SSVQ Annual Congress 2021

Obesity: Motivational Approach

October 28th, 2021

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University of Toronto

DISCLOSURE OF RELATIONSHIPS WITH COMMERCIAL INTERESTS

CONSULTING FEES	ADVISORY BOARD FEES	SPEAKER HONORARIA
Novo Nordisk	Novo Nordisk	Novo Nordisk
Bausch Health	Bausch Health	Bausch Health

Cognitive restructuring – The mechanism of CBT

Definition :

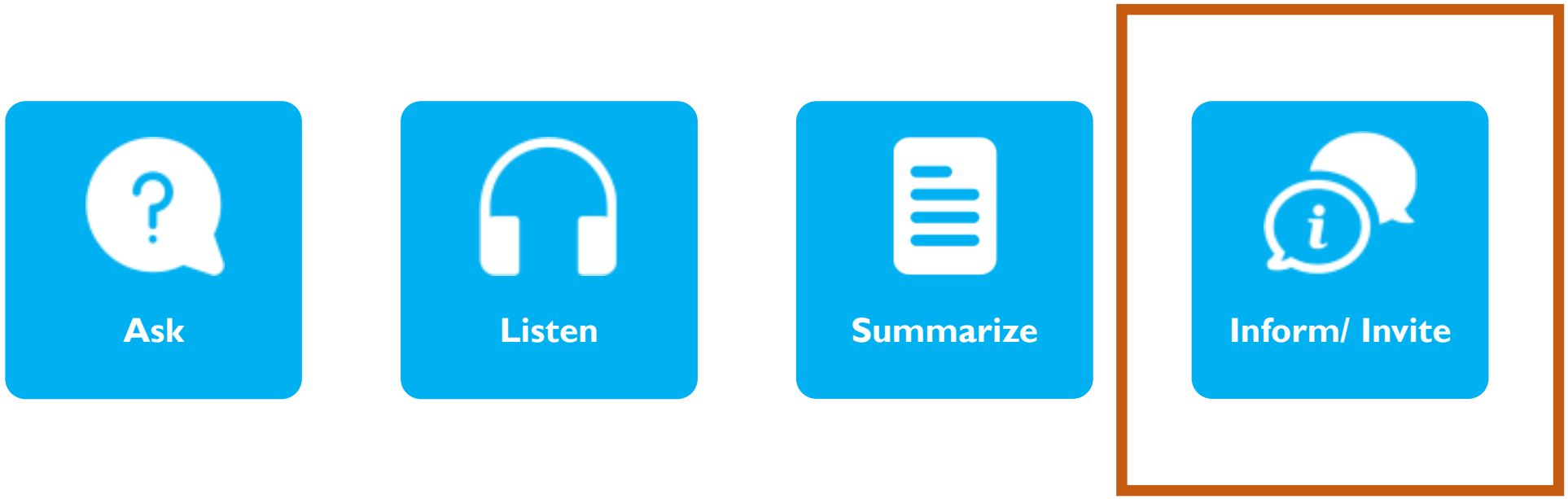
Cognitive restructuring is the mechanism of CBT and a **technique** that has been successfully used to help people change the way they think.

Obesity: Motivational Approach

Objectives:

1. Understand the neurobiological principles associated with weight gain.
2. Understand the subconscious aspects of appetite control and the external agents that can modulate it.
3. Determine the conscious aspects of our behavior that modulate overconsumption of food.

MODULE CONVERSATIONS (MOTIVATIONAL INTERVIEWING)



New Canadian Guidelines

- Obesity is a complex chronic disease in which abnormal or excess body fat (adiposity) impairs health, increases the risk of long-term medical complications and reduces lifespan.
- People living with obesity face substantial **bias and stigma**, which contribute to increased morbidity and mortality independent of weight or body mass index.
- Obesity care should be based on evidence-based principles, move beyond simplistic approaches of "eat less, move more," and address the **root drivers** of obesity.

GUIDELINE CPD

Obesity in adults: a clinical practice guideline

Sean Wharton MD, David C.W. Lau MD PhD, Michael Vallis PhD RPsych, Arya M. Sharma MD PhD, Laurent Biertho MD, Denise Campbell-Scherer MD PhD, Kristi Adamo PhD, Angela Alberga PhD, Rhonda Bell PhD, Normand Boulé PhD, Elaine Boyling PhD, Jennifer Brown RD MSc, Betty Calam MD, Carol Clarke RD MHSc, Lindsay Crowshoe MD, Dennis Divalentino MD, Mary Forhan OT PhD, Yoni Freedhoff MD, Michel Gagner MD, Stephen Glazer MD, Cindy Grand MPH, Michael Green MD MPH, Margaret Hahn MD PhD, Raed Hawa MD MSc, Rita Henderson PhD, Dennis Hong MD, Pam Hung MScOT BSc, Ian Janssen PhD, Kristen Jacklin PhD, Carlene Johnson-Stoklossa RD MSc, Amy Kemp BKin BA, Sara Kirk PhD, Jennifer Kuk PhD, Marie-France Langlois MD, Scott Lear PhD, Ashley McInnes PhD, David Macklin MD, Leen Naji MD, Priya Manjoo MD, Marie-Philippe Morin MD, Kara Nerenberg MD MSc, Ian Patton PhD, Sue Pedersen MD, Leticia Pereira PhD, Helena Piccinini-Vallis MD PhD, Megha Poddar MD, Paul Poirier MD, Denis Prud'homme MD MSc, Ximena Ramos Salas PhD, Christian Rueda-Clausen MD PhD, Shelly Russell-Mayhew PhD RPsych, Judy Shiau MD, Diana Sherifali RN PhD, John Sievenpiper MD PhD, Sanjeev Sockalingam MD MHPE, Valerie Taylor MD PhD, Ellen Toth MD, Laurie Twells PhD, Richard Tytus MD, Shahebina Walji MD, Leah Walker BA RCT, Sonja Wicklum MD

