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ORIGINAL RESEARCH

Why We Never Eat Alone: The Overlooked Role of Microbes and Partners in Obesity Debates in Bioethics

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Abstract Debates about obesity in bioethics tend to unfold in predictable epicycles between individual choices and behaviours (e.g., restraint, diet, exercise) and the oppressive socio-economic structures constraining them (e.g., food deserts, advertising). Here, we argue that recent work from two cutting-edge research programmes in microbiology and social psychology can advance this conceptual stalemate in the literature. We begin in section 1 by discussing two promising lines of obesity research involving the human microbiome and relationship partners. Then, in section 2, we show how this research has made viable novel strategies for fighting obesity, including microbial therapies and dyad-level interventions. Finally, in section 3, we consider objections to our account and conclude by arguing that attention to the most immediate features of our biological and social environment offers a middle ground solution, while also raising important new issues for bioethicists.

Keywords Obesity in bioethics · Human microbiome · Relationship science · Social psychology · Food ethics

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Introduction

Obesity has been called "the most difficult and elusive public health problem this country [the United States] has ever encountered" (Callahan 2013a, 34). As is well known, two-thirds of Americans are now classified as overweight or obese (Ogden , Carroll, Kit, and Flegal 2014). While there is debate over the nature of this classification as a problem or as an epidemic (Lyons 2009), there is no escaping the fact that obesity has been continuously associated with negative health outcomes including heart disease, stroke, type 2 diabetes, and certain kinds of cancer. The estimated annual medical cost of obesity in the United States was $147 billion in 2008 U.S. dollars; the medical costs for people who have obesity were $1,429 higher than those of normal weight (Center for Disease Control and Prevention [CDC] 2012; for a critique see also Dean 2014). Additionally, individuals with obesity face stigmatization and discrimination in the areas of employment, education, healthcare, and elsewhere (Puhl and Brownell 2001).

If there is anything resembling consensus among clinicians, it is that "long lists of interventions have been recommended, but most experts agree that no single intervention will be successful on its own. The required prescription is comprehensive, multilevel interventions" (Bassett and Perl 2004, 1477). What was true a decade ago still holds today: "the time is now ripe, and more urgent than ever, to implement a new, multilevel approach to understanding the basis of the obesity epidemic and how to reverse it (Huang and Glass 2008, 1811).

Despite this widespread agreement, the bioethics literature on obesity has largely remained entrenched in a

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dichotomous framework, pitting (various forms of) individual responsibility against (various forms of) environmental/social determinants of health. While there are exceptions, debates in bioethics still tend to unfold in predictable epicycles between individual choices and behaviours (e.g., restraint, diet, exercise) and the oppressive socio-economic structures constraining them (e.g., food deserts, advertising).

To take just one example, consider the lively exchanges surrounding Daniel Callahan's high-profile article (2013a). While Callahan does advocate for some public health measures, most of his discussion (and the criticisms of it) centre around individual responsibility. For example, "it will be imperative, first, to persuade them [obese individuals] that they ought to want a good diet and exercise for themselves" (37). He similarly claims that we ought "to induce people who are overweight or obese to put some uncomfortable questions to themselves" (39).

Many critics (rightly, in our view) decry these individualizing tendencies and the stigmatizing effects stemming from them (Tomiyama and Mann 2013; Goldberg and Puhl 2013; Walter and Barnhill 2013; Schmidt 2013). The substance of Callahan's response (2013b) to his critics is less illuminating for our purposes than his conceptual framing, which exemplifies the dichotomous nature of the debate:

> Some years ago in the South, there was a tension between two theological factions, the "help its" and the "can't help its." The "helps its" believed in free will and the "can't help its" in predestination. That same tension is visible in debates on obesity: the former believe we have some real choice about what we eat and whether we exercise, and the latter place almost all the emphasis on the obesogenic society in which we live, stealing our freedom from us. (Callahan, 2013a, 9)

Here, we are not assessing whether Callahan has succeeded in striking a balance between these factions. Instead, the intuition underlying the present paper is that the terms of this debate are profoundly misguided.

The present paper has three aims. First, we will introduce into the bioethics literature two new bodies of empirical research on obesity which have received scant attention to date (section 1). Second, we will argue that while this empirical evidence is still preliminary, it is suggestive enough to demand a re-examination of the conceptualizations that bioethicists have used to talk about the causes of, and treatments for, obesity (section 2). Third, we will suggest that attention to these new lines of empirical research has the potential to both suggest new clinical intervention strategies for obesity and also advance stalemated debates about obesity in the bioethics literature. More specifically, we argue that recognizing the role of our most immediate *biological* environment—our microbiome—can increase predictive and explanatory power while also highlighting novel sites of intervention. Similarly, recognizing and maximizing the role of our *social* partners can bolster predictive and explanatory resources while also suggesting new intervention strategies. We then conclude by considering objections to our account (section 3).

