Advantages and Limitations of NHANES-Dataset Driven Childhood Obesity Guidelines: an Interdisciplinary Case Study

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Abstract

Significant time, energy, and money is spent towards addressing childhood obesity, yet there is hardly scientific consensus surrounding both species-level causes of obesity, and individual factors that lead to the development of obesity. In the face of the controversial new American Academy of Pediatrics (AAP) guidelines from early 2023 that now recommend surgery and medication for certain groups of obese children, I propose that an interdisciplinary synthesis of current obesity research and assessment of the AAP's research practices can offer a meaningful critique on whether or not such drastic, individualistic measures are warranted based on controversial measures of excess adiposity like BMI. An analysis of the history of obesity shows that policy often does not follow scientific consensus. I then present a synthesis of various schools of thought on obesity, showing how the issue largely lies outside the control of the individual. Finally, I propose investigating the role of the NHANES database in the AAP clinical guideline formulation, and find that applying an interdisciplinary scope of analysis demonstrates how such epidemiological databases are being used to bolster clinical guideline formulation that still focuses on poorly understood correlations between BMI and cardiometabolic outcomes, despite the proclaimed focus on the "whole child" and community in the guidelines–suggesting a need for updated research practices to truly incorporate a more holistic, community based approach to childhood obesity guidelines.

I. What is obesity?

In early 2023, the American Academy of Pediatrics released "the first edition of the American Academy of Pediatrics clinical practice guideline for evaluation and management of children and adolescents with overweight and obesity", in which the academy recommends major changes to the treatment of what they deem to be an "epidemic" (Hampl et al., 2023, Introduction and sec VII, A). Most notably, obesity screening for

children now begins at age 2 instead of age 6, and adolescents age 12 and up can be recommended for both medication to manage obesity, as well as bariatric surgery (Morrill, 2023). Previous guidance adopted a "wait and watch" approach, and the agency plans to release further information related to prevention, but this has not yet been made available to the public (Curley, 2023; Rodriguez, 2023). The guidelines have inspired significant backlash from those who say that the new guidelines are excessively stigmatizing and can lead to disordered eating concerns in a pediatric population (Sole-Smith, 2023). Given the stark decrease in age at which children are recommended to be screened, "some argue AAP guidance for children as young as 2 years old may ruin their relationship with food and exercise without giving them a chance to develop intuitive eating skills" (Rodriguez, 2023). Additional concerns include the lack of long term data on weight loss medications and bariatric surgery, especially in the pediatric population, given that both are major medical interventions and carry the risk of significant side effects and complications (Rodriguez, 2023).

While the guidelines have inspired backlash for their extremism, this is only the latest example in the clash of politics, policy, and science with regards to obesity. Obesity is defined by the CDC as a "common and serious [...] chronic disease" ("Overweight & Obesity," 2022). It is also claimed on the CDC website that obesity "can lead to type 2 diabetes, heart disease, and some cancers" ("About Overweight & Obesity," 2023). However, obesity was not always defined as a disease by major medical associations. In 2013, the American Medical Association (AMA) House of Delegates voted on whether or not to reclassify obesity from a "complex disorder" to a disease (Fryhofer, 2013; Pollack, 2013). The AMA's Council on Science and Public Health had studied the issue for a year prior, and recommended *against* this change for a host of reasons (2013). Given this dissonance of scientific opinion, one may question whether the semantic difference of being classified as a disease or not matters?

One of the primary hesitancies of the council in declaring obesity as a disease was the nebulous nature of "disease" itself–an interesting comparison can be drawn between obesity and other "diseases" that are diagnosed based on a measured criterion alone, such as hypertension or hyperglycemia. In obesity, there is currently no solid set of symptoms or criteria for diagnosis beyond BMI. High BMI does not always predict metabolic syndrome, which is arguably more of a defined disease state/a cluster of diseases including hypertension, high triglycerides, etc. that predict risk of cardiovascular disease, stroke, and more. This phenomenon is sometimes called the obesity paradox ("National Heart, Lung, and Blood Institute," 2022).

While "disease" may be poorly defined, this semantic difference has implications for how society views obesity, how we invest time and money into the effort, what the target of anti-obesity interventions are (ie. body size vs healthy behaviors), and for the stigma that fat people

experience. The same AMA report indicates that while disease classification could bring about potential positive increases in awareness, "urgency," and may pressure the FDA to move forward on approving drugs for treating obesity, there are also potential negative consequences, such as an overemphasis on body size and modifying body size or BMI through medication and surgery instead of focusing on healthy behaviors (2013). In modern society, disease is viewed as an undesirable state that requires cure, which is somewhat at odds with the growing body of literature that body size may not be as mutable as we think it is, and importantly, it may not be equally mutable in all individuals.

