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Psychological and Behavioural Aspects: Appetite in Context

Jason Halford & Carl Roberts

Behavioral and psychological consideration of Weight Management

Overview

- Hunger and satiety signalling from our gut to our brain
- Central psychological influences: Hedonics & Neurobiology of Reward
- People living with obesity have a biological vulnerability for weight gain
- Dieting intensifies biological vulnerability
- Broader psychology of weight-gain – depression, stress
- Obesity is a disease

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Appetite Expression

- Human eating is a complex and varied behaviour
- Hunger, food craving, and the anticipation and enjoyment of eating are all ***psychological experiences*** that are underpinned by interactions between peripheral and central/neuro-biology
- Appetite regulation involves a complex interplay between hunger/satiety, reward processes and cognitive control processes (Roberts, Christiansen & Halford, 2017)
- In the brain these processes are ascribed to interacting hypothalamic and mesocorticolimbic neuro-circuitry (Morales & Berridge, 2020).

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Hunger and satiety signalling from our gut to our brain

- The brain integrates numerous signals from the body in order to determine the energy requirement of the body and to modify the experience of hunger and initiate the relevant behavioural actions in response to this.
- We feel hungry --> we eat
- We have started to understand the biology that underpins short term feelings of hunger

Defining concepts in appetite (Psychological experiences)

Hunger

- The drive to consume, eliciting and sustaining a behavioural response (***eating***) in response to a biological need.

Satiation

- Processes during a meal that generate negative feedback leading to its termination (***within-meal inhibition***).

Satiety

- The end state of satisfaction. The further suppression of the drive to consume and post meal intake (***between-meal inhibition***).

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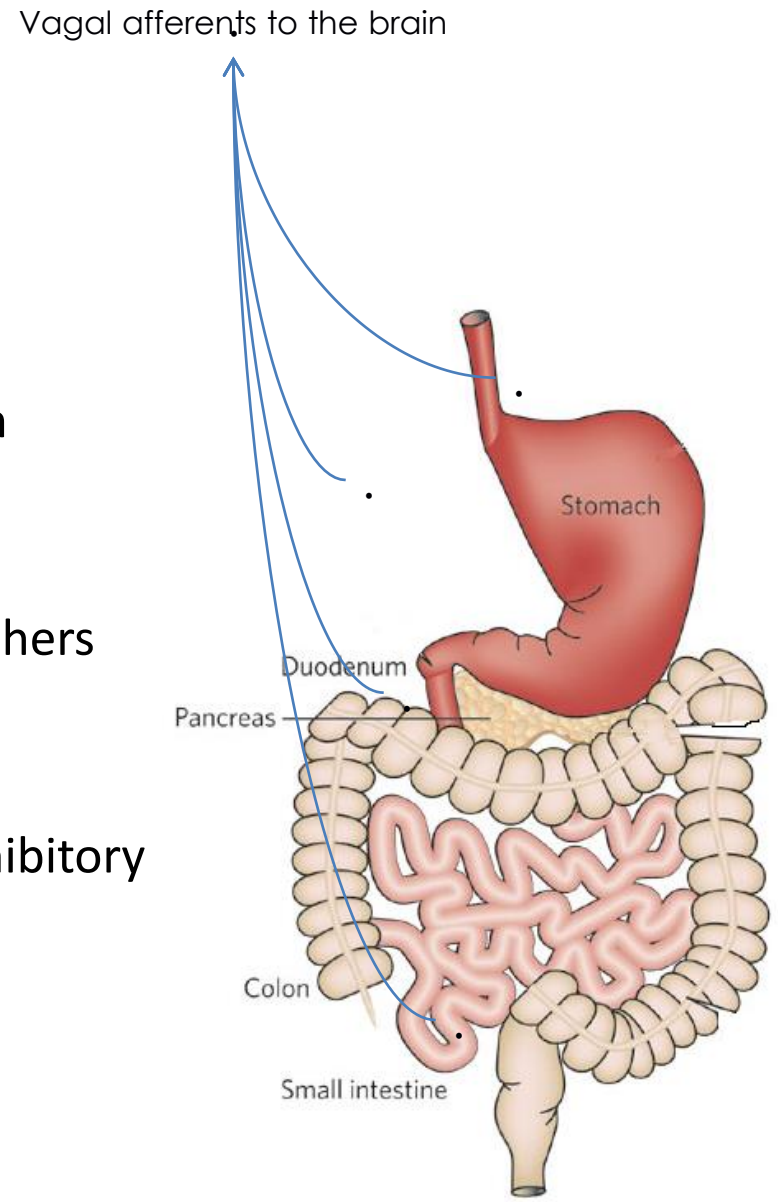
Short term appetite regulation Satiety-related hormones

- After we eat signals are generated by nutrients in the gastrointestinal tract (hormone release from stomach, intestine, & colon)
- Hormones (satiety-related peptides) signal to the brain (hypothalamus)
- Some signals exert their effects during the meal and others after the meal
- Some signals are stimulatory (ghrelin) but most are inhibitory (CCK, GLP-1)



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Short term satiety signals are modulated by other “long term” signals

- Insulin secreted by the pancreas and leptin secreted by adipocytes – these are signals that inhibit neurons that promote energy intake and excite neurons that reduce energy intake in the brain
- These signals modulate how responsive the brain is to satiety signals from food
- These long term signals change in relation to weight gain i.e. as we gain weight these reduce the “short term” satiety signals to the brain.
- Taken together – there is a complex biology that belies our experience of hunger

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Central psychological influences: Hedonics & Neurobiology of Reward

Physiological models of food intake give relatively little attention to ***psychological influences*** such as the enjoyment and pleasure of eating. Which can be termed FOOD HEDONICS.

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Hedonic eating – food is rewarding

- Whilst there is an impressive gut-brain system designed to achieve energy homeostasis – Given prevalence of people living with high obesity, it is clear that these signals are unable to prevent weight gain in modern environment.

- Palatability and pleasure are arguably THE MOST powerful motivators of food intake – the rewarding nature of food easily overrides homeostatic control of appetite.