Microbes and Obesity

A number of recent studies in microbiology have shown that biological organisms, from the simplest to the most complex like ourselves, integrate within their own functioning an entire community of bacteria, protists, fungi, and viruses (Gordon and Klaenhammer 2011; Knight 2015). This community, an organism's *microbiome*, varies from body site to body site and also from individual organism to individual organism, up to the point that it captures one of the most individualizing aspects of our biochemistry—our unique microbial signature (Franzosa et al. 2015). Once the size (being only 47% human by cell count; Sender, Fuchs, and Milo 2016) and the genetic diversity (exceeding human genes by a factor of 100; Xu and Gordon 2003; Turnbaugh et al. 2007) of this microbial community was properly documented, scientists explored the roles that these intimate microorganisms play in our own physiological functioning, as well as the ways in which they influence our health and overall well-being (Cho and Blaser 2012; Cryan and Dinan 2012; Relman 2015).

An emerging conclusion is that human phenotypic traits (and organismal traits in general) can no longer be predicated *solely* in relation to the human genome. We need to supplement our conception of what it means to be an individual organism by integrating our second—microbial—genome (Grice and Segre 2012). As numerous scientists and philosophers have argued, this integration is particularly relevant when we strive to understand important physiological functions like our immune (Pradeau 2012) or our metabolic/digestive system (Neish 2009; Turnbaugh et al. 2008; O'Malley and Stotz 2011).

While biologists have shown that certain organisms can offload in part, or even completely outsource, their digestive mechanism (see Brune 2014 for a review), recent research on the gut microbiome has further deepened our knowledge of the crucial role it plays in in human digestion. Thanks to groundbreaking work by Jeffrey Gordon and colleagues, we have gathered a better understanding of the central role that our gut microbiota play in energy extraction and fat storage (Bäckhed et al. 2004; Turnbaugh et al. 2006).

If the gut microbiota played no role in regulation, energy extraction, and storage, then an identical diet would make no difference between a normal and a gnotobiotic (germ-free) organism. Bäckhed et al. (2004) tested this hypothesis on mice. Their experiments show that conventionally raised mice gain 42% more body fat than the germ-free mice. Moreover, the introduction of gut microbiota into a germ-free mouse produces a rapid increase (57%) in their total body fat content. There are multiple mechanisms that could explain the higher metabolic rates that are observed in conventionally raised mice as opposed to their microorganism-free counterparts (e.g., microbiota lead to increase hepatic production of triglycerides in the host and promotes their storage in adipocytes).

In the case of human organisms, even though the composition of the gut microbiome and the diet cannot be controlled as closely as in experiments with mice, there is evidence that one's microbiome may contribute to the host energy balance (Ley et al. 2006). The overall conclusion is that we have nutrient sharing relations with our symbionts and that our gut microbiome plays a central role in efficiently processing our food and depositing the energy in fat-storage tissue.

In a 2013 experiment, Ridaura and colleagues colonized genetically identical germ-free mice with uncultured microbiota from four pairs of human twins, where one twin was lean (Ln) and the other one was obese (Ob). The hypothesis was that given an identical diet, mice receiving the gut microbiota from the Ob co-twin would exhibit greater body mass and adiposity. The results confirmed this prediction and also identified—thanks to a co-housing condition—particularly important avenues for intervention.[1] When the Ln and Ob mice shared their cage and—given their coprophagic nature, had access to their own faeces—the co-housed mice *both* stopped the development of adiposity in the Ob mice and altered their gut microbiota to look like the gut profile of the Ln mice.

While the invasion effects and the phenotypic change to a lean condition were diet-specific, these findings demonstrate that gut microbiota play an important role in the extraction and storage of energy from our food intake and that diet-microbiota interactions are modifiable and eventually transmissible. Certainly, the question of translating those findings for humans remains open. Still, anticipatory analyses about such translational work and the attendant ethical implications are squarely within the purview of bioethical inquiry.

This microbial view of the human organism is both preliminary and provocative. Regardless, the kinds of evidence presented here have led theorists to rethink important aspects of the nature of biological individuality and of biological selves (Gilbert, Sapp, and Tauber 2012; Rees, Bosch, and Douglas 2018). While research on the human microbiome and obesity will continue to mature, and while we ought to remain *extremely* cautious about the strength of the causal claims that link probiotics to obesity (e.g., Raoult 2009; Delzenne and Reid 2009), there is a growing consensus that microbiome data significantly improves prediction accuracy for many human traits, including obesity, in comparison to models that use only host genetic and environmental data (Rothschild et al. 2018). Given the recent trajectory of this research, we have every reason to believe that work on the human microbiome will feature more prominently in discussions of obesity and, as such, that bioethicists ought to take heed.

