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Article

Obesity, Stigma, and Responsibility: Should the Concept of Agency be Abandoned?

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Abstract: Appeals to individual agency and responsibility are increasingly viewed as antithetical to the goals of reducing stigma towards overweight and obesity and are sometimes even framed as anathema to civil discussion in academia. The current paper argues that this is a naïve view of agency and responsibility and, contrary to helping prevent or reduce stigma, removing these concepts from our conversations around obesity may instead worsen outcomes for those most at risk. This paper provides background for what follows and an introduction to the topic, before detailing and responding to the most common arguments for the futility of agency: from subconscious processes; from biological determinism; from free will; from obesity as a disease; and from framing and stigma. It then considers the impact on research of this proposed framing / perspective. The final section considers three key shifts in conceptualisation which I believe are necessary to highlight the importance of agency in weight management, whilst also providing the best care possible to patients and society at large. The proposed conceptual shifts are: agency is necessary but often not sufficient as it is constrained; diseases are not created equal; and there are multiple pathways to obesity. Acknowledging these fundamental realities can help us avoid the schism currently developing in researchers' and clinicians' conceptions of overweight and obesity.

Keywords: obesity; stigma; agency; responsibility; addiction; biological determinism

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Background

The editors of this journal have suggested I provide some background to the journey from conception to publication for what follows, and I agree that it is important to add context to just how quickly perceptions towards the views contained in this article shifted from *not at all contentious* to *too controversial to publish* among my co-authors. I reference my co-authors' positions and opinions throughout, summarising the thoughts they shared

via email or in-person conversation. A full account would be far too lengthy and thus not possible here, so I will simply outline as briefly as possible just a few of the key events which I believe to be indicative of the current climate in the field of Appetite and Obesity Research.

My initial goal was to write a short commentary-style riposte to a new (at the time) paper[1] which argued that we need to 'rethink agency' when it comes to overweight and obesity (see Section 1 'Introduction'). Upon learning about my proposal at a meeting, five colleagues across two universities joined the paper as co-authors and suggested it be a larger article written more generally (a good suggestion, which I happily accepted). The reception from the team to the initial draft was perhaps the most positive I have ever received – Professors 1, 2, and 3 opined that the paper was a joy to read, containing excellent arguments, and that few edits were required (personal communications: 3 December 2021 to 30 March 2022). Additionally, at first inspection the paper was explicitly considered uncontentious (Professor 3 and Lecturer; personal communications: 30 March 2022 to 31 March 2022).

Professor 3 bowed out due to lack of input; I was happy to still include them as co-author as they had engaged fully with an ongoing discussion, but I respected their decision (personal communication: 30 March 2022). Then, a request to soften some of the language by one co-author led to a relatively heated debate regarding the lack of sensitivity offered in the paper; two more absconded, citing that their work with patient groups could be compromised and that the potential backlash (possibly resulting in losing out on career opportunities) from the research community was too great (Professor 4 and Lecturer; personal communications: 2 April 2022).

A domino effect had now taken hold and new issues were being discovered by one of the remaining two co-authors which they had previously not considered problematic. Specifically, Professor 1 felt that the paper had changed too much in the wrong direction and pointed to specific sentences they thought too egregious and in need of removal; I pointed out how odd this new perception was given their previous praise of these very same sentences (evidenced by Track Changes in the Word document) just months prior, and that the only changes made since their last read were in fact made by Professor 1 themselves (personal communications: 3 April 2022 to 4 April 2022). It seemed to me that at least one of the remaining co-authors was now combing back through the paper in search of previously uncontested – even previously praiseworthy – text so it could now be censored. This shift occurred over five months. During a subsequent conversation both co-authors provided context for their reticence, explaining that their discomfort was born from the extent to which certain ideologies had crept into our research field, ideologies which were both impenetrable and hostile to criticism and which made them fearful of backlash (Professors 1 and 2, personal communication: 6 April 2022).

Eventually, we finalised the paper for submission to a call for papers. However, even though the call seemingly fit our article perfectly – literally containing the words 'reframing obesity' in the title – our paper was rejected by editors who cited lack of a clear fit. We remarked on how strange this response was given the clear suitability of our paper (Professor 1, personal communication: 6 June 2022); unfortunately, we received very similar responses from another four publications. At this point, Professor 1 decided to disengage from the project (personal communication: 14 October 2022). I

[1] Andrew Grannell, Finian Fallon, Werd Al-Najim and Carel le Roux, (2021). 'Obesity and Responsibility: Is It Time to Rethink Agency?' *Obesity Reviews* 22(8): e13270.

next suggested the *Journal of Controversial Ideas*; Professor 2 asked for time to consider whether they should publish under a pseudonym given the current political climate in academia (personal communication, 22 March 2023). Eventually, Professor 2 decided not to move forward as co-author without further overhauls to the paper and/or the recruitment of more co-authors, which I rejected based on the already drawn-out and exhausting nature of the process (21 April 2023). It had been more than two years since I first put pen to paper. I would like to end this personal recounting by stating that I do not believe anything written below to be scientifically, philosophically, or morally controversial, and neither did any of my five co-authors upon first reading. Of course, *controversy* is by its very nature subjective, but the key issue I wish to highlight in the above is that regardless of controversy, academics should both *feel* and *be* free to publish on contentious topics without fear of reprisal from their peers or institutions.

1. Introduction

In academia, concepts like 'personal agency', 'self-control', and 'willpower' (i.e., the capacity to influence one's own actions) are increasingly viewed as antiquated, unimportant, and even taboo when thinking about overweight and obesity.[2,3,4] Indeed, this notion rapidly escaped the confines of the academy to be widely spread by celebrities, activist groups, cultural icons, and even clinicians all expressing some version of this viewpoint.[5,6,7,8,9] I will refer to this henceforth as the position of *agentic futility* or *futility of will* – the viewpoint that agency/self-control/willpower simply does not matter all that much for weight management, and that even discussing or investigating such factors may cause unnecessary harm. In addition to the potential direct pitfalls of this reframing (the focus of this article), there are also potential indirect effects spawned from ideological offshoots related to such a reframing, but which are beyond the scope of this

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2 Ibid.

3 Johanna Ralston et al., (2018). 'Time for a New Obesity Narrative'. *The Lancet* 392: 1384. link to the article; accessed 1 December 2021.

4 Harry Rutter, Louise Marshall and Adam Briggs, (30 July 2020). 'Obesity: Tackling the Causes of the Causes' *The BMJ Opinion*. link to the article; accessed 1 December 2021.

5 Laura Backstrom, (2020). 'Shifting the Blame Frame: Agency and the Parent–Child Relationship in an Anti-Obesity Campaign'. *Childhood* 27(2): 203–219. link to the article.

6 Kiana Docherty, (2020a). 'The Toxic World of Tess Holliday and Fat Activism | Politics, Lies... and Health?' *YouTube*. link to the article; accessed 28 November 2021.