- Food reward drives eating in the absence of hunger

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Hedonic eating – food pleasure

- We have evolved efficient motivational mechanisms that direct us toward food sources (wanting), encourage overconsumption (liking) and permit storage of excess energy as a protection against food shortage.
- Our food reward system can override satiety signalling allowing us to eat when we do not feel hunger and undermine the ability to exert control overeating
- The prevalence of people living with obesity is an indication of the efficiency of these hedonic processes in governing eating motivation.
- Arguably weight-gain is the natural response to our changing food environment

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Defining concepts in appetite: hedonic and motivational factors (Psychological experiences)

- **Wanting**
  - The hedonic motivation to consume a specific food, manifesting explicitly (craving) or implicitly (incentive salience)

- **Liking**
  - The sensory pleasure elicited by contact with food contributing to the hedonic motivation to consume (**wanting**)

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Neurobiology of reward

- Wanting and liking are separable psychological processes with overlapping but distinct neuro-circuitry

- Wanting - located primarily in the mesocorticolimbic reward pathway comprising the nucleus accumbens shells, dorsal striatum and amygdala (Morales & Berridge, 2020).

- Neuro-chemically ‘wanting’ is understood to be underpinned primarily by dopamine, glutamate, opioid and endocannabinoid neurotransmission (Berridge et al., 2010)

- Liking’ (pleasure) on the other hand is primarily generated in a sub-region of the mesocorticolimbic reward pathway (nucleus accumbens)

- Notably, dopamine does not appear to have a primary role in liking, instead ‘liking’ is primarily underpinned neuro-chemically by opioid and endocannabinoid activity (Kirkham, 2009).

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People living with obesity have a biological vulnerability for weight gain

Behaviour associated with adiposity – underpinned by our (neuro)biology

Satiety signalling

- Inadequate impact of food
- Increased eating rate and a failure to develop normal satiation during the course of a meal
- weakened satiety signals after a meal (feel hungrier quicker)

Reward

- Greater responsiveness to food cues (sights and smell of food)
- Heightened hedonic responses to palatable food (eating in pleasurable)
- Craving

Less control over eating

- Experiences of uncontrolled hunger and greater disinhibition of eating behaviour

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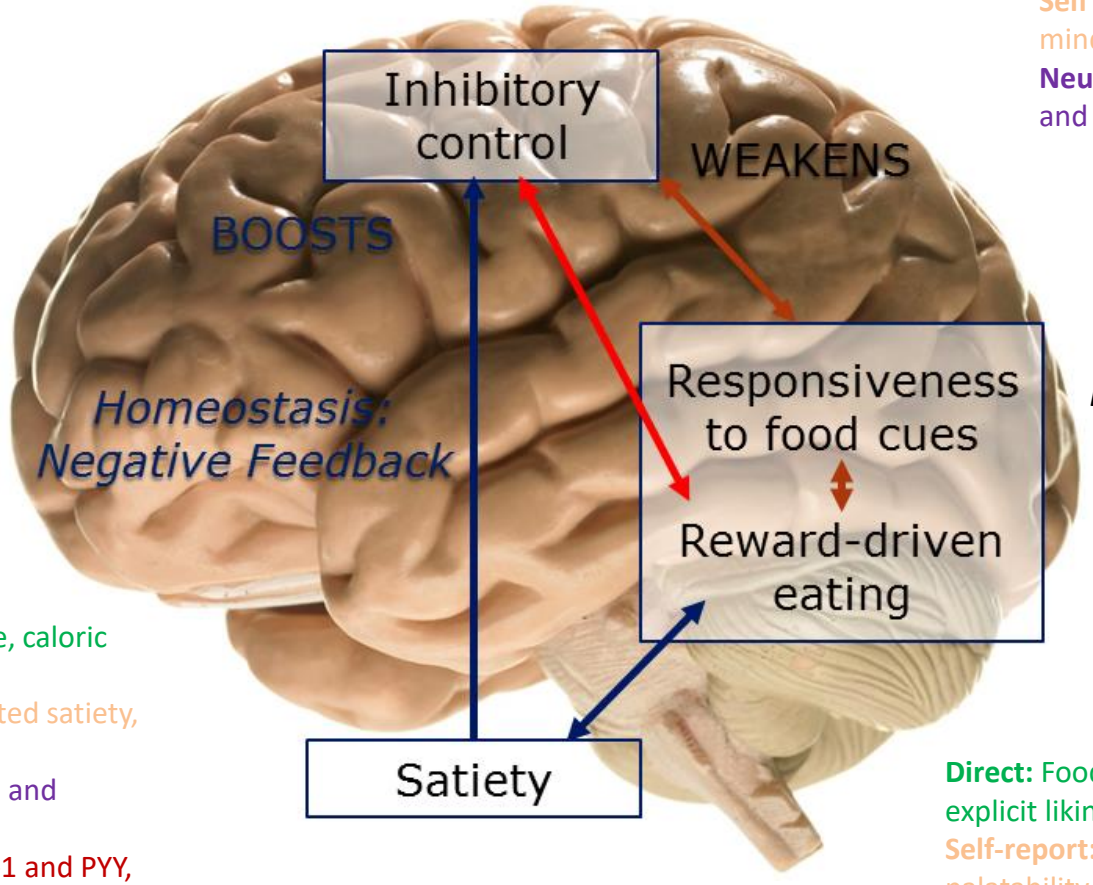
Appetite regulation involves a complex interplay between satiety, reward and inhibitory control

Regulatory control (satiety) and reward:

Dual System Model of CNS integration



Direct: Cue specific inhibitory control task.
Self report: Power of food, external eating, mindful eating.
Neurophysiological markers: anterior cingulate and dorsolateral PFC

Hedonic Drive: Positive Feedback

Direct: ad libitum intake, eating rate, caloric compensation.
Self-report: Appetite ratings, expected satiety, perceived hunger, satiety quotient.
Neurophysiological markers: insula and hypothalamus.
Physiological makers: Ghrelin, GLP-1 and PYY, metabolic factors, energy balance.

Direct: Food Choice, macronutrient intake, implicit & explicit liking and wanting, visual probe, eye tracking.
Self-report: palatability, taste, pleasure, expected palatability, food preference, cravings,.
Neurophysiological markers: activation and connectivity mesolimbic DA system