Relationships and Obesity

In the previous section, we argued that an emerging body of research in microbiology suggests that the most immediate elements of our internal biological environment—our microbiome—play a crucial role in obesity that has not yet been adequately appreciated by bioethicists. In this section, we develop a structurally similar argument, but at the social rather than biological level.

When it comes to romantic relationships, is it true that opposites attract? Maybe. But a study by Zajonc et al. (1987) suggests that over time, spouses do come to look more and more like each other. The researchers collected two sets of photographs from twelve married

[1] For obvious reasons, a key variable in early studies on mice involved keeping the two populations separate—no cohousing condition between germ free versus conventionally raised—to rule out the transfer of microbiota through faecal matter.

couples: one set of each spouse taken during their first year of marriage, and a second set taken twenty-five years later. Half of the participants saw the "young" subset and the other half saw the "old" subset, such that no participant judged the same couple's "young" and "old" photographs. Participants then rated the photos in terms of resemblance and likelihood of being married.

In the photographs taken after 25 years of marriage, spouses were rated as significantly more similar and likely to be married. Moreover, in a follow-up study, Zajonc and colleagues report that the more couples were rated as resembling each other, the greater their self-reported happiness, frequency of sharing worries and concerns, and perceived similarity of attitudes.

What could explain these surprising effects? The authors briefly consider and then reject the hypothesis that convergence of appearance is due to a shared diet. We will have more to say about this below. But we think this is illustrative of a larger set of empirical findings that are especially important for obesity research. Namely, that there are *dense patterns of reciprocal interactions within close relationships*. Not only does this lead to convergence in physical appearance, but also to significant concordances in a wide range of health-relevant outcomes.

The literature on health concordance within couples is vast (see Meyler, Stimpson, and Peek 2007 for a review), and so our focus here is limited to obesity-relevant domains. But the consensus in the broader literature is that couples are strongly associated with respect to physical health outcomes, mental health outcomes, and health behaviour.

It is well known that mortality rates are lower for members of married couples compared to unmarried individuals. One popular theory of concordance (e.g., Umberson 1992) suggests that this is due to social control, where spouses monitor and regulate their partner's health behaviours. In a meta-analysis of thirty-five studies containing data from over 8,000 participants, Craddock et al. (2015) found a positive overall relationship between health behaviour and social control ($d = .14$), with a larger effect size estimate ($d = .31$) between health behaviour and *positive* social control (e.g., positive reinforcement, modelling) as opposed to negative social control, such as disapproval and pressing. Similar results were reported for psychological well-being.

Perhaps the most famous result is that when one spouse develops obesity, the likelihood of the other spouse developing obesity increases by 37% (Christakis and Fowler 2007). Indeed, in a meta-analysis of seventy-one papers covering more than 100,000 couples and reporting over 420,000 correlations, Di Castelnuovo et al. (2009) found that the second most strongly correlated factor within couples is body mass index ($r = .23$). On the surface, this might seem unsurprising, given that married and cohabitating couples often purchase food, cook meals, or eat together. But this simplistic observation glosses too quickly over the profound influence close partners exercise over one another in this domain.

In one study, (Markey, Markey, and Gray 2007) researchers asked participants to generate their own thoughts about how their romantic partners influenced their health (rather than using a standardized questionnaire with preselected health categories). The top three most influential domains reported were eating, physical activity, and self-esteem. That is, couples are not only aware of the influence of their partner in health-relevant domains, *but they seemed especially attuned to these influences in the two domains at the centre of obesity research: diet and exercise*.

Homish and Leonard (2008) report similar results: one spouse's behaviour before marriage predicts the other's spouse's behaviour over the course of marriage. Individuals whose partners regularly exercised in the year before marriage were significantly more likely to engage in regular exercise over the first four years of marriage. The same was true for both healthy and unhealthy eating (Bove, Sobal, and Rauschenbach 2003). But it is not the case that the partners just passively converge on their eating habits. Further evidence of dense interdependence has been found in the domain of *changing* health behaviours implicated in obesity. Franks et al. (2012) showed that the desire to make changes to diet and exercise was associated not only with one's own readiness to change but also with partner readiness to make the same change. Thus, wives who indicated readiness to eat a healthier diet and get more exercise were less likely to endorse confidence to change when their husbands reported a lower stage of readiness. Similarly, husbands who indicated readiness were less likely to endorse being confident in their ability to lose weight when their wives were at a lower stage.