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CMAJ Podcasts: author interview at https://www.cmaj.ca/lookup/doi/10.1503/cmaj.191707/tab-related-content

Obesity is a complex chronic disease in which abnormal or excess body fat (adiposity) impairs health, increases the risk of long-term medical complications and reduces lifespan.[1] Epidemiologic studies define obesity using the body mass index (BMI; weight/height^2), which can stratify obesity-related health risks at the population level. Obesity is operationally defined as a BMI exceeding 30 kg/m^2 and is subclassified into class 1 (30–34.9), class 2 (35–39.9) and class 3 (≥ 40). At the population level, health complications from excess body fat increase as BMI increases.[2] At the individual level, complications occur because of excess adiposity, location and distribution of adiposity and many other factors, including environmental, genetic, biologic and socioeconomic factors (Box 1).[2]

Over the past 3 decades, the prevalence of obesity has steadily increased throughout the world,[3] and in Canada, it has increased threefold since 1985.[3] Importantly, severe obesity has increased more than fourfold and, in 2016, affected an estimated 1.9 million Canadian adults.[3]

Obesity has become a major public health issue that increases health care costs[14,15] and negatively affects physical and psychological health.[16] People with obesity experience pervasive weight bias and stigma, which contributes (independent of weight or BMI) to increased morbidity and mortality.[17]

KEY POINTS

- Obesity is a prevalent, complex, progressive and relapsing chronic disease, characterized by abnormal or excessive body fat (adiposity), that impairs health.
- People living with obesity face substantial bias and stigma, which contribute to increased morbidity and mortality independent of weight or body mass index.
- This guideline update reflects substantial advances in the epidemiology, determinants, pathophysiology, assessment, prevention and treatment of obesity, and shifts the focus of obesity management toward improving patient-centred health outcomes, rather than weight loss alone.
- Obesity care should be based on evidence-based principles of chronic disease management, must validate patients' lived experiences, move beyond simplistic approaches of "eat less, move more," and address the root drivers of obesity.
- People living with obesity should have access to evidence-informed interventions, including medical nutrition therapy, physical activity, psychological interventions, pharmacotherapy and surgery.

Obesity is caused by the complex interplay of multiple genetic, metabolic, behavioural and environmental factors, with the latter thought to be the proximate cause of the substantial

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Sean Wharton, et al. Obesity in adults: a clinical practice guideline CMAJ Aug 2020, 192 (31)

When behaviour meets biology

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DOI: 10.1111/cob.12443

COMMENTARY

Clinical OBESITY WILEY

When behaviour meets biology: if obesity is a chronic medical disease what is obesity management?

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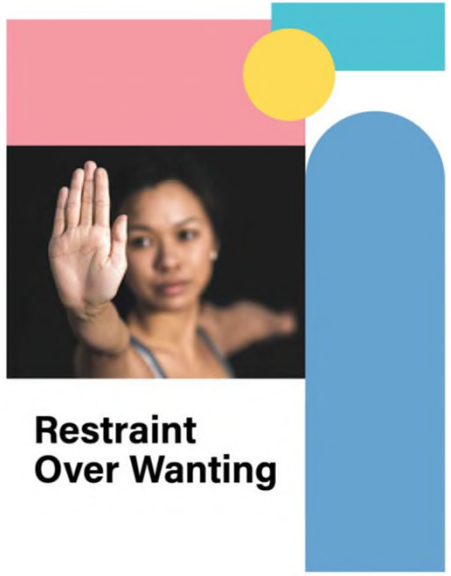
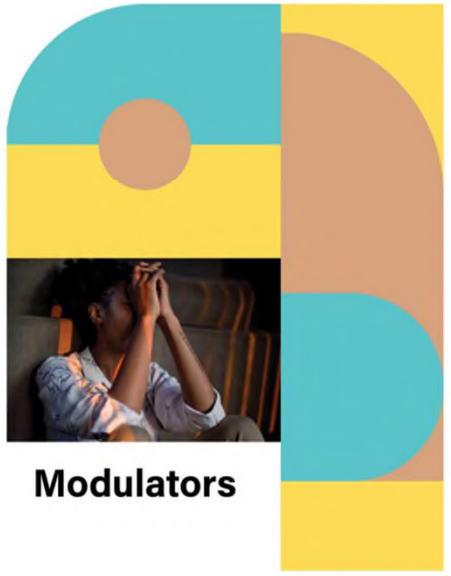
1 | INTRODUCTION

Excess weight gain has become a pandemic in our contemporary societies[1] and weight loss has long been an obsession. Until recently, there was very little attention paid to obesity as a disease in medicine and those seeking weight loss were inundated with a variety of unscientific messages. These messages varied from benevolent (commercial continue with an intervention. A reframing of what obesity management means to a person living with obesity is required. In this paper, such a reframe is presented, the goal of which is to engage a person in a weight management plan that is realistic, evidence based as well as motivating and reinforcing.

