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Does Near-Roadway Air Pollution Contribute to Childhood Obesity?

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Abstract

Exposure to man-made combustion products, including secondhand tobacco smoke, maternal smoking during pregnancy, and near-roadway air pollution (NRAP), have been associated with increased body mass index and obesity in children and have been shown to result in excess weight gain in animal models. Potential mechanisms include pro-inflammatory central nervous system effects of airborne particles on appetite control, resulting in increased caloric intake, or changes in basal metabolism due to effects on mitochondria and brown adipose tissue. Combustion-derived polycyclic aromatic hydrocarbons have also been linked to altered lipid metabolism, epigenetic effects on PPAR γ expression, particle-induced estrogenic effects, and alterations in the distribution of visceral fat. Emerging evidence that a broad spectrum of environmental chemicals have “obesogenic” properties and alter the metabolic profile of adipose tissue challenges the prevailing model that the childhood obesity epidemic is explained solely by increased caloric density of food and decreased physical activity. Research on environmental obesogens could identify novel targets for intervention and yield public health benefits, since NRAP and SHS exposure are both common in populations most at-risk for development of obesity.

Keywords

Near-Roadway Air pollution; Built Environment

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The prevailing model of the epidemic of childhood obesity is that increased energy intake combined with a sedentary lifestyle results in positive energy balance and excess fat storage.¹ Increased caloric density of food found in fast food restaurants and decreased opportunities for physical activity common in inner-city areas with high rates of obesity are likely important determinants of positive energy balance. However, emerging evidence indicates that other factors may affect food consumption, absorption, basal metabolism and adipose deposition, which could therefore be additional contributors to the epidemic. These factors include the *in utero* environment,² neonatal feeding practices,³ dietary composition,¹ and the gut microbiome.⁴ Furthermore, recent evidence from studies in animal models also supports the premise that a broad spectrum of environmental chemicals and chemical mixtures have “obesogenic” properties and alter the metabolic profile of adipose tissue.⁵

Animal models and human epidemiological investigations indicate that maternal smoking during pregnancy causes subsequent childhood obesity.⁶ Recently, prospective epidemiological studies have found associations of childhood obesity and trajectory of body mass index (BMI, calculated as weight in kg/[height in m]²) with another anthropogenic combustion source, near-roadway air pollution (NRAP). NRAP is a complex mixture, including particulate and gaseous combustion products in fresh vehicle emissions, debris from tires and brake wear, and metals from engine wear.⁷ Among Southern California children, increased NRAP exposure was associated with a higher BMI trajectory over the course of primary school.⁸ In older children followed through adolescence, exposure to NRAP (but not regional urban particulate matter), secondhand tobacco smoke (SHS) and maternal smoking during pregnancy were associated with BMI growth trajectory and attained BMI at the conclusion of follow-up at age 18.⁹ More notably, synergistic effects of co-exposure to SHS and high NRAP were estimated to account for 6 kg of excess body weight in an obese young adult male.

While evidence points to nicotine as a driver of obesity associated with *in utero* exposure to maternal smoking,⁶ the synergistic effects of SHS and NRAP suggest that similar biological pathways may be involved in each. As there is no nicotine in NRAP, other products of these two anthropogenic combustion sources may be responsible for the observed associations, if they are indeed causal. Combustion-related polyaromatic hydrocarbons, for example, are present in both NRAP and SHS. Among children in a New York City pregnancy cohort, being in the top tertile of the polyaromatic hydrocarbon exposure distribution was associated with greater than 2-fold increased risk of obesity at age 7.¹⁰

Together, these observations challenge the view that childhood obesity is due solely to increased caloric intake and reduced physical activity, or even food composition and feeding practices. In addition, they cast doubt on the usual explanation that *in utero* exposure to nicotine is the only cause of the well-recognized obesogenic effects of early life tobacco smoke exposure.