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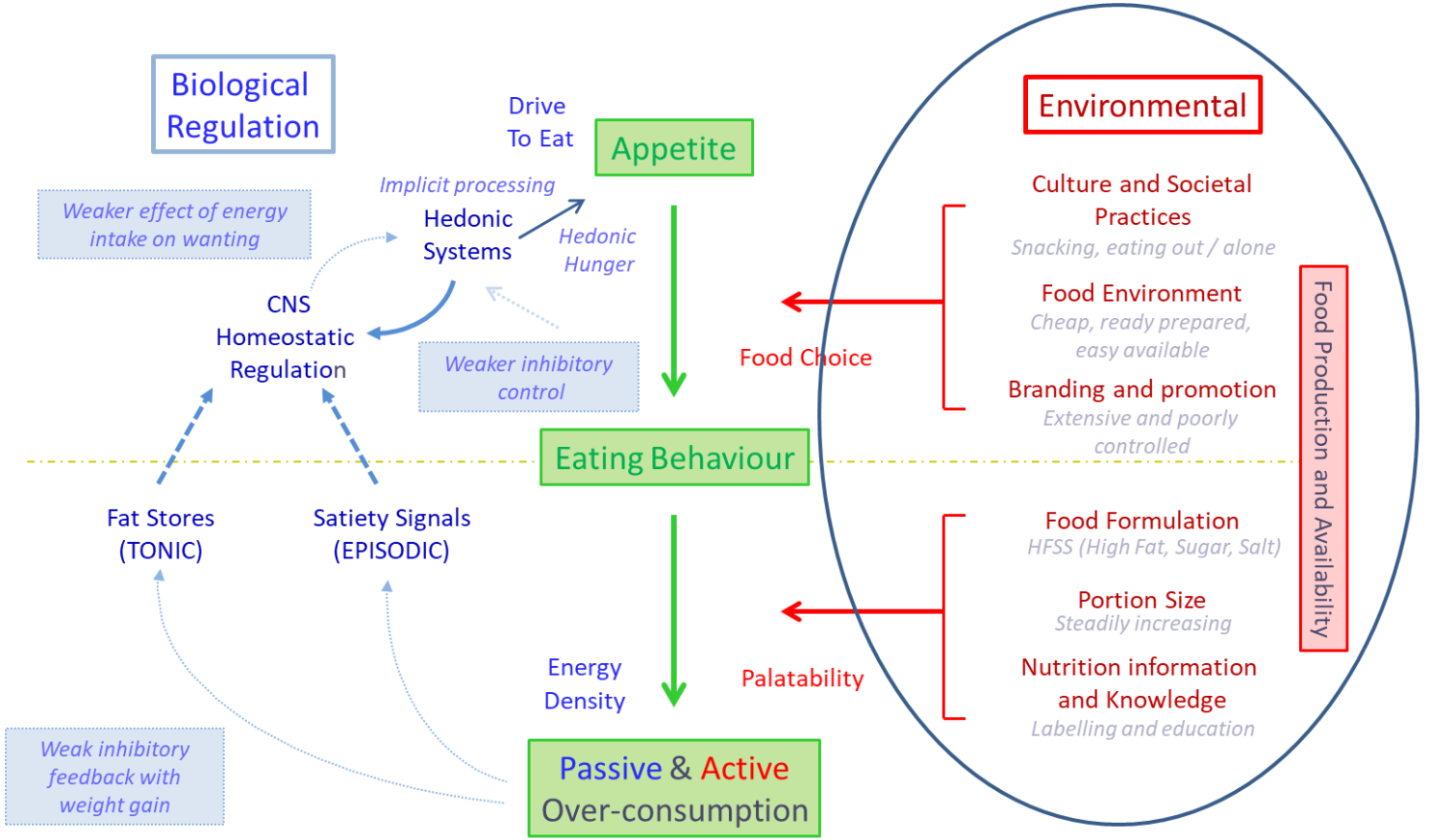
Interaction between biology and environment in the control of appetite and energy intake

- If we understand that individuals with obesity have weakened biological regulation of hunger / satiety, and also are highly responsive to food cues and hedonics then exerting control over eating already requires constant exertion

- This becomes problematic in modern westernised “obesogenic” environments whereby energy-dense foods are easily accessed, and palatable foods are heavily marketed



Blundell Cica 1993 adapted by Finlayson

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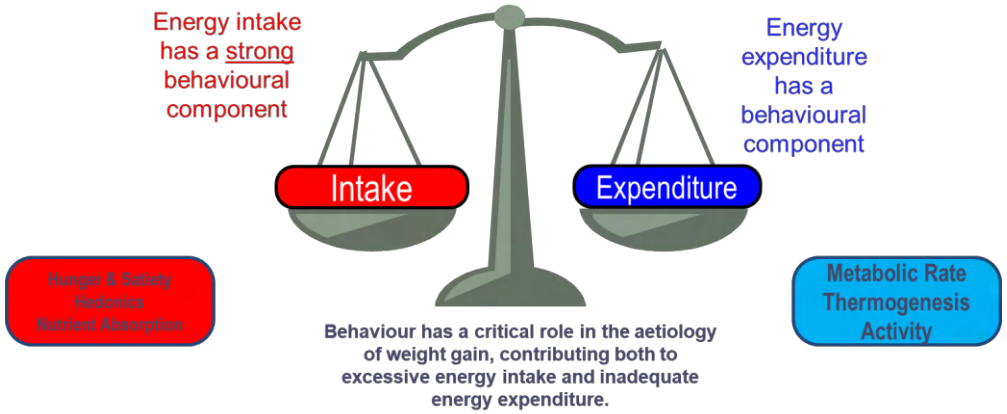
Role of Behaviour in Weight Control: The Energy Balance Equation



Fails to capture how difficult behaviour change is to achieve

- Due to weak satiety signalling from the gut, or powerful hedonic processes behaviour change is incredibly difficult
- not only because individuals with obesity have a biological vulnerability driving behaviour, but also, that patterns of eating and activity are shaped by life-long learning, and behaviour modification must also tackle entrenched habits.

- Maintaining healthier behaviours in an environment that promotes weight gain, demands constant exertion. The obesogenic environment primes individuals to relapse into their pre-intervention behavioural repertoire.
- Problematically, appetite regulation is asymmetrical, in that the body defends against under-consumption irrespective of current weight status or energy reserves.

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Interim Summary

1. Eating behaviour is influenced by the regulatory (*satiation and satiety*) and hedonic (*liking and wanting*) components of appetite

2. The former is driven by signals generated by ingestion (*largely but not exclusively physiological*), the latter by palatability of foods and the wider food environment.

3. The concept of energy balance fails to capture the difficulty of lasting behaviour change especially in an obesogenic environment.

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DIETING AND ENERGY RESTRICTION

Psychology of Deprivation and Physiological Consequences of Energy Deficit

The Challenge of Dieting.

Psychology of Deprivation and Physiological Consequences of Energy Deficit.