A core driver of behaviour is reinforcement.[11] If a behaviour results in positive outcomes (adding something positive or removing

Knowledge Translation MODULES





INTERNALIZED WEIGHT BIAS

INTERNALIZED WEIGHT BIAS *OBJECTIVES*

Learn to invite/inform your patient that obesity is

1. a real disease,
2. not your fault
3. something for which treatment exists

INTERNALIZED WEIGHT BIAS *DEFINITION*

Weight Bias refers to negative attitudes and stereotypes about obesity and people living with obesity - judging a person's values, skills, abilities, or personality based on their body weight or shape.

You know what their problem is?

They are unmotivated

They have no willpower

They are lazy

INTERNALIZED WEIGHT BIAS *THE PROBLEM*

Survey of 13,996 adults currently engaged in weight management Modified Weight Bias Internalization Scale (WBIS-M)

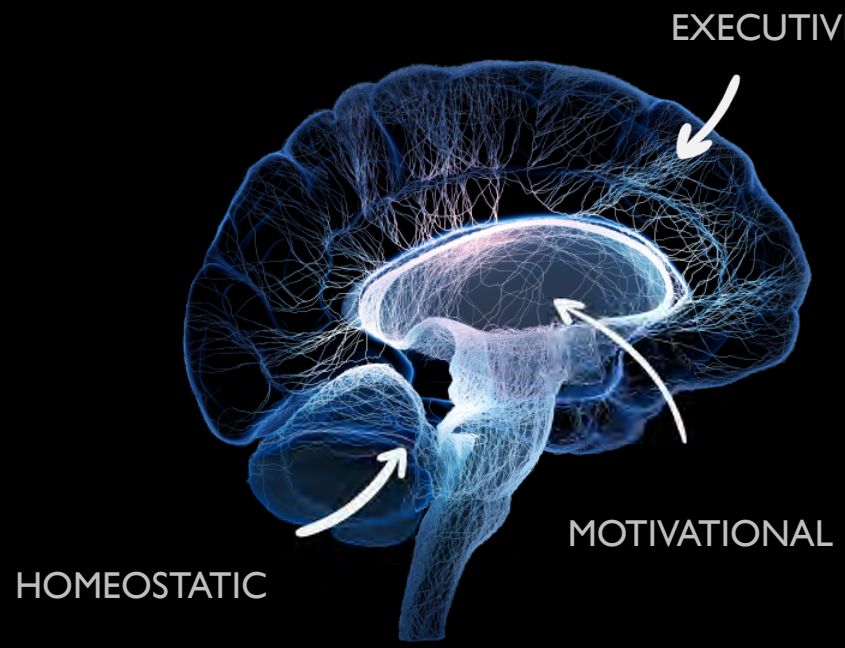
WBIS-M scores were associated with:

I am am lazy, unmotivated and have no willpower

- greater weight gain in the past year
- poorer mental and physical HRQOL,
- less eating and physical activity self-efficacy
- Greater eating as coping strategy
- more avoidance of going to the gym
- poorer body image
- greater perceived stress

Rebecca L. Pearl - relevence and correlates of weight bias internalization in weight management: A multinational study: Population Health 13 (2021)

INTERNALIZED WEIGHT BIAS *REAL DISEASE*



- 70% heritable - *genes expressed in the brain
- Personal appetite system
- Three layers
- Regulates weight, appetite and metabolism
- Evolved for calorie scarce environment
- Progressively defends against fat loss

*Yeo GSH, Heisler LK. Unraveling the brain regulation of appetite: lessons from genetics. Nat Neurosci. 2012 Oct;15(10):1343–9.

THE COLLISION

- This ultra-processed
- Sugar-fat-salt-filled
- Ultra portioned,
- Ultra available,
- Advertised to look healthy,
- Brought to-your-door food environment

THE HUMAN APPETITE SYSTEM

INTERNALIZED WEIGHT BIAS *HERITABILITY*

- A systematic review that identified 88 independent estimates of BMI heritability from twin studies (total 140,525 twins)

- BMI heritability estimates from twin studies =0.75 (75%)

frontiers in ENDOCRINOLOGY

ORIGINAL RESEARCH ARTICLE

Variability in the heritability of body mass index: a systematic review and meta-regression

