantly lower on the DASS-21 after 28 days of IF, indicating reductions in adverse mood experiences (low mood, anxiety and stress).

Table 4: Change in eating pathology, binge-eating, food craving and mood after 28 days of 5:2 IF

Measure	Mean (SD)		Test used	t statistic	df	p	95% CI		Effect size (d)
	Baseline	Final					Lower	Upper	
EDEQ Global score	2.11 (1.11)	1.87 (0.94)	Paired samples t-test	3.708	80	<.001**	0.11	0.37	.430
EDEQ Binge frequency	3.73 (5.63)	1.74 (4.27)	Paired samples t-test	3.739	80	<.001**	0.93	3.05	.414
BEDT score	47.48 (18.30)	41.38 (15.44)	Paired samples t-test	5.458	79	<.001**	3.88	8.32	.635
State Food Craving score	29.79 (12.65)	26.01 (11.11)	Paired samples t-test	2.534	80	0.14*	0.80	6.76	.282
DASS-21 score	31.72 (6.61)	29.78 (6.98)	Paired samples t-test	2.567	80	.012*	0.44	3.44	.285

**= significant at p<.05, **=significant at p<.01*

NOTE: There were no differences between the results of parametric and non-parametric analysis. To allow more complete data reporting, results for parametric tests are reported here. Results of non-parametric tests are available in Appendix G.

Summary:

After 28 days of intermittent fasting, those that completed the study reported significant reductions in global eating pathology on the EDEQ, in frequency of bingeing reported on the EDEQ, in binge eating symptoms reported on the BEDT, in food craving and in symptoms of low mood, anxiety and stress on the DASS-21. Participants consumed significantly fewer calories on fasting days. There were no differences in reported calorie intake on days before the diet and non-fasting days after starting the diet.

3.3 Interactions with risk factors

Categorical variables:

Gender: There was a main effect of gender on global EDEQ score, with females scoring higher than males at both timepoints (Mean difference = .640, standard error =.303, partial eta^2= .053) (see Table 5). There were no significant interactions between gender and any of the outcome variables.

Dieting history: There was a main effect of diet history on global EDEQ score and BEDT score, with a history of dieting associated with higher disordered eating symptomatology reported on both measures**.** There were significant interactions between dieting history and EDEQ score, binge frequency and food craving. Exploration of pairwise comparisons showed that the group of participants who had dieted in the past (N=66) showed significant reductions in EDEQ score (Mean difference=-2.223, SD= .401, p<.001, d=.554), binge frequency (Mean difference =-2.636, SD= .568, p<.001, d=.737) and food craving (Mean difference =-5.227, SD=1.625, p=.002, d=.391), while those who had never dieted in the past (N=12) showed no significant changes on EDEQ (Mean difference =.300, SD= .842, p=.723, d=-.074), binge frequency (Mean difference =.867, SD= 1.191, p=.469, d=-.167) or food craving (Mean difference =2.600, SD= 3.409, p=.448, d=.-.205) after starting IF.

Continuous variables:

Age: There were no significant effects, or interactions, of age on any of the outcome variables.

Weight dissatisfaction: Significant interactions were found between weight dissatisfaction at baseline and change in binge eating frequency on the EDEQ and BEDT score. Further exploration of these associations using Pearson's correlations showed that greater weight dissatisfaction was positively correlated with reduction of binge eating frequency (r(81)=.248, p=.026) and reduction of binge eating disorder symptoms on the BEDT (r(80)=.392, p>.001).

Weight suppression: There were no significant effects of, or interactions with, weight suppression on any of the outcome variables.

Self-esteem: Global EDEQ score and BEDT score showed evidence of interactions with self-esteem scores on the SES. Reduction in global score on the EDEQ was negatively correlated with self-esteem ($r(81)= -.327$, $p=.003$), with those reporting lower self-esteem at baseline showing greater reductions in disordered eating symptomology. Reduction in binge eating disorder symptoms on the BEDT was negatively correlated with self esteem ($r(80)= -.342$, $p=.002$), indicating those reporting lower self esteem reported greater reductions in binge-eating disorder symptoms.

Proportion of self-esteem dependent on weight and shape: There was a significant interaction between change in binge-eating symptom score on the BEDT and the percentage of self esteem dependent on weight and shape as indicated on the SAWBS at baseline. Reduction in BEDT score was positively correlated with the proportion of self esteem dependent on shape and weight ($r(71)=.319$, $p=.007$).

Dichotomous thinking: There were interactions between dichotomous thinking about eating, as measured on the DTED, and change in scores on the EDEQ and the BEDT. Dichotomous thinking score was positively correlated with reduction in EDEQ score ($r(81)=.252$, $p=.023$) and BEDT score ($r(80)=.309$, $p=.005$).

Summary:

A range of risk factors for disordered eating showed significant interactions with study outcomes. Dieting history, weight dissatisfaction, global self esteem, the proportion of self esteem dependent on weight and shape, and the level of dichotomous thinking about food and weight, all showed evidence of interactions with global eating pathology and/or binge eating.

Table 5: Main effects of and interactions with risk factor variables

Outcome	Risk factor measure	Test used	Interaction				Between-subjects main effects		
			f	*df*	*df error*	*p*	*f*	*df*	*p*
EDE-Q Global score	Age	ANCOVA	.245	1	79	.622			
	Gender	Mixed ANOVA	1.294	1	79	.259	4.465	1	.038*
	Diet history	Mixed ANOVA	6.104	1	79	.016*	12.157	1	.001***
	Dissatisfaction	ANCOVA	3.735	1	79	.057			
	Suppression	ANCOVA	.855	1	79	.358			
	SES	ANCOVA	10.082	1	79	.002**			
	SAWBS	ANCOVA	2.322	1	70	.132			
	DTED	ANCOVA	4.192	1	79	.044*			
EDEQ Binge-eating frequency	Age	ANCOVA	.031	1	79	.861			
	Gender	Mixed ANOVA	.437	1	79	.510	.459	1	.500
	Diet history	Mixed ANOVA	7.048	1	79	.010**	.092	1	.762
	Dissatisfaction	ANCOVA	19.13	1	79	<.001***			
	Suppression	ANCOVA	.953	1	79	.332			
	SES	ANCOVA	2.143	1	79	.147			
	SAWBS	ANCOVA	4.953	1	70	.029*			
	DTED	ANCOVA	3.157	1	79	.079			
BEDT score	Age	ANCOVA	1.918	1	78	.170			
	Gender	Mixed ANOVA	.050	1	78	.824	.306	1	.582
	Diet history	Mixed ANOVA	1.422	1	78	.237	4.959	1	.029*
	Dissatisfaction	ANCOVA	13.759	1	78	<.001***			
	Suppression	ANCOVA	.305	1	78	.582			
	SES	ANCOVA	10.546	1	78	.002**			
	SAWBS	ANCOVA	7.722	1	69	.007**			
	DTED	ANCOVA	8.551	1	78	.005**			
State Food Craving scale score	Age	ANCOVA	.119	1	79	.731			
	Gender	Mixed ANOVA	2.078	1	79	.153	.178	1	.674
	Diet history	Mixed ANOVA	4.296	1	79	.041*	.003	1	.954
	Dissatisfaction	ANCOVA	.050	1	79	.824			
	Suppression	ANCOVA	.232	1	79	.632			
	SES	ANCOVA	.403	1	79	.527			
	SAWBS	ANCOVA	1.576	1	70	.231			
	DTED	ANCOVA	2.698	1	79	.104			
DASS-21 score	Age	ANCOVA	.825	1	79	.367			
	Gender	Mixed ANOVA	.069	1	79	.793	1.305	1	.257
	Diet history	Mixed ANOVA	2.764	1	79	.100	.054	1	.817
	Dissatisfaction	ANCOVA	1.489	1	79	.226			
	Suppression	ANCOVA	1.111	1	79	.295			
	SES	ANCOVA	2.439	1	79	.122			
	SAWBS	ANCOVA	1.131	1	70	.291			
	DTED	ANCOVA	.222	1	79	.639			

= significant at p<.05, **=significant at p<.01, *=significant at p<.001*

4.0 Discussion

4.1 Key findings

Participants lost on average 1.92kilos over 28 days of 5:2 intermittent fasting. This weight loss is very similar to that observed in other studies of intermittent fasting over a four week period (Harvey et al., 2011; Hoddy et al., 2015, Laessle et al., 1996). Participants that completed the study reported reduced global disordered eating, reduced frequency of binge eating, and reduced symptoms of binge eating disorder. These effects all remained significant after Bonferroni correction. They also reported reduced state food craving, and lower levels of depression, anxiety and stress. As would be expected, participants consumed fewer calories on fasting days. There were no differences in calorie consumption between days prior to the diet and non-fasting days during the diet. There were a number of interactions with risk factors for disordered eating and outcome measures. Females reported higher levels of disordered eating throughout the study. The group of participants who had dieted in the past reported reductions in global disordered eating on the EDEQ, binge eating frequency, and food craving. This interaction remained significant following Bonferroni correction. Greater weight dissatisfaction at baseline was positively correlated with reduction in global disordered eating, binge eating frequency, and binge eating disorder symptoms on the BEDT. Interactions with BEDT score and binge eating frequency, but not EDEQ score, remained significant following Bonferonni correction. Dichotomous thinking score positively correlated with reduction in disordered eating and symptoms of binge eating disorder. Reduction of binge eating symptoms on the BEDT also positively correlated with the proportion of self esteem dependent on shape and weight, and negatively correlated with self esteem.