7 Kiana Docherty, (2020b). 'Never Take "Health" Advice From This Fat Activist | Virgie Tovar' *YouTube*. link to the article; accessed 26 May 2021.

8 Lindo Bacon, (2021). 'Health at Every Size® Principles Help Us Advance Social Justice, Create an Inclusive and Respectful Community, and Support People of All Sizes in Finding Compassionate Ways to Take Care of Themselves'. *Health At Every Size*. link to the article; accessed 5 November 2021.

9 This Morning, (8 February 2021). 'The Fat Doctor Clashes With Weight Loss Guru | This Morning' *YouTube*. link to the article; accessed 4 November 2021.

paper.[10,11,12,13,14,15,16,17] Here, I examine a variety of arguments for the agentic-futility framing of obesity – including the role of biological drivers, obesity as a disease state, free will, stigma, and others – in a bid to exculpate the view that self-control can and must play a role in successful weight management.

Throughout this article, I structure my reasoning around the arguments of recent papers which tackle the issue of where responsibility for overweight ultimately lies.[18,19,20,21] Note that 'responsibility' is often a more common framing than 'agency', 'self-control', or 'willpower' in the cited papers, and I would like to briefly disentangle these terms here.[22] 'Responsibility' can refer both to moral responsibility (spiking someone's drink with drugs) and prudential responsibility (consuming large amounts of drugs); in the former, the action is immoral as it will likely confer negative (potentially life-threatening) outcomes for another person as a direct result of the action, and thus the actor is morally responsible. In the latter, the action is imprudent as it will likely confer negative (potentially life-threatening) outcomes for the actor alone, and thus the actor is prudentially responsible. (I refer only to *direct* action-outcome events and not indirect second- and third-order effects such as effects on family, wider society, etc.) In this paper, I refer only to prudential responsibility, not moral, and so questions of 'blame' (a moral notion) do not apply; here, I agree with a recent article on the subject:[23] '…*no one* is to be blamed, but *everyone* has … responsibility.'

I present papers containing the most up-to-date arguments regarding agency and obesity which expertly encapsulate the core reasons for the proposed shift away from agency-based conceptions of weight management. The authors offer several arguments which, presented in a short extract format, are compelling: given that genetic and neural factors influence the drive to eat, that these create greater vulnerability in some people more than others, that the obesogenic environment compounds these biological vulnerabilities, and that stigma serves only to worsen the plight of those who are already vulnerable, we should abolish any framing of overweight and obesity as due to personal agency or responsibility. (Actual quotations from papers will be given in the sections below to illustrate the argument in authors' own words.) The argument framed in this way is persuasive, but as I aim to show, digging into the details of each argument separately reveals nuances that call into question the soundness of the conclusions.

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10 Docherty, 2020a.
11 Docherty, 2020b.
12 Bacon, 2021.
13 James Lindsay, (8 April 2020). 'Fatphobia' *New Discourses*. link to the article; accessed 8 April 2021.
14 James Lindsay, (2020a). 'Health At Every Size'. *New Discourses.* link to the article; accessed 8 August 2021.
15 James Lindsay, (2020b). 'Fat Studies' *New Discourses.* link to the article; accessed 4 February 2021.
16 Helen Pluckrose, James Lindsay and Peter Boghossian, (20 August 2020) 'Academic Grievance Studies and the Corruption of Scholarship'. *New Discourses.* link to the article; accessed 4 January 2022.
17 James Lindsay, (2020c). 'Body Positivity'. *New Discourses*. link to the article; accessed 30 June 2021.
18 Grannell et al., 2021.
19 Ralston et al., 2018.
20 Rutter, Marshall and Briggs, 2020.
21 John P. H. Wilding, Vicki Mooney and Richard Pile, (2019). 'Should Obesity Be Recognised as a Disease?' *The BMJ* 366. link to the article.
22 Thanks goes to the editor whose expertise provided clarification on this point.
23 Elliot M. Berry, (2020). 'The Obesity Pandemic – Whose Responsibility? No Blame, No Shame, Not More of the Same'. *Frontiers in Nutrition* 7(2): 3. link to the article; accessed 1 December 2021.

My key contentions are: (i) the arguments put forward are insufficient to warrant the denial of the importance of agency in eating behaviour, (ii) better accounts can be found of 'constrained agency' in vulnerable groups, and (iii) the proposals put forward in these papers[24,25,26] do not consider the potential adverse effects of the denial of agency in controlling noncommunicable disease (NCD). Possible adverse effects include the inculcation of a mindset wherein energy-balance behaviours (i.e., attempts to manage *calories in vs. calories out*) play little-to-no role in the development, prevention, nor treatment of obesity, which could be extremely counterproductive and detrimental to patients.

2. Argument from 'Subconscious Processes'

One of the most powerful arguments proposed is that, since key components of appetite like hunger are governed by subcortical brain structures, the drivers of eating behaviour preclude conscious experience and thus agency:[27]

> Thus, subcortical regions of the brain on their own appear capable of directing behaviour in response to homeostatic perturbation…projections to the cortex as seen in homo sapiens generate a conscious percept and thus experience offering an evolutionary advantage enabling better chance of survival and more sophisticated behaviours.
>
> Therefore, our point … is that for some individuals, for a given set of environmental and physiological parameters, the drive to eat may be greater than the ability to manage this drive.

While I agree that evolutionarily old and important functions like eating behaviour are regulated by subcortical regions over which individuals have little-to-no conscious control, I take issue with the implication that the individual is powerless to intervene, as well as with the limited scope with which the authors describe appetitive behaviour. Before diving into the more substantive biological arguments, I would ask the reader to consider the following examples (and associated questions): (i) groups of protesters going on hunger strike for days or even weeks at a time (*what is the likelihood they all possess a unique biology which allows them – and them alone – to override their hunger drive?*); (ii) millions if not billions of religious individuals observe fasting for weeks at a time every year (*are such religious peoples biologically distinct from non-religious ones, or else does their faith somehow give them greater capacity to ignore biological processes during religious festivals?*).