Governments and private agencies are allocating significant time and resources to childhood obesity and obesity in general. Despite this, many obesity prevention programs for children and adolescents show modest or no results (Stice et al., 2006). Given the significant backlash to the AAP guidelines specifically focused on medical and surgical interventions for children, it is worth investigating whether or not the research in this area surrounding correlations between BMI and negative cardiometabolic outcomes is robust enough to support such drastic interventions as medication and surgery? Using an interdisciplinary analysis to look at a primary dataset used for decision making (NHANES), I will argue that the nature of most obesity research may not support such measures in place of more holistic prevention measures, especially because research has shown that BMI may only be a mediating variable for other fundamental causes of metabolic dysregulation in adolescents, and that analysis of NHANES data that controls for confounding variables shows the association between BMI and comorbid conditions isn't clear cut. I will also show that additional lenses of analysis on current data and expanding data collection to include variables such as weight stigma can begin to provide a more complete picture of the complex relationship between BMI and comorbid conditions. This is particularly relevant given that the AAP guidelines state that BMI is the widely accepted screening and diagnostic tool of choice in pediatric populations, despite their own admission of its limitations in certain groups (Hampl et al. 2023, sec. VII, A). This work highlights the urgent need for interdisciplinary research and assessment of current research practices and the translation of research to policy decisions, to promote sustainable and impactful solutions.

II. Interdisciplinary inquiry into causes of obesity

An interdisciplinary approach looking at the potential *causes* of obesity can help shed some light on the locus of control of body size, and therefore help inform where prevention efforts should be centered.

Genetics and epigenetics have long tried to disentangle whether there is a specific genetic component to obesity. While no "obesity gene" has been unearthed in humans, twin studies have found that "genetic variants contribute to [anywhere between] 45-80% of BMI variation" (Symonds et

al., 2013, p. 90). Studies on twins raised apart showed that genetics seemed to play a larger role in determining body size than the eating and lifestyle promoted by either set of parents (Mann, 2015, pp. 17-31). Advances in genetic science such as the genome wide association study (GWAS) that looks at the genome sequences of a large population at the level of single nucleotide polymorphisms (SNPs), or single base pair differences among people, haven't yielded much with regards to association to BMI. GWAS studies have found SNPs that explain only up to 5% of body variation between people (Symonds et al., 2013; Mahmoud, 2022, Introduction). One emerging area of studying is epigenetics, or non-coding changes to DNA that impacts regulation of gene expression, and could help explain the heritable component of body size. There is evidence that increased methylation of certain genes associated with metabolism such as leptin and insulin can negatively influence body weight (Mahmoud, 2022, sec 2.1). There is also evidence that environmental exposures, such as to phthalates (added to many consumer products to make plastics more durable) can alter the methylation of several genes that impact metabolism ("Phthalates Factsheet," 2021; Mahmoud, 2022, sec 3.1). Further, observational studies in humans and experimental studies in animals show that epigenetic changes due to traumatic events such as famine during a critical window during pregnancy can be passed down to children, which can lead to increased risk of obesity (King & Skinner, 2020).

As the genetic perspective supports, there is evidence for heritable variation among body size, which raises the question about how evolutionary processes may be playing a role in larger body size. Leading evolutionary theories about the causes of obesity include a mismatch between the environment that we are most adapted for, given our hunter-gatherer ancestors were much more active and ate a less processed diet, and the modern day obesogenic environment where sedentary lifestyles and processed foods predominate (Power, 2012). Other hypotheses that have been advanced include the popular thrifty gene (given periods of famine in the past, those who could store more fat would theoretically be able to survive and reproduce more effectively) (Speakman, 2013). A number of oppositions have been raised against the thrifty gene hypothesis, notably that if this holds true, then all individuals in the modern environment should theoretically be obese (Speakman, 2013). Hypotheses that hold more weight include obesity as a byproduct of another gene that underwent recent positive selection, or as a set of genes that have been subject to genetic drift–explaining why some people may be prone to obesity whereas others are not (Speakman, 2013).

Other physiological/metabolic research on weight loss and metabolism suggests there may be strong safeguards to preventing drastic changes in body size. Set point theory argues that the body has a range of fat tissue content that it maintains, and attempts to lose weight, such as through dieting, will be resisted through changes in the hormonal profile.

Indeed, studies support long-term changes to hormonal profile even one year after weight loss–including a decrease in satiety-associated hormones and an increase in hunger associated hormones (Sumithran et al., 2011, Results). Several factors during pregnancy and early life can impact long-term physiological functioning. There is an association between maternal obesity and maternal gestational diabetes and obesity (Hampl et al., 2023, sec. VIII, D.1.c). Additional associations under investigation include antibiotic use early in life, as this can disrupt the gut microbiome, which is thought to play an essential role in regulating our metabolism (Hampl et al., 2023, sec. VIII, D3).