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Evidence for a major role of genetic factors in the determination of body mass index (BMI) comes from studies of related individuals. Despite consistent evidence for a heritable component of BMI, estimates of BMI heritability vary widely between studies and the reasons for this remain unclear. While some variation is natural due to differences between populations and settings, study design factors may also explain some of the heterogeneity. We performed a systematic review that identified 88 independent estimates of BMI heritability from twin studies (total 140,525 twins) and 27 estimates from family studies (42,988 family members). BMI heritability estimates from twin studies ranged from 0.47 to 0.90 (5th/50th/95th centiles: 0.58/0.75/0.87) and were generally higher than those from family studies (range: 0.24–0.81; 5th/50th/95th centiles: 0.25/0.46/0.68). Meta-regression of the results from twin studies showed that BMI heritability estimates were 0.07 ($P = 0.001$) higher in children than in adults; estimates increased with mean age among childhood studies (+0.012/year, $P = 0.002$), but decreased with mean age in adult studies (−0.002/year, $P = 0.002$). Heritability estimates derived from AE twin models (which assume no contribution of shared environment) were 0.12 higher than those from ACE models ($P < 0.001$), whilst lower estimates were associated with self reported versus DNA-based determination of zygosity (−0.04, $P = 0.02$), and with self reported versus measured BMI (−0.05, $P = 0.03$). Although the observed differences in heritability according to aspects of study design are relatively small, together, the above factors explained 47% of the heterogeneity in estimates of BMI heritability from twin studies. In summary, while some variation in BMI heritability is expected due to population-level differences, study design factors explained nearly half the heterogeneity reported in twin studies. The genetic contribution to BMI appears to vary with age and may have a greater influence during childhood than adult life.

Keywords: body mass index, twin study, family study, heritability

INTRODUCTION

Studies of twins and families have quantified the contribution of genetic variation to inter-individual differences in body mass index (BMI). In the last comprehensive review of BMI heritability, Maes et al. (1997) reported that the proportion of phenotypic variance (V_P) that can be attributed to genetic factors (h^2) ranged from 0.40 to 0.90 in twin studies and 0.20 to 0.50 in family studies, demonstrating the wide variation in the magnitude of BMI heritability observed both within and between these study designs (Maes et al., 1997). Genome-wide association studies (GWAS) have so far identified 32 loci robustly associated with adult BMI (Frayling et al., 2007; Loos et al., 2008; Thorleifsson et al., 2009; Willer et al., 2009; Speliotes et al., 2010). Despite highly statistically significant associations, these 32 loci account for less than 2% of the total V_P in BMI. Sub-genome-wide significant variants may be able to explain a substantial portion of the unexplained genetic variance of complex traits. However, even when considering such variants, the variance explained remains lower than estimates of heritability (Yang et al., 2011) and much attention has been focused on finding the so-called "missing heritability" (Manolio et al., 2009).

Twin studies are used to quantify genetic and environmental contributions to variation in BMI by comparing intra-pair concordance between monozygotic (MZ) twins and dizygotic (DZ) twins. Assignment of zygosity (MZ or DZ) to twin pairs is achieved either using questionnaires or more accurate DNA-based methods. Twin studies model the V_P to be the composite of up to four components: (A) additive genetic factors; (D) non-additive or dominant genetic factors; (C) shared environmental factors; and (E) non-shared environmental factors (Neale and Cardon, 1992; Rijsdijk and Sham, 2002). Heritability is usually reported as the proportion of overall V_P that can be attributed to additive genetic factors ($h^2 = A/V_P$), as dominant genetic factors (D) are confounded with shared environmental factors (C) and cannot be estimated in the same model. The "best estimate" of heritability is calculated from the statistically best fitting and most parsimonious combination of the three remaining variance components (A, C, and E), determined by sequentially removing components from

Abbreviations: BMI, body mass index; DZ, dizygotic; MZ, monozygotic.

Elks. Variability in the heritability of body mass index: a systematic review and meta-regression Frontiers in endocrinology, 2012 -

INTERNALIZED WEIGHT BIAS *CNS*

- The central nervous system (CNS) and neuronal pathways that control the hedonic aspects of food intake have emerged as the major drivers of body weight for both monogenic and polygenic obesity.

- Genes that are either enriched or exclusively expressed within the brain and CNS have a central role in obesity.

REVIEWS

The genetics of obesity: from discovery to biology

Ruth J. F. Loos and Giles S. H. Yeo

Abstract | The prevalence of obesity has tripled over the past four decades, imposing an enormous burden on people's health. Polygenic (or common) obesity and rare, severe, early-onset monogenic obesity are often polarized as distinct diseases. However, gene discovery studies for both forms of obesity show that they have shared genetic and biological underpinnings, pointing to a key role for the brain in the control of body weight. Genome-wide association studies (GWAS) with increasing sample sizes and advances in sequencing technology are the main drivers behind a recent flurry of new discoveries. However, it is the post-GWAS, cross-disciplinary collaborations, which combine new omics technologies and analytical approaches, that have started to facilitate translation of genetic loci into meaningful biology and new avenues for treatment.

Obesogenic environment: An environment that promotes weight gain

Obesity is associated with premature mortality and is a serious public health threat that accounts for a large proportion of the worldwide non-communicable disease burden, including type 2 diabetes, cardiovascular disease, hypertension and certain cancers[1,2]. Mechanical issues resulting from substantially increased weight, such as osteoarthritis and sleep apnoea, also affect people's quality of life[3]. The impact of obesity on communicable disease, in particular viral infection[4], has recently been highlighted by the discovery that individuals with obesity are at increased risk of hospitalization and severe illness from COVID-19 [REF.5–7].