Studies into restriction and dieting dating as far back as the Minnesota Semi-Starvation study (Keys et al., 1950) have lead to a near consensus in the eating disorder field that attempts to restrict calorie intake can lead to difficulties with disordered eating, binge-eating, preoccupation with food and food craving, stress and low mood, along with weight gain (Polivy

& Herman, 2002; Stice, 2002; Stice et al., 2008; Vitousek, Manke, et al., 2004; Wilson et al., 1997). Participants in this study, despite regularly engaging in extreme restriction of their calorie intake (down to 500 calories for women/650 calories for men on two days each week), demonstrated no such predicted difficulties, and indeed appeared to even take benefit from this intermittent 'fasting', with reductions on all outcome measures. Interpretation of this finding is complicated by the high drop-out rate of the study, with almost half of the participants completing baseline measures failing to complete measures at the end of the IF period. There were few differences between the group of participants who completed the study and those that dropped out at baseline. However, it is highly plausible that those experiencing adverse effects as a result of IF would be more likely to drop out, while those experiencing benefits would continue with the diet and the study. Indeed, anecdotally, several participants dropping out cited adverse effects of the diet, such as feeling unwell, finding it impossible to stick the restricted calorie intake while over-eating at other times, and lacking energy to meet the demands of their daily life, as their reason for doing so.

If the results are not simply an artefact of participant attrition, they may reflect differences between naturalistic, voluntary dieting over a short period of time, and the forms of extreme, involuntary and/or long term restriction studied in starvation and fasting studies, and in clinical populations. A growing number of studies, along with the experiences of many 'everyday' dieters, suggest that not all attempts to restrict food intake result in adverse psychological difficulties, and not all dieters will experience difficulties (Stice, 2002; Williamson et al., 2008b). The makeup of this sample, consisting primarily of adults in their 40s and 50s with no history of ED despite having dieted previously, are clearly different from clinical populations, and might be considered a group at lower risk of going on to develop difficulties with disordered or binge eating from following any diet. Adverse effects may take time to emerge, as the initial sense of motivation and achievement wears off, and motivational and biological processes to protect against starvation kick in (Stice et al., 2008; Tapper & Pothos, 2010). A brief (28 days) study duration

was chosen to minimise participant attrition; is possible that a longer follow-up period may have allowed the identification of iatrogenic effects taking longer to emerge. However, a similar study by Williamson et al. (2008), looking at the impact of following a calorie restricted diet in a population of overweight dieters with a similar mean age, also identified no increases in eating disorder symptoms over a period as long as one year.

As few researchers have studied naturalistic dieting of any kind in this way, it is possible that the results of this study are not unique to IF but would also be found in studies tracking followers of any diet over a short time period. Alternatively, these findings may reflect some difference between IF and other forms of 'continuous' dieting and restriction. For example, eating normally on 'non-fasting' days may prevent the activation of biological mechanisms, or the flexibility of the diet (including the ability to 'switch' days if you are struggling to fast on a particular day) and lack of strict 'rules' on non-fasting days may mean there is a reduced risk of cognitive 'abstinence violation', which is thought to trigger episodes of bingeing in some individuals when dietary rules are violated (Wilson et al., 1997). Tentative evidence of some difference between IF and other weight loss diets comes from the results of an unpublished thesis study which compared individuals already undertaking 5:2 IF to a group of dieters following any other diet (Teng, 2015). They found that the IF group scored lower on measures of eating pathology while there were no differences between the groups on measures of binge eating or mood. However, it is impossible to establish whether these differences are a result of differences between the diets or reflect pre-existing differences in the individuals choosing to follow them.

Further credence to this 'IF is different' account is offered by the identified interactions with risk factor variables. Consistently, where interactions were found, they appeared to demonstrate that those scoring higher on risk factors for disordered eating, (weight dissatisfaction, dieting history, low self-esteem, self-esteem being highly dependent on weight and shape, and dichotomous 'all-or-nothing' thinking about weight and eating) experienced

greater reductions in disordered and binge-eating over the 28 day IF period. These findings are in contrast to research demonstrating that calorie restriction and dieting are particularly likely to have adverse consequences for these 'at risk' individuals (Fairburn et al., 2005). When interpreting these interactions, it is important to note that all of these factors were associated with increased disordered eating and binge eating at baseline. It cannot be ruled out that these findings reflect some form of regression to the mean, or may be common to the early days of any diet, perhaps as a positive reaction to weight loss. Alternatively, there may be something about IF that preferentially benefits those who would otherwise be considered 'at risk' of disordered or binge eating, while those not at risk experience no benefit. Interestingly, looking more closely at the data related to dieting history, the small group of participants (n=12) who had never dieted in the past experienced no significant reductions in global disordered eating, binge eating frequency or food craving. In fact, in this group mean scores on all of these measures were slightly increased after 28 days of IF, however these changes were non-significant. It is unclear whether this simply reflects random 'noise' in the data, or whether, had this group been larger, the accompanying increase in statistical power would have revealed that those who had never dieted in the past actually experienced some adverse effects after IF. If so, this would have important implications for clinicians providing advice regarding the benefits and risks of IF.

4.2 Strengths and Limitations

This study represents an attempt to study the psychological and behavioural effects of IF in healthy dieters (self-selected, predominantly non-obese, no history of eating disorders, no specific health condition) in a naturalistic way. The online protocol allowed the recruitment of a sample likely to be largely representative of those choosing to attempt IF currently, with participants covering a wide age range and living in several different countries across the world. Unlike other studies of IF, and indeed most studies of dieting and/or fasting, participants in this study were predominantly not obese, had chosen independently to follow IF, and were

responsible for making their own food choices throughout the study. The findings of this study may therefore be widely applicable to every day dieters choosing to follow IF.

However, in this context, the lack of an alternative weight loss diet control represents a major challenge to interpretation of the results of this study. The lack of comparable studies investigating the effect of alternative diets means it is impossible to establish whether the results seen in this study are unique to IF, or common to all effective weight loss diets over this short time period. The decision not to strictly track compliance with the diet was taken in an attempt to minimise participant burden and to ensure that dieting behaviour more closely modelled the behaviour of individuals independently choosing to follow a 5:2 IF diet in a self-directed way (in line with the experience of most dieters). This means that it is impossible to know whether participants were adhering to the IF pattern, failing to adequately restrict, or indeed choosing to restrict on additional days. Conclusions can therefore only be drawn about the impact of *attempting* to follow the 5:2 IF eating pattern. Some support indicating that compliance with the diet may have been high comes from the similar pattern of weight loss to that observed in other IF studies (Harvie et al. 2011). Food diary data also demonstrated that eating patterns were consistent with the '5:2' pattern, although these are of course subject to reporting error. The remote, naturalistic design also means that measures relied on self-report, and there was no means of 'follow up' to assess the impact of the diet over longer timescales.

The most serious limitation of this study is the high drop-out rate. With follow up data unavailable for almost half of the participants that completed baseline measures, it is impossible to establish whether this group would have reported the same apparent benefits of IF, experienced no change over the 28 day period, or might have in fact reported adverse effects of IF, such as increases in disordered and binge eating. This severely limits the strength of the conclusions that can be drawn from this study regarding the safety of IF diets, or indeed their potential utility. Attrition rates were much higher than those in other studies of IF, which made use of more intensive, directive designs, based around single universities and clinics and

involving in person contact with researcher at each stage (Harvey et al., 2011; Hoddy et al., 2015; Laessle et al., 1996). They are equivalent to those observed by Geraghty, Torres, Leykin, Perez-Stable & Munoz (2012), who investigated attrition rates from a large internet-based health promotion intervention. They found attrition rates of 61% over one-month for participants receiving automated email follow up (up to three emails), while for participants receiving more assertive 'live' follow up (personalised email and up to 10 telephone calls) attrition was as low as 10% over 3 months.

4.3 Implications

Clinical implications:

Overweight and obesity, and related health conditions, represent a major challenge to public health, and there is a pressing need to identify safe and effective means for individuals to achieve and maintain healthy body weight. While research into the potential health benefits of IF is ongoing, the results of this study suggest that healthy adults may be able to use IF as a means to lose weight without the experiencing adverse psychological and behavioural side-effects thought to be associated with attempts at calorie restriction. It may be that IF represents a qualitatively different approach to weight loss, with a reduced risk of developing difficulties with disordered or binge eating. While the absence of alternative weight loss control and high drop-out rate of this study limit the strength of conclusions that can be drawn, if further studies were to identify real differences between IF and other forms of continuous dieting, or differing responses for different participant groups, this might have important implications for theories regarding dietary restraint, and the models of eating disorders characterised by binge eating (such as binge eating disorder and bulimia nervosa) which have arisen out of them (Wilson et al., 1997).

Further research:

To allow stronger conclusions to be drawn about the safety and potential benefits of IF, future studies into IF should make an effort to address the major limitations of this study. A larger scale and more intensive study should involve more assertive follow up of participants, including gathering data for those who 'drop out' of the diet, and tracking changes over a longer diet period. To allow conclusions about the similarities/differences between IF and continuous diets, an alternative weight loss diet control group should be included. Studies should also look carefully at participant groups that might be considered more likely to develop difficulties with disordered eating, such as young adult women, to ensure that the diet is 'safe' for this group.

Studies following independent dieters in a more naturalistic way could add much to the literature surrounding dieting in general, as well as IF specifically. While the overall statistical power of this study was high, larger scale studies would also allow the recruitment of larger numbers of participants from under-represented groups in this population, such as males and those with no dieting history, and allow more detailed analyses of interaction and mediation. These studies would benefit from looking closely at the characteristics of both the diets and participant groups used, including any potential risk or protective factors, to throw light onto questions such as why restriction and dieting seems to lead to bingeing and EDs for some people but not others, or whether certain diets are more risky for this than others.