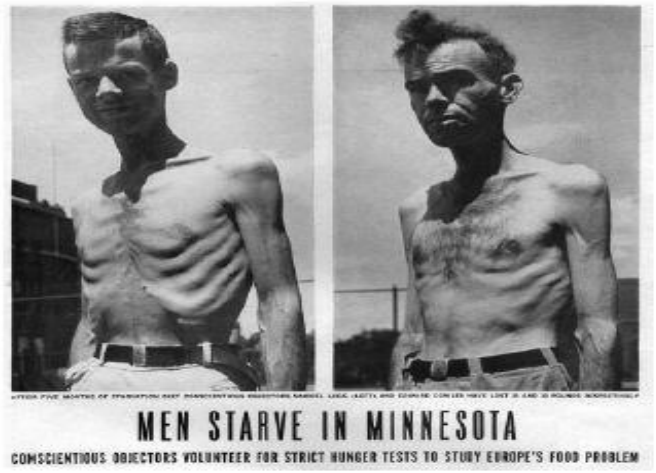

FIGURE 2 *Life* magazine photograph of conscientious objectors during starvation experiment. July 30, 1945. Volume 19, Number 5, p. 43. Credit: Wallace Kirkland/Time Life Pictures/Getty Images.

Obsession with food, increased response to food cues, cravings, loss of concentration and dysphoric mood all contribute to failure in dieting

Energy restriction and weight loss reduce satiety hormone levels – so change may outlast the diet

Hunger is a barrier to and a consequence of dieting

1. Increase in preoccupation with food
2. Relentless thoughts of food and eating inhibited concentration on usual daily activities
3. Serious difficulties adhering with diet when confronted with unlimited access to food

Biological mechanisms act to increase appetite

- During and after weight loss

After weight reduction, the brain is stimulated to increase caloric intake by changes in levels of circulating hormones

↓ Leptin

↑ Ghrelin

↓ GLP-1

↑ Increased appetite
↑ Increased preference for energy-dense foods (high-fat/sugary foods)

GLP-1, glucagon-like peptide-1

Eckel RH. *N Engl J Med*. 2008;358:1941–1950; Murphy et al. *Nature*. 2006;444:854–859

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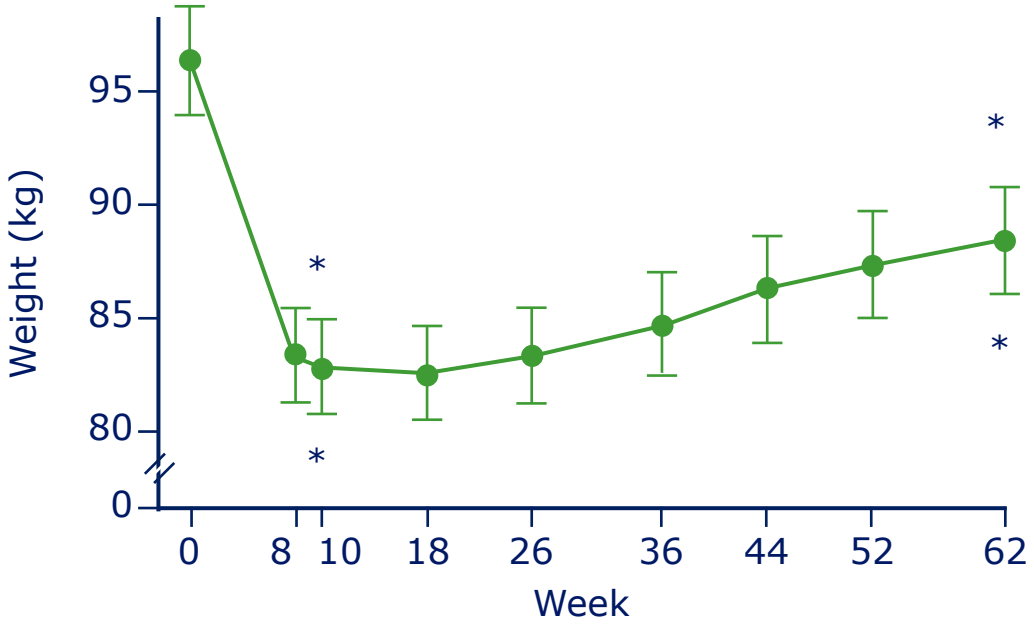
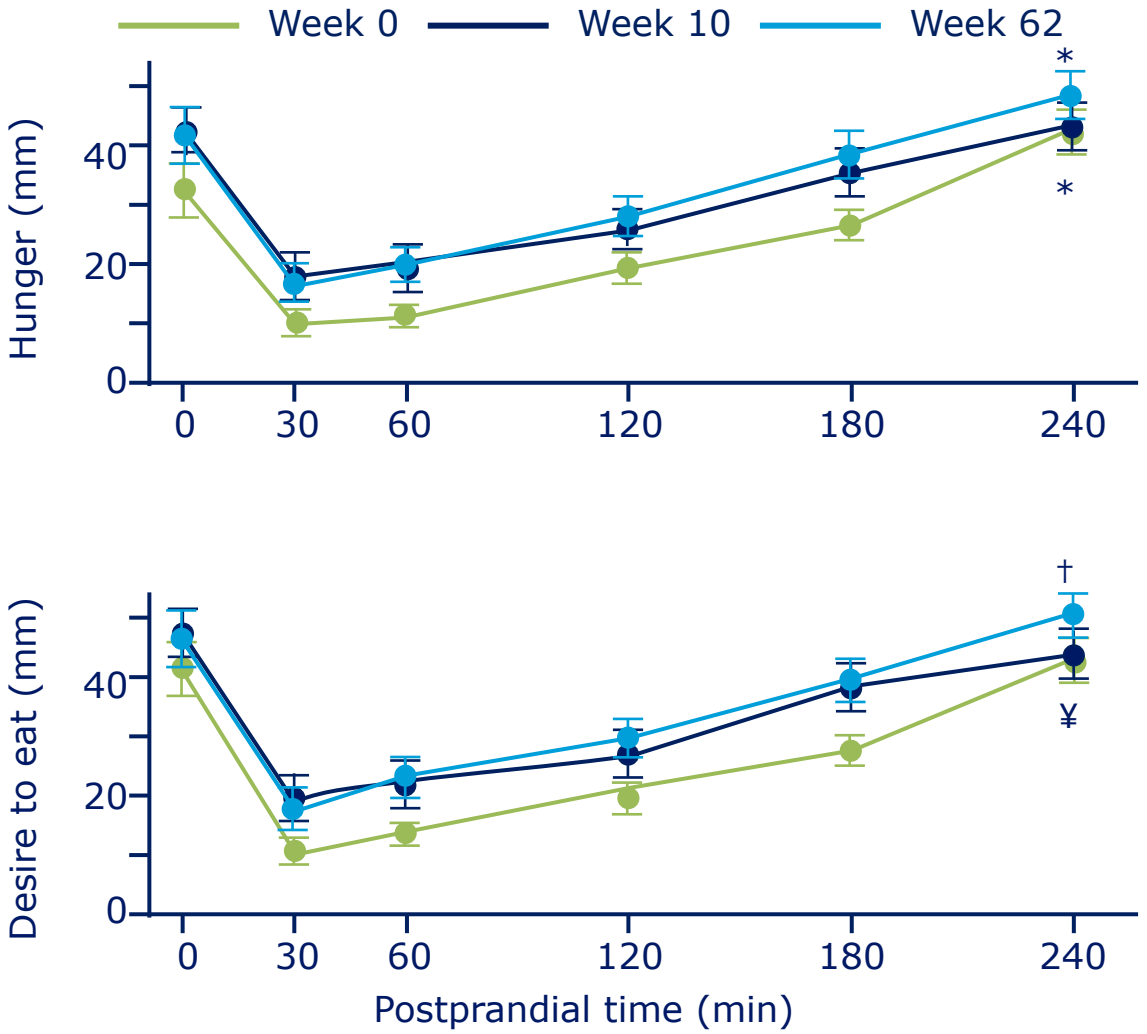
Hunger increases in response to weight loss