On the basis of the latest data from the NCD Risk Factor Collaboration, in 2016 almost 2 billion adults (39% of the world's adult population) were estimated to be overweight (defined by a body mass index (BMI) of ≥25 kg m^{-2}), 671 million (12% of the world's adult population) of whom had obesity (BMI ≥30 kg m^{-2}) — a tripling in the prevalence of obesity since 1975 [REF.8, FIG. 1]. Although the rate of increase in obesity seems to be declining in most high-income countries, it continues to rise in many low-income and middle-income countries and prevalence remains high globally[8]. If current trends continue, it is expected that 1 billion adults (nearly 20% of the world population) will have obesity by 2025. Particularly alarming is the global rise in obesity among children and adolescents; more than 7% had obesity in 2016 compared with less than 1% in 1975 [REF.9].

Although changes in the environment have undoubtedly driven the rapid increase in prevalence, obesity results from an interaction between environmental and innate biological factors. Crucially, there is a strong genetic component underlying the large interindividual variation in body weight that determines people's response to this 'obesogenic' environment. Twin, family and adoption studies have estimated the heritability of obesity to be between 40% and 70%[2,10]. As a consequence, genetic approaches can be leveraged to characterize the underlying physiological and molecular mechanisms that control body weight.

Classically, we have considered obesity in two broad categories [FIG. 2]: so-called monogenic obesity, which is inherited in a Mendelian pattern, is typically rare, early-onset and severe and involves either small or large chromosomal deletions or single-gene defects; and polygenic obesity (also known as common obesity), which is the result of hundreds of polymorphisms that each have a small effect. Polygenic obesity follows a pattern of heritability that is similar to other complex traits and diseases. Although often considered to be two distinct forms, gene discovery studies of monogenic and polygenic obesity have converged on what seems to be broadly similar underlying biology. Specifically, the central nervous system (CNS) and neuronal pathways that control the hedonic aspects of food intake have emerged as the major drivers of body weight for both monogenic and polygenic obesity. Furthermore, early evidence shows that the expression of mutations causing monogenic obesity may — at least in part — be influenced by the individual's polygenic susceptibility to obesity[11].

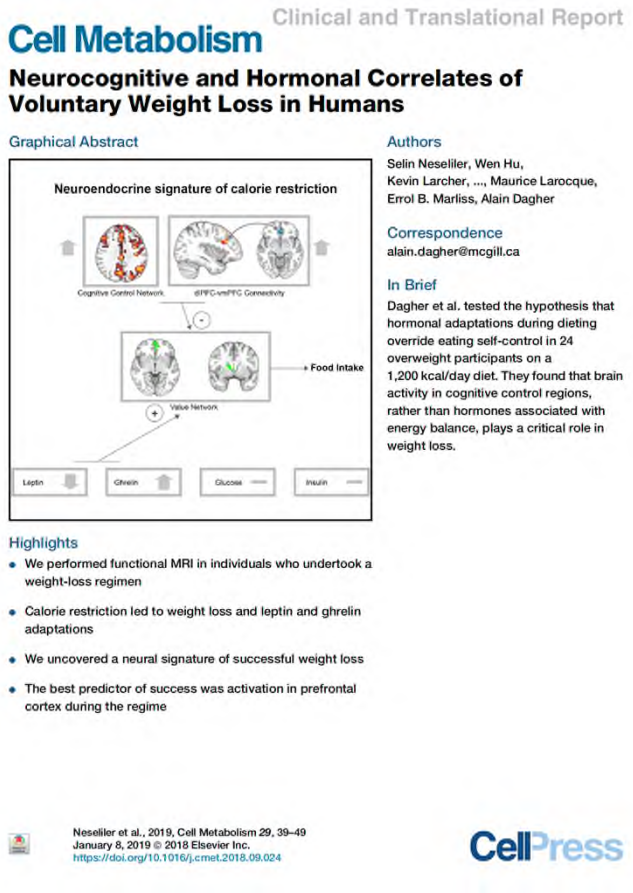
In this Review, we summarize more than 20 years of genetic studies that have characterized the molecules and mechanisms that control body weight, specifically focusing on overall obesity and adiposity, rather than fat distribution or central adiposity. Although most of the current insights into the underlying biology have been derived from monogenic forms of obesity, recent years have witnessed several successful variant-to-function translations for polygenic forms of obesity. We also explore how the ubiquity of whole-exome sequencing

NATURE REVIEWS | GENETICS

Loos, R.J.F., Yeo, G.S.H. The genetics of obesity: from discovery to biology. Nat Rev Genet (2021)

INTERNALIZED WEIGHT BIAS *CNS*

- Functional MRI in individuals who undertook a weight-loss regimen
- Calorie restriction led to weight loss and leptin and ghrelin adaptations
- “Neural Signature” the best predictor of success was activation in prefrontal cortex during the regime
- Changes were associated with food cue reactivity in reward-related brain regions



Cell Metabolism Clinical and Translational Report

Neurocognitive and Hormonal Correlates of Voluntary Weight Loss in Humans

Graphical Abstract

Neuroendocrine signature of calorie restriction

Cognitive Control Network

dlPFC-vmPFC Connectivity

+ Food Intake

Value Network

Leptin

Ghrelin

Glucose

Insulin

Authors

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In Brief

Dagher et al. tested the hypothesis that hormonal adaptations during dieting override eating self-control in 24 overweight participants on a 1,200 kcal/day diet. They found that brain activity in cognitive control regions, rather than hormones associated with energy balance, plays a critical role in weight loss.