Regarding the limited scope, the arguments made for subcortical drivers focus solely on 'hunger' and 'satiety' ('appetite sensations'), regulated by hypothalamic nuclei[28]. First, there is no mention of how factors like macronutrient composition, energy density, and food texture – over which individuals have at least some control – may influence

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[24] Grannell et al., 2021.
[25] Ralston et al., 2018.
[26] Rutter, Marshall and Briggs, 2020.
[27] Grannell et al., 2021, p. 3.
[28] Juliana Austin and Daniel Marks, (2009). 'Hormonal Regulators of Appetite'. *International Journal of Pediatric Endocrinology*. link to the article; accessed 22 March 2023.

hunger and satiety,[29,30,31,32] although better data are needed.[33,34] Second, whilst hypothalamic control of hunger is one important component of a complex neurobiological system underpinning eating behaviour, reducing all appetite expression to hunger is far too simplistic. Appetite control involves (at minimum) a complex interplay between hunger/satiety, reward processing, and inhibitory/behavioural control processes operating in a context of environmental cues perpetually shaping learned responses (i.e., habits).[35]

There is also a well-developed literature on the neurobiology of pleasure and desire which is omitted: decades of neuroscience research have focused on the separable psychological reward components 'wanting' and 'liking'.[36] 'Liking' is understood to be the conscious pleasure we experience; it is linked to 'hedonic hotspots', i.e., subregions of the reward system causally amplifying hedonic impact via the nucleus accumbens (NAcc) medial shells, insula cortex, and orbitofrontal cortex (OFC) (subcortical and cortical regions).[37] Similarly, 'wanting' (incentive motivation/salience) incorporates both subcortical and cortical areas (ventral tegmental area [VTA], NAcc/ventral striatum, amygdala, and prefrontal cortex [PFC]). Both wanting and liking can involve conscious and subconscious processing, and incorporating subjective feelings *and* objective hedonic reactions can help us better understand how the conscious and subconscious combine to drive behaviour[38].

The above is not mere pedantry, it is vital to include for several reasons: (i) at the level of the individual, reward is one of the greatest drivers of obesity, and reward neurocircuitry primes cravings for primarily energy-dense foods; (ii) the conscious experience of pleasure conditions us to respond to the sights and smells of foods in our obesogenic environment (i.e., advertising works by inducing wanting, not hunger); and (iii) subcortical reward regions *necessarily* interact with cortical areas like the PFC, which means behavioural control (i.e., agency) is at least theoretically possible. Crucially, these interactions likely shape acquired reward preferences and

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29 Miriam Clegg and Amir Shafat, (2010). 'Energy and Macronutrient Composition of Breakfast Affect Gastric Emptying of Lunch and Subsequent Food Intake, Satiety and Satiation'. *Appetite* 54: 517.

30 Kênia M. B. de Carvalho et al., (2020). 'Dietary Protein and Appetite Sensations in Individuals with Overweight and Obesity: A Systematic Review'. *European Journal of Nutrition* 59: 2317. link to the article; accessed 2 December 2021.

31 Ali Kohanmoo, Shiva Faghih and Masoumeh Akhlaghi, (2020). 'Effect of Short- and Long-Term Protein Consumption on Appetite and Appetite-Regulating Gastrointestinal Hormones, a Systematic Review and Meta-Analysis of Randomized Controlled Trials'. *Physiology & Behavior* 226: 113123.

32 Ecaterina Stribiţcaia and others, (2020) 'Food Texture Influences on Satiety: Systematic Review and Meta-Analysis'. *Scientific Reports* 10(1). link to the article; accessed 2 December 2021.

33 Seyedeh Parisa Moosavian and Fahimeh Haghighatdoost, (2020). 'Dietary Energy Density and Appetite: A Systematic Review and Meta-Analysis of Clinical Trials'. *Nutrition* 69: 110551.

34 Andrew Warrilow et al., (2018) 'Dietary Fat, Fibre, Satiation, and Satiety – a Systematic Review of Acute Studies'. *European Journal of Clinical Nutrition* 73(3): 333. link to the article; accessed 2 December 2021.

35 Carl A. Roberts, Paul Christiansen and Jason C. G. Halford, (2017). 'Tailoring Pharmacotherapy to Specific Eating Behaviours in Obesity: Can Recommendations for Personalised Therapy Be Made from the Current Data?' *Acta Diabetologica* 54: 715. link to the article; accessed 29 September 2021.

36 Ileana Morales and Kent C. Berridge, (2020). '"Liking" and "Wanting" in Eating and Food Reward: Brain Mechanisms and Clinical Implications'. *Physiology & Behavior* 227: 113.

37 Ibid.

38 Kent C. Berridge and Morten L. Kringelbach, (2015). 'Pleasure Systems in the Brain'. *Neuron* 86(3): 646. link to the article; accessed 29 September 2021.

behaviours such as eating habits.[39,40] Additionally, obesity has a multivariate aetiology and while some individuals may be driven by hunger (i.e., 'hypothalamic vulnerability'), many more struggle with the reward-related (wanting/liking) components, as indicated by the clinical superiority of drugs that target satiety *and* reward (e.g., Liraglutide, Semaglutide, Phentermine/Topiramate, Bupropion/Naltrexone) compared to satiety alone (e.g., Lorcaserin).[41,42]

Regarding the argument of powerlessness ('for some individuals, for a given set of environmental and physiological parameters, the drive to eat may be greater than the ability to manage this drive'[43]), this is a misplaced generalisation of the experiences of very few people to the majority. Indeed, this description fits severe cases like sufferers of Prader-Willi syndrome or pituitary disorders like Cushing's disease, but – definitionally – this does not apply to most cases of overweight and obesity, and neither does the dire prognosis of powerlessness. There is an additional failure to consider that the drive to eat is heavily influenced by the size and composition of the body which, if changed, can in turn alter the drive.[44]

Even if the overly pessimistic view outlined in the above quotations is accepted, there remains an acknowledgement that subcortical-cortical projections allow for conscious experience of the outcomes of subcortical activity (e.g., hunger, desire), as well as allowing 'more sophisticated behaviours' to improve 'survival'. That is, even this view allows the cortex to grant individuals the ability to evaluate *and* the potential to regulate subconscious impulses. Neurobiologically, the mesocorticolimbic loop (widely understood as the 'reward system') comprises projections from the VTA to the striatum (both subcortical) and then to the prefrontal cortices (cortical): cognitively, this reflects the interplay between drivers like *incentive salience/cue-induced motivation* (subcortical) and regulators like *control in decision making* (cortical).[45] Thus, given the brain's plasticity, 'the drive to eat' being greater than 'the ability to manage' is not a fixed state, and so allows the scales to swing in the opposite direction to better manage subconscious drives.[46] Indeed, evidence from the weight management and famine literature suggest that this manageability exists on a sliding scale.[47] Thus, agentic action can lead to improvements even when constrained.

To borrow from the example of substance dependence as a state arguably even more reliant on subcortical drives: at a neural level, since shifts in goal-directed to habitual

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39 Shelly B. Flagel and Terry E. Robinson, (2017). 'Neurobiological Basis of Individual Variation in Stimulus-Reward Learning'. *Current Opinion in Behavioral Sciences* 13: 178. link to the article.

40 Francesco Versace, George Kypriotakis, Karen Basen-Engquist and Susan M Schembre (2015). 'Heterogeneity in Brain Reactivity to Pleasant and Food Cues: Evidence of Sign-Tracking in Humans'. *Social Cognitive and Affective Neuroscience* 11: 604.

41 Rohan Khera et al., (2016). 'Association of Pharmacological Treatments for Obesity With Weight Loss and Adverse Events: A Systematic Review and Meta-Analysis'. *JAMA* 315: 2424. link to the article; accessed 30 September 2021.