- 50 individuals with overweight/obesity lost weight on a 10-week VLCD
- Appetite was measured using VAS scores at 0, 10 and 62 weeks



*p<0.001, ¥p=0.008, †p=0.09 vs mean at baseline (week 0)

Sumithran *et al. N Engl J Med* 2011;365:1597–604



Corresponding increases in ghrelin and reductions in PYY response

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Food cue reactivity, cravings in dieters

Food cue responsiveness

- Hunger heightens perception of food cues.[2]
- Lower food cue reactivity predicts more successful weight loss in dieters.[3,4]

Cravings

- Dieters experience stronger cravings that are harder to resist and typically for the foods being restricted.[5]

Therefore, FCR and cravings act as barriers to weight loss success

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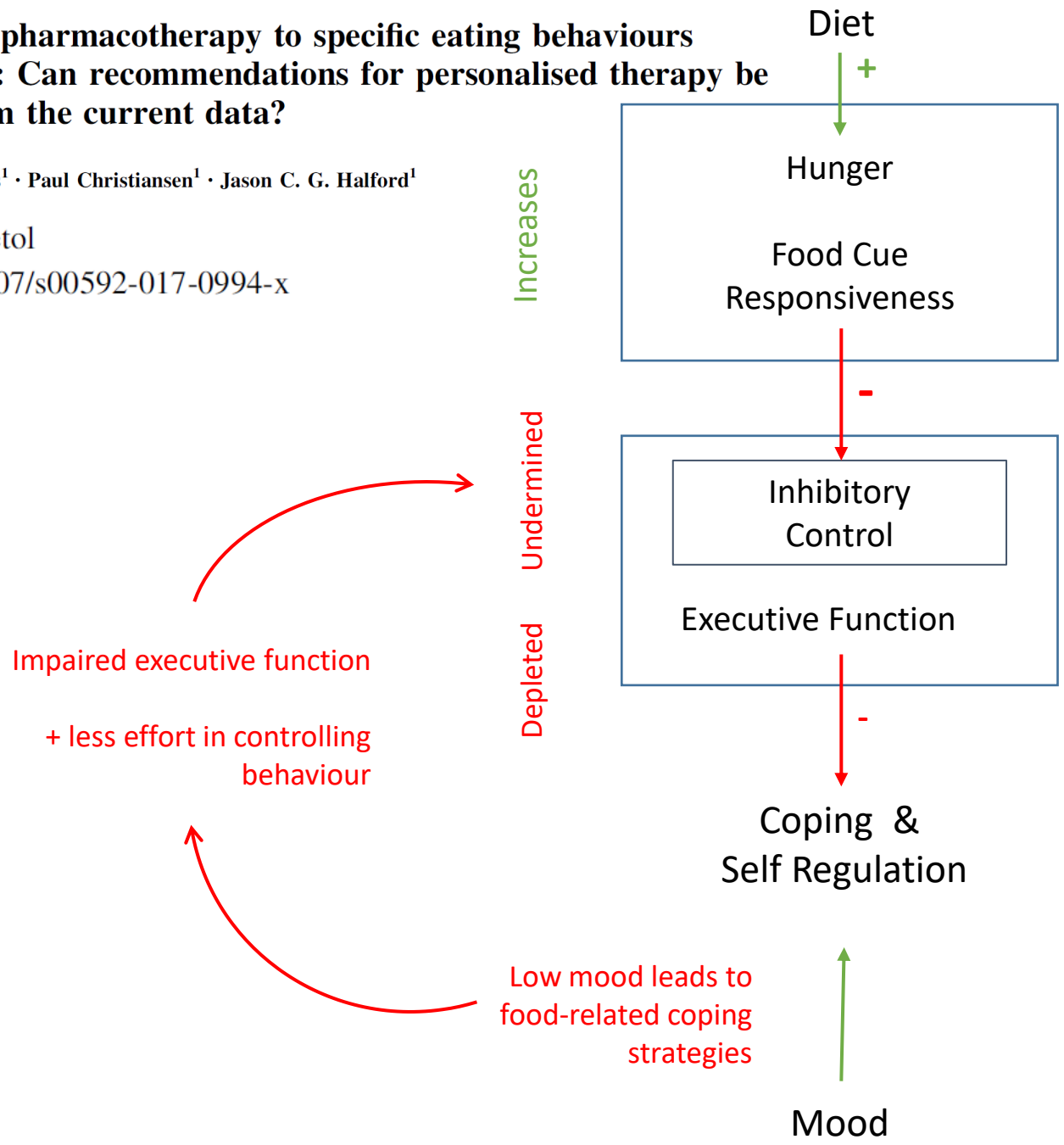
- 1. Nijs et al (2008) Eating Behaviors 9, 462-70; 2. Piech et al (2010) Appetite 54; 579-82;
- 3. Murdaugh et al, (2012) Neuroimage 59(3); 2709-21; 4. Ouwehand & Papies (2010) Appetite 55; 55-60;
  5. Massey and Hill (2012) Appetite 58 (3) 781-5; 6. Meule et al (2012) Appetite 38 (1) 88-97,

Tailoring pharmacotherapy to specific eating behaviours in obesity: Can recommendations for personalised therapy be made from the current data?