4.4 Conclusions

Starting a 5:2 intermittent fasting diet did not lead to increases in disordered eating, binge-eating, food craving, or mood difficulties in healthy adult dieters, and in fact appeared to result in improvements in these outcomes. Participants scoring higher on risk factors for disordered eating reported greater reductions in disordered and binge-eating over the 28 day IF period. The findings of this study may have important implications for those attempting to maintain a healthy body weight, and for models of dietary restraint and binge eating. The strength of these

conclusions is limited by the high drop-out rate and absence of alternative weight loss diet control.

5.0 References

American Psychiatric Association. (1994). *Diagnostic Statistical Manual of Mental Disorders (4th Ed.)*. Washington: American Psychiatric Association.

Antony, M. M. Bieling, P. J., Cox, B. J., Enns, M. W., Swinson, R. P., Haynes, S. N. (1998). Psychometric properties of the 42-item and 21-item versions of the Depression Anxiety Stress Scales in clinical groups and a community sample. *Psychological Assessment, 10*(2), 176-181.

Appleton, K. M., & Baker, S. (2015). Distraction, not hunger, is associated with lower mood and lower perceived work performance on fast compared to non-fast days during intermittent fasting. *Journal of Health Psychology*, *20*(6), 702–11. doi:10.1177/1359105315573430

Button, E. J., Sonuga-Barke, E. J., Davies, J., & Thompson, M. (1996). A prospective study of self-esteem in the prediction of eating problems in adolescent school-girls: Questionnaire findings. *British Journal of Clinical Psychology*, *35*(2), 193–203.

Byrne, S. M., Allen, K. L., Dove, E. R., Watt, F. J., & Nathan, P. R. (2008). The reliability and validity of the dichotomous thinking in eating disorders scale. *Eating Behaviors*, *9*(2), 154–62. doi:10.1016/j.eatbeh.2007.07.002

Cepeda-Benito, A., Gleaves, D., Williams, T., & Erath, S. (2000). The development and validation of the state and trait food-cravings questionnaires. *Behaviour Therapy*, *31*(1), 151–173. doi:10.1016/S0005-7894(00)80009-X

CR Society International. (2002). CR Society Home Page. Retrieved April 21, 2016, from http://www.crsociety.org/

Fairburn, C. G., & Beglin, S. J. (1994). Assessment of eating disorders: Interview or self report questionnaire? *International Journal of Eating Disorders*, *16*, 363–370.

Fairburn, C. G., Cooper, Z., Doll, H. a, & Davies, B. A. (2005). Identifying dieters who will develop an eating disorder: a prospective, population-based study. *The American Journal of Psychiatry*, *162*(12), 2249–55. doi:10.1176/appi.ajp.162.12.2249

Geller, J., Johnston, C., & Madsen, K. (1997). The Role of Shape and Weight in Self-Concept: The Shape and Weight Based Self-Esteem Inventory. *Cognitive Therapy and Research*, *21*(1), 5–24. doi:10.1023/A:10218124937

Geraghty, A. W. A., Torres, L. D., Leykin, Y., Perez-Stable, E. J. & Munoz R, F. (2012). Understanding attrition from international internet health interventions: a step towards global eHealth. *Health Promotion International, 28 (3).* doi:10.1093/heapro/das029

Harvie, M. N., Pegington, M., Mattson, M. P., Frystyk, J., Dillon, B., Cuzick, J., Howell, A. (2011). The effects of intermittent or continuus energy restriction on weight loss and metabolic disease risk markers: a randomised trial in young overweight women. *International Journal of Obesity*, *35*(5), 714–727. doi:10.1038/ijo.2010.171.The

Harwell, M., Rubinsteing, E., Hayes, W., & Olds, C. (1992). Summarising Monte Carlo results in methodological research: The one- and two- factor fixed effects ANOVA cases. *Journal of Educational Statistics*, *17*, 315–339.

Herman, C. P., & Polivy, J. (1975). Restrained and unrestrained eating. *Journal of Personality*, *43*, 647–660.

Herman, C. P., & Polivy, J. (1984). A boundary model for theregulation of eating. In A. Stunkard & E. Stellar (Eds.), *Eating and its disorders* (pp. 141–156). New York: Raven.

Hoddy, K. K., Kroeger, C. M., Trepanowski, J. F., Barnosky, A. R., Bhutani, S., & Varady, K. A. (2015). Safety of alternate day fasting and effect on disordered eating behaviors. *Nutrition Journal*, *14*, 44. doi:10.1186/s12937-015-0029-9

Johnson, J. B., Laub, D. R., & John, S. (2006). The effect on health of alternate day calorie restriction: Eating less and more than needed on alternate days prolongs life. *Medical Hypotheses, 67* (2), 209–211. doi:10.1016/j.mehy.2006.01.030

Keys, A., Brozek, J., Henschel, A., Mickelson, O., & Taylor, H. L. (1950). *The biology of human starvation*. Minneapolis: University of Minnesota Press.

Laessle, R. G., Platte, P., Schweiger, U., & Pirke, K. M. (1996). Biological and Psychological Correlates of Intermittent Dieting Behavior in Young Women . A Model for Bulimia Nervosa. *Physiology & Behaviour*, *60*(1), 1–5.

Lovibond, S. H., & Lovibond, P. F. (1995). *Manual for the Depression Anxiety Stress Scales* (2nd Editio.). Sydney: Psychology Foundation.

Lowe, M. R. (1993). The effects of dieting on eating behavior: A three-factor model. *Psychological Bulletin*, *114*, 100–121.

Mahony, K. (2016). Nutrition and cognition: Exploring their relationship from two sides of the same coin. *DClinPsy Thesis Submission*.

Mizes, J. S., Heffner, M., Madison, J. K., & Varnado-Sullivan, P. (2004). The validity of subjective measures of body image disturbance. *Eating Behaviors*, *5*(1), 55–66. doi:10.1016/S1471-0153(03)00056-4

Mond, J.M., Hay, P.J., Rodgers, B., Owen, C. & Beumont, P.J.V. (2004). Validity of the Eating Disorder Examination Questionnaire (EDE-Q) in screening for eating disorders in community samples. *Behaviour Research and Therapy*,42(5), 551-567

Mosley, M., & Spencer, M. (2013). *The Fast Diet: The Secret of Intermittent Fasting - Lose Weight, Stay Healthy, Live Longer*. London: Short Books.

MyFitnessPal. (2011). MyFitnessPal. *LLC*. Retrieved from www.myfitnesspal.com

Ogden, J. (2010). *The Psychology of Eating: From Healthy to Disordered Behaviour. 2nd Edition.* Wiley-Blackwell.

Polivy, J., & Herman, C. P. (2002). Causes of eating disorders. *Annual Review of Psychology, 53*, 187–213.

Qualtrics. (2005). Qualtrics. Utah: Qualtrics. Retrieved from http://www.qualtrics.com

Rose, S., Vaewsorn, A., Rosselli-Navarra, F., Wilson, G. T. & Weissman, R. S. (2013). Test-retest reliability of the Eating Disorder Examination-Questionnaire (EDE-Q) in a college sample. *Journal of Eating Disorders*, 1, 42-52

Rosenberg, M. (1965). Society and the adolescent *self image*. Princeton, NJ: Princeton University Press.

Schmider, E., Ziegler, M., Danay, E., Beyer, L., & Bühner, M. (2010). Is It Really Robust? *Methodology*, *6*(4), 147–151. doi:10.1027/1614-2241/a000016

Seimon, R. V, Roekenes, J. A., Zibellini, J., Zhu, B., Gibson, A. A., Hills, A. P., … Sainsbury, A. (2015). Do intermittent diets provide physiological benefits over continuous diets for

weight loss? A systematic review of clinical trials. *Molecular and Cellular Endocrinology*, *418 Pt 2*, 153–72. doi:10.1016/j.mce.2015.09.014

Sinclair, S. J., Blais, M. A., Gansler, D. A., Sandberg, E., Bistis, K. & Locicero, A., (2010) Psychometric properties of the Rosenberg Self-Esteem Scale: Overall and across demographic groups living within the United States. *Evaluation & the Health Professions*, *33*(1), 56-80

Stice, E. (2001). A prospective test of the dual-pathway model of bulimic pathology: Mediating effects of dieting and negative affect. *Journal of Abnormal Psychology*, *110*(1), 124–135. doi:10.1037/0021-843X.110.1.124

Stice, E. (2002). Risk and maintenance factors for eating pathology: A meta-analytic review. *Psychological Bulletin*, *128*(5), 825–848. doi:10.1037//0033-2909.128.5.825

Stice, E., Davis, K., Miller, N. P., & Marti, C. N. (2008). Fasting increases risk for onset of binge eating and bulimic pathology: a 5-year prospective study. *Journal of Abnormal Psychology*, *117*(4), 941–6. doi:10.1037/a0013644

Stice, E., Marti, N., & Durant, S. (2011). Risk Factors for Onset of Eating Disorders: Evidence of Multiple Risk Pathways from an 8-Year Prospective Study. *Behaviour Research and Therapy*, *49*(10), 622–627. doi:10.1016/j.brat.2011.06.009.Risk

Tapper, K., & Pothos, E. M. (2010). Development and validation of a Food Preoccupation Questionnaire. *Eating Behaviors*, *11*(1), 45–53. doi:10.1016/j.eatbeh.2009.09.003

Telch, C. F., & Agras, W. S. (1993). The effects of a very low calorie diet on binge eating. *Behavior Therapy, 24,* 177–193.

Teng, T. (2015). The 5:2 intermittent fasting diet: Feasibility and dieters' eating disorder pathology, psychological functioning and experiences. University College London, DClinPsy Thesis. *Unpublished.*

Thelen, M. H., Farmer, J., Wonderlich, D. A., & Smith, M. (1991). A revision of the bulimia test: The BULIT-R. *Psychological Assessment*, *3*, 119–124.