Psychological factors can also impact production of the stress hormone cortisol, which in turn is thought to promote eating and weight gain. Experiencing stigma is a large driver of increased cortisol, which includes weight stigma (to be discussed in depth later). Beyond such biological drivers, there is an important role of social determinants of health in determining who becomes obese. There is some association between socioeconomic status and obesity, which follows logically from the understanding that those living in high-stress environments due to structural discrimination may have less ability to make ideal lifestyle choices and may lack access to medical care (Ogden et al., 2010; Mayor, 2017).

These perspectives highlight the limited ability of individuals to change their body size. Given large structural changes to the environment and the lack of resources that many have to deal with these changes, as well as physiologic regulatory systems that resist body size changes, potential epigenetic and genetic links, it appears that the locus of control of the body size lies largely outside the individual.

III. How is obesity measured? Brief introduction to NHANES

In the United States, obesity is primarily measured through body mass index, or the ratio of an individual's weight to height squared. BMI is endorsed in the AAP guidelines not only as the diagnostic and screening tool of choice for pediatricians due to the low cost and ease of implementation, but also because it can be used to "evaluate the success or impact of interventions" (Hampl et al. 2023, sec. VII, A). Statistics on obesity in the United States on the CDC website come from data obtained from the National Health and Nutrition Examination Survey (NHANES), a national database that surveys individuals of all ages on nutritional and health status ("About the National Health and Nutrition Examination Survey," 2022). The cross-sectional study methodology includes a physical assessment of various characteristics, such as weight and height, as well as collection of samples for biomarker analysis. Home interviews of participants are also collected in order to characterize their health habits, diet, and environmental risk factors. The NHANES program began in the 1960s, but "in 1999, the survey became a yearly program that visits

15 counties across the country, and samples 5,000 individuals ("About the National Health and Nutrition Examination Survey," 2022, Overview).

NHANES data are used by the CDC to "determine prevalence of major diseases and risk factors for disease," and to come up with standards for various physiological measurements like BMI. Because NHANES data are widely used in research due to their public availability and sound methodology, given their ability to influence significant health policy, it is vital to understand the appropriate scope of analysis that can be conducted with such data. For example, the NHANES data released every two years are a cross-sectional dataset, and because this survey does not follow the same people through time, nor does it visit the same counties from year to year, it is not possible to draw causal relationships between X variable and Y health outcome, though correlations between variables throughout years can certainly be explored (Lee, 2021).

Numerous researchers working with NHANES have also noted the blunt nature of the categories which often make it difficult to capture the full nuance of a topic. According to Gabrielle Benoit, the interview protocol asks the interviewer to note down what they think the interviewee's gender is–which hides the distinction between gender/sex and also limits the entire spectrum of sexual and gender diversity to interviewer discretion (personal communication, March 7, 2023). Dr. Elizabeth Miller has also spoken on her work disentangling the role of race and low birth weight using NHANES–she notes that racial/ethnic categorizations are limited to: white, Black, Hispanic, and other–for many years there was not an option for Asian American (Ocobock & Lynn, 2023). Thus it is difficult to truly control for and account for racial/ethnic factors with such blunt categorization, and such limitations must be kept in mind during policymaking.

IV. The new AAP guidelines and validity of weight loss research

As discussed above, the hope of the medical establishment is that classifying obesity as a "disease" reduces stigma by deemphasizing the role of personal choice. Despite this change, "a study found about two-thirds of Americans report experiencing weight stigma from doctors" (Rodriguez, 2023). Notably, the AAP guidelines spend ample time discussing obesity as a "complex" disease with "genetic, physiologic, socioeconomic, and environmental contributors" that move beyond the realm of "personal choice" (Hampl et al., 2023, sec I). There is extensive discussion on social determinants of health that contribute to obesity, including:

Policy Factors	Neighborhood and community factors	Family and home environment factors	Individual factors
1. Marketing of unhealthy foods 2. Under-resourced communities 3. Food insecurity	1. School environment 2. Lack of fresh food access 3. Fast food proximity 4. Access to safe physical activity 5. Environmental health	1. Parenting feeding style 2. Sugar-sweetened beverages 3. Portion sizes 4. Snacking behavior 5. Dining out and family meals 6. Screen time 7. Sedentary behavior 8. Sleep duration 9. Environmental smoke exposure 10. Psychosocial stress 11. Adverse childhood experiences	1. Genetic factors ● Monogenetic syndromes and polygenetic effects ● Epigenetic effects 2. Prenatal risk ● Parental obesity ● Maternal weight gain ● Gestational diabetes ● Maternal smoking 3. Postnatal risk ● Birth weight ● Early breastfeeding cessation and formula feeding ● Rapid weight gain during infancy and early childhood ● Early use of antibiotics 4. Childhood risk ● Endocrine disorders ● Children and youth with special health care needs ● Children with autism spectrum disorder ● Children with developmental and physical disabilities ● Children with myelomeningocele ● Attention-deficit/hyperactivity disorder ● Weight-promoting appetitive traits ● Medication use (weight-promoting medications) ● Depression