42 Carl A. Roberts, Paul Christiansen and Jason C. G. Halford, (2019). 'Pharmaceutical Approaches to Weight Management: Behavioural Mechanisms of Action'. *Current Opinion in Physiology* 12: 26.

43 Grannell et al., 2021, p. 3.

44 R. James Stubbs and Jake Turicchi, (2021). 'From Famine to Therapeutic Weight Loss: Hunger, Psychological Responses, and Energy Balance-Related Behaviors'. *Obesity Reviews* 22: e13191. link to the article; accessed 2 December 2021.

45 Roberts, Christiansen and Halford, 2019.

46 Marc Lewis, (2017). 'Addiction and the Brain: Development, Not Disease'. *Neuroethics* 10: 7. link to the article; accessed 6 August 2021.

47 Stubbs and Turicchi, 2021.

behaviour are governed by subcortical-cortical interconnections between the OFC and dorsolateral striatum, respectively,[48,49,50] it is also possible for bad habits to be overridden by goal-oriented focus and substituted for better habits.[51,52,53,54,55,56] (A key difference is that long-term abstinence causes the craving and seeking of an addictive substance to eventually abate, while the opposite is true for food. Thus, I would obviously strongly recommend against long-term abstinence from food in any case, so this difference has little relevance.) At a cognitive level, drug-dependent patients who are trying to abstain often develop an *approach-avoidance* response to drug cues, consciously averting their attention away after initially being reflexively (subconsciously) drawn to the cue.[57,58] At a behavioural level, implementation intentions – self-regulated strategies using carefully thought-out if-then plans to solidify wanted habits and avoid unwanted ones – allow individuals to consciously plan to avoid and overcome risky situations.[59] The key is that no state is fixed, and as is evident neurobiologically, cognitively, behaviourally, and experientially, improvement in outcomes can and does happen.

3. Argument from 'Biological Determinism'

A related argument is that appetitive behaviour is biologically driven *in general* (i.e., genes, brain, and everything in-between):

> Biological determinism appears to play a strong role in dictating behaviour with regard to signals such as hunger and satiety.[60]
>
> This approach [the idea that we can control our eating behaviour] disregards the complex interplay between factors not within individuals' control (e.g., epigenetic, biological, psychosocial) …[61]

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48 Jay A. Gottfried, John O'Doherty and Raymond J. Dolan, (2003). 'Encoding Predictive Reward Value in Human Amygdala and Orbitofrontal Cortex'. *Science* 301: 1104.

49 Christina M. Gremel and Rui M. Costa, (2013). 'Orbitofrontal and Striatal Circuits Dynamically Encode the Shift between Goal-Directed and Habitual Actions'. *Nature Communications* 4: 2264. link to the article; accessed 20 March 2014.

50 John P. O'Doherty, (2007). 'Lights, Camembert, Action! The Role of Human Orbitofrontal Cortex in Encoding Stimuli, Rewards, and Choices'. *Annals of the New York Academy of Sciences* 1121: 254.

51 Lewis, 2017.

52 Marieke A. Adriaanse and Aukje Verhoeven, (2018). 'Breaking Habits Using Implementation Intentions' in *The Psychology of Habit: Theory, Mechanisms, Change, and Contexts*, pp. 169–188 (Cham: Springer). link to the article; accessed 4 August 2021.

53 Laura H. Corbit, Billy C. Chieng and Bernard W. Balleine, (2014). 'Effects of Repeated Cocaine Exposure on Habit Learning and Reversal by N-Acetylcysteine'. *Neuropsychopharmacology* 39(8): 1893. link to the article; accessed 4 August 2021.

54 Peter M. Gollwitzer and Paschal Sheeran, (2006). 'Implementation Intentions and Goal Achievement: A Meta-analysis of Effects and Processes'. *Advances in Experimental Social Psychology* 38: 69.

55 Amy Malaguti et al., (2020). 'Effectiveness of the Use of Implementation Intentions on Reduction of Substance Use: A Meta-Analysis'). *Drug and Alcohol Dependence* 214: 108120.

56 Leona Tam, Richard P. Bagozzi and Jelena Spanjol, (2010). 'When Planning Is Not Enough: The Self-Regulatory Effect of Implementation Intentions on Changing Snacking Habits' *Health Psychology* 29: 284.

57 Matt Field et al., (2013). 'Attentional Biases in Abstinent Alcoholics and Their Association with Craving'. *Psychology of Addictive Behaviors* 27: 71. link to the article; accessed 22 July 2021.

58 Matt Field et al., (2016). 'The Role of Attentional Bias in Obesity and Addiction'. *Health Psychology* 35: 767.

59 Malaguti et al., 2020.

60 Grannell et al., 2021, p. 4.

61 Ralston et al., 2018, p. 1385.

As with the argument in Section 2, this applies to every human experience. We have known since at least Darwin (1859)[62] that individuals differ, that these differences are largely coded biologically, and that if traits confer survival advantages (such as driving the individual to consume more energy) that on average those traits increase in frequency in subsequent generations. More modern behavioural genetics results show that across all traits ever studied in humans, genetic variation accounts for around half of all variation in trait phenotype (*heritability*) across 39 countries.[63] Such findings gave rise to the first two laws of behavioural genetics: (i) all human behavioural traits are heritable, and (ii) the effect of being raised in the same family is smaller than the effect of genes.[64] Thus, although I would not use the term 'biological determinism' (opting instead for 'underpinnings' or 'drives'), I fully agree that biology (and every other contributing factor) needs to be included in our scientific models and in our sociopolitical conversations. However, if the implication of the above quotations (and surrounding paragraphs) is that if biology is involved then choice and responsibility cannot be, then that would require biological determinism in all other domains of human experience as well.

4. Argument from 'Free Will'

The inevitable culmination of the first two arguments – from subconscious processes and from biological determinism – is that, if free will exists, it is drastically impaired in decisions relating to food:[65]

> …empirical evidence shows we perhaps do not have full control over our decision making around food … Building on the classic Libet studies, functional magnetic resonance imaging (fMRI) has been used to investigate the … moment of a conscious decision to engage in movement revealing subconscious brain activity emerges before the decision was made. Indeed, up to 8s prior to the conscious decision to move it was possible to determine which hand would be moved.

Rather than engage in the specifics of Libet-style experiments[66,67,68] and their criticisms,[69,70] I will focus on the logic on display via exploration of one's own conscious

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62 Charles Robert Darwin, (1859). *On the Origin of Species by Means of Natural Selection, or Preservation of Favoured Races in the Struggle for Life* (John Murray ed., 1st edn).

63 Tinca J. C. Polderman et al., (2015). 'Meta-Analysis of the Heritability of Human Traits Based on Fifty Years of Twin Studies'. *Nature Genetics* 47: 702. link to the article.

64 Eric Turkheimer, (2009). 'Three Laws of Behavior Genetics and What They Mean'. *Current Directions in Psychological Science* 9: 160.