Tinsley, G. M., & La Bounty, P. M. (2015). Effects of intermittent fasting on body composition and clinical health markers in humans. *Nutrition Reviews*, *73*(10), 661–74. doi:10.1093/nutrit/nuv041

Van den Eynde, F., Koskina, A., Syrad, H., Guillame, S., Broadbent, H., Campbell, I., & Schmidt, U. (2012). State and trait food craving in people with bulimic eating disorders. *Eating Behaviours*, *13*(4), 414–417.

Vander Wal, J. S., Stein, R. I., & Blashill, A. J. (2011). The EDE-Q, BULIT-R, and BEDT as self-report measures of binge eating disorder. *Eating Behaviors*, *12*(4), 267–71. doi:10.1016/j.eatbeh.2011.07.006

Varady, K., & Hellerstein, M. (2007). Alternate-day fasting and chronic disease prevention : a review of human and animal trials. *The American Journal of Clinical Nutrition*, *86*, 7–13.

Vitousek, K. B., & Hollon, S. D. (1990). The investigation of schematic content and processing in eating disorders. *Cognitive Therapy and Research*, *14*(2), 191–214. doi:10.1007/BF01176209

Vitousek, K. M., Gray, J. A., & Grubbs, M. (2004). Invited Article Caloric Restriction for Longevity : I . Paradigm , Protocols and Physiological Findings in Animal Research. *European Eating Disorders Review*, *12*, 279–299.

Vitousek, K. M., Manke, F. P., Gray, J. A., & Vitousek, M. N. (2004). Invited Article Caloric Restriction for Longevity : II — The Systematic Neglect of Behavioural and Psychological Outcomes in Animal Research. *European Eating Disorders Review*, *12*, 338–360.

Williamson, D. a, Martin, C. K., Anton, S. D., York-Crowe, E., Han, H., Redman, L., & Ravussin, E. (2008a). Is caloric restriction associated with development of eating-disorder symptoms? Results from the CALERIE trial. *Health Psychology : Official Journal of the Division of Health Psychology, American Psychological Association*, *27*(1 Suppl), S32–42. doi:10.1037/0278-6133.27.1.S32

Williamson, D. a, Martin, C. K., Anton, S. D., York-Crowe, E., Han, H., Redman, L., & Ravussin, E. (2008b). Is caloric restriction associated with development of eating-disorder symptoms? Results from the CALERIE trial. *Health Psychology : Official Journal of the Division of Health Psychology, American Psychological Association*, *27*, S32–42. doi:10.1037/0278-6133.27.1.S32

Wilson, G. T., Fairburn, C. G., & Agras, S. (1997). Cognitive-behavioural therapy for bulimia nervosa (2nd Ed.). In *Handbook of treatment for eating disorders* (pp. 67–93). New York: Guildford Press.

Part 3: Critical Appraisal

This appraisal will reflect on the process of designing, undertaking and making sense of the research outlined in this thesis, with reference to the perspective of the scientist practitioner. The philosophical and theoretical issues underlying my choice of literature review topic will be considered, along with the opportunities and challenges for the clinical and theoretical application of the literature, and the implications for my own research design process. The assumptions brought to my choice of topic for the empirical paper will be considered, along with decisions made about research design and the experience of recruiting and retaining participants for the study. The potential clinical significance of the results will be explored. Finally, reflections are made on the implications of this research experience for my thinking both as a clinician and researcher.

Literature Review

Choice of topic

My decision to review the eating disorder literature for studies identifying protective factors was informed by an interest in positive psychology and resilience (Masten, Best, & Garmezy, 1991; Seligman & Csikszentmihalyi, 2000). Working within the 'scientist practitioner' framework, clinical psychologists are encouraged not only to ensure that their clinical work is informed by empirical research, but also to use their clinical experience to develop research questions and inform research (Raimy, 1950). As clinical work inevitably brings us in to contact with individuals or groups experiencing distress and difficulties with functioning, there is a danger of developing a 'problem-focussed' view, and of neglecting to ask questions about the experience of those who do not experience distress, who develop and behave in ways that are adaptive, and who demonstrate resilience in the face of adverse experiences (Wood & Tarrier, 2010). This may represent a missed opportunity as researchers to identify factors promoting adaptive development and wellbeing, which could otherwise be of use to efforts to prevent difficulties such as eating disorders and disordered eating, to develop effective interventions and to support recovery (Steck, Abrams, & Phelps, 2004).

A focus on strengths, resources and abilities, and an approach to change which prioritises supporting individuals and groups to discover and make use of these, is a key part of many systemic and community psychology approaches (Freedman & Combs, 1996; Schueller, 2009; Selekman, 1993). These approaches also highlight the importance of moving beyond a focus on individual determinants of development and behaviour, to identify influences at the level of systems: families, communities and social groups, and wider socio-cultural influences (Bronfenbrenner, 1979). While many, if not most, clinical psychologists would acknowledge the importance of these factors, the nature of the majority of therapeutic work involves a primary focus on the individual, and many therapeutic approaches therefore prioritise intrapersonal factors in their attempts to ameliorate distress and promote adaptive functioning. The role of social and cultural 'risk factors' in the development of eating disorders and disordered eating is well acknowledged, and attempts have been made to integrate this knowledge into models, interventions and prevention programmes. However, a relative neglect of the role of 'positive institutions' to act as protective factors may represent a real missed opportunity (Austin, 2000; Neumark-Sztainer, 2011; Schueller, 2009; Steck et al., 2004). To allow work of this kind to develop in a coherent way, a clear picture is needed of the field of research so far. This was the reasoning underlying my decision to review the literature on protective factors in social systems and environments.

Making sense of the literature: Opportunities and challenges

When reviewing the literature, I was pleased to discover that many researchers had begun to identify factors in families, schools and peer groups which could act as protective factors against eating disorders or disordered eating. It appears that there are things that families, schools, peer groups and communities can do that will help to protect young people against the development of these difficulties and support resilience. The potential of this literature to inform clinical prevention and intervention efforts is, however, severely limited by its fragmented nature and common weaknesses in methodology. I was left with no sense of a coherent attempt to

identify and study protective factors, with many studies naming 'protective factors' solely on the basis of a post-hoc identification of an inverse cross-sectional correlation with maladaptive outcomes. Inevitably, the interests and assumptions of researchers have influenced the potential protective factors studied; certain systems of influence, such as families, and specific factors, such as family meals, have been the topic larger numbers of studies, while others, such as schools, peer groups and communities, have been almost entirely neglected. Variables are often poorly defined, and very few of the studies have attempted to consider or investigate the processes and mechanisms underlying the associations found.

Moving beyond these methodological issues, the literature also highlights the practical and ethical challenges inherent in attempts to prevent difficulties and promote positive development and functioning. Many of the 'protective factors' identified were non-specific to eating disorders and disordered eating. 'Developmental assets' such as family support, caring schools, and communities which value youth are factors associated with a wide range of positive outcomes for children and young people (French et al. 2001). Some might argue that awareness of these factors creates an ethical challenge for practitioners who continue to work to treat distress and psychopathology at the individual level, suggesting that our efforts should instead be focussed on prevention and promotion of wellbeing at the community level (Orford, 2008). However, there are clear practical challenges to work of this kind, with no coherent picture of what such a comprehensive approach would look like. It also raises ethical questions about how much psychologists and other health professionals should seek to involve themselves in peoples' everyday lives, as well as the potential for the diversion of limited resources away from those experiencing the most distress and difficulty (Wood & Tarrier, 2010).

Implications for my own research

Reviewing the strengths and weakness in this literature was useful in informing my own research process. Flaws in the methodology of many studies, such as the use of non-standardised and validated measures of disordered eating, and a lack of clearly stated a-priori hypotheses,

reduced their utility to future researchers and clinicians, and I was keen to avoid these weaknesses in my own design. It also highlighted that while research in 'healthy' individuals can be valuable in informing our understanding of clinical populations, there are key differences between 'protective' and 'risk' factors influencing the development and onset of difficulties and factors which maintain them or promote recovery, and limits to the applicability of research with healthy adults to clinical populations.

Empirical Paper

Choice of topic and assumptions

My choice to study intermittent fasting (IF) or '5:2' diets, and the assumptions I held prior to undertaking this research, were undeniably informed by my perspective as a scientist practitioner with a clinical interest in working with those with eating disorders. Having seen accounts of the diet in the media, including a high profile BBC documentary, which made bold claims about the potential health benefits of intermittent fasting, my initial reaction was one of scepticism and concern (Dart, 2012). Within the eating disorder field, there is a commonly held view that 'diets don't work', that dieting is a clear risk factor for the development of eating disorders, and that attempts to restrict calorie intake are likely to result in difficulties with disordered and binge eating. These views are informed by well known empirical research dating back to the 1940s, commonly used clinical models of eating disorders, and clinical experience (Fairburn et al., 2013; Keys, Brozek, Henschel, Mickelson, & Taylor, 1950; Wilson & Fairburn, 1993). In addition to a general suspicion of weight-loss diets, the apparently 'abnormal' nature of the eating pattern proposed by this diet, combining severe restriction of calorie intake on some days with a complete lack of nutritional restrictions and an exhortation to 'eat whatever you want' on others, appeared to bear superficial similarity to eating patterns seen in disorders such as bulimia nervosa and binge eating disorder.