Carl A. Roberts[1] · Paul Christiansen[1] · Jason C. G. Halford[1]

Acta Diabetol
DOI 10.1007/s00592-017-0994-x



- Dieting increases hunger and food cue responsiveness, undermining inhibitory control and other executive functions and in turn, the ability to cope and maintain the diet (self-regulation)
- Dieting has negative effects on mood which reduces self-regulation of controlling behaviour and reintroduce food-related coping strategies
- Negative mood reduces inhibitory control and other executive functions producing a cycle whereby ability to control behaviour and self-regulation is undermined

Modified from Roberts et al. (2017)

Interim Summary

1. Individuals living with obesity tend to demonstrate weaker satiety signalling and increased responsiveness to the food environment. This will weaken inhibitory control (*the ability to resist*).

2. Dieting also tends to weaken satiety and increase responsive to the food environment (*irrespective of weight status*)

3. Individuals with obesity have a biological vulnerability which is intensified by dieting, that makes long lasting behaviour change very difficult. Individual weight management plans require patient input to tailor interventions to individual needs

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BROADER PSYCHOLOGY OF WEIGHT-GAIN

Live Events, Stress and Coping

Depression,

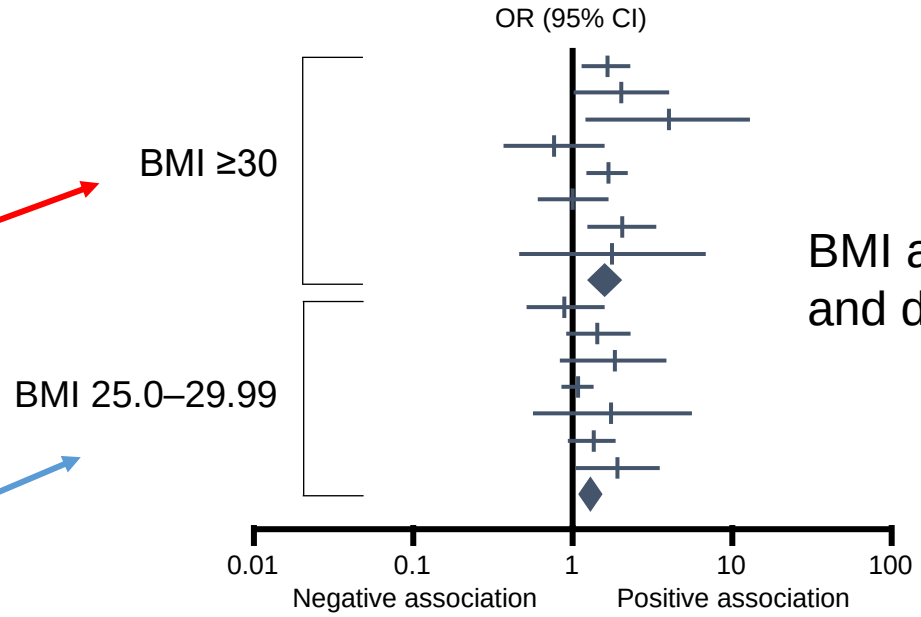
Overweight, Obesity, and Depression

A Systematic Review and Meta-analysis of Longitudinal Studies

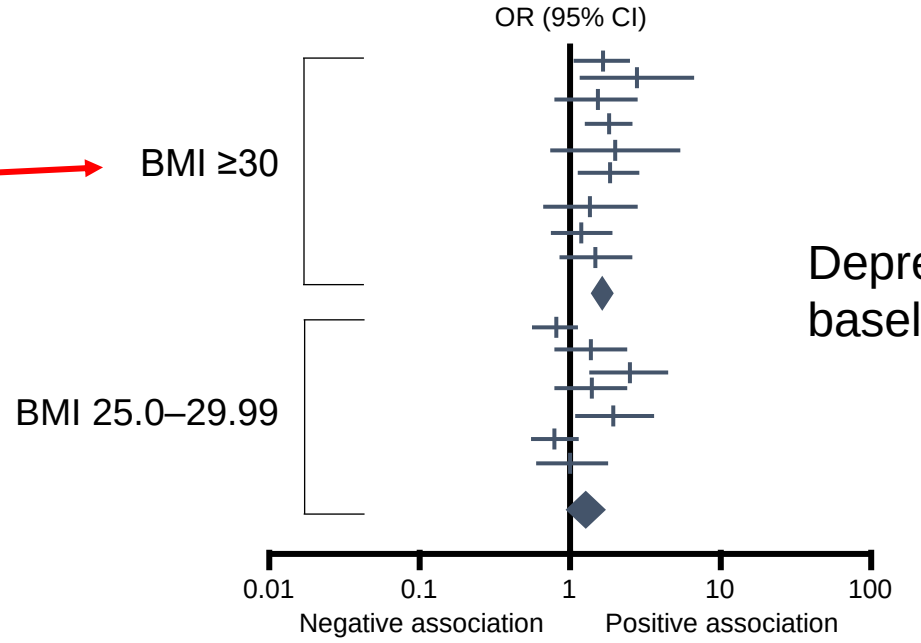
Floriana S. Luppino, MD; Leonore M. de Wit, MS; Paul F. Bouvy, MD, PhD; Theo Stijnen, PhD; Pim Cuijpers, PhD; Brenda W. J. H. Penninx, PhD; Frans G. Zitman, MD, PhD

1. Obesity at baseline increased the risk of onset of depression at follow up.
2. Overweight increased the risk of onset of depression at follow-up.
3. Baseline depression increased the odds for developing obesity.