65 Grannell et al., 2021, p. 5.

66 Moritz Nicolai Braun, Janet Wessler and Malte Friese, (2021). 'A Meta-Analysis of Libet-Style Experiments'. *Neuroscience & Biobehavioral Reviews* 128: 182.

67 Benjamin Libet, (1985). 'Unconscious Cerebral Initiative and the Role of Conscious Will in Voluntary Action'. *Behavioral and Brain Sciences* 8: 529. link to the article; accessed 5 August 2021.

68 Benjamin Libet et al., (1993). 'Time of Conscious Intention to Act in Relation to Onset of Cerebral Activity (Readiness-Potential)', in *Neurophysiology of Consciousness*, p. 249 (Boston, MA: Birkhäuser). link to the article; accessed 5 August 2021.

69 Daniel C. Dennett, (1984). *Elbow Room : The Varieties of Free Will Worth Wanting* (Cambridge, MA: MIT Press).

70 Paul G. Nestor, (2019). 'In Defense of Free Will: Neuroscience and Criminal Responsibility'. *International Journal of Law and Psychiatry* 65: 101344.

experience.[71,72] But first it is worth noting that while Grannell et al. (2021)[73] point out that these experiments use simple movement decisions in their methodologies, they imply that only (or mostly) 'decision making around food' lacks free will – their reasoning for this is already rebutted in the first two arguments (i.e., that the subconscious processing and neurobiology which applies to eating also applies to many other facets of life for which we still believe in agency).

Investigating our conscious experience of free will via the subcortical-cortical distinction drawn earlier is straightforward and I would like the reader to follow along:

> *Conjure any number into consciousness. Which number did you pick? Does it hold special significance, or did it appear at random for seemingly no reason? Did you know what number you were going to pick before you were consciously aware of it? Whatever the number and whatever the reason, the answer to 'did you know the number before you were aware of it' is of course 'no'.*

The number simply appears in consciousness from nowhere, or to use a more material description, it is projected from the subcortical unconscious to the conscious cortex. My point here is not to argue for or against free will, but to point out that whatever is true, it is true for all thoughts, feelings, and emotions, and is not esoterically bound to appetite.

5. Argument from 'Obesity as a Disease'

Another common argument[74,75,76] is that obesity is a disease which some people are more vulnerable to developing than others and which defies attempts to reverse via physiological resistance (i.e., evolution has designed us to not lose weight easily). While I can agree with the conception of obesity as a disease state that some are more vulnerable to than others, this conception does not preclude the role of agency. As noted in Section 3, virtually all traits are heritable, every individual is innately unique,[77] and thus every individual is uniquely vulnerable and/or advantaged in all endeavours. Susceptibility to obesity is not unique. It is also important to differentiate between NCDs which are truly inherent (i.e., unavoidable regardless of behaviour or environment, such as trisomy 21), and those like obesity which in most cases are not. Currently, no such distinction is being made in the literature, which imposes a sense of fatalism for obesity which does not actually exist (see Sections 7 and 8).

The claim of physiological resistance may appear more applicable to obesity, substance use disorders, and behavioural compulsions, but this appearance is at least partly due to the overwhelming concentration of research on such topics (due to their overwhelming impact on people and society), which has allowed our understanding of their physiological underpinnings to grow. If we had devoted as much research to any other human experience, we would also possess more in-depth models of the 'physiology

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71 Richard Oerton, (2012). *The Nonsense of Free Will: Facing up to a False Belief* (1st edn, Kibworth Beauchamp: Matador).
72 Sam Harris, (2012). *FREE WILL* (1st edn, Free Press).
73 Grannell et al., 2021.
74 Ibid.
75 Ralston et al., 2018.
76 Wilding, Mooney and Pile, 2019.
77 Kevin J. Mitchell, (2007). 'The Genetics of Brain Wiring: From Molecule to Mind'. *PLOS Biology* 5: e113. link to the article; accessed 28 July 2021.

of …' sleep, exercise, learning, mindset, social relationships, romantic relationships, and so on. All these domains are deeply social, cultural, and systemic, but they are also personal and biological. Experiences that span a lifetime (many of which are habitually ingrained) are multivariate and typically possess immense inertia – altering almost any of them in any substantial or long-term way requires either tremendous individual effort, the reshaping of society, or both. This point alone should be enough to garner compassion for anyone struggling with weight management (or management of similar problems, as outlined below).

Sleep is influenced by sociocultural and economic factors such as a culture's occupational demands, types of social activity (exercise vs. alcohol consumption), timing of social activities (daytime vs. night-time), urban vs. rural living, shift work, the comfortableness of the bed one can afford, etc. It is also influenced by physiology: chronotype – early birds vs. night owls – is influenced by genetics and grey matter volume, and is considered a stable, trait-like construct, despite night owls experiencing much worse outcomes and thus having real reasons to alter their sleep patterns.[78,79,80,81] Despite this resistance (both environmental and physiological), and until such time that society can be reshaped to abolish shift work or science is able to biologically alter circadian typology, there are recommended steps an *individual agent* can take to improve sleep immediately, such as changes to exercise, bedtime routine, and light exposure.[82] Incidentally, sleep is deeply interconnected to neurological reward networks and behaviours, and to food choice specifically.[83,84]

A more straightforward analogue to obesity may be asthma: sufferers are vulnerable to worse health outcomes, the disease can be made worse by both biological and environmental factors, and it is physiologically resistant to change. However, while medical treatment is vital, a growing evidence base suggests that individuals who increase their physical activity levels are more likely to get their symptoms under control, build stronger lung function, and potentially better manage the disease in the long term.[85,86,87]

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78 Ana Adan et al., (2012). 'Circadian Typology: A Comprehensive Review'. *Chronobiology International* 29: 1153. link to the article; accessed 3 August 2021.

79 Stella J. M. Druiven et al., (2020). 'Stability of Chronotype over a 7-Year Follow-up Period and Its Association with Severity of Depressive and Anxiety Symptoms'. *Depression and Anxiety* 37: 466. link to the article; accessed 3 August 2021.

80 David A. Kalmbach et al., (2017). 'Genetic Basis of Chronotype in Humans: Insights From Three Landmark GWAS'. *Sleep* 40. link to the article; accessed 3 August 2021.

81 Jessica Rosenberg et al., (2018). 'Chronotype Differences in Cortical Thickness: Grey Matter Reflects When You Go to Bed'. *Brain Structure and Function* 223: 3411. link to the article; accessed 4 August 2021.

82 Alyssa M. Rivera and Andrew D. Huberman, (2020). 'Neuroscience: A Chromatic Retinal Circuit Encodes Sunrise and Sunset for the Brain'. *Current Biology* 30: R316.

83 Lauren D. Asarnow, Stephanie M. Greer, Mathew P. Walker and Allison G. Harvey, (2017). 'The Impact of Sleep Improvement on Food Choices in Adolescents With Late Bedtimes'. *Journal of Adolescent Health* 60: 570. link to the article; accessed 2 December 2021.

