Trajectories of Child Obesity and Child Obesity Among Children in the US

McEniry, Garcia, Aldea, Huangfu, Guo, Beltrán-Sanchez, Palloni (2025)

Introduction

Empirical research demonstrates that obesity is a phenotype influenced by genetics, maternal effects (embryonic and fetal growth developmental processes), and by environments to which individuals are exposed. These 'obesogenic' environments encompass macro- as well as micro-settings environments. The former are determined by conditions over which individuals have little if any influence (built-in environments, type of food available, green spaces, neighborhood poverty levels). The latter are mostly shaped by parents, siblings and other close kin who co-reside in children' s households. These environments influence the impact of four classes of exposures that modify individuals' risk of obesity: diet, physical activity, sleep patterns, stress levels, environmental exposures. However, indirect genetic effects ("genetic nurture" or effects of parental genes that influence their children phenotype but are not passed down to their children) can also have great importance (Baud et al., 2022; Young et al., 2019) as does niche construction, namely, when organisms actively modify the environments to which they are exposed to and elements of these can be potentially transmitted across generations

We study child obesity as a result of genetic, indirect genetic, and cultural effects and, in addition, interactions between genetic and cultural factors (GxE interactions). Importantly, we also examine the possibility that some of these effects may be transmitted intergenerationally so that obesity among today's children may translate in child obesity among their offspring.

Genetic transmission entails allelic variants that are associated with the phenotype whose impact is reflected in polygenic risk scores (PRS) for obesity. Vertical cultural heredity refers to parent-to-offspring transmission of a phenotype via non-genetic pathways that include imitation, copying, learning (Cavalli-Sforza,L.L. & Feldman, M.W., 1981). Vertical cultural heredity includes indirect genetic effects (Baud et al., 2022; Kong et al., 2018; Yengo et al., 2018; Young et al., 2019) that have become part of research in the field of niche construction (Bonduriansky & Day, 2009; Odling-Smee et al., 2003; Jablonka & Lamb, 2006). Finally, GxE interactions can augment or

attenuate the impact of genetic and indirect genetic effects on the child phenotype. To our knowledge ours is the first attempt to examine child obesity and the joint outcomes of these domains. Admittedly, our conceptual framework is incomplete since it ignores the role played by peers (horizontal cultural transmission) and by influencers and teachers (oblique cultural transmission).

We use parental and child phenotypes for obesity (BMI and waist circumference) along with parental and child genotypes in the form of waist circumference polygenic scores (only for PSID) and sociodemographic variables. At the household level we examine the role played by sociocultural characteristics (e.g. parental education, diet, physical activity, household stresses, food security and poverty). At the neighborhood level, we include neighborhood characteristics (e.g. neighborhood poverty). Preliminary results show a strong association between PRS, cultural elements, poverty and child obesity. We describe how our results fit into a larger multiple dataset project to predict the load of chronic illness, disability, and mortality associated with child obesity.

Data and Methods

PSID-CDS (Child Development Supplement) collects extensive longitudinal data on a representative sample of children along with their primary caregivers. We use the CDS beginning in 2014 when genomic data were collected (n=1174) and children were 5-17 years old and follow up them up in 2019 and 2021. We use two waves from **L.A. FANS** (wave one 2000-2002 and wave two 2006-2008) which has extensive information on neighborhoods and household behavioral patterns regarding diet and physical activity and which collected information mostly on migrant children. Child obesity was obtained in the second wave; the study does not have genetic information.

We use these two data sets and estimate structural equation models (SEM) with a single latent variable (see below) to predict child obesity as a function of individual, household and neighborhood characteristics. The model specification defines the latent construct as a function of the above-described obesity-related domains. We define a cultural risk score (CRS) as the normalized factor scores retrieved from the SEM. The CRS is simply a summary measures of the total effects of obesity-related domains. It is analogous to the PRS score in that it assigns to each individual the weighted average

effect of multiple BMI-related domains which, in the case of the PRS, are multiple allelic loci known to be related to BMI.