FIGURE 1: Chart adapted from Hampl et al., 2023, sec. VIII, B (Table 1)

It is important to distinguish individual factors (things that may impact an individual and vary from person to person within a community) from an individual locus of control, which would indicate that an individual would be able to affect meaningful change in that realm if they so choose. What is striking about the above list is that very few, if any, of the factors listed would be within a child's individual locus of control. Perhaps older children may exercise some level of control over their screen time or diet, but even these are mediated by complex processes, including family environment, access to resources, socialization, and more. None of these factors stand alone in their contribution to children's body size, and very few of them are mutable by an individual child themselves. Much of the critique surrounding these new policies centers around the fact that while the AAP themselves states that these new clinical guidelines take into account the holistic nature of the disease, the guidelines themselves still focus on measuring obesity through imperfect measures such as BMI and employing individualistic, medical interventions.

An interdisciplinary perspective begets the understanding that the locus of control of body size does not lie within the individual. It seems that AAP guidelines understand this assertion to some degree, given their above synthesis of evidence from various fields. An important question to ask then becomes, given the AAP's understanding of these dynamic and complex processes that determine body size, are the so-called risks of overweight/obesity great enough to warrant sometimes irreversible intervention, like surgery? Is the risk to the individual *so great* that pharmaceutical intervention must be employed for children? While the AAP plans to speak separately on prevention, such information has not yet been released and the lag of timing between releases speaks to potential priority differences between treatment and prevention.

Though not the focus of this thesis, it is of note that the AAP claims that numerous studies, including RCTs, show that "despite the complex nature of this disease, obesity treatment can be successful" (Hampl et al., 2023, sec I). One of the cited papers for this assertion found that "there was no direct evidence on the benefits or harms of screening children and adolescents for excess weight" (O'Connor et al., 2017, p. 2427). Furthermore, while some of the "lifestyle-based" interventions with a certain amount of patient contact showed some modest reduction in BMI, medication based treatment (such as with metformin) showed "small or no benefit for cardiometabolic outcomes" leading the authors to conclude that "the clinical significance of the small benefit of medication use is unclear" (O'Connor et al., 2017, p. 2427). The AAP guidelines use correlations between higher BMI and indicators of metabolic health as a significant justification for why more aggressive treatment must be pursued for obesity, yet one of the studies they cite to claim that obesity treatment is

successful demerits their own claim about the necessity of medication in such a treatment regimen.

Further, data on whether long-term weight loss is sustainable is ambivalent at best. Hunger et al. found that RCTs with randomly assigned diet/non-diet groups show no significant difference in weight (2020, p. 79). Similar studies cast doubt on the role of long-term (12 month) exercise interventions in significant weight loss (Hunger et al., 2020, p.79). This is not to say that health *promoting* behaviors are not worth pursuing–but simply that weight loss as a goal may not be sustainable. An analysis of observational weight loss studies done by Mann et al. also demonstrates that "the follow-up rates in these studies were quite low (see Table 1). Overall, 33% of the original participants in these diet studies returned for their long-term follow-up. [...] It is generally believed that low follow-up rates bias the results of diet studies, making the diets appear to be more effective than they were, because individuals who gain back large amounts of their weight are particularly unlikely to show up for follow-up tests" (2007). Additional longitudinal studies (including rigorous twin studies spanning up to 15 years) analyzed by Mann and colleagues showed that dieting actually was a significant predictor of weight *gain* (2007). While diets do initially lead to weight loss, the long term effects of repeated attempts to lose weight (and the inevitable increase following weight loss), termed "weight cycling" can lead to cardiometabolic dysfunction, and increase the risk for disorders such as type 2 diabetes and obesity (Rhee, 2017, p. 239).

Hunger and colleagues also introduce evidence from various studies that suggest that body size may be a mediating variable, not the *cause* of ill health. They cite work on: liposuction failing to drastically improve cardiometabolic biomarker status, obesity as a possible *side effect* of metabolic disease/dysfunction, studies that control for sedentary behavior that erase the effect of BMI in predicting mortality, and more (2020). This thesis does not attempt to resolve the long standing debate over whether or not weight loss is possible and sustainable, but simply calls attention to the conclusions that the AAP uses to drive policy, and point to the work being done to ensure that conclusions being drawn about weight and health are methodologically sound. Doing so may require us to question what seems like incontrovertible truths in the realm of weight science.

The AAP paper is both a dossier on policy recommendations and current obesity science, but also a research endeavor. The taskforce that contributed to the paper had two main goals in mind: "What are effective clinically based treatments for pediatric obesity? What is the risk of comorbidities among children with obesity?" (Hampl et al., 2023, sec IV, B). The conclusions from this research in turn guided the updated policy recommendations. Given the conflicting evidence both on the validity of weight loss as a sustainable and achievable goal and the apparent mismatch between the preponderance of evidence from an interdisciplinary perspective suggesting that control over body size is

limited and the recommendation of a stricter individual approach (medication and surgery)–it is vital to examine the assumptions present in goal two regarding comorbidities. I propose a follow up question to this: does the data on BMI association and comorbidities still strongly support individualistic measures like surgery if confounding variables or other lenses of analysis are applied to the same data to add nuance?