Many more studies could be brought to bear which support the concordance between relationship partners in various health domains. Indeed, the research on concordance for obesity in parent–child dyads is equally vast in this regard (see Skouteris et al. 2012 for a review). What ties all of this research together, however, is the widespread recognition that these findings point

toward novel approaches to fighting obesity. Nearly every paper cited in this section concludes with something like the following:

> … research and practice interventions that recognize and maximize the role of the social environment in the adoption and maintenance of health behaviors may be more effective than those that focus solely on individuals alone. (Franks et al. 2012, 330)

It is worth noting here that even the researchers emphasizing the crucial role of environmental factors for obesity still fall into the easy and intuitive framework—individual *versus* social factors. Our claim is that these results, while not definitive, are still suggestive enough (especially when taken in the aggregate) to call into question this intuitive framing of obesity discourses around the individual/social dichotomy. This is, of course, a promissory claim of sorts, whose ultimate value will be assessed on the basis of future theoretical and empirical work. Still, the very nature of much bioethical inquiry is to consider the likely ethical implications of future research. In the next section, we introduce more empirical evidence to show how the conceptual strategy we have been advocating pays off in terms of new clinical intervention strategies.

Probiotics and Obesity: A Possible Way Forward?

Microbiologists have provided us sufficient evidence to show that the manipulation of an organism's microbiome could either provide the organism in question with an additional immune protection (e.g., the use of Janthinobacterium lividum against the amphibian mega-killer Batrachochytrium dendrobatis fungus [Harris et al. 2009]) or with the very possibility of digesting specific foods (e.g., the use of Synergistes jonesii for goats so they can feed on the Leucaena shrub [Jones and Megarrity 1986]). Those studies stood as proof of concept that the deliberate administration, in adequate amounts, of certain microorganisms as a probiotic can boost an organism metabolic and immune functions, and thus, "confer a health benefit to the host" (Hill et al. 2014).

They also anticipated that it would be only a matter of time until medicine would integrate them into its regular practice by thinking of microbes as an effective form of therapy. Some might even—rightly, we think—claim that the centuries-long history of safe use of probiotics (from Metchnikoff's sour milk to the U.S. Food and Drug Administration (FDA) guidelines for live biotherapeutics in 2016) represent the unfolding of that critical vision of the role of probiotics in human health (O'Toole, Marchesi, and Hill 2017).

Certainly, there are many scientific, ethical, and policy-driven questions that still require robust answers in order to properly translate this knowledge into medicine and to develop a probiotic that would not only slow down tissue adiposity but potentially demonstrate a reversing effect as it was shown in mice. However, the development of novel culturing methods, metagenome sequencing techniques, and genome editing tools is bringing the probiotics era significantly closer to our daily reality (O'Toole, Marchesi, and Hill 2017). All these factors combined have created the conditions for numerous microbial biologists to think of obesity differently (from a microbial perspective) and to point out an important fact about human metabolism: the presence of Bacteroides species is correlated with a reduction in body fat (Ridaura et al. 2013) and offers a certain protection against obesity (Turnbaugh et al. 2006).

Data gathered from experiments on mice fed on a high fat diet with supplements of Lactobacillus paracasei shows a decrease in fat mass (Aronsson et al. 2010). Studies including Akkermansia muciniphila, which resides in the mucus layer, have shown that treatment with this bacterium is correlated with improved metabolic functions, especially with reversing fat-mass gain in high-fat diet induced disorders (Everard et al. 2013). Given data gathered from animal models, it makes sense to continue this research on human subjects and see if the administration (via oral delivery or transplant) of certain probiotics would produce specific health benefits, such as body mass loss or control of adiposity.

We must emphasize that the results from human studies are decidedly mixed, though we believe that this should not diminish the importance of our forward-looking bioethical analysis. While the administration of Lactobacillus gasseri (1011 CFU/day) has led to reduced body weight in randomized double-blind interventions ($n = 87$) (Kadooka et al. 2010), in other studies, no significant relationship between this bacteria and reduced adiposity was reported (Sanz et al. 2013). The case of Bacteroides is similar in this regard.

As Ley (2010) reports, in some studies the loss of abundance of this taxon is related to a gaining weight phenotype, while in others the relation is either neutral or negative. There are numerous explanations for these

potential discrepancies. Whether one reads into this the lack of standardization of approaches that characterize the gut microbiota or a difficulty for the microorganisms administered in probiotics to effectively colonize the gut is beyond the scope of this article. We believe that these intriguing findings and the experiments that target the manipulation of our microbiome as a potential solution for obesity converge with our overall argument. More importantly, these studies bring to light the epistemic limits of the easy but misleading dichotomy between individual versus environment. From a microbial perspective, individuality is not cast solely in terms of inherited human genetics, but it also entails that parts of our most immediate biological environment can be so intimately connected to our bodies as to be become part and parcel of our basic physiological functioning (Morar and Skorburg 2018).