84 Ninad Gujar, Seung-Schik Yoo, Peter Hu and Matthew P. Walker, (2011. 'Sleep Deprivation Amplifies Reactivity of Brain Reward Networks, Biasing the Appraisal of Positive Emotional Experiences'. *Journal of Neuroscience* 31: 4466. link to the article; accessed 2 December 2021.

85 Kimberly M. Avallone and Alison C. McLeish, (2013). 'Asthma and Aerobic Exercise: A Review of the Empirical Literature'. *Journal of Asthma* 50: 109. link to the article; accessed 29 September 2021.

86 Stefano R. Del Giacco, Davide Firinu, Leif Bjermer and Kai-Håkon Carlsen, (2015). 'Exercise and Asthma: An Overview'. *European Clinical Respiratory Journal* 2: 27984. link to the article; accessed 29 September 2021.

87 Sirpa A. M. Heikkinen and others, (2017). 'Effects of Regular Exercise on Asthma Control in Young Adults'. *Journal of Asthma* 55: 726. link to the article; accessed 29 September 2021.

As with the treatment of obesity, successful long-term management of both sleep and asthma requires one's own will to be prominent (perhaps in addition to medical treatment), and to take that away by telling sufferers that willpower/agency has little bearing on outcomes could be fatal.

6. Argument from 'Framing and Stigma'

The core reason given[88,89,90,91] for the desire to expunge agency from the causal chain of obesity is that it introduces the possibility (or increases the likelihood) of stigma:

> This stigma is anchored in the belief that obesity is directly due to limited self-control and an inability to consciously make correct choices regarding diet and exercise.[92]
>
> …we remain assailed by the rhetoric of 'choice' and 'lifestyle', and campaigns exhorting us to change our behaviour… [93]

There are three problems with this view. First, it is a view Grannell et al. (2021) seem to implicitly hold given that in their very final paragraph they state that:

> it remains the patient's responsibility to adhere to treatment that proves effective …[94]

This is at odds with their castigation of 'self-control' given the necessary link between self-control and prudential responsibility (see Section 1). Moreover, given the safe assumption that these authors do not believe they stigmatise sufferers of obesity, alongside the above evidence that they believe in (prudential) responsibility (which I think they may be mistaking for moral responsibility), this would strongly imply that one can simultaneously believe in agentic causes (i.e., self-control) *and* show compassion absent any hint of blame.[95] In fact, this is my view, too. Thus, one need not throw the agentic baby out with the stigmatising bathwater.

A second issue is the claim that any public stigma flows directly from conceptions of agency, and that tying obesity more closely to biology will help solve this problem: 'Importantly, the understanding that appetite is regulated in subcortical regions of the brain beyond conscious experience challenges the idea that obesity is the individuals' fault'.[96] While I entirely understand this logic – and the arguments of the cited review[97] – findings from the fields of addiction and criminology show that things are not so straightforward.

Biological or medicalised explanations may reduce perceptions of volition and/or responsibility (depending on explanation type), but reduction in blame is marginal.[98]

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88 Grannell et al., 2021.
89 Ralston et al., 2018.
90 Rutter, Marshall and Briggs, 2020.
91 Wilding, Mooney and Pile, 2019.
92 Grannell et al., 2021, p. 4.
93 Rutter, Marshall and Briggs, 2020, para 3.
94 Grannell et al. 2021, p. 6.
95 Eric Racine, Sebastian Sattler and Alice Escande, (2017).'Free Will and the Brain Disease Model of Addiction: The Not So Seductive Allure of Neuroscience and Its Modest Impact on the Attribution of Free Will to People with an Addiction'. *Frontiers in Psychology* 8(1). link to the article; accessed 6 August 2021.
96 Grannell et al., 2021, p. 4.
97 Rebecca M. Puhl and Chelsea A. Heuer, (2010). 'Obesity Stigma: Important Considerations for Public Health'. A*merican Journal of Public Health* 100: 1019. link to the article; accessed 6 August 2021.
98 Rachel McKenzie, Barry Schwartz and John R. Monterosso, (2021). 'Effects of Addiction Science on Conceived Freewill and Responsibility'. *Addictive Behaviors* 120: 106955.

Specifically, framing addiction as a 'brain disease' is unlikely to alter stigma but may redirect research funding from socioeconomic causes to those strictly biological.[99] Furthermore, it is common for service providers and experts in addiction to hold a range of diverse views concurrently, namely the disease model, social model, free will model, etc. Service providers also identify both positive (reduced stigma) and negative (learned helplessness) consequences to the disease model.[100] In criminology, genetic and neuroscientific testimony can sometimes reduce perceptions of responsibility, but perhaps paradoxically it is simultaneously either untethered to or may even exacerbate attributions of choice and blame, and can even result in harsher sentencing.[101,102,103]

Similar evidence exists for obesity. For example, if obesity were to be reframed as an addiction – the current view of which is as a chronic *brain disease*[104,105] – this may reinforce stigma and maintain focus on individual-level rather than population-level interventions.[106,107] Worryingly, almost two decades ago addiction researchers were highlighting the problematic potential of understanding addiction as a disease as possibly leading to 'great and desperate cures'.[108] Unfortunately, the time has come for such concern to be directed towards obesity, given recent recommendations from the American Academy of Pediatrics to prescribe pharmacotherapy for children and surgery for adolescents.[109] 'Food addiction' is commonly viewed simultaneously as more disease-like than smoking and as being caused by individual choices to a greater extent than alcoholism.[110] Crucially, a recent randomised study concluded that the 'food addiction' framing resulted in higher internalised weight stigma with no effect on

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[99] Molly J. Dingel, Katrina Karkazis and Barbara A. Koenig, (2011). 'Framing Nicotine Addiction as a "Disease of the Brain": Social and Ethical Consequences'. *Social Science Quarterly* 92: 1363. link to the article; accessed 6 August 2021.

[100] Anthony I. Barnett et al., (2018). 'Drug and Alcohol Treatment Providers' Views about the Disease Model of Addiction and Its Impact on Clinical Practice: A Systematic Review'. *Drug and Alcohol Review* 37: 697. link to the article; accessed 6 August 2021.

[101] Darby Aono, Gideon Yaffe and Hedy Kober, (2019). 'Neuroscientific Evidence in the Courtroom: A Review'. *Cognitive Research: Principles and Implications* 4. link to the article; accessed 6 August 2021.

[102] Paul S. Appelbaum, Nicholas Scurich and Raymond Raad, (2015). 'Effects of Behavioral Genetic Evidence on Perceptions of Criminal Responsibility and Appropriate Punishment'. *Psychology, Public Policy, and Law* 21(2): 134. link to the article; accessed 6 August 2021.

[103] Robert Blakey and Tobias P. Kremsmayer, (2018). 'Unable or Unwilling to Exercise Self-Control? The Impact of Neuroscience on Perceptions of Impulsive Offenders' *Frontiers in Psychology* 8: 1. link to the article; accessed 6 August 2021.