Highlights

- We performed functional MRI in individuals who undertook a weight-loss regimen
- Calorie restriction led to weight loss and leptin and ghrelin adaptations
- We uncovered a neural signature of successful weight loss
- The best predictor of success was activation in prefrontal cortex during the regime

Neseliler et al., 2019, Cell Metabolism 29, 39–49
January 8, 2019 © 2018 Elsevier Inc.
https://doi.org/10.1016/j.cmet.2018.09.024

CellPress

Dagher et al., Neurocognitive and Hormonal Correlates of Voluntary Weight Loss in Humans Cell Metabolism 29, 39–49 January 8, 2019

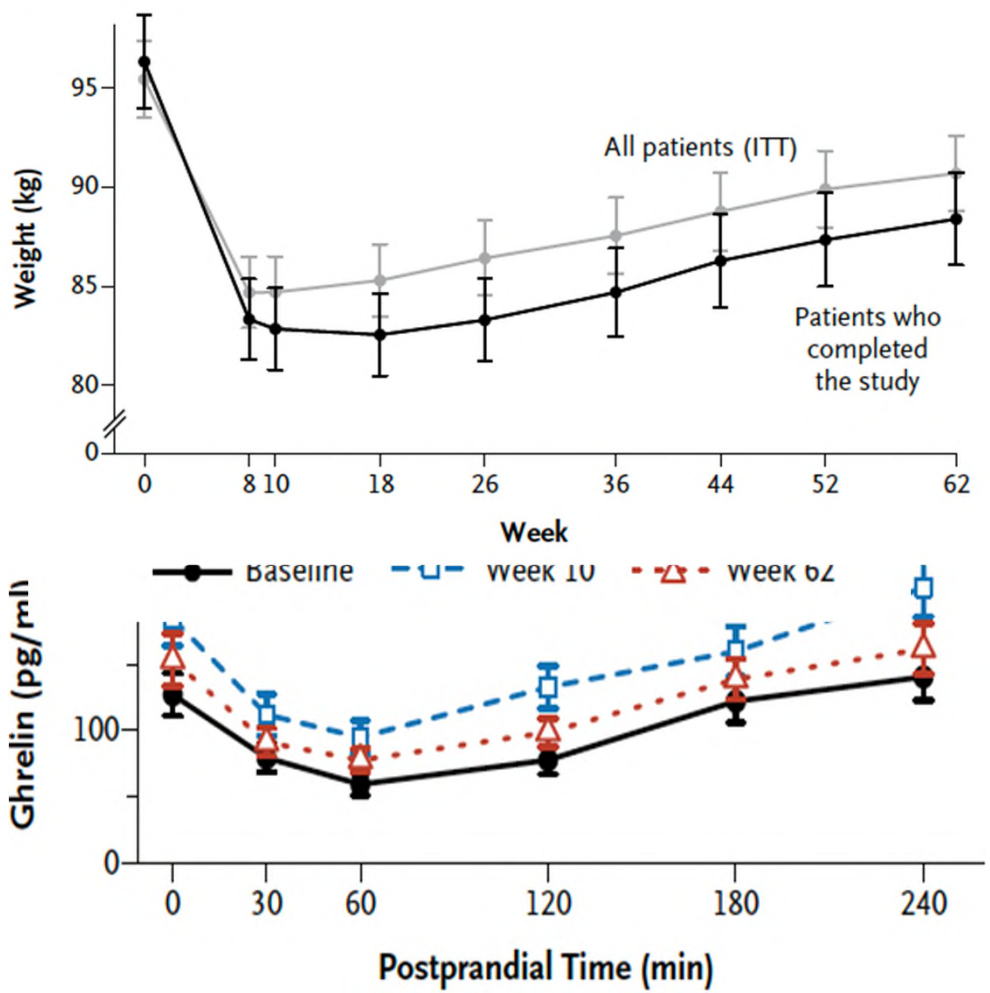
INTERNALIZED WEIGHT BIAS *PROGRESSIVE*

- Set point / settling point theory
- Reward sensitization
- Dissolution of the connectome

INTERNALIZED WEIGHT BIAS *PROGRESSIVE*

Significant hormonal changes that favour weight regain and were still evident a year later