While my stated hypotheses for the research were open ended, I was aware of a strong personal assumption that attempts to follow 5:2 IF were likely to result in negative consequences

for dieters, predicting increases in eating psychopathology, binge eating, preoccupation with food and food craving, irritability, depression and anxiety. My conversations with other clinicians working with those with eating disorders suggested that I was not alone in this view. These assumptions appear to form part of a wider pattern of scepticism which has contributed to a lack of interdisciplinary collaboration between researchers conducting biological and medical research into areas such as obesity and weight loss, and those researching and working with eating disorders (Vitousek, Gray, & Grubbs, 2004; Vitousek, Manke, Gray, & Vitousek, 2004). This divide creates areas of weakness in the field, for example a tendency by diet and calorie restriction researchers to neglect the study of psychological and behavioural variables, and confusion and distress for individuals who are told by some health professionals that they must diet to lose weight and improve health, and by others that dieting is harmful and they must at best aim for weight stabilisation.

Study design

The process of designing empirical research studies involves inevitable trade-offs between what would be 'optimal' from a validity perspective, allowing the researcher to draw firm conclusions, and what is practically feasible within the scope of the research project (Barker, Pistrang, & Elliot, 2002). In designing this research, a number of decisions were made to increase the feasibility of the research design. Due to the lack of previous studies into the psychological impact of IF, and my assumptions about the potential harms of this way of eating, a decision was taken from an ethical standpoint that was not appropriate to ask individuals to follow the diet simply for the purposes of the study. It was therefore necessary to recruit a sample of participants who had decided independently to follow IF. Based on the assumptions outlined previously, it could be suggested that individuals with pre-existing difficulties with disordered eating might be more likely to choose to follow a diet. A repeated measures design was therefore chosen, with each participant completing baseline measures prior starting the diet, to allow some inference of causality should their scores on key outcomes change. This created a limited time

window, between taking the decision to start IF and actually doing so, in which we could recruit participants for the study. Participants also needed to remain in the study long enough to complete follow up measures.

Recruitment and retention of participants therefore became a primary driver of decision making. The study was conducted completely online, allowing participant recruitment from far beyond the immediate geographical area. To allow this, and to minimise participant burden, easy to complete self-report measures were chosen. A study length of 28 days was chosen with the hope that this was long enough for changes to be observed while reducing the opportunity for participants to 'give up' and drop out. Finally, I decided it would be impractical to simultaneously recruit a sufficient number of followers of other diets, meaning that no alternative weight-loss diet control was included in the study. All of these decisions present some challenges to the conclusions which can be drawn.

Participant recruitment and retention

Having assumed that recruitment of participants would be a major challenge for the study, I was surprised by the rapidity and ease with which we were able to initially recruit 176 participant volunteers. This was down to a pro-active and targeted online recruitment strategy, involving use of social media and email to make direct contact with media personalities and websites associated with '5:2', gaining their support to place our advertisement in places where it would be seen by individuals considering the diet. This strategy was highly successful, and could be a valuable approach for other researchers.

Our use of this recruitment strategy had an impact on the demographic makeup of our self-selected sample. An area of strength was the international profile of the sample, with participants recruited from six continents. Despite this, the vast majority of participants reported their ethnicity as White or Caucasian. The participant group were highly educated, with the vast majority of participants educated to at least degree level, and many holding post-graduate qualifications and working in research. Many were very enthusiastic about the diet, and had spent

time looking at research into the claimed health benefits of the diet beyond the potential for weight loss. While we recruited participants aged 18 – 65 years, the mean age for participants was 45.22 years, much older than the typical age of onset for eating disorders (Micali, Hagberg, Petersen, & Treasure, 2013). For ethical reasons, potential participants were excluded from the study if they reported a history of any eating disorder. This meant that it is possible to consider our participant group 'resilient' in the sense that they had so far in their lives avoided developing clinical levels of disordered eating. This does limit the applicability of our findings to clinical populations.

As expected, attrition from the study over the 28 day period was relatively high. The terms of the ethical approval for the study stated that "participants have the right to withdraw from the study at any time, without giving a reason". This meant that for many of the participants no information was available about their experience of the diet or their reasons for dropping out. The potential for these participants to be the very ones who experienced a negative impact of IF is a major challenge to the strength of conclusions about the potential benefits and risks of following the IF diet.

Making sense of findings: Potential clinical implications

Holding the assumptions outlined above, the results of the study were a complete surprise for me. Far from experiencing harmful psychological effects of the diet, participants who completed the study reported reductions in disordered and binge eating, food craving, and mood symptoms. Going further than this, the very participant characteristics which I might have expected to be associated with an increase in difficulties, known 'risk factors' for eating disorders such as low self-esteem and weight dissatisfaction, were associated with greater reductions in eating pathology. In every sense, these results were the opposite of expected. Careful consideration was therefore required of the conclusions that could be drawn, and any potential clinical implications.

A number of possible explanations for this unexpected result were considered. As described above, the results may be an artefact of the study design and research process. They may reflect participant attrition, the short study period, or the nature of the participant group. The absence of an alternative weight loss diet control, in the context of a dearth of other research into naturalistic dieting, means it not possible to say whether the same pattern of results would be observed with a this healthy adult participant group following any diet. If this is the case, this presents an interesting challenge to the view from the ED perspective of diets and attempts at restriction as ineffective and potentially harmful. In our society, where attempts at dieting to lose weight are so common as to be considered 'normative' behaviour, it is undeniable that very many, if not the majority, of dieters do not go on to develop eating disorders, and to seek support from clinicians working in this field (Rodin, Silberstein, & Striegel-Moore, 1984). This challenge carries with it, however, an inherent opportunity to learn from these 'resilient' individuals. If researchers can identify protective factors at play in these dieters, it is possible to consider whether these are factors which can be promoted by attempts to prevent difficulties, or incorporated into attempts to intervene with those who have already developed them.

If, instead, further studies making comparisons to other diets were to find that there does appear to be something 'different' about IF, opportunities are presented to analyse the characteristics of the approach which diverge from other diets, for example the absence of a requirement for consistent restriction, the flexibility of the diet, or the lack of strict diet rules (Stice, Davis, Miller, & Marti, 2008; Tapper & Pothos, 2010; Wilson, Fairburn, & Agras, 1997). An understanding of this would have potentially important clinical implications, for example in supporting obese individuals to lose weight in a way that minimises the potential risk of developing difficulties with binge eating, or in understanding and supporting those experiencing difficulties with binge eating.

Personal Reflections

Undertaking this research highlighted the value of the research process in the potential for the findings to surprise and challenge assumptions. Reflecting on my own assumptions and experience of surprise has also made me aware of some of the opportunities and challenges inherent in the scientist practitioner identity. As a clinician conducting research, clinical knowledge and experience will unavoidably influence assumptions and inform the research questions asked, and this can be viewed as a key asset of the clinical psychologist (Raimy, 1950). However, there is a danger that the nature of much clinical work, coming into contact with those experiencing distress and difficulty, often as an individual within a therapy room, might blind us somewhat to the experience of those who are functioning adaptively, responding with resilience and experiencing high levels of wellbeing. A reminder to move beyond this problem-focussed mindset to consider the strengths, resources, and protective factors available to individuals and communities and promoting positive outcomes, presents a powerful opportunity for my work both as a researcher and a clinician.

References

Austin, S. (2000). Prevention research in eating disorders: theory and new directions. *Psychological Medicine*, *30*(06), 1249–1262.

Barker, C., Pistrang, N., & Elliot, R. (2002). *Research Methods in Clinical Psychology*. Chichester: Wiley.

Bronfenbrenner, U. (1979). *The Ecology of Human Development: Experiments by Nature and Design*. Cambridge, MA: Harvard University Press.

Dart, K. (2012). Horizon: Eat, Fast and Live Longer. UK: BBC.

Fairburn, C. G., Cooper, Z., Doll, H. A., O'Connor, M. E., Palmer, R. L., & Dalle Grave, R. (2013). Enhanced cognitive behaviour therapy for adults with anorexia nervosa: a UK-Italy study. *Behaviour Research and Therapy*, *51*(1), R2–8. doi:10.1016/j.brat.2012.09.010

Freedman, J., & Combs, G. (1996). *Narrative Therapy and the Social Construction of Preferred Realities*. New York: Norton.

French, S. A., Leffert, N., Story, M., Neumark-Sztainer, D., Hannan, P., & Benson, P. L. (2001). Adolescent binge/purge and weight loss behaviors: Associations with developmental assets. *Journal of Adolescent Health*, *28*(3), 211–221.

Keys, A., Brozek, J., Henschel, A., Mickelson, O., & Taylor, H. L. (1950). *The biology of human starvation*. Minneapolis: University of Minnesota Press.

Masten, A. N. N. S., Best, K. M., & Garmezy, N. (1991). Resilience and development :

Contributions from the study of children who overcome adversity. *Development and Psychopathology*, *2*(1990), 425–444.

Micali, N., Hagberg, K. W., Petersen, I., & Treasure, J. L. (2013). The incidence of eating disorders in the UK in 2000-2009: findings from the General Practice Research Database. *BMJ Open*, *3*(5). doi:10.1136/bmjopen-2013-002646

Neumark-Sztainer, D. (2011). Prevention of Eating Disorders in Children and Adolescents. In D. Le Grange & J. Lock (Eds.), *Eating Disorders in Children and Adolescents: A Clinical Handbook* (p. 512). Guilford Press.

Orford, J. (2008). *Community Psychology: Challenges, Controversies and Emerging Consensus*. Chichester: Wiley.

Raimy, V. C. (1950). *Training in clinical psychology*. New York: Prentice-Hall.

Rodin, J., Silberstein, L., & Striegel-Moore, R. (1984). Women and weight: a normative discontent. *Nebraska Symposium on Motivation. Nebraska Symposium on Motivation*, *32*, 267–307.

Schueller, S. M. (2009). Promoting wellness: integrating community and positive psychology. *Journal of Community Psychology*, *37*(7), 922–937. doi:10.1002/jcop.20334

Selekman, M. D. (1993). *Pathways to change*. New York: Guilford.