Conclusion: a reciprocal link between depression and obesity



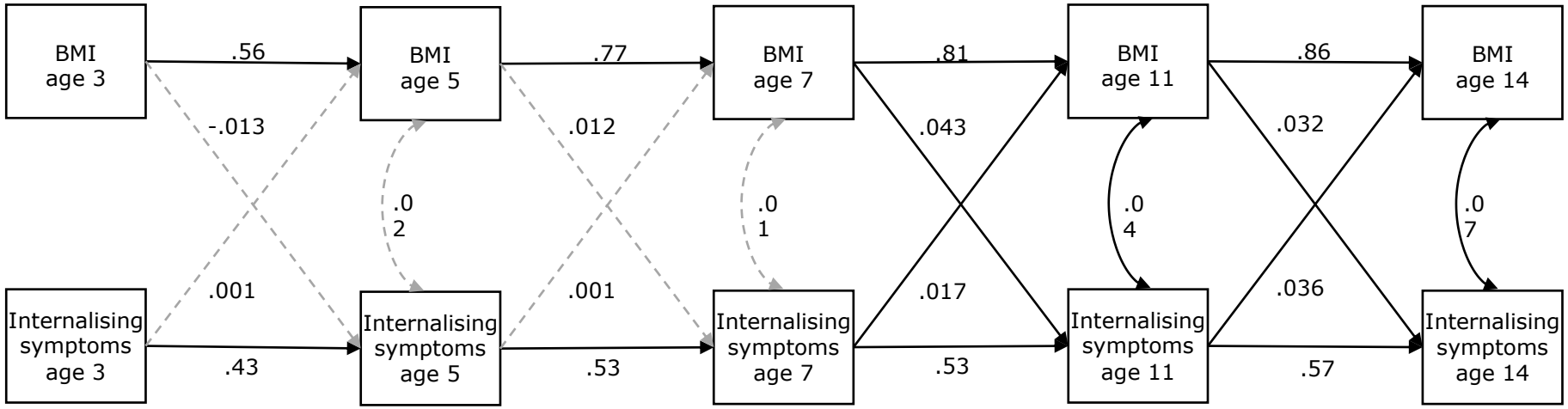
BMI at baseline and depression



Depression at baseline and BMI

Luppino FS et al. Arch Gen Psychiatry 2010;67:220–229.

Early Origins of Depression and Obesity (Millennium Cohort Study, N=17,215)



Patalay & Hardman, 2019 JAMA Ped

Model Fit: RMSEA=.046, CFI=.94, TLI=.90, SRMR=.048

- BMI and internalisation increase from early childhood (weak associate and not a risk factor for each other) - high level clinical symptoms increase from 5% in three year olds to 18% in children with obesity
- Bidirectional emerges between 7 and 14 (Full baseline model) - Associations explained in part by socio-economic risk

With self reported depressive symptoms at age 14, of those identified as obese based on BMI 25% have high depressive symptoms (35% female, 15% male)

Personal circumstance plays a role in weight gain as well as mental health issues

1. **Current events:** Traumatic life events such relationship break ups or widowhood or other forms of loss can effect body weight (Jeffry & Rick, 2002; Eng et al 2005).

2. **Past trauma:** Serious and sustained abuse and neglect may focus self care behaviour in adulthood.
   - Childhood maltreatment/neglect predict excessive weight gain in adolescence (Hessey et al 2006; Bentley & Widom, Lissau, 2009 & Sorensen, 1994).
   - Childhood physical/sexual abuse associated with obesity in women (Midei et al 2010).
   - Childhood bullying, rejection or emotionally abuse is associated with obesity in men (Gundstad et al. 2006).
   - Physical and verbal abuse up to 18 years old associated with later obesity (Williamson et al. 2002).
   - Severity rather than the type of trauma is associated with the likelihood of becoming obese (D'Argeno et al. 2009).

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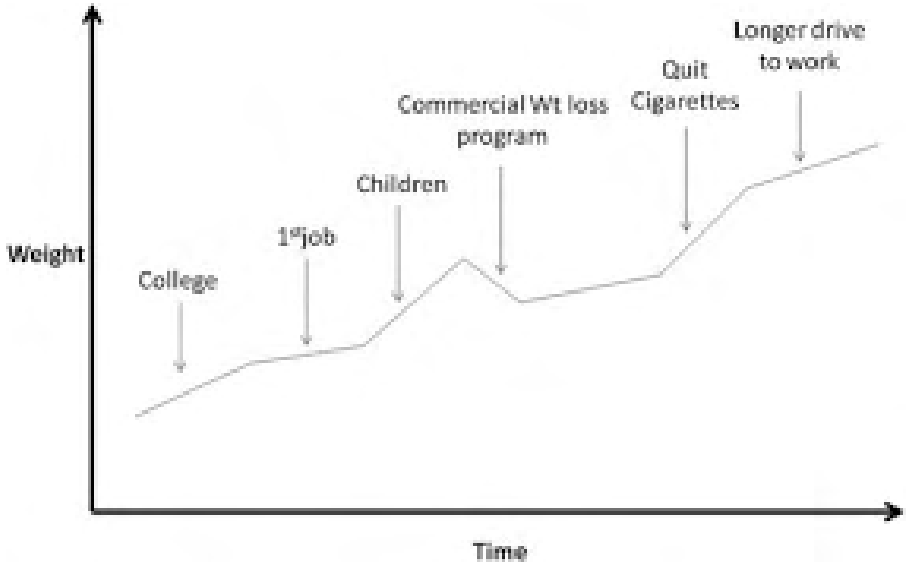
Coping strategies and stress



Members of a slimming club, n=538

Weight Loss	%	Weight Gain	%
Relationship problem	18.9	Pregnancy	23.5
Pregnancy	17.1	Feeling comfortable in relationship	14.3
Special event	13.3	Death of someone close	11
Own illness	10.2	Relationship problem	11
Death of someone close	5.3	Stress	6.4
Reaching certain age	3.6	Disability after injury	4.4
Seeing picture of self	3.1	Illness	3.7
Traveling	3.1	Giving up smoking	3.5
Falling in love	2.7	Depression	3.3
University	1.9	University	3.1

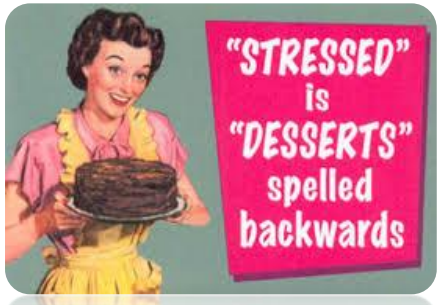
Ogden J et al. *Psychol Health Med*. 2009;14:239-249.

Stress, Mood and Weight Management

- The effects of stress and mood on dietary restraint and weight management success are widely acknowledged
- Repeated exposures to stressful life situations are associated with a greater preference for energy dense and nutrient dense foods rich in sugar and fat

- Coping strategies and stress: Coping is the mediator, as if your coping strategy has always been eating then when you are under stress then you are more likely to go to your learnt coping strategy

Stress management and appropriate / inappropriate coping mechanisms are critical factors