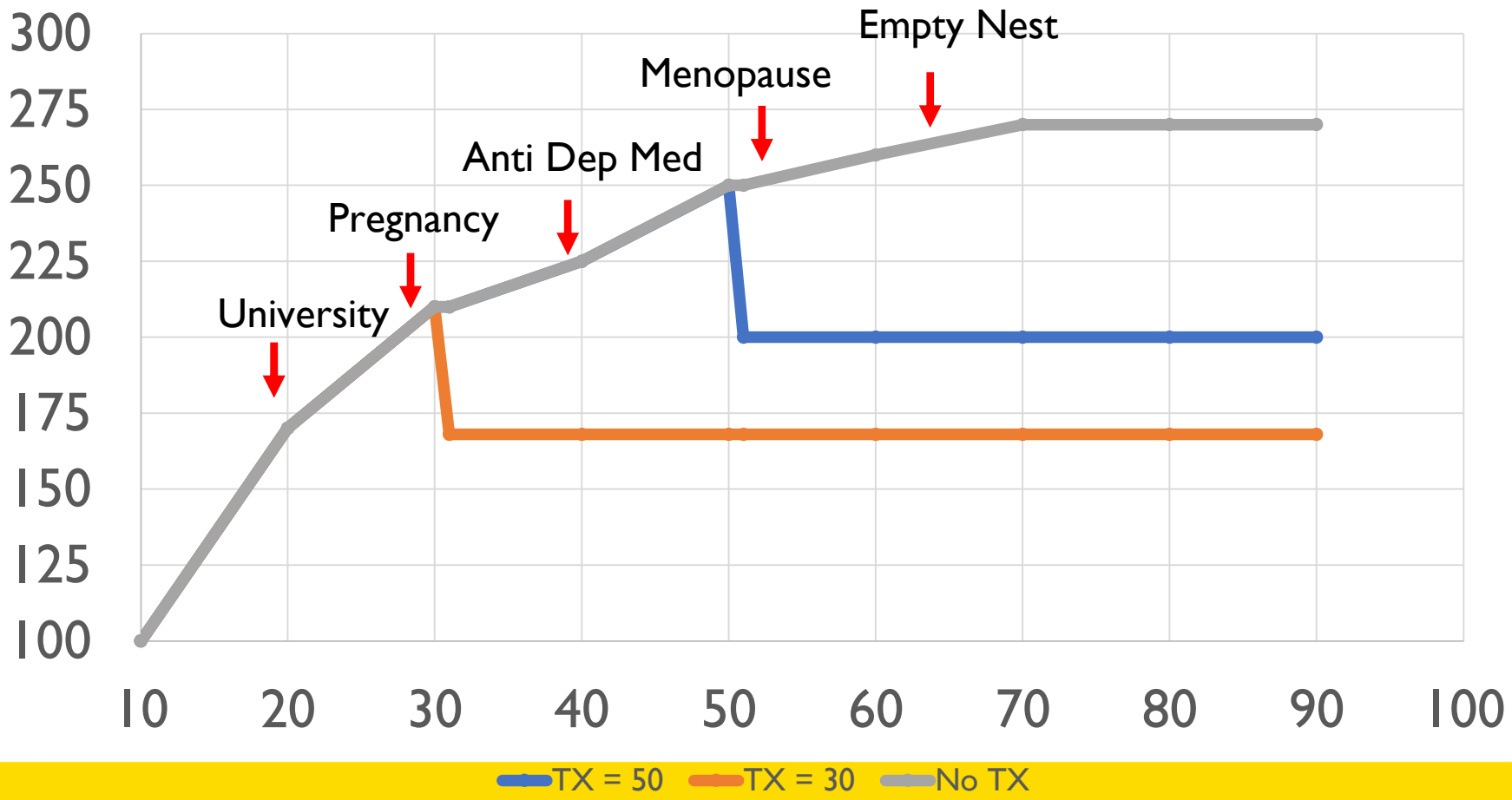
- ↓ Insulin ↓ Leptin
- ↓ PYY ↓ CCK
- ↓ GLP-1 ↑ Ghrelin



Adapted from Sumithran P et al. N Engl J Med. 2011;365:1597-604.

INTERNALIZED WEIGHT BIAS *PROGRESSIVE*

Theoretical 5 foot 6 inches tall female



INTERNALIZED WEIGHT BIAS *TREATMENT*

Behavioural Therapy

HOMEOSTATIC

MOTIVATIONAL

Medication

Surgery

Elks. Frontiers in endocrinology, 2012 - Yengo, L.. Molec Gen., 2018. Yeo GSH. Nat Rev Neurosci, 2012.

INTERNALIZED WEIGHT BIAS *INVITATION*

WOULD YOU CONSIDER…

Real disease

That struggling with weight is a real condition that is mostly genetic, is centered in the brain, is strongly influenced by the environment and is progressive.

Not your fault

That your past weight loss efforts were difficult not because of some flaw in your character, a lack of willpower or motivation, the wrong diet or not enough activity, but instead, because you were struggling with a real condition untreated.

Treatment exists

Three treatments exist for this condition. At the foundation is a **behavioural treatment**. Often, safe and effective medication may be added as another treatment, and safe and effective surgery may be added as well.

INTERNALIZED WEIGHT BIAS *EVIDENCE*

Self-bias is common and may affect outcomes. Assessing for internalized weight bias is recommended to aid with reducing bias and encouraging achievable expectations

LEVEL 2 – GRADE B

Coping strategies consistent with the principles of cognitive behaviour therapy and acceptance and commitment therapy can help mitigate against internalized weight bias

LEVEL 1B – GRADE A

Pearl RL, Puhl RM. The distinct effects of internalizing weight bias: An experi- mental study. Body Image. 2016;17:38-42. doi:10.1016/j.bodyim.2016.02.002. Mensinger JL, Calogero RM, Tylka TL. Internalized weight stigma moderates eating behavior outcomes in women with high BMI participating in a healthy living program. Appetite. 2016;102:32-43. doi:10.1016/j.appet.2016.01.033. Murakami JM, Latner JD. Weight acceptance versus body dissatisfaction: Effects on stigma, perceived self-esteem, and perceived psychopathology. Eat Behav. 2015;19:163-167. doi:10.1016/j.eatbeh.2015.09.010. Lee M, Ata RN, Brannick MT. Malleability of weight-biased attitudes and beliefs: A meta-analysis of weight bias reduction interventions. Body Image. 2014;11(3):251-259. doi:10.1016/j.bodyim.2014.03.003. Schvey NA, Puhl RM, Brownell KD. The impact of weight stigma on caloric consumption. Obesity. 2011;19(10):1957-1962. doi:10.1038/oby.2011.204.

