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High birth weight modifies association between adolescent physical activity and cardiometabolic health in women and not men

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ABSTRACT

Recent evidence suggests that adverse prenatal development alters physiological response to physical activity, but longitudinal epidemiologic evidence is scant. This study tested the hypothesis that lower physical activity during adolescence and young adulthood is more strongly associated with later cardiovascular disease (CVD) risk and diabetes or prediabetes (DM/PDM) in women and men who were born with high or low birth weight (HBW, LBW), compared to normal birth weight (NBW). We analyzed data from the National Longitudinal Study of Adolescent to Adult Health, a cohort study of US adolescents followed into adulthood (1994–2009). Using sex-stratified multivariable regression, 30-year CVD risk score (calculated using objective measures; n = 12,775) and prevalent DM/PDM (n = 15,138) at 24–32 years of age were each modeled as a function of birth weight category, self-reported moderate-to-vigorous physical activity frequency in adolescence (MVPA1) and young adulthood (MVPA3), and MVPA–birth weight interactions. Greater MVPA1 was associated with lower 30-year CVD risk score and DM/PDM risk in HBW women but not NBW or LBW women. Associations between MVPA1 and 30-year CVD risk or DM/PDM were not modified by HBW in men; or by LBW in women or men. Additionally, birth weight did not modify estimated effects of MVPA3. Findings suggest that frequent MVPA in adolescence may be a particularly important cardiometabolic risk reduction strategy in girls born HBW; however, we found no evidence that birth weight and MVPA interact in cardiometabolic disease risk in men, for MVPA in adulthood, or for LBW.

1. Introduction

Cardiovascular disease (CVD) is the leading cause of death worldwide, accounting for an estimated 17.5 million deaths in 2012 (World Health Organization, 2015). Diabetes affects 422 million people globally and is a major risk factor for CVD (World Health Organization, 2016). Physical activity in adolescence, independent of adult activity, may affect CVD and type 2 diabetes (DM2) later in life (Nechuta et al., 2015). Recently, prenatal development has emerged as another important factor in the pathogenesis of CVD, DM2, and other chronic diseases.

A growing body of evidence suggests that intrauterine growth restriction and fetal overnutrition—often indicated by low and high birth weight (LBW, HBW), respectively—may alter physiological response to physical activity. This emerging field builds on evidence that LBW is associated with hypertension, diabetes and insulin resistance, coronary heart disease, and renal disease (Harder et al., 2007; Huxley et al., 2007; Mu et al., 2012; Pfab et al., 2006; Vos et al., 2006; Whincup et al., 2008), and that HBW is a risk factor for later obesity (Johnsson et al., 2014; Schellong et al., 2012; Skilton et al., 2014) and diabetes (Harder et al., 2007; Johnsson et al., 2014). It considers how physical activity interacts with LBW and HBW in the prevention of cardiometabolic disease. In cross-sectional observational studies, associations of physical activity with DM2 (Eriksson et al., 2004), metabolic measures (Laaksonen et al., 2003; Labayen et al., 2013; Ortega et al., 2011), and BMI (Boone-Heinonen et al., 2016) were stronger in LBW or HBW participants. These findings are supported by experimental evidence showing stronger metabolic responses to short-term periods of bed rest (Mortensen et al., 2014) or exercise (Mortensen et al., 2013) in men born with LBW, and by animal studies showing amplified response to exercise in prenatally overnourished offspring (Bahari et al., 2013; Rajia et al., 2013).

To our knowledge, no longitudinal epidemiologic studies have investigated the extent to which children born with LBW or HBW are

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more sensitive to the harmful effects of physical inactivity on developing cardiometabolic disease later in life. The objective of this study was to test the hypothesis that less frequent moderate-to-vigorous physical activity (MVPA) during adolescence and young adulthood is more strongly associated with later CVD risk and diabetes/prediabetes in participants who were born HBW or LBW, compared to normal birth weight (NBW), in a representative United States (US) cohort.

2. Methods

2.1. Study population and data collection

The National Longitudinal Study of Adolescent to Adult Health (Add Health) is a prospective cohort study of > 20,000 adolescents followed into adulthood (Harris et al., 2009). The core sample represents all adolescents attending US public, private, and parochial schools, grades 7–12, in the 1994–1995 school year, with oversampled minority groups. The Wave I in-home participant and parental interview was conducted in the 1994–95 academic year (11–21 years of age; n = 20,745). Wave II included all eligible adolescents who would have been in school in 1995–96 (excluding those who graduated in 1995) (n = 14,738). All located Wave I respondents were eligible for Waves III (2001–2002; n = 15,197) and IV (2008–2009; n = 15,701). All waves included an in-home interview, and biospecimens were collected in Wave IV.

Our analytic sample included participants who did not have physical activity limitations at Wave I (57 individuals excluded) and who were not pregnant at Wave IV (519 individuals excluded). After excluding those missing the outcome variable for each model, final sample sizes were 12,775 (CVD models) and 15,138 (diabetes models). Data were analyzed in 2015–17. This study was determined exempt by the Oregon Health & Science University Institutional Review Board.

Trained interviewers collected questionnaire and physical exam data from participants at each wave (Harris, 2013). In Wave I, a parent of each participant also provided demographic and health information about themselves and their adolescent child. In Wave IV, interviewers measured blood pressure and collected blood via finger prick.