[104] Lewis, 2017.

[105] Sally Satel and Scott O. Lilienfeld, 'Addiction and the Brain-Disease Fallacy' (2014) 4 Frontiers in Psychiatry 1. link to the article; accessed 6 August 2021.

[106] Nicolas Rasmussen, 'Stigma and the Addiction Paradigm for Obesity: Lessons from 1950s America' (2015) 110 Addiction 217. link to the article; accessed 6 August 2021.

[107] Daniel Z. Buchman, Judy Illes and Peter B. Reiner, (2010). 'The Paradox of Addiction Neuroscience'. *Neuroethics* 4: 65.

[108] Wayne Hall, (2006). 'Stereotactic Neurosurgical Treatment of Addiction: Minimizing the Chances of Another "Great and Desperate Cure"'. *Addiction* 101: 1.

[109] Sarah E.Hampl et al., (2023). 'Executive Summary: Clinical Practice Guideline for the Evaluation and Treatment of Children and Adolescents with Obesity'. *Pediatrics* 151: e2022060641.

[110] Jenny A. DePierre, Rebecca M Puhl and Joerg Luedicke, (2014). 'Public Perceptions of Food Addiction: A Comparison with Alcohol and Tobacco'. *Journal of Substance Use* 19: 1. link to the article; accessed 6 August 2021.

externalised weight stigma (vs. a 'calorie-balance' framing).[111] Moreover, while public support for the notion of 'food addiction' has grown, the public nevertheless views individual choice and responsibility as the primary causes of and treatment options for obesity.[112] Somewhat paradoxically, a better way to increase support for broader obesity-reduction policies while also increasing empathy for sufferers is to emphasise *both* personal responsibility and social and environmental determinants.[113,114,115] This messaging is also more effective at increasing intentions to eat more fruits and vegetables, exercise, and/or alter diet in overweight/obese populations.[116] I am not the first to suggest a more balanced, multivariate perspective.[117,118,119]

The third problem is that, while framing obesity as a biological disease (even a chronic brain disease like addiction) untethered from one's choices might reduce stigma (or not, as outlined above), it might also encourage learned helplessness and aggravate the very problem it attempts to solve. Grannell et al. (2021)[120] unintentionally reveal this when they argue 'there is strong confirmation and survivorship bias behind these [public health messages about self-regulation of diet and exercise] as this approach works in approximately two in every 10 people'. The article they reference[121] deals with weight stigma, but messages regarding agency and control need not be stigmatising; they can instead be empowering, motivating, and empathy-inducing.[122,123,124,125] Further, the quote concedes that self-regulation messaging 'works for … two in every 10 people' but implies that this is inadequate. This is inexplicable: if a drug was discovered tomorrow that helped reduce overweight/obesity in 20% of the population it would be considered miraculous.

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111 Lindsey Parnarouskis Riley J. Jouppi, Jenna R. Cummings and Ashley N. Gearhardt (2021). 'A Randomized Study of Effects of Obesity Framing on Weight Stigma'. *Obesity* 29: 1625. link to the article; accessed 1 December 2021.

112 Natalia M. Lee et al., (2013). 'Public Views on Food Addiction and Obesity: Implications for Policy and Treatment'. *PLOS ONE* 8: e74836. link to the article; accessed 6 August 2021.

113 Jeff Niederdeppe et al., (2014). 'Narrative Persuasion, Causality, Complex Integration, and Support for Obesity Policy'. *Health Communication* 29: 431. link to the article; accessed 6 August 2021.

114 Jeff Niederdeppe, Sungjong Roh and Michael A. Shapiro, (2015). 'Acknowledging Individual Responsibility While Emphasizing Social Determinants in Narratives to Promote Obesity-Reducing Public Policy: A Randomized Experiment'. *PLoS ONE* 10: 1. link to the article; accessed 6 August 2021.

115 Paul H. Thibodeau et al., (2017). 'Narratives for Obesity: Effects of Weight Loss and Attribution on Empathy and Policy Support'. *Health Education & Behavior* 44: 638. link to the article; accessed 6 August 2021.

116 Jeff Niederdeppe, Sungjong Roh, Michael A. Shapiro and Hye Kyung Kim, (2013). 'Effects of Messages Emphasizing Environmental Determinants of Obesity on Intentions to Engage in Diet and Exercise Behaviors'. *Preventing Chronic Disease* 10. link to the article; accessed 6 August 2021.

117 Berry, 2020.

118 Christina A Roberto et al., (2015). 'Patchy Progress on Obesity Prevention: Emerging Examples, Entrenched Barriers, and New Thinking'. *The Lancet* 385: 2400. link to the article; accessed 1 December 2021.

119 Fiona Sim, (2017). 'Obesity: Personal Responsibility or Environmental Curse?' *The BMJ Opinion*. link to the article; accessed 4 April 2022.

120 Grannell et al., 2021, p. 4.

121 A. Janet Tomiyama et al., (2018). 'How and Why Weight Stigma Drives the Obesity "Epidemic" and Harms Health'. *BMC Medicine* 16: 123. link to the article; accessed 11 August 2021.

122 Niederdeppe et al., 2014.

123 Niederdeppe, Roh and Shapiro, 2015.

124 Thibodeau et al., 2017.

125 Niederdeppe et al., 2017.

The evidence that self-monitoring, self-control, and/or self-motivation works – is even actively required – for weight loss and maintenance is unequivocal.[126,127,128,129,130,131] Self-efficacy – a belief in one's own capacity to achieve a specific goal – is particularly important for weight-loss maintenance,[132,133] but this would appear difficult or even impossible to countenance in a world where agency and self-control are futile in any attempt to manage one's weight. In fact, self-control beliefs and perceived behavioural control are directly tied to self-efficacy, as well as to actual weight-management behaviours such as exercise.[134,135,136,137]

7. Impact on Research

The reframing sought by some even extends to the moral legitimacy of research questions, which can have a chilling effect on scientists' willingness to be associated with certain research topics or research-based claims (see the 'Background' section at the beginning of this paper). Accusing a research group[138] of stigmatisation for investigating cognitive predictors of weight loss, Grannell et al. (2021) write:

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[126] C. A. Befort et al., (2008). 'Weight Maintenance, Behaviors and Barriers among Previous Participants of a University-Based Weight Control Program'. *International Journal of Obesity* 32: 519. link to the article; accessed 12 August 2021.

[127] K. Elfhag and S. Rössner, (2005). 'Who Succeeds in Maintaining Weight Loss? A Conceptual Review of Factors Associated with Weight Loss Maintenance and Weight Regain' *Obesity Reviews* 6: 67. link to the article; accessed 12 August 2021.

[128] Kelly H. Webber et al. (2010). 'Motivation and Its Relationship to Adherence to Self-Monitoring and Weight Loss in a 16-Week Internet Behavioral Weight Loss Intervention'. *Journal of Nutrition Education and Behavior* 42: 161. link to the article; accessed 12 August 2021.