One should certainly not get carried away and simply assume that we have found in our microbiome, especially in our metabolic relation with *Bacteroides, Lactobacillus, or Akkermansia*, the silver bullet to solve obesity. That view would merely trade one simplistic solution ("it's all about our individual choices" or "it's all about the environment") for another ("it's all about the presence of specific taxa in the gut"). However, minimizing the role of certain taxa in obesity would surely stand against the evidence that we have presented so far.[2]

Moreover, as Ridaura et al. (2013) have shown, our microbiome contributes to our energy balance and thus could protect us from gaining weight *only* against *specific* diets. This intriguing finding points out the extent to which our fast dichotomies are simply missing something crucial about the important interactions among all levels of biological and social organization (Robert 2017). There is no individual on one side, the microbiome on the other, and somehow an intrapersonal/social environment on top of them. This is why Walker and Parkhill (2013, 1070) note that "future microbiota-based therapies for an obese individual will require an alteration in diet to aid colonization by beneficial microbes." The success of a probiotic solution stands in the symbiotic convergence of reduced caloric consumption, increased fibre intake, and diverse microbial communities that are associated with lean phenotypes.

Dyadic Approaches to Obesity

In section 1.2 we presented evidence from the social psychological literature suggesting that relationship partners exert unique, persistent, and reciprocal influence on one another in obesity-relevant domains such as diet and exercise. Echoing a sentiment commonly expressed in this literature, Jackson, Steptoe, and Wardle (2015, 390) note: "given that partners have a mutual influence on one another's behavior, behavior change interventions could be more effective if they targeted couples as opposed to individuals." While this speculation certainly fits the theoretical framework we are advocating, the questions raised are empirical ones: Do these wider sites of intervention—which are currently obscured by framing in terms of individual versus social environment—actually open up more effective treatments? We provide some evidence that suggests a cautious but affirmative answer.

In a multisite randomized controlled trial involving 357 overweight or obese couples, Gorin et al. (2008) explored whether a weight loss intervention delivered to one spouse might have a "ripple effect" on the untreated spouse. In the treatment condition—an intensive lifestyle intervention (ILI)—one spouse attended group and individual sessions aimed at behavioural skills training. The control also involved just one spouse, but in a treatment-as-usual condition. While untreated spouses did not differ at baseline, at a twelve-month follow-up, untreated spouses in the ILI condition lost significantly more weight than untreated spouses in the control condition. In other words, the effects from the lifestyle intervention had a ripple effect on the untreated spouse. Similarly, more ILI spouses lost greater than 5% of total body weight and more stayed at or below their baseline than did spouses in the control condition. These results led the authors to conclude:

> … by assessing only individual participants, the existing literature on behavioral weight loss treatment may have underestimated the reach and cost effectiveness of these interventions … treating one spouse can have a beneficial, and clinically

[2] In fact one could argue that, in their search for mechanistic explanations, the early studies on probiotics and obesity have downplayed the complexity of ecological relations between host and microbiome and between microbe and microbe as if there is a universal probiotic that shields us from gaining weight. A more realistic approach would not assume that bacteria in our gut exist in a vacuum, but that our metabolic responses are a function of community composition and of the relative abundances among various taxa (Ley et al. 2006).

> significant, impact on the weight of the untreated spouse. This benefit was achieved without any additional cost to treatment providers. (Gorin et al. 2008, 1683)

A number of related studies (Golan et al. 2010; Schierberl Scherr, McClure Brenchley, and Gorin 2013; Cornelius, Gettens, and Gorin 2016; Gorin et al. 2017, 2018) have found further evidence of this "ripple effect" whereby treating one spouse yields a significant outcome for the untreated spouse. For example, Golan et al. (2010) find that at a six-month follow-up after treatment, spouses whose partners were treated lost significantly more weight than spouses whose partner was not treated.

In perhaps the most impressive study in this vein, Gorin et al. (2018) replicate these "ripple effects" in a randomized controlled trial involving 130 couples. Participants were randomized to either a Weight Watchers condition (a widely available weight-loss programme) or a self-guided control condition. At a three-month follow-up, as expected, participants in the Weight Watchers condition lost significantly more weight than the self-guided control condition, but at a six-month follow-up there was no difference between conditions. Surprisingly, *across both treatment and control conditions, untreated spouses lost weight at three months*. This suggests that ripple effects are present across more and less structured approaches to weight loss.

The authors point out that current guidelines for managing obesity (Jensen et al. 2014) suggest 3% weight loss to achieve measurable health benefits. Across both treatment and control conditions, *this criterion was met by 32% of untreated spouses*. Thus, through the various pathways present in close relationships, the effects of an intervention on one partner can be propagated to the other. Gorin et al. (2018, 503) thus conclude that their results add to the growing literature suggesting that treatments should "effectively harness household and social dynamics to promote clinically significant weight loss that could improve the reach and cost effectiveness of weight management programs."