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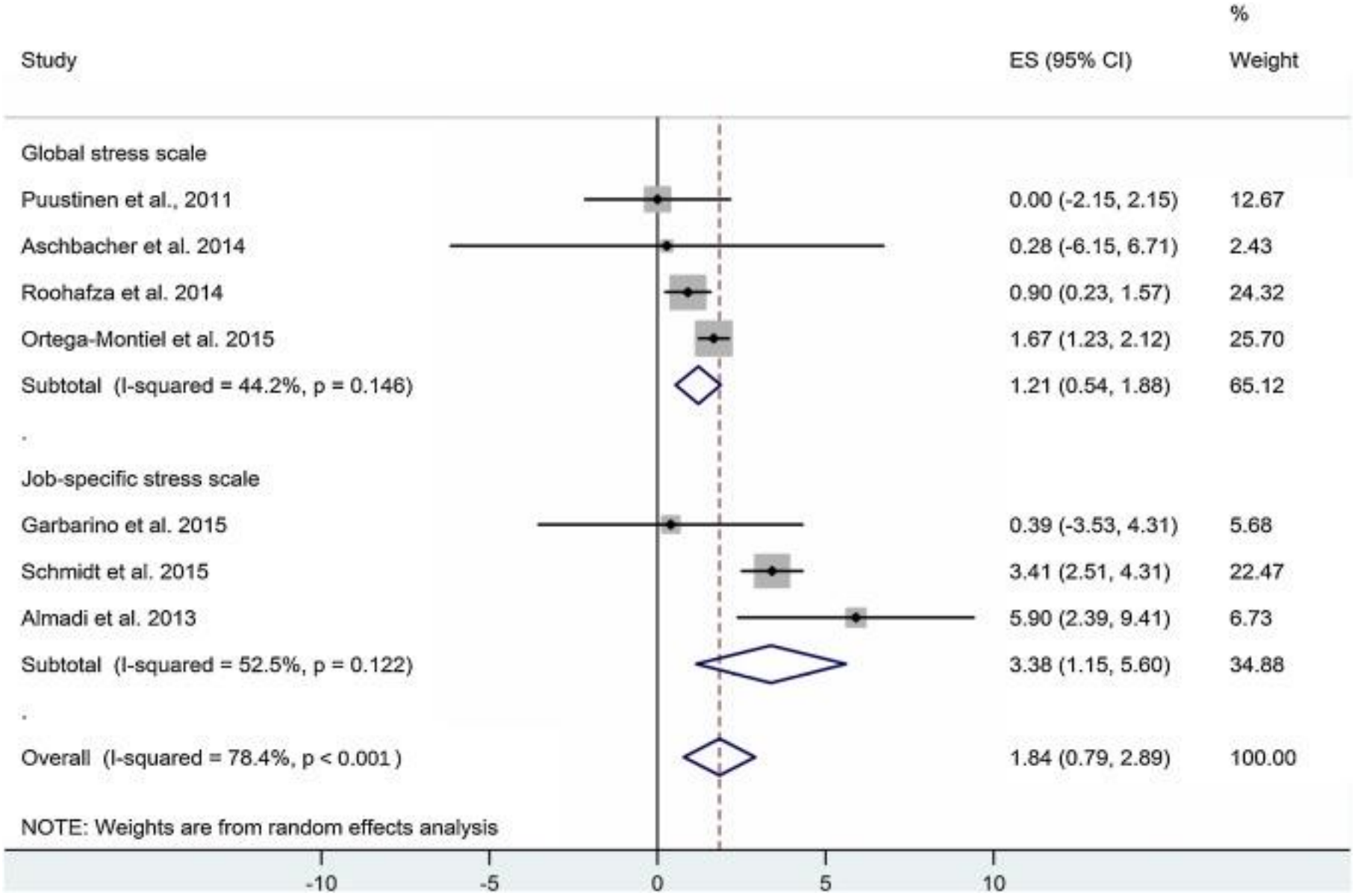
1. McElroy et al. *J Clin Psychiatry* 2004;65:634-51 2. Greeno et al. *Psychol Bull* 1994;115:444-64
2. 3. Marniemi et al. *J Intern Med* 2002;251:35-43 4. Torres et al. *Nutrition* 2007;23:887-94

Perceived (global and job specific) stress correlates with visceral obesity (WC) Tenk et al 2018

Fig. 3. Forest plot representing the differences in waist circumference values of low- and high-stress groups with regard to global and job-specific stress.

Squares show the difference in mean values with the grey area reflecting the weight assigned to the study. Horizontal bars indicate 95% confidence intervals (95% CI). The diamond shows the overall effect size (ES) with its corresponding 95% CI.



Psychoneuroendocrinology 95 (2018) 63–73

Occurrence of temptations and lapses – role of coping

Temptation – *a sudden urge to break diet at which you were close to the brink*

- **Appetite** – *greater* hunger, *less* fullness
- **Situation** – anywhere, any time
- **Mood** – *greater*: sadness, deprivation, stress, and boredom; *Less*: Relaxed, content, feeling of being in control

Coping responses:

- distinguish *temptations* from *lapses* in individuals undergoing behavioural weight loss programme.
- Appropriate *strategies* in place to resist *temptations* predicted *self-efficacy*

Coping response

Lapse – *an incident where you broke your diet*

- **Appetite** – *greater* hunger, *less* satisfied
- **Situation** – home, evening, and weekends
- **Mood** – *greater*: sadness, deprivation, stress, and nervousness; *Less*: Relaxed, and feelings of being in control
- **Abstinence Violations** – *greater* worry; *Less* prepared to resist, less confidence in success, and will power

- Followed by diminished *confidence* and *self-efficacy*, feelings of *failure* and *guilt*
- Associated with diminished *coping*
- Frequency of *lapses* negatively associated with *initial*, and *overall* weight loss

Need to i) reduce exposure to temptations, ii) prevent them triggering lapses and iii) manage the consequences of lapses

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Carel et al, 2001 Eating Behav. 2:307-321; Carel et al, 2004 j. Con. Clin. Psych, McKee et al 2014 Ann. Behav. Med. 48:300-310; Forman et al 2017 Ann Behav Med 51:741 753

Summary

- Appetite consists of interactive processes of hunger, wanting, liking, satiation and satiety which all impact on inhibitory/behavioural control (the individuals ability to resist).

- People living with obesity have a biological vulnerability for weight gain – weakened satiety signalling, intensified reward signalling.

- Dieting and energy restriction intensifies biological vulnerabilities and further compromises appetite control. It only takes a momentary lapse in otherwise constant exertion to undermine a diet

- The Broader psychology – stress and coping style mediate the link between depression and BMI through temptations and lapses. We need help to develop better coping mechanisms - For example –being aware of stress triggers, and engaging in self care

- Interventions targeting satiety, reward, inhibitory control, and personal psychological triggers (e.g. stress, mood etc) could provide personalised approaches to weight-management allowing patients to gain perceived control and self-efficacy important to long-term success.

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Thank you for listening

- Obesity is a chronic disease – not a choice
- We need to reduce stigma – use less stigmatising language

- Questions?

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Disclosures

- Grant support from Unilever (Carl Roberts)
- Consultancy with Boehringer Ingelheim (Carl Roberts & Jason Halford)

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