[129] R. R. Wing and James O. Hill, (2001). 'Successful Weight Loss Maintenance'. *Annual Review of Nutrition* 21: 323. link to the article; accessed 12 August 2021.

[130] R. R. Wing and S .Phelan, (2005). 'Long-Term Weight Loss Maintenance'. *American Journal of Clinical Nutrition* 82: 222S. link to the article; accessed 12 August 2021.

[131] Yaguang Zheng et al., (2015). 'Self-Weighing in Weight Management: A Systematic Literature Review'. *Obesity* 23: 256. link to the article; accessed 12 August 2021.

[132] Lora E. Burke et al., (2015). 'The SELF Trial: A Self-Efficacy-Based Behavioral Intervention Trial for Weight Loss Maintenance'. *Obesity* 23: 2175. link to the article; accessed 12 August 2021.

[133] Pedro J. Teixeira et al., (2010). 'Mediators of Weight Loss and Weight Loss Maintenance in Middle-Aged Women'. *Obesity* 18: 725. link to the article; accessed 12 August 2021.

[134] Icek Ajzen, (2002). 'Perceived Behavioral Control, Self-Efficacy, Locus of Control, and the Theory of Planned Behavior'. *Journal of Applied Social Psychology* 32: 665. link to the article; accessed 12 August 2021.

[135] James J. Annesi, Ping H. Johnson and Kristin L. McEwen, (2015). 'Changes in Self-Efficacy for Exercise and Improved Nutrition Fostered by Increased Self-Regulation Among Adults With Obesity'. *Journal of Primary Prevention* 36: 311. link to the article; accessed 12 August 2021.

[136] James J. Annesi and Linda L. Vaughn, (2017). 'Directionality in the Relationship of Self-Regulation, Self-Efficacy, and Mood Changes in Facilitating Improved Physical Activity and Nutrition Behaviors: Extending Behavioral Theory to Improve Weight-Loss Treatment Effects'. *Journal of Nutrition Education and Behavior* 49: 505.

[137] Navin Kaushal, Béatrice Bérubé, Martin S. Hagger and Louis Bherer, (2021). 'Investigating the Role of Self-Control Beliefs in Predicting Exercise Behaviour: A Longitudinal Study'. *British Journal of Health Psychology* 26: 1155. link to the article; accessed 12 August 2021.

[138] Ryoko Sawamoto et al., (2017). 'Predictors of Successful Long-Term Weight Loss Maintenance: A Two-Year Follow-Up'. *Biopsychosocial Medicine* 11: 14. link to the article; accessed 12 August 2021.

> This narrative [of responsibility, willpower, and behaviour control] has even made its way into the literature … as weight loss is commonly described as being due to cognitive factors. This weight stigma …[139]

Adoption of such a view alongside the castigation of scientists with a particular focus will lead to a dearth of research into specific aetiologies, which will seriously impair our attempts to both prevent and treat obesity. The two core pathways to behaviour change are (i) altering energy balance patterns shaped by entrenched, lifelong conditioning, and (ii) maintaining these healthier patterns.[140] Even when combined with pharmacotherapy, these pathways must be adhered to and, as reviewed above, both require self-efficacy which is itself best informed and guided by patients' experiences of their personal barriers to change.[141] Removing peoples' decisions, patterns of behaviour, self-reported reasons for acting or not acting – i.e., agency – from both research and treatment plans is a recipe for disaster. Moreover, the censuring of researchers based on the faulty claim that their ideas lead to stigmatisation is both unscientific and dangerous.

8. Can Obesity and Agency Be Reconciled?

I hope to have effectively refuted the core arguments of the *futility of will* framing of obesity's development, prevention, and management, but it is insufficient to simply counter an opposing view; one should also aim to provide an alternative. My ultimate goal here is to provide a framework for compassionately *and* accurately reflecting the reality of overweight and obesity in order to provide researchers, clinicians, and patients with the optimal resources with which to think effectively about, research, treat, and manage obesity. I believe coproduction of health models involves joint (not necessarily equal) responsibility on the part of the patient and practitioner working together to solve difficult and nuanced problems. The biomedical model is clearly too simplistic; it is the combination of cognitive behaviour-change approaches and the practice, development, and acquisition of new habits in the face of old habits that determines the success of efforts to change behaviour.[142] The fact that the Appetite & Obesity field has focused mainly on social cognitive theory until the last couple of decades means that reactive components of behaviour change interventions (such as emotional and stress responses to treatment) are under-researched.

This goal is ultimately identical to those who wish to downplay agency (in whatever guise it appears: choice, self-control, responsibility, etc.) in that I wish to redirect focus to a more nuanced, multivariate, and compassionate model of obesity, as well as encouraging more policy interventions at the population level (systemic change) as opposed to just at the individual level (health messaging/behaviour change attempts). To move this conversation forward, however, I believe certain facts must be acknowledged and certain distinctions made:

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[139] Grannell et al., 2021, p. 4.
[140] Roberts, Christiansen and Halford, 2017.
[141] Ibid.
[142] Menna Price, Suzanne Higgs, James Maw and Michelle Lee, (2016). 'A Dual-Process Approach to Exploring the Role of Delay Discounting in Obesity'. *Physiology & Behavior* 162: 46. link to the article; accessed 3 December 2021.

1. *Agency exists but is constrained:* If agency exists for other human activities, it also exists for eating. This does not preclude constraints on agency; indeed, given the known influence of biological and environmental factors a 'constrained agency' model of behaviour is most reasonable, with such constraints becoming even more important in reward-related behaviour. Notably, different constraints exist for different people in different contexts and to differing extents.
2. *Diseases are not created equal:* NCDs like obesity – the cause of which is multivariate – need to be explicitly distinguished from NCDs with more direct and inevitable biological causal pathways for which agency is entirely absent (e.g., 'inherent diseases' like single-gene disorders). They are also distinct from NCDs which have multivariate aetiologies, but for which the treatment (not cause) is minimally affected by lifestyle changes (e.g., cancer).
3. *There are multiple pathways to obesity:* What we might call 'state obesity' (with a multivariate causal chain) is qualitatively different from 'trait obesity' which might be the product of diseases like Prader-Willi syndrome or hypothyroidism. (We need not pretend this division is black-and-white, but the distribution on the continuum is heavily skewed, with most people at the 'state' end of the obesity spectrum rather than at the 'trait' end.[143]) Neither is easy to overcome, but agency is clearly less constrained in state obesity than in trait obesity.

9. Conclusion

Adding on to the diagnosis of obesity an unavoidable and unassailable biological component which strips away agency could spawn a self-conceptualisation which impedes treatment. Framings of the self and the world are not abstract entities, they actively determine an individual's choice to act (or not) and thus help shape their future. While I, of course, agree that some individuals' choices are much more difficult and hard-won than others due to their biology and environment, devaluing agency is both theoretically untenable and practically counterproductive.

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[143] Stubbs and Turicchi, 2021.