INTERNALIZED WEIGHT BIAS *EVIDENCE*

Healthcare providers should assess their own attitudes and beliefs regarding obesity and consider how their attitudes and beliefs may influence care delivery

LEVEL 1A - GRADE A

Healthcare providers should recognize that internalized weight bias (bias towards oneself) in people living with obesity can affect behavioural and health outcomes

LEVEL 2A - GRADE B

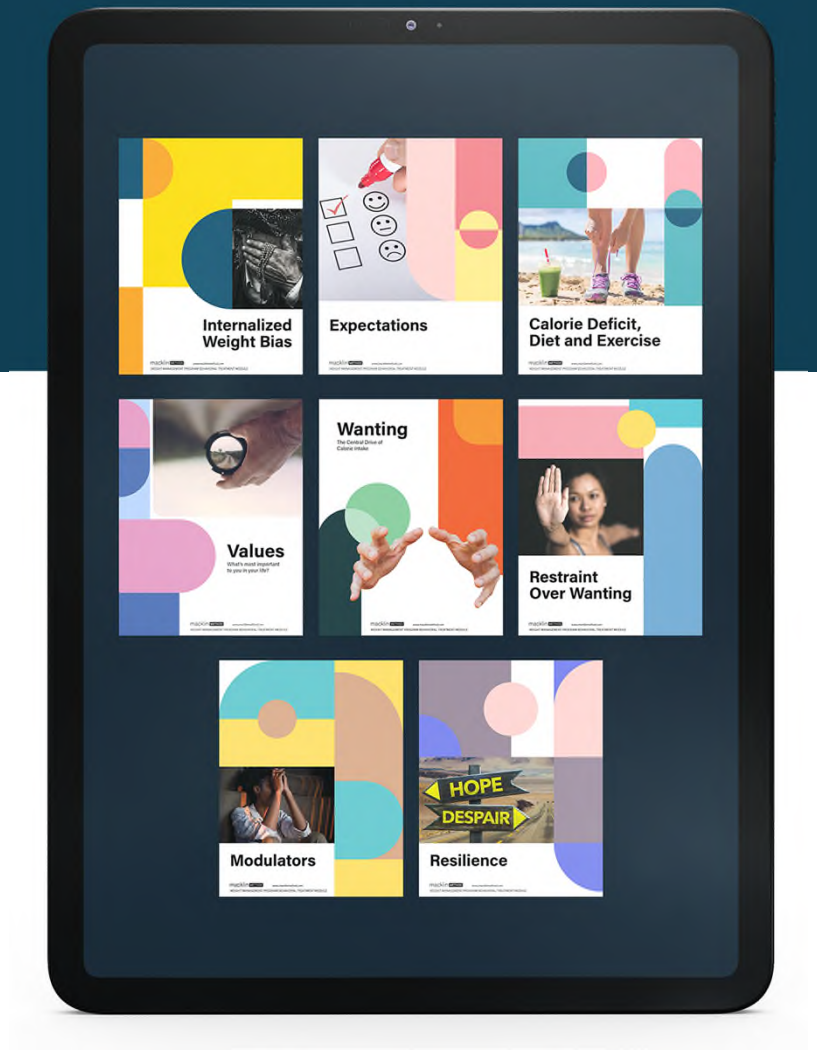
RLee M, Ata RN, Brannick Malleability of weight-biased attitudes and beliefs: A meta-analysis of weight bias reduction interventions. Body Image. 2014;11(3):251-259. doi:10.1016/j.bodyim.2014.03.003. Pearl RL, Puhl RM. The distinct effects of internalizing weight bias: An experimental Body Image. 2016;17:38-42. doi:10.1016/j.bodyim.2016.02.002. Murakami JM, Latner Weight acceptance versus body dissatisfaction: Effects on stigma, perceived self-esteem, and perceived psychopathology. Eat Behav. 2015;19:163-167. doi:10.1016/j.eatbeh.2015.09.010. Mensinger JL, Calogero RM, Tylka Internalized weight stigma moderates eating behavior outcomes in women with high BMI participating in a healthy living program. Appetite. 2016;102:32-43. doi:10.1016/j.appet.2016.01.033. Olson CL, Schumaker HD, Yawn Overweight women delay medical care. Arch Fam Med. 1994;3(10):888-892. doi:10.1001/archfami.3.10.888.

Summary

1. Obesity is a real medical condition that is mostly genetic, is centered in the brain, is strongly influenced by the environment and is progressive.

2. People living with obesity are subject to bias, stigma and discrimination leading to impaired quality of life and these serve as an obstacle to treatment for a disease that is not their fault.

3. Effective, ethical, expert treatment exists for this condition characterized by three pillars; Behavioural/CBT therapy, Medication and Surgery



macklin METHOD

WEIGHT MANAGEMENT PROGRAM
BEHAVIORAL TREATMENT MODULES

These modules represent the basics and principles of obesity treatment

www.macklinmethod.com

VICKI MOONEY AND HER DAUGHTER MIA
Vicki lives with obesity
Spain

Thanks . . . Questions?