2.2. Study variables

2.2.1. Primary outcome: 30-year CVD risk score

Given the low incidence of cardiovascular events in young adults (Blackwell & Lucas, 2015), we used an algorithm developed by Pencina et al. that predicts 30-year risk of coronary death, myocardial infarction, fatal or non-fatal stroke, coronary insufficiency, angina pectoris, transient ischemic attack, intermittent claudication, or congestive heart failure (Pencina et al., 2009). This algorithm was derived from a younger age group (20–59 years) than other CVD risk-prediction methods and accounted for competing risk of non-cardiovascular death. The model incorporated Wave IV age, sex, systolic blood pressure, total cholesterol, high-density lipoprotein cholesterol (HDL-C), smoking, diabetes, and hypertension medication use.

Participants were classified as having diabetes if they met one or more of the following criteria at Wave IV: Hemoglobin A1c (HbA1c) of ≥ 6.5%, fasting blood glucose ≥ 126 mg/dL, non-fasting glucose ≥ 200 mg/dL (American Diabetes Association, 2015), self-reported history of diabetes except during pregnancy, or self-reported use of diabetes medication in the previous four weeks (Whitsel et al., 2012). Hypertension medication status was ascertained from self-reported use in the previous four weeks at Wave IV. Due to concerns of limited precision, Add Health reported deciles for total cholesterol and HDL-C rather than absolute values (Whitsel et al., 2013). To obtain absolute values required by the CVD risk algorithm, we converted deciles of these variables to median values of deciles derived from NHANES 2007–8 and 2009–10 participants (Supplemental Table S1) (NHANES, 2007–2008; NHANES, 2009–2010).

Sex was derived from self-reported sex/gender at the most recent wave for which it was available. We interpret this variable as sex, rather than gender, because it aligns with the term used in the Add Health interview question from the etiologically relevant period (adolescence), and because biological sex differences are most relevant to prenatal and developmental programming effects. We defined current smoking at Wave IV as at least one cigarette per day on each of the previous 30 days.

2.2.2. Secondary outcome: diabetes or prediabetes

We examined prevalent diabetes (DM) and prediabetes (PDM) as components of CVD risk that have greater relevance to a young population compared to hypertension or hyperlipidemia, which tend to manifest later in adulthood. Diabetes was defined as described above. We defined prediabetes as HbA1c of 5.7–6.4% or fasting glucose of 100–125 mg/dL at Wave IV in non-diabetic participants (American Diabetes Association, 2015).

2.2.3. Primary exposure: MVPA at Waves I and III

Self-reported weekly frequency of MVPA was collected with a standard, interviewer-administered activity recall (Supplemental Table S2) (Add Health, n.d.). The questionnaire included a wide range of activities, from walking and yard work to running and team sports, which were modified to accommodate life-stage differences between waves. Therefore, Wave III MVPA (33 activities per week maximum) was scaled to be comparable to Wave I MVPA (16.5 activities per week maximum) by multiplying by (16.5/33), as others have done (Gordon-Larsen et al., 2004; Richardson et al., 2014). We analyzed MVPA as a continuous variable in order to examine associations with incremental MVPA differences across the full range of MVPA frequency.

2.2.4. Effect modifier: birth weight

Birth weight of participants was assessed in the Wave I parent interview and recorded to the nearest ounce. Gestational age at birth was not recorded in Add Health, precluding the ascertainment of size for gestational age. A three-level variable was constructed based on clinically relevant thresholds that are consistent with prior literature (Boulet et al., 2003; Committee on Scientific Evaluation of WIC Nutrition Risk Criteria - Institute of Medicine, 1996; Martin et al., 2011): LBW, < 2.5 kg (5 lb., 8 oz); NBW, 2.5–4 kg; HBW, > 4 kg (8 lb., 13 oz). Sensitivity analyses with higher HBW threshold were not possible due to small numbers of participants > 4.5 kg (n = 111 male, n = 63 female).

A priori confounders were age, smoking at Waves I and III, race/ethnicity (Hispanic, white, Black, Asian, or other), parental educational attainment at Wave I (no college, some college, or college degree or higher), and household income at Wave I. Although age was a component of the CVD risk algorithm, it was included as a covariate to adjust for its expected confounding effect.

2.3. Statistical analysis

Statistical analysis was performed in Stata version 13.1 (StataCorp, College Station, Texas). All analyses were stratified by sex due to previously observed sex–birth weight and sex–behavioral interactions (Boone-Heinonen et al., 2016; Gamborg et al., 2007; Intapad et al., 2014; Moritz et al., 2010). Established survey procedures were used to address sample weights and the clustered sampling design of Add Health (Chen & Chantala, 2014). We used multiple imputation to address missing values of independent variables (Appendix A); results were similar with pre-imputation data (Supplemental Table S3).

We used multivariable regression analyses to model log-transformed 30-year CVD risk score (linear regression) and prevalent DM or PDM (ordinal logistic regression) as a function of self-reported weekly frequency of MVPA at Wave I (MVPA1) or Wave III (MVPA3), LBW and HBW, interactions between birth weight category and MVPA1 or MVPA3, and a priori confounders. A three-level diabetes outcome (DM,