V. NHANES analysis and case study within AAP guidelines

To do this, I propose looking specifically at the use of NHANES in this decision-making process. While NHANES was not the only dataset consulted in the AAP's research, it provides data that official United States agencies use for understanding obesity trends (as discussed above), and it is additionally methodologically rigorous, spans a large period of time, and collects a significant breadth of information that lends itself to research and analysis. The nature of the data also makes it a popular choice to look at associations between BMI and other variables. The AAP also concurrently released a paper entitled "Appraisal of Clinical Care Practices for Child Obesity Treatment. Part II: Comorbidities," which provides a complete list of studies and analyses included in their attempt to answer the question about associations between BMI and comorbidities (Skinner et al., 2023).

My first aim was to investigate to what extent NHANES data is used in included studies in this paper:

Studies included in their analysis are presented by category of comorbidity. Out of 21 total categories (ie. dyslipidemia, HDL, LDL, depression), NHANES data is present in studies in 12 categories. Looking further into NHANES methodology shows that there is no data collected relating to: HOMA-IR, alanine aminotransferase, aspartate aminotransferase, and non-alcoholic fatty liver disease, which excludes 4 categories.

Comorbidity categories for which NHANES data is present–17
Categories for which NHANES data is used in included studies–12

Category Name	**Total studies**	**USA studies**	**How many used NHANES data**	**% of USA studies using NHANES**
Abnormal HDL	n = 39	21	7	33.3
Abnormal triglycerides	n = 38	21	6	28.6

Abnormal LDL	n = 26	14	1	7.1
Abnormal total cholesterol	n = 23	14	3	21.4
Dyslipidemia	n = 6	4	1	25
Abnormal HbA1C	n = 7	5	2	40
Abnormal glucose	n = 31	22	8	36.4
Prevalence of metabolic syndrome	n = 16	5	3	60
Abnormal systolic BP	n = 21	8	2	25
Abnormal diastolic BP	n = 19	8	2	25
Prevalence of hypertension	n = 61	34	7	20.5
Prevalence of asthma	n = 26	12	2	16.7

FIGURE 2: *Presence of NHANES-based studies in Skinner et al., 2023*

Average use of NHANES in included USA studies: 28.25%

Based on this analysis, it appears that though data is pooled from worldwide sources, the USA-specific studies rely significantly on NHANES as a dataset–around 30% of data in USA studies in the 12 categories where NHANES data was present came from NHANES.

The supplementary paper itself highlights various limitations of studies that use NHANES or NHANES-like datasets: “the cross-sectional design of these studies prevented an examination of within-individual changes in comorbidity prevalence as it relates to fat accumulation and obesity and comorbidity incidence across the age range. This limitation makes it difficult for a primary care provider to determine when during a young patient’s life these screenings are most efficient, useful, and necessary” (Skinner et al., 2023, Discussion). This directly contradicts the guidance issued in Hampl et al. that BMI is a useful tool for providers to screen and diagnose obesity, as well as determine the efficacy of obesity interventions. The review also highlights that the non-NHANES studies

come from specific screening studies in one geographical location, or perhaps clinics specifically for child obesity, which can lead to somewhat biased interpretations of relationships between BMI and comorbidities. The lack of a clear definition of pathology in some instances means that while BMI-comorbid variable relationships may exist, it is not clear how to translate these correlative relationships into clinical guidelines for practice, which calls into question the theoretical basis of these new clinical guidelines. While in many cases correlative studies are used as the basis for clinical guidance, it is important to note that further analysis of NHANES data has shown that there are mediating or confounding variables that complicate the relationship between BMI and comorbid conditions.

There is undoubtedly a correlative relationship between BMI and numerous comorbidities in children, as shown by NHANES data. I have also demonstrated the extent to which NHANES data and similar datasets play a key role in AAP decision making. Given some of the previous exploration of both NHANES limitations, the general lack of individual control over one's body size, and the complex science surrounding obesity and its nebulous relationship to poor health, these lines of thought can be connected to offer several critiques on the rationale and decision making of the AAP:

1. BMI as the primary measure of excess adiposity in NHANES studies and in AAP clinical decision making guidelines

BMI was initially adopted in the US in the 1970s, though critics at the time noted that it was a "poor representation of one's body fat" (Nuttall, 2015, p. 119). Recent analyses using NHANES data on adults have shown "a relatively poor correlation" between BMI and body fat in both men and women, by using bioelectrical impedance to estimate actual body fat (Nuttall, 2015, p. 120). The AAP study states that BMI is a validated clinical tool for both screening and diagnosis, and is an accepted "proxy" for excess adiposity, however AAP themselves offer the following disclaimer about the use of BMI: "BMI does not directly measure body composition and fat content and may under- or over-detect excess adiposity in certain racial and ethnic groups [and in] children and adolescents [with] high fat-free mass" (Hampl et al., 2023, sec. VII, A). While it is reasonable to assume that clinicians would assess individual cases and make tailored recommendations, the question remains to what extent BMI is clinically relevant if it is not an equal predictor of adiposity in all demographic groups, which is striking given such a focus on social determinants of health in the AAP paper, including structural racism. If this is the recommended clinical tool for physicians to use in discerning the necessity for and efficacy of treatments, it becomes necessary that the tool is validated across populations to be broadly applicable.

In work done by Ruiz et al. on the Hispanic Health Paradox, the seemingly paradoxical longevity of Hispanic individuals in the United

States in spite of economic, social, and physiological risk factors, Ruiz et al. importantly suggest that these replicable and robust data on the Hispanic health paradox both with regards to specific disease processes and overall mortality "raise questions about the generalizability of existing knowledge from one racial/ethnic group to another [and] contest the validity of existing risk prediction models" (2016, p. 463). This assertion provides a challenge to the AAP's defense of the use of BMI with their contradictory acknowledgement that BMI may not be equally predictive across all racial/ethnic lines. There are clearly additional sociocultural, genetic/epigenetic, or lifestyle factors that could buffer against some of these supposedly "negative" consequences of BMI in different racial/ethnic groups and improve prognosis, thus it is worth questioning the extent to which surgery and medication should become first-line treatments for a problem that is not yet fully understood and characterized across demographic groups.

If BMI is to be the primary screening and diagnosis tool for overweight/obesity, as well as a measure that helps support associations between body size and comorbidities, it is also critical that the AAP and the research that they base their guidelines on bear the burden of proof that other confounding variables could not also have explanatory power in the BMI-comorbid condition relationship. Doing so would help support clinical guidance formation that privileges this measurement above all other possible diagnostic criteria to determine the necessity for medical or surgical intervention. However, as mentioned previously, there is a body of work suggesting that BMI is a mediating variable rather than a cause of comorbidity–analyses have shown that cardiovascular fitness (regardless of BMI) and controlling for sedentary behavior are better predictors of mortality. Controlling for activity level erased the effects of BMI in one study (Hunger et al., 2020). Given the AAP's reliance on BMI as a clinical screening and diagnostic tool, and reliance on studies that correlate BMI with comorbidities (such as the NHANES-based studies), it is important to question the assumptions and generalizability of such studies to clinical practice that may not address confounds that could lead to comorbid conditions. Understanding such correlations with large datasets such as NHANES are important to derive population-based trends, but BMI is clearly not generalizable to all body types, and ethnic/racial groups. BMI-comorbidity correlations, such as those drawn from NHANES data, can hide nuances that impact prognosis, such as those driven by demographic differences. A health equity approach should consider that such correlations are only jumping off points for further research, and that additional demographic-specific research is needed to validate BMI in the clinical setting for all groups.

2. Unsubstantiated claims about growth patterns

One of the supporting arguments for the AAP guidelines includes a comparison of current BMI distribution among children in comparison to

CDC growth charts, which were formulated using NHANES data from the 1960s-1990s. The AAP claims that "The CDC Growth Charts provide a historical comparison of children's weight status relative to a time before the current obesity epidemic during that healthier growth patterns predominated" (Hampl et al., 2023, sec. VII, A). There is a lack of substantiation or evidence given for the claim that "healthier growth patterns" predominated during this time period. It is unclear what is meant by this assertion–is this based on measures of cardiometabolic health within the NHANES data? A comparison to an outside standard of children's health?

While it may be true that the trends across decades of NHANES data show that overall body mass is increasing among children, NHANES data has also been used to show that children's health is *improving* in critical ways. NHANES data was used to argue for lead bans in gasoline and food packaging, and NHANES data continues to show just how effective these policies have been at reducing lead content in children's blood ("About the National Health and Nutrition Examination Survey," 2022). The critical piece here is that NHANES data can be framed from multiple angles. Because of the amount of data available, research supports both improvements and potential deteriorations of children's health, given what information is presented. It is also worth repeating that NHANES is a cross-sectional dataset, and it does not follow the same group of children through time, nor does it follow children from the same geographic location each year, so it is difficult to claim that any specific time period and the predominant lifestyle at that time period could be the cause of healthier growth patterns. There is not a direct X → Y relationship between the thirty year span listed above, and children's healthy growth patterns.