In both studies we define child obesity as a dichotomous variable defined in accordance to CDC guidelines as a children-specific function of BMI. In the case of PSID, we also use an alternative measure of adiposity, waist circumference. Only PSID data include genomic information in the form of polygenic risk scores for waist circumference for children and their primary caregivers. This opens up the possibility of exploring variability in estimates induced by including and excluding genetic indicators and to use this to evaluate results from the L.A. FANS study where such indicators are absent. Both studies include slightly different indicators of parental sociodemographic variables (maternal and paternal education), parental obesity and, when available, parental PRS for obesity, household level factors (diet, physical activity, household stressors, food security and poverty) and, finally, neighborhood level characteristics (neighborhood poverty).

A preliminary SEM with PSID data includes the educational level of the household head (number of years attained) and household food security (1=high to 4=very low food security) along with the school grade of the child, PRS for waist circumference for both primary caregivers and children, waist circumference for primary caregivers, and child obesity based on BMI.

The L.A. FANS study has a smaller sample size and does not include genetic indicators. As in the case of PSID, we define obesity using CDC guidelines based on child BMI. Our preliminary models are based on Wave Two where respondents were measured for height and weight. We specify the latent variable as a function of the education level of the primary caregiver for the child (categorical variable 1=lowest to 4=highest), diet information containing is self-reports from the caregiver, and language of the interview (1=Spanish, 0=English).

Results and Next Steps

Preliminary results using **PSID-CDS 2014** showed significant associations between obesity of the primary caregiver measured using waist circumference, the child's PRS, cultural elements (e.g. parental education, food security) and child obesity. Adult waist circumference was associated with culture and adult PRS scores were significantly associated with child PRS scores. Our final model will use longitudinal

data from PSID-CDS 2014-2021 and include the stress level of the primary caregiver, an indicator of achieving CDC guidelines for moderate/rigorous exercise, and a predicted estimate for waist circumference as another measure of obesity. We will also examine the correlation in changes in exposure (e.g. diet, exercise) to changes in BMI using PSID-CDS 2014-2021 data.

Similarly, results using **L.A. FANS 2006-2008** show significant associations between adult obesity, culture and child obesity. Our final model will include more information on individuals, households and neighborhoods from Wave One and include lagged effects from Wave One.

References

Baud, A., McPeek, S., Chen, N., & Hughes, K. A. (2022). Indirect Genetic Effects: A Cross-disciplinary Perspective on Empirical Studies. Journal of Heredity, 113(1), 1–15. https://doi.org/10.1093/jhered/esab059.

Bonduriansky, R. & Day, T. (2009). Nongenetic Inheritance and Its Evolutonary Implications. Annual Review of Ecology, Evolution and Systematics, 40:103-125. https://doi.org/10.1146/annurev.ecolsys.39.110707.173441

Cavalli-Sforza,L.L. & Feldman, M.W. (1981). Cultural Transmission and Evolution: A Quantitative Approach. Princeton University Press.

Jablonka, E., & Lamb, M. J. (2006). Evolution in Four Dimensions: Genetic, Epigenetic, Behavioral, and Symbolic Variation in the History of Life. The {MIT} Press.

Kong, A., Thorleifsson, G., Frigge, M. L., Vilhjalmsson, B. J., Young, A. I., Thorgeirsson, T. E., Benonisdottir, S., Oddsson, A., Halldorsson, B. V., Masson, G., Gudbjartsson, D. F., Helgason, A., Bjornsdottir, G., Thorsteinsdottir, U., & Stefansson, K. (2018). The nature of nurture: Effects of parental genotypes. Science, 359(6374), 424–428. https://doi.org/10.1126/science.aan6877

Odling-Smee, F. J., Laland, K. N., & Feldman, M. W. (2003). Niche construction: The neglected process in evolution. Princeton University Press.

Yengo, L., Robinson, M. R., Keller, M. C., Kemper, K. E., Yang, Y., Trzaskowski, M., Gratten, J., Turley, P., Cesarini, D., Benjamin, D. J., Wray, N. R., Goddard, M. E., Yang, J., & Visscher, P. M. (2018). Imprint of assortative mating on the human genome. Nature Human Behaviour, 2(12), 948–954. https://doi.org/10.1038/s41562-018-0476-3

Young, A. I., Benonisdottir, S., Przeworski, M., & Kong, A. (2019). Deconstructing the sources of genotype-phenotype associations in humans. Science, 365(6460), 1396–1400. https://doi.org/10.1126/science.aax3710