Seligman, M. E. P., & Csikszentmihalyi, M. (2000). Positive psychology: An introduction. *American Psychologist*, *55*, 5–14. doi:10.1037//0003-066X.55.1.5

Steck, E. L., Abrams, L. M., & Phelps, L. (2004). Positive Psychology in the Prevention of Eating Disorders. *Special Issue: Positive Psychology and Wellness in Children.*, *41*(1), 111–117.

Stice, E., Davis, K., Miller, N. P., & Marti, C. N. (2008). Fasting increases risk for onset of binge eating and bulimic pathology: a 5-year prospective study. *Journal of Abnormal Psychology*, *117*(4), 941–6. doi:10.1037/a0013644

Tapper, K., & Pothos, E. M. (2010). Development and validation of a Food Preoccupation Questionnaire. *Eating Behaviors*, *11*(1), 45–53. doi:10.1016/j.eatbeh.2009.09.003

Vitousek, K. M., Gray, J. A., & Grubbs, M. (2004). Invited Article Caloric Restriction for Longevity: I . Paradigm , Protocols and Physiological Findings in Animal Research. *European Eating Disorders Review*, *12*, 279–299.

Vitousek, K. M., Manke, F. P., Gray, J. A., & Vitousek, M. N. (2004). Invited Article Caloric Restriction for Longevity : II — The Systematic Neglect of Behavioural and Psychological Outcomes in Animal Research. *European Eating Disorders Review*, *12*, 338–360.

Wilson, G. T., & Fairburn, C. G. (1993). Cognitive treatments for eating disorders. *Journal of Consulting and Clinical Psychology*, *61*(2), 261–269. doi:10.1037//0022-006X.61.2.261

Wilson, G. T., Fairburn, C. G., & Agras, S. (1997). Cognitive-behavioural therapy for bulimia nervosa (2nd Ed.). In *Handbook of treatment for eating disorders* (pp. 67–93). New York: Guildford Press.

Wood, A. M., & Tarrier, N. (2010). Positive Clinical Psychology: A new vision and strategy for integrated research and practice. *Clinical Psychology Review*, *30*(7), 819–829. doi:10.1016/j.cpr.2010.06.003

Appendices

Appendix A:

Standard Quality Assessment Criteria (QualSyst) for reviewing quantitative studies

Kmet, Lee, & Cook (2004)*Standard Quality Assessment Criteria for Evaluating Primary Research Papers from a Variety of Fields*. Alberta: Alberta Heritage Foundation for Medical Research.

Criteria		YES (2)	PARTIAL (1)	NO (0)	N/A
1	Question / objective sufficiently described?				
2	Study design evident and appropriate?				
3	Method of subject/comparison group selection or source of information/input variables described and appropriate?				
4	Subject (and comparison group, if applicable) characteristics sufficiently described?				
5	If interventional and random allocation was possible, was it described?				
6	If interventional and blinding of investigators was possible, was it reported?				
7	If interventional and blinding of subjects was possible, was it reported?				
8	Outcome and (if applicable) exposure measure(s) well defined and robust to measurement / misclassification bias? Means of assessment reported?				
9	Sample size appropriate?				
10	Analytic methods described/justified and appropriate?				
11	Some estimate of variance is reported for the main results?				
12	Controlled for confounding?				
13	Results reported in sufficient detail?				
14	Conclusions supported by the results?				

To score:

Yes = 2, Partial = 1, No = 0.

Items not applicable to a particular study design marked "n/a" and excluded from calculation of summary score.

Summary score calculated by summing total score obtained across relevant items and dividing by the total possible score (total score / no. of items x 2).

Raw scores from QualSyst for each paper

Study	Item Number														Total	No of items	QualSyst Score
	1	2	3	4	5	6	7	8	9	10	11	12	13	14			
Ackard & Neumark-Sztainer (2001)	2	2	1	2	N/A	N/A	N/A	2	2	2	1	N/A	2	2	18	10	.900
Ahren, Chiesa, Koupil, Magnusson, Dalman & Goodman (2013)	2	2	2	2	N/A	N/A	N/A	2	2	2	2	N/A	2	2	20	10	1.000
Allen, Gibson, McLean, Davis & Byrne (2014)	2	2	2	2	N/A	N/A	N/A	2	2	2	2	N/A	2	2	20	10	1.000
Berge, Maclehose, Loth, Eisenberg, Bucchianeri, Neumark-Sztainer (2013)	2	2	2	2	N/A	N/A	N/A	1	2	2	2	N/A	2	1	18	10	.900
Berge, Wall, Larson, Eisenberg, Loth, Neumark-Sztainer (2014)	2	2	2	2	N/A	N/A	N/A	1	2	2	2	N/A	2	2	19	10	.950
Bertoli, Leone, Ponissi, Bedogni, Beggio, Strepparava & Battezzati (2015)	2	2	2	2	N/A	N/A	N/A	2	2	2	2	N/A	2	2	20	10	1.000
Brown & Keel (2012)	2	2	1	2	N/A	N/A	N/A	2	1	2	2	N/A	2	2	18	10	.900
Chandy, Harris, Blum, Resnick (1994)	2	2	2	2	N/A	N/A	N/A	1	2	1	0	N/A	2	2	16	10	.800
Chandy, Harris, Blum, Resnick (1995) [+]	2	2	2	2	N/A	N/A	N/A	1	2	1	0	N/A	2	2	16	10	.800
Cordero & Israel (2009)	2	2	1	2	N/A	N/A	N/A	2	2	2	2	N/A	2	2	19	10	.950
Croll, Neumark-Sztainer, Story & Ireland (2002)	2	2	2	2	N/A	N/A	N/A	1	2	2	0	N/A	2	2	17	10	.850
Ferreiro, Seoana & Senra (2012)	2	2	2	2	N/A	N/A	N/A	2	2	2	0	N/A	2	2	18	10	.900
Fonseca, Ireland & Resnick (2002)	2	2	2	2	N/A	N/A	N/A	1	2	2	2	N/A	2	2	19	10	.950

Francisco, Narcisco & Alarcao (2013)	2	2	1	1	N/A	N/A	N/A	2	2	2	2	N/A	2	2	18	10	.900
French, Leffert, Story, Neumark-Sztainer, Hannan & Benson (2001)	2	2	1	2	N/A	N/A	N/A	1	2	2	2	N/A	2	2	18	10	.900
Fulkerson, Story, Mellin, Leffert, Neumark-Sztainer & French (2006)	2	2	1	2	N/A	N/A	N/A	1	2	2	2	N/A	2	2	18	10	.900
Haines, Gilman, Rifas-Shiman, Field & Austin (2009)	2	2	1	2	N/A	N/A	N/A	1	2	2	2	N/A	2	2	18	10	.900
Haines, Kleinman, Rifas-Shiman, Field & Byrn Austin (2010)	2	2	1	1	N/A	N/A	N/A	1	2	2	2	N/A	2	2	17	10	.850
Kunstman, Smith & Maner (2014)	2	2	1	2	1	2	2	2	2	2	2	1	2	2	25	14	.887
Lampis, Agus & Cacciarru (2014)	2	2	2	2	N/A	N/A	N/A	2	2	2	0	N/A	2	2	18	10	.900
Loth, Wall, Choi, Bucchianeri, Quick, Larson, Neumark-Sztainer (2015)	2	2	2	2	N/A	N/A	N/A	1	2	2	2	N/A	2	2	19	10	.900
Mazur, Dzielska & Malkowska-Szkutnik (2011)	2	2	2	1	N/A	N/A	N/A	2	2	2	2	N/A	2	1	18	10	.900
McVey, Pepler, Davis, Flett & Abdolell (2002)	2	2	1	2	N/A	N/A	N/A	2	2	2	2	N/A	2	2	19	10	.950
Neumark-Sztainer, Eisenberg, Fulkerson, Story & Larson (2008)	2	2	2	2	N/A	N/A	N/A	1	2	2	2	N/A	2	2	19	10	.950
Neumark-Sztainer, Wall, Story & Falkerson (2004)	2	2	2	2	N/A	N/A	N/A	1	2	2	2	N/A	2	2	19	10	.950
Neumark-Sztainer, Wall, Story & Sherwood, (2009)	2	2	2	2	N/A	N/A	N/A	1	2	2	2	N/A	2	2	19	10	.950
Neumark-Sztainer, Wall, Haines, Story, Sherwood & Van der Berg (2007)	2	2	2	2	N/A	N/A	N/A	1	2	2	0	N/A	2	2	17	10	.850

Nicholls & Viner (2009)	2	2	2	1	N/A	N/A	N/A	2	2	2	2	N/A	2	2	19	10	.950
Perkins, Luster & Yank (2002)	2	2	2	2	N/A	N/A	N/A	1	2	2	2	N/A	2	2	19	10	.950
Schirk, Lehman, Perry, Ornstein, & McCall-Hosenfeld (2015)					N/A	N/A	N/A					N/A					1.000
Scoffier, Maiano, & D'Arripe-Longueville (2010)	2	2	1	2	N/A	N/A	N/A	2	2	2	2	N/A	2	2	19	10	.950
Twamley & Davis (1999)	2	2	1	2	N/A	N/A	N/A	2	1	2	2	N/A	2	2	18	10	.900
Wang, Peterson, Richmond, Spadano-Gasbarro, Greaney, Mezgebu McCormick & Byrn Austin (2013)	2	2	2	2	N/A	N/A	N/A	1	2	2	2	N/A	2	2	19	10	.950

Appendix B: Participant Recruitment Advertisements

UCL

Are you thinking of starting the 5:2 Diet?

Are you curious about the impact it has on our thinking, feelings or behaviour?

Would you like to help us find out more?