3. BMI as a potential byproduct, not a cause of disease

As seen previously in this paper, some scholarship suggests that BMI is only a mediating variable, or side effect of more fundamental causes of disease (Hunger et al., 2020). There is emerging support for such a hypothesis that comes from novel analyses of NHANES data, which determines that while a correlational relationship does exist between BMI and comorbid conditions, a more holistic analysis is needed to address the true fundamental causes of disease. These analyses conducted on the same data available in NHANES datasets shows that BMI may not be a strong enough predictor of disease to base diagnostic and clinical guidance upon.

What is meant by the term "fundamental cause?" A seminal paper by Link and Phelan outlines that epidemiological studies are successful at identifying "proximal causes" of disease, such as diet, exercise, etc. However, these proximal causes of disease are precipitated by fundamental causes, notably "socioeconomic status [SES] and social support" (Link & Phelan, 1995, p. 80). There is a large body of evidence

showing that controlling for other health factors, SES and social support are strong predictors of morbidity and mortality in multiple contexts, and that SES and social support embody access to resources, stress buffering, and mediate exposure to environmental and social risks. Thus, eliminating or mitigating proximal causes of disease will only cause a transformation, rather than an elimination of the problem of health disparities, which has largely been seen even with the transition to modern medicine or with countries who have universal healthcare. Health disparities and health issues persist, and specifically persist along class, race, gender, and status lines.

A 2019 analysis of NHANES data by Gaston and Tulve showed that urinary phthalate burden was positively correlated with the presence of metabolic syndrome in adolescents, and there is evidence linking phthalates to metabolic dysfunction through several proposed causal pathways, though the evidence on phthalates as a *cause* of obesity is limited (2019). However, "low SES is often associated with increased obesity, MetS, and chemical exposures" (Gaston & Tulve, 2019, p. 196), demonstrating that socioeconomic status may be the fundamental cause leading to both increased body mass *as well as* environmental exposures to phthalates that cause metabolic syndrome. While phthalates may not cause increased body mass themselves, a focus solely on BMI without taking into account other criteria that could help support a diagnosis of obesity or metabolic syndrome would hide the fundamental cause (low SES) and other proximal causes (phthalate burden) of comorbidities.

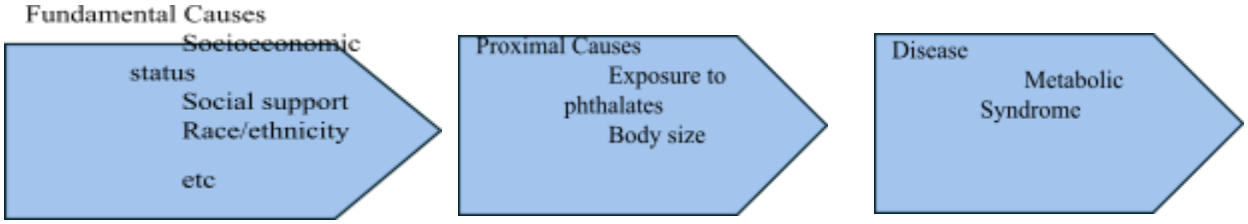

FIGURE 3: Example relationship between fundamental causes, proximal causes, and disease state (adapted from Cassels, 2023)

4. NHANES data can be analyzed from multiple angles

This paper has explored various uses of the NHANES dataset in the analysis of the relationship between body size and comorbidities. While many of the AAP studies have found correlative links between increased BMI and various comorbidities, other NHANES-based analyses (with the caveat that they were done on adults) cited above showed that BMI was a relatively poor predictor of poor cardiometabolic health. NHANES data can also show links between environmental exposures (phthalate burden) which has some proposed causal links to metabolic syndrome, and indeed there is a positive correlation between these measures in an analysis of NHANES data based on adolescents. An analysis of NHANES data done

by Tomiyama et al. shows that analyzing biomarkers and physiological measurements associated with metabolic syndrome such as blood pressure, C-reactive protein, cholesterol, etc. demonstrates that "nearly half of overweight individuals, 29% of obese individuals and even 16% of obesity type 2/3 individuals were metabolically healthy (2016, p. 883). Moreover, over 30% of normal weight individuals were cardiometabolically unhealthy" (Tomiyama et al., 2016, p. 883). The authors noted that a motivating factor for this study was a proposal by the United States Equal Employment Opportunity Commission to allow "employers to penalize employees up to 30% of health insurance costs if they fail to meet 'health' criteria, such as reaching a specified body mass index" (Tomiyama et al., 2016, p. 883). Such a policy demonstrates another attempt to connect BMI specifically to negative health outcomes. While this analysis was performed on adults, up to 30% of obese individuals are misclassified as metabolically unhealthy, which suggests that BMI should be a limited factor (if considered at all) in considering pediatric bariatric surgery. It also opens up another possibility for analysis of childhood NHANES data to see if a similar metabolic misclassification exists among children and adolescents. Furthermore, it bolsters the point that while robust and methodologically sound, drawing simple associations between BMI and other variables with NHANES data can hide other nuance present in the dataset, and that additional angles of analysis can be performed on the same dataset and yield different interpretations, all of which should be considered from a clinical guidance and policymaking standpoint to truly yield the best results in treating the condition holistically