To be sure, many of these effects are small, and there are well-founded worries about self-selection biases in the literature (e.g., highly supportive relationship partners consent to participate in the studies). As is the case with most weight loss interventions, these behavioural interventions do not necessarily achieve long-term weight loss or maintenance. Still, we are encouraged by the fact that similar effects have been observed for parent–child dyads, where an obesity intervention targeting the parent can lead to weight loss in the untreated child (e.g., Boutelle et al. 2017). Similar results have also been reported in the literature on bariatric surgery, such that family members of patients undergoing gastric bypass surgery report weight loss and improved healthy eating (Woodard et al. 2011).

Future empirical research will be needed to establish the reliability and generalizability of these effects. For our purposes, however, this body of work does establish the conceptual and clinical limitations of thinking of individuals apart from their most immediate social environment.

Again, rather than a silver bullet, we think of these findings as providing new set of (perhaps underutilized) tools to the kit for fighting obesity. Moreover, many of the strategies described here suggest that these new sites of intervention can be more cost-effective while also producing clinically significant results.

We contend that these kinds of studies have not received sufficient attention in the bioethics literature, at least in part because they sit uneasily in the individual versus environment dichotomy which has set the terms for the debates. The intervention strategies described throughout this section, however, do have important conceptual implications for these long-standing debates in the bioethics literature, and we now return to a discussion of them.

Potential Objections

So far, we have claimed that recent conversations about obesity are dominated by a series of fast dichotomies such as help its versus can't help its or individual responsibility versus social determinants of health. But is this representative of the obesity literature? We think so. Some scholars defend the individualistic framework since they believe that we have real choice about what we eat and whether we exercise (Spike 2018; Anderson-Shaw 2018). Other socially-minded researchers place almost all the emphasis on the obesogenic society in which we live, up to the point of calling into question our very choice for lean phenotypes (e.g., Maginot and Rhee 2018; Humbyrd 2018).

In a recent *Lancet* series, Roberto et al. (2015, 2400) pointed out that, "although obesity is acknowledged as a complex issue, many debates about its causes and solutions are based on overly simplistic dichotomies." Kleinert

and Horton (2015, 2327) similarly note that the obesity debate is "becoming increasingly polarized with false and unhelpful dichotomies." They list examples such as personal versus collective responsibilities for obesity, individual versus structural causes of obesity, blaming individuals versus obesogenic society, obesity as disease versus new normal, lack of activity versus overconsumption, intervening on individual behaviours versus industry practices—and the list could go on. While there are surely exceptions to this rule, and while these dichotomies operate differently in different contexts, we agree with Roberto et al. (2015) in their overall assessment that the obesity literature (broadly construed at the intersection of bioethics, public health, public policy, etc.) is too often framed in terms of (various versions of) an individual/environment dichotomy.

Still, we recognize that in subsuming the dichotomies listed above under a general "individual/environment" heading, we run the risk of conflating distinct concepts in the literature (e.g., blame, responsibility, cause, intervention) operating in different registers (e.g., biology, psychology, public health, public policy). There are at least three reasons, however, that the potential payoffs outweigh this risk.

First, in light of the characterizations offered by numerous scholars (e.g., Callahan 2013a, b; Roberto et al. 2015; Kleinert and Horton 2015), it seems fair to suggest that various instantiations of the individual/environment dichotomy have figured prominently in theorizing about obesity. Moreover, popular discourse surrounding obesity further entrenches the dichotomy (e.g., Lawrence 2004; Greener, Douglas, and van Teijlingen 2010).

Second, we are confident that we can mitigate the risk of conflation by limiting the scope of our claims, since the evidence we have presented speaks to certain features of the biological and social aspects of the individual/environment interface. It would be most welcome if others find the evidence and argumentative structure applicable to different domains, but our arguments our aimed at showing how blurring the boundaries between individuals and the most immediate features of their biological and social environments can reveal promising new approaches to fighting obesity.

Third, because we draw from diverse theoretical and empirical sources, some conceptual slippage is inevitable. But insofar as bioethicists are committed—as we think they should be—to integrating multilevel, interdisciplinary perspectives into their inquiries, we think this is a feature, not a bug.

Finding a Middle Ground?

Another key benefit of the research programmes we introduced above is that they do not fit neatly into the dichotomies—e.g., Callahan's "help its" and "can't help its"—which have tended to characterize debates about obesity in bioethics. And, *in virtue of this*, they may offer a new way forward. The simplest version of our claim is that there is more room than is commonly supposed between the individual responsibility theorists and social determinants theorists.

In order to develop this claim, we first want to emphasize as strongly as possible that attempting to stake out this middle ground by no means entails ignoring or downplaying the insights from the individual and social poles in the debate. Much ink has already been spilled about the ways in which a myopic focus on individual responsibility in the obesity debates is not only inadequate, but harmful and counterproductive (see e.g., Reiheld 2015; Mayes 2015). Indeed, we endorse many of these arguments.