We are researchers, looking to understand more about the psychological effects of the 5:2 diet/ intermittent fasting in healthy adults. We will be investigating the diet's impact on mood, cognition (thinking), and eating behaviour. We would like to compare how people answer certain questions before and after starting the diet, and how they perform on certain online tasks on fasting and non-fasting days.

We are looking for people (aged 18-65) who have not yet started the 5:2 diet, but intend to do so for at least a month, to take part in our research. Participation will involve completing questionnaires, and doing online experimental tasks. You can participate from anywhere as the study can be completed online and over the phone.

All participants will be entered into a prize draws to win Amazon vouchers. There will be 12 vouchers in total to be won (ranging from £20-£100). For each person taking part in the study we will also donate £1 to charity and, and you will be able to vote for the charity this money goes to. Once we have completed the study, all participants will also receive a summary of the findings.

If you think you might be interested in taking part, or would like more information, **please contact us to find out more:**

[redacted]

We look forward to hearing from you,

Jasmin Langdon-Daly and Kate Mahony
UCL DClinPsy Programme

Appendix C: Outcome Measures – Food Diary

Time/Date Fast (F)/ Non fast (NF)	Situation (where, who with etc.)	Hunger level (0-10)	Mood (%,	Food/ drink	Amount	Sense of loss of control (Yes/No)	Any other comments
Example: 7pm 22nd August (NF)	*Dinner in a restaurant with friends*	*6*	*Happy (70%) Tired (40%)*	*Italian style pizza with ham, mushrooms, cheese and tomato White wine*	*1 10" pizza 1 large glass*	*No*	
8.30am 23rd August (F)	*Breakfast at my desk in office, alone, checking emails*	*4*	*Bored (50%) Annoyed (20%)*	*Ryvita with cottage cheese Tea (breakfast tea) with milk*	*2 ryvita, 2 tbsp cottage cheese 1 mug tea, splash milk*	*No*	

Appendix D: Participant Information Sheets, Consent Forms and Instructions

Participant Information Sheet

Information Sheet for Interested Participants
Investigating the Impact of Intermittent Fasting (5:2) Diets on Cognition, Behaviour and Emotional Wellbeing

This study is approved by the UCL Research Ethics Committee (Project ID Number): 6377/001

Name and Contact Details: Jasmin Langdon-Daly & Kate Mahony [redacted]
Dr Lucy Serpell
Work Address: Research Department of Clinical Educational and Health Psychology
1-19 Torrington Place, University College London, London WC1E 7HB

We would like to invite you to participate in this research project. You may be able to participate if you are an adult who is about to start the 5:2 diet. You should only participate if you want to. Before you decide whether you want to take part, it is important for you to read the following information carefully. If anything is not clear or if you would like more information, please contact us on the email addresses above.

Information for participants

The 5:2 diet is a form of intermittent fasting: one eats normally for five days per week and reduces calorie intake to 25% of normal requirements, usually 500 calories per day, for two days per week. Although the 5:2 diet is gaining popularity, there is not much research on its impact on people's thoughts, feelings and abilities. The aim of this study is to find out about the impact of starting the 5:2 diet on mood, eating behaviour and ability to do certain mental tasks.

We are looking for healthy adults (18-65 yrs) who are planning to start the 5:2 diet but have not yet begun, to take part in our study.

We would like to compare how people answer certain questions before and after starting the diet, and how they perform on certain online tasks on fasting and non-fasting days. If you decide to participate, before you start the diet we will ask you to complete a food diary for one week and fill out a series of questionnaires online which will ask about your mood, eating behaviours, self-esteem and thinking about food, shape and weight (around 15 minutes). Once you have begun dieting, we will then ask you to complete online tasks (lasting about 20 minutes) at least twice, on different days. After one month of dieting, you will be asked to complete the same food diary and similar questionnaires again.

As an acknowledgement of your contribution participants will be entered into a prize draw to win Amazon vouchers once you have completed the baseline measures. Upon finishing the study, you will be entered into another draw to win more prizes. There will be 12 vouchers in total to be won (£20-£100). For each person taking part in the study we will also donate £1 to charity and, and you will be able to vote for the charity this money goes to. Once we have completed the study, all participants will also receive a summary of the findings.

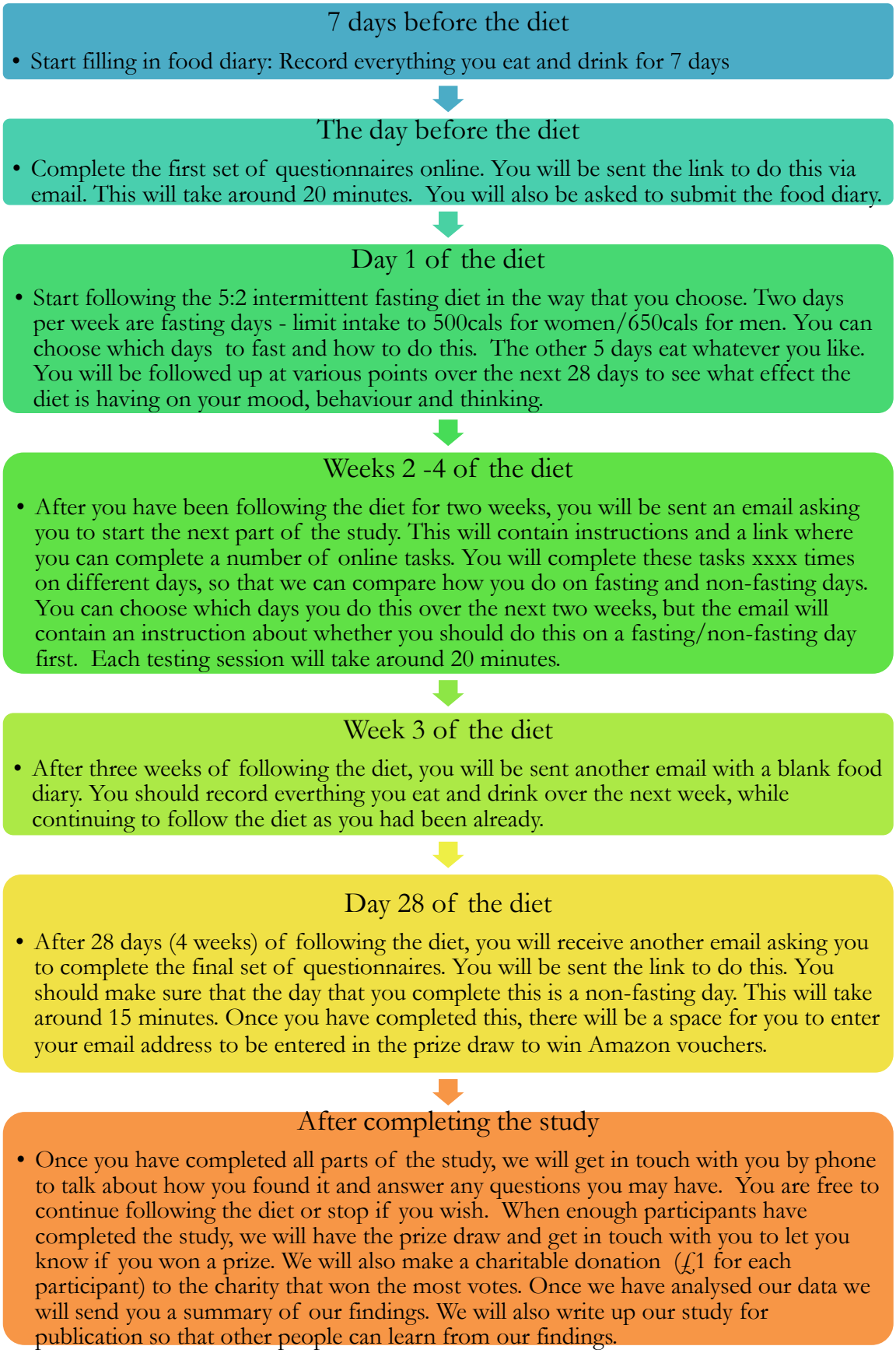
It is up to you to decide whether or not to take part. If you decide to take part you are still free to withdraw from the study at any time during the testing session and without giving a reason. This will not affect your rights in any way.
If you would like to take part, please read the consent form below, and if you agree to the terms then please sign to indicate your consent. After this, you will be asked to choose a date to start the diet and be sent instructions about the next steps. You will be contacted by the researchers to remind you to complete the various steps, and can contact us at any point with questions.

All information provided (along with your personal details) will be kept confidential, and anonymised. Your personally identifiable details will not be linked to your individual responses. No information about you will be disclosed to a third party.

Your participation will contribute significantly to our understanding regarding dieting and psychological well-being. Thank you very much for your time.

All data will be collected and stored in accordance with the Data Protection Act 1998.

Participant Information Sheet: Study Process



Informed Consent Form for Participants in Research Studies

Please complete this form after you have read the Information Sheet and/or listened to an explanation about the research.

Title of Project: Investigating the Impact of Intermittent Fasting (5:2) Diets on Cognition, Behaviour and Emotional Wellbeing

This study has been approved by the UCL Research Ethics Committee [Project ID Number: 3529/001]

Thank you for your interest in taking part in this research. Before you agree to take part the researcher must explain the project to you.

I confirm that I have read the Information Sheet, and that I have had an opportunity to ask the researcher any questions or raise any concerns about the project with her, and have had these answered satisfactorily.

I understand what taking part in the study involves.

I understand that participation is voluntary, and I am free to withdraw from the study at any time, without giving a reason.

I understand that I must not take part if I have health conditions which make dieting inappropriate, e.g. pregnancy or diabetes, and that I am free to stop the diet at any point should I feel unwell or uncomfortable.