5. Large datasets missing the experience of weight stigma

Finally, it is important to consider potential causal mechanisms of metabolic dysfunction and/or higher weight that may be missing entirely from datasets such as NHANES, and therefore, may not be considered during policymaking and clinical guideline formulation. An emerging area of research spearheaded by Dr. Janet Tomiyama suggests that weight stigma can be a driving factor of obesity and metabolic dysfunction itself. Her COBWEBS model suggests that weight stigma, a form of social/evaluative stigma which is known to increase cortisol, leads to increased cortisol and hunger, weight gain, and this feeds back into the cycle of stigma ("The Science of Fat Shaming," 2021). Experimentally, Tomiyama et al. have also shown a causal relationship between perceived weight stigma and salivary cortisol/increased eating ("The Science of Fat Shaming," 2021). There is some evidence showing that experiencing weight stigma may increase the risk for all-cause mortality by 60% (Tomiyama et al., 2018, p. 2).This model emphasizes the role of perceived stigma, regardless of one's BMI, if they perceive themselves a target of weight stigma, there is an association with a host of "biological markers of poorer health, including unhealthy blood pressure, C-reactive protein,

HDL cholesterol, triglycerides, glucose, and HbA1c levels," which are also key markers of metabolic syndrome, thus presenting a confounding dimension to the BMI–metabolic syndrome relationship (Tomiyama et al., 2018, p. 2).

With regards to children specifically, one longitudinal study of Australian children found that parents' perception of their children as overweight (regardless of their actual weight) led to more weight gain in the children (Robinson & Sutin, 2016). A longitudinal study on over 2000 girls done by the NHLBI showed that children who experienced "weight labeling" or negative weight-based comments at age 10 significantly predicted obesity at age 19, controlling for BMI and other factors (Hunger and Tomiyama, 2014). Scholars studying weight stigma have argued that epidemiological datasets could benefit from the inclusion of the experience of weight stigma. Datasets like NHANES already probe for depression, adverse childhood experiences, diet/exercise. Given the significant and growing body of evidence on the causal impact of weight stigma on weight gain and negative health consequences, it is critical that NHANES expand their realm of inquiry to more accurately assess the full range of variables that impact weight.

VI. Conclusion and recommendations for future research

This analysis has demonstrated that: NHANES based studies associating BMI and comorbidities make up almost ⅓ of USA based studies included in the AAP inquiry on the relationship between obesity and comorbidities. Given this data, the AAP has concluded that there is a significant enough risk of comorbidities with overweight/obesity such that additional medical/surgical treatment should be pursued for some higher weight categories, using BMI as the diagnostic/screening tool. Several limitations of analyses using NHANES were also outlined, including: controversy about the use/generalizability of BMI as a clinical tool across all demographics and as a measure of excess adiposity and the ability for NHANES data can be analyzed from different angles to argue that simple BMI-comorbidity associations hide the nuance present in BMI categories, to name a few. Further analyses of NHANES reveal that there are additional confounding variables that could be impacting the BMI–biomarker relationship, such as proximal causes of SES leading to *both* increased body size and environmental exposure to phthalates, and the phthalate burden leading to metabolic syndrome. Finally, datasets such as NHANES must update their methodology to include factors such as weight stigma, which could be a significant driver of increased body size.

While there are undoubtedly correlations between BMI and comorbid conditions that are well supported by NHANES data, what becomes clear with an interdisciplinary analysis of NHANES limitations and current obesity research is that these findings may be used to support clinical guidance that is narrowly focused only on BMI as the diagnostic/screening

tool, and individualistic measures such as medication and surgery as the treatment. In order to meet the AAP's own benchmark of treating this condition holistically, there is an urgent need both for better research practices that seek to understand confounding factors in the development of obesity and comorbid conditions and for greater attention on the analysis and policy conclusions that come from such research. This paper has suggested several tangible future directions for research that fit within existing frameworks such as NHANES, including incorporating measures of weight stigma into the dataset, looking at proximal causes, investigating environmental obesogens, analyzing whether children are subject to the same metabolic misclassification as adults, investigating the predictive power of BMI for prognosis across different demographic groups just to name a few.

Obesity is clearly not a one-size-fits-all problem–there is hardly scientific consensus surrounding the species-level causes, and individual causes. An interdisciplinary perspective also provides a critical lens on obesity as a pathology in general, and though body sizes are on the rise, this paper also outlines the limited control that individuals have over body size, especially children. Thus it may be worth inquiring whether prevention and treatment are separate endeavors, or if "prevention" (addressing environmental and social factors, improving access to nutrition for all) is in fact also the most effective treatment for childhood obesity/overweight.

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