But we also worry that these theorists sometimes employ polarizing language which too quickly dismisses individual responsibility theorists and obscures possible avenues of compromise and collaboration. To take just two examples, Reiheld (2015, 246) argues that obesity campaigns "must target structural factors rather than focusing on individuals," implying that the two may be mutually exclusive. Brownell et al. (2010) antagonistically evoke caricatures of "totalitarianism" and the "nanny state" to describe their opponents' objections.

In pointing this out, our goal is decidedly *not* to underplay the stigmatizing effect that numerous obesity prevention campaigns or advertising clips have had in targeting certain populations by deploying negative emotions towards their eating habits (Eller 2014; Abu-Odeh 2014; Kukla 2018; Kelly and Morar 2018). Nor do we mean to distract ourselves from the important social inequalities and injustices that play a significant role in the obesity discourse and often undergird policy decisions that are not appropriately responding to endemic issues of poverty and access to certain public primary goods such as transportation and healthcare (Hoffman 2010; Guthman 2011). Nor do we intend to paper over the ways in which, as feminist philosophers have pointed out, discourses around dieting and exercise often perpetuate forms of gender oppression (e.g., Bordo 1993). We want to keep all of this in mind while also highlighting that dichotomizing tendencies may

risk erasing both new sites of clinical intervention and shared conceptual resources which do not fit neatly within the individual versus environment framing. Our proposed middle-ground solutions are entirely consistent with rejecting stigmatizing, marginalizing, and other harmful language about obesity.[3]

We contend, then, that the evidence presented in Sects. 1 and 2 undermines these fast dichotomies between individual choice and social environment and suggests a more nuanced approach to the debate. Synthesizing this evidence through two further examples below, we show how the distinctions between "individual" and "environmental" processes are cross-cutting.

First, consider the ways in which we understood our metabolism and its relationship to obesity before we were able to characterize the significant role of our microbiome in digestion. As O'Malley and Stotz (2011) rightly show, obese phenotypes, even when they were correlated with "obesogenic environments" (Chadwick and O'Connor 2014), would still be primarily explained *either* in terms of weight-gaining genetic predispositions that were supposedly highly heritable (e.g., thrifty gene hypothesis, Neel 1962) *or* in terms of epigenetics and developmental factors that would emphasize prenatal and poor childhood conditions that would later in life trigger certain metabolic processes leading to obesity (Diamond 2004).

These styles of scientific explanation certainly have contributed to a more complex understanding of obesity, but at the same time, they reinforce the very dichotomy that has led us to the present conceptual stalemate. Not only have these explanations focused on a narrow conception of the organism, but they have also parsed out the influence to obese phenotypes *only* in terms of individual dispositions versus environmental factors. A view of obesity that meaningfully considers our microbiome brings an important adjustment to those accounts of obesity by providing us with an *intermediate* level of genetic influence that is neither fixed within the limits of our human genome nor completely extended to the whole environmental metagenome.

[3] In addition to the fact that social and political aspects of obesity are consistent with research on the human microbiome, we also suggest that when our proposals are presented in tandem, they provide an even stronger sense of how experiences of injustice can *truly* get under one's skin and profoundly alter biological processes. Hertzman and Boyce (2010, 330) call this "biological embedding," and one's microbiome is a privileged site to witness the causal effects of socioeconomic factors on one's own biology.

The human microbiome demands a more nuanced discussion of the role of genetics and obesity, and it also makes us aware of the profound impacts that our eating habits have on our own bodies and *on our own genetics*. Perhaps the paradigmatic case of a "can't help its" argument is that genetics predispose some toward obesity. But if we fully take into consideration the role that our microbiome plays in our digestive system, we see that certain diets, especially those with high levels of saturated fats, will not only make it harder for individuals to avoid obesity but—as we know now—have a selective impact on our gut microbiota, which will become even more efficient at extracting energy from such a food intake (Turnbaugh et al. 2009).

There is thus a sense in which certain diets tune our microbiome, our second genome, towards obesity, but this genetic aspect may not be entirely beyond our control. When we are eating, as Fishbach and Sonnenburg (2011) claim, we are always eating for two, given that our metabolism constantly sets up important interspecies interactions in our gut. This renewed attention to the role of symbiotic relations provides us with a novel way of thinking about obesity and with an opportunity to move beyond the "either/or" tendencies of this conversation. It is in this *middle ground*, between genes and environment (indeed, even gene by environment interactions), where we believe significant progress can be made in bioethics and (perhaps) in health policy.

A similar strategy applies to our most immediate social environment. We agree with many of Callahan's (2013a) critics that there are strong reasons to reject social stigma as an approach to obesity prevention. However, the evidence presented in Sects. 1.2 and 2.2, in conjunction with a growing body of research in social psychology concerning *transactive goal dynamics* (e.g., Finkel, Fitzsimmons, and van Dellen, forthcoming; Fitzsimmons, Finkel, and van Dellen 2015; Fitzsimons and Finkel 2015), offers an alternative way to conceive of social influence in the context of obesity that does not lead to victim-blaming or stigmatization.