I consent to the processing of my personal information for the purposes of this study.

I understand that such information will be treated as strictly confidential and handled in accordance with the provisions of the Data Protection Act 1998.

I agree to take part in this study.

Signed: Date:

Consent to Complete Testing Session Message

You are completing this testing session as you have agreed to take part in our study. The aim of this study is to find out about the impact of starting the 5:2 diet on mood, eating behaviour and ability to do certain mental tasks. The 5:2 diet is a form of intermittent fasting. It involves having two fasting days per week, where you eat no more than 500cals for women and 650cals for men, and then eating what you like for the rest of the week.We would like to compare how people answer certain questions before and after starting the diet, and how they perform on certain online tasks on fasting and non-fasting days.

Over the next few pages you will be asked to answer some questions about your mood, eating patterns and other things. This should take around 15 minutes. All of your responses are confidential, are stored securely, and can be identified only by your participant number. Remember that you are free to withdraw from the study at any point without giving a reason.

All data will be collected and stored in accordance with the Data Protection Act 1998.

Do you consent to taking part in this testing session?

(If not please close the browser and do not continue)

- Yes

Participant Instruction Emails

Initial Study Instructions

Dear XXXX,

Thank you very much for agreeing to give your time to take part in our intermittent fasting study. Remember that you can decide that you no longer wish to take part at any point in the future without having to give a reason.

Attached to this email is a document which has an outline of the whole study so you have a clear idea of what you will need to do and when. We will also email you to remind you when you need to complete the next part of the study. All of the study can be completed online, via the links that we send you.

The first thing that you need to do is complete the Consent Form, and email back to us as soon as you can (either a scanned copy of this completed form or fill it in using an electronic signature).

We agreed that you would begin the food diary tomorrow, **Thursday 16th July,** complete the first set of questionnaires on **Wednesday 22nd July** and then start following the intermittent fasting 5:2 diet the day after that on **Thursday 23rd July 2015**. You are free to follow the diet in the way that you choose, and to decide which two days of the week you wish to 'fast' (meaning limiting your intake to 500cals for women and 650 cals for men). We would ask you to complete the first set of cognitive tasks on a **Non-Fasting day**, and the second set on a **Fasting day** in the evening (6-10pm) at the same time each time.

You have been assigned the **participant number XXX.** Please use this number whenever you complete part of the study, and do not use your name. This will help us to link your data together while ensuring that it remains anonymous and secure. When password protecting documents, the password is: xxxxxx

We have attached a blank food diary. Could you please fill this in in the week up until you complete the first set of questionnaires, when there will be an opportunity to submit it. Please write your participant number but not your name on this diary.

In the meantime, please use this Doodle Poll to vote for the charity that you would like us to make the donation to at the end of the study. We will donate £1 for each participant to the charity receiving the most votes:

https://doodle.com/6vmee26rtct6u3pe

If you have any questions or concerns, or feel that you need more information to help you complete the study, please do not hesitate to get in contact with us.

Thank you again for your help,
Jasmin and Kate

Trainee Clinical Psychologists
UCL DClinPsy Programme
1-19 Torrington Place
London WC1E 7HB

Baseline Questionnaire Testing Session Instructions

Dear XXX,

It is now time for you to complete the first set of questionnaires as part of our intermittent fasting study. You can complete this by clicking on the link below. This should take about 20 minutes.

https://uclpsych.eu.qualtrics.com/SE/?SID=SV_0oBSGtnliRaoALj

Please make sure that you enter your participant number, which is **XXX.** You should not enter your name. At the end of completing the questionnaires, you should send me the food diary with the information about what you have eaten and drunk in the last week. I have also attached a questionnaire called the Shape and Weight Based Self-Esteem Scale (SAWBS),which is in a format that is difficult to complete online. Could you complete this, either using Microsoft Word or by printing it, completing it and scanning it, and then email it at the same time as the food diary. Both of these documents should have your participant number written on them and should be password protected using the password diet (see below for instructions about how to do this). If you have difficulties emailing these documents you can freepost them to me (see address below).

Once you have completed these questionnaires, you can start to follow the 5:2 intermittent fasting diet in the way that you choose. This involves having two fasting days per week, where you eat no more than 500cals for women and 650cals for men, and then eating what you like for the rest of the week. You can choose which days you want to fast and how you want to plan your meals on those days.

We will be in touch again in a few weeks to let you know about the next part of the study. In the meantime if you have any questions, please do get in touch.

Thank you for your time,

Jasmin and Kate

Email: [redacted]
Post: FREEPOST University College London, London WC1E 6BT
Department of Clinical, Educational and Health Psychology
(Attention: Jasmin Langon-Daly, DClinPsy trainee)
(Please let me know if you have posted so I know to check the FREEPOST)

Final Questionnaire Testing Session Instructions

Dear XXXXX

It is now time for you to complete the final set of questionnaires as part of our intermittent fasting study. You can complete this by clicking on the link below. This should take about 15 minutes.
https://jfe.qualtrics.com/form/SV_a3LaxH4MHBYinn7

Please do take the time to complete these, so that we can use your data from the start of the study. It is important that you complete these questionnaires on a *Non-Fasting Day*.

Please make sure that you enter your participant number, which is **XXX**. You should not enter your name. At the end of completing the questionnaires, you should email or post me your food diary with the information about what you have eaten and drunk in the last week. This document should have your participant number written on it and should be password protected using the password that we have agreed (see below for instructions / postal address).

Once you have completed these questionnaires, you have finished all parts of the study. THANK YOU! As a token of our thanks you will be entered into the Prize Draw to win Amazon vouchers. Once you have completed the study, you are free to continue following the diet or stop.

If you would like to speak to us about how you found the study, or if you have any remaining questions, please email us or let us know a good time to call.

Once we have collected data from all the participants, we will hold the Prize Draw and contact you if you have won a prize. We will also donate £1 per participant to the charity that received the most votes. Once we have analysed all of the data, we will send you a summary of our findings. We
will also aim to have our study published so that more people can benefit from what we have learnt.

Thank you again for your time and support,

Jasmin and Kate
UCL DClinPsy Programme

Appendix E: Ethical Approval for Study

UCL RESEARCH ETHICS COMMITTEE
ACADEMIC SERVICES

Dr Lucy Serpell
Department of Clinical, Educational and Health Psychology
UCL

28 January 2015

Dear Dr Serpell

Notification of Ethical Approval
Project ID 6377/001: Investigating the impact of intermittent fasting diets on cognition, behaviour and emotional well-being

I am pleased to confirm in my capacity as Chair of the UCL Research Ethics Committee (REC) that your study has been approved by the UCL REC for the duration of the project i.e. **until September 2016**.

Approval is subject to the following conditions:

1. You must seek Chair's approval for proposed amendments to the research for which this approval has been given. Ethical approval is specific to this project and must not be treated as applicable to research of a similar nature. Each research project is reviewed separately and if there are significant changes to the research protocol you should seek confirmation of continued ethical approval by completing the 'Amendment Approval Request Form': http://ethics.grad.ucl.ac.uk/responsibilities.php

2. It is your responsibility to report to the Committee any unanticipated problems or adverse events involving risks to participants or others. Both non-serious and serious adverse events must be reported.

   Reporting Non-Serious Adverse Events
   For non-serious adverse events you will need to inform Helen Dougal, Ethics Committee Administrator [redacted] within ten days of an adverse incident occurring and provide a full written report that should include any amendments to the participant information sheet and study protocol. The Chair or Vice-Chair of the Ethics Committee will confirm that the incident is non-serious and report to the Committee at the next meeting. The final view of the Committee will be communicated to you.

   Reporting Serious Adverse Events
   The Ethics Committee should be notified of all serious adverse events via the Ethics Committee Administrator immediately the incident occurs. Where the adverse incident is unexpected and serious, the Chair or Vice-Chair will decide whether the study should be terminated pending the opinion of an independent expert. The adverse event will be considered at the next Committee meeting and a decision will be made on the need to change the information leaflet and/or study protocol.

On completion of the research you must submit a brief report (a maximum of two sides of A4) of your findings/concluding comments to the Committee, which includes in particular issues relating to the ethical implications of the research.

Appendix F: Details of Collaboration in Joint Project

The empirical research outlined in Part Two of this thesis was undertaken as part of joint project with Kate Mahony, another trainee clinical psychologist at UCL. Her part of the project studied the same group of participants following 5:2 IF, using a repeated measures design to compare performance on a range of cognitive tasks on fasting vs non-fasting days. The details of this part of the project are outlined in her thesis submission: Mahony, K. (2016). Nutrition and cognition: Exploring their relationship from two sides of the same coin. Clinical Psychology Doctorate Thesis.

Aspects of research undertaken independently:

- Review of literature
- Research proposal
- Selection of measures
- Study design for this part of the project
- Data collection and processing
- Data analysis
- Write up of empirical paper

Aspects of research undertaken jointly:

- Agreeing overall study protocol
- Research governance tasks (application for ethical approval, funding, risk assessment, data protection)
- Recruitment of participants
- Correspondence with participants

Appendix G: Results of Non-Parametric Alternative Analyses

Measure	Mean (SD)		Test used	Z statistic	*p*
	Baseline	Final			
EDEQ Binge frequency	3.73 (5.63)	1.74 (4.27)	Wilcoxon signed rank test	-4.07	<.001**
BEDT score	47.48 (18.30)	41.38 (15.44)	Wilcoxon signed rank test	-5.142	<.001**
State Food Craving score	29.79 (12.65)	26.01 (11.11)	Wilcoxon signed rank test	-2.545	.011*
DASS-21 score	31.72 (6.61)	29.78 (6.98)	Wilcoxon signed rank test	-2.887	.004**

**= significant at p<.05, **= significant at p(.001)*