For example, Fitzsimmons, Finkel, and van Dellen (2015) detail the pathways and mechanisms through which social influence (especially within close relationships) can undermine *or* promote goal pursuit in areas such as diet and exercise (indeed many of their examples are in precisely these domains). The key insight is that relationship partners exert "such a great deal of mutual influence in each other's goals, pursuits, and outcomes

that the partners' self-regulatory systems become inextricably linked, part of a complex and messy web of interdependence," and this, in turn leads to the suggestion that "relationship partners are best conceptualized not as mostly independent goal pursuers who occasionally influence each other, but instead, as *interdependent subparts of one self-regulating system*" (Fitzsimmons, Finkel, and van Dellen 2015, 648, emphasis added).

Here, paradigmatically social relationships have an individualizing element, such that conceptualizing the dyad as a unit yields novel predictive and explanatory resources for central research questions in the obesity literature. The importance of self-regulation and goals is clear in this regard. But it is also worth noting that this dyad-as-unit strategy has shown promise in other relationship contexts, such as family-based interventions for obesity and parent–child dyads (e.g., Boutelle et al. 2017; Best et al. 2016; Loveman et al. 2015). Similar dyad-level effects have also been observed in other related domains, including diabetes and blood glucose monitoring (Anderson et al. 1997; Berg et al. 2013), sleep apnea (Baron et al. 2011), arthritis (Hemphill et al. 2016), and pain management (Wilson, Martire, and Sliwinski 2017). Moreover, our account provides novel resources in support of an emerging trend in the bioethics literature which acknowledges the social character of eating (e.g., Kukla 2018; Stohr 2018).

We suggest that these strategies—which foreground the dense, reciprocal interactions between individuals and their biological and social environments—could perhaps provide a detente between two long-entrenched camps in the obesity debates: individual choice (roughly, Callahan's "can help its") versus environmental/social drivers of obesity (roughly, Callahan's "can't help its"). We think there is more room for common ground than is often supposed between these poles.

On the account we are advancing, aspects of individual choice (such as food decisions and exercise) can be acknowledged while still granting a profound level of environmental influence (from microbes and relationship partners) that does not necessarily extend into notoriously unwieldy (but no less important) territory of social, economic, and political structures (Morar and Skorburg 2018).

The examples of metabolism and transactive goals offer resources to question the received framework in the obesity debates in bioethics, and they push us to complicate the picture by integrating truly multilevel analyses. Taking these results seriously provides an avenue through which bioethicists can make good on promises of being truly interactionist and comprehensive and not merely falling back on either individualism or social determinism when it is suitable for their conclusions. If there is any solution to the problem of obesity, it will most likely stem from the synergistic efforts that would capture the organic, psychological, social, and political complexities and injustices that underlie this phenomenon.

Conclusion

We think there is reason to be optimistic. In a recent review, Barnhill and Doggett (2018, 7) note the growing consensus that:

> Obesity needs to be seen not (just) as the result of individual choices, but as a response to a food environment pervaded with processed foods that are high in sugar, fat, sodium, and calories, that are cheap, that are aggressively marketed as desirable, that are designed to be hard to resist, and that do in fact undermine our self-regulatory capacities.

We agree. And in this paper, we have tried to make a novel contribution by introducing recent research in microbiology and social psychology. These research programmes make salient new sites of intervention and also suggest a fertile middle ground—comprised of our most immediate biological and social environment—which could help to undermine the dichotomous thinking which all too often obstructs the comprehensive, interactionist, multilevel approaches that are so badly needed to fight obesity.

These approaches, in turn, raise new medical, moral, legal, and political issues. For example, our discussion not only sounds the alarm—once again—against the worrisome overuse of antibiotics in our current medical practices (Blaser 2014) but also raises questions about the ethical significance of microbiome research with respect to ownership, privacy, human subject research, and biobanking (see Rhodes, Gligorov, and Schwab 2013).

Moreover, if features of our biological and social environments are indeed integral for crucial human functions, then there is a sense in which they are a part of our healthy functioning and more importantly, of *who we are*. Might microbial therapies pose threats to our

self-identity and to our sense of agency? If the dyad-level interventions discussed here turn out to be a particularly promising avenue of intervention, could certain constraints on one partner be justified to promote the health of the other?

The research we have introduced here invites us to reconsider many traditional conceptions of human health, human agency, and ultimately, human nature, as well as the normative roles that these conceptions have played in bioethics. It is our hope that the arguments presented in this paper can motivate a new, middle-path approach to debates about obesity in bioethics which will begin to address these and related questions.

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