There are several possible mechanisms for obesogenic effects of combustion products. NRAP-associated weight gain in animals could be mediated by effects on the brain,¹¹ possibly through effects on appetite or on anxiety-associated overeating. For example, mice exposed *in utero* to diesel exhaust particulate, a model near-roadway air pollutant, had an

increase in fetal brain inflammatory cytokines and subsequent increased adult microglial activation and increased anxiety induced by a high fat diet, than mice exposed to filtered air during gestation¹¹. Exposed mice gained more weight during early life, and the early life exposure to diesel exhaust particulate also primed mice to gain more weight when placed on a high fat diet later in life.

Alternative explanations include NRAP-induced changes in basal metabolism. Polyaromatic hydrocarbons inhibit catecholamine-induced lipolysis, resulting in weight gain in experimental animals (in the absence of increased caloric intake).¹² Animal studies have also shown that early life exposure to urban particulate matter causes mitochondrial damage and increased accumulation of white adipose tissue relative to metabolically active brown adipose tissue,^{13–15} potentially skewing overall metabolic balance from energy utilization more towards storage. In utero exposure to particles also results in less DNA methylation and higher gene expression of PPAR γ , effects of which were shown to extend transgenerationally.¹⁶ Ambient particulate matter has also been found to have estrogenic effects,¹⁷ and in utero exposure to environmental estrogens cause subsequent obesity in an animal model.¹⁸ Finally, exposure to urban particulate matter led to marked increases in visceral adipose tissue and adipose tissue inflammation, both of which are considered to be determinants of the development of the associated insulin resistance and metabolic abnormalities.¹⁵ However, urban background particle-exposed animals in general did not gain more weight compared with those exposed to filtered air. This raises the intriguing possibility that NRAP causes obesity, while urban background particulate contributes to obesity-associated metabolic and inflammatory sequelae, if not obesity itself.

In conclusion, new epidemiological findings and animal models suggest there may be large obesogenic effects of NRAP. The results of such studies could have large public health implications, since NRAP and SHS exposure are both prevalent in the most at-risk populations. It is likely that neither regional urban exposure to particulate matter nor SHS by themselves are important causes of the epidemic of childhood obesity, at least in the United States, because levels of regional pollution and smoking rates have both declined over the course of the epidemic. However, there are many toxicologically relevant compounds in the complex mixture of NRAP, which has not been well characterized, and there has been a large temporal increase in number of vehicle miles traveled over the past several decades corresponding well to the time course of the epidemic.¹⁹ Heavy NRAP-generating traffic corridors are ubiquitous in poor urban areas where the epidemic is most acute. A role for NRAP in the epidemic, perhaps in combination with increased caloric intake and changing dietary composition in an environment not conducive to physical activity, or in synergy with stable or decreasing levels of tobacco smoke exposure, merits further investigation to identify novel mechanisms of obesogenic effects and potential targets for intervention.

References

1. Ludwig DS, Friedman MI. Increasing adiposity: consequence or cause of overeating? *Jama*. 2014; 311(21):2167–8. [PubMed: 24839118]
2. Ross MG, Desai M. Developmental programming of offspring obesity, adipogenesis, and appetite. *Clinical obstetrics and gynecology*. 2013; 56(3):529–36. [PubMed: 23751877]

3. Patel MS, Srinivasan M, Laychock SG. Metabolic programming: Role of nutrition in the immediate postnatal life. *Journal of inherited metabolic disease*. 2009; 32(2):218–28. [PubMed: 19096914]
4. Bervoets L, Van Hoorenbeeck K, Kortleven I, et al. Differences in gut microbiota composition between obese and lean children: a cross-sectional study. *Gut pathogens*. 2013; 5(1):10. [PubMed: 23631345]
5. Blumberg B, Iguchi T, Odermatt A. Endocrine disrupting chemicals. *J Steroid Biochem Mol Biol*. 2011; 127(1–2):1–3. [PubMed: 21839836]
6. Thayer KA, Heindel JJ, Bucher JR, Gallo MA. Role of environmental chemicals in diabetes and obesity: a National Toxicology Program workshop review. *Environmental health perspectives*. 2012; 120(6):779–89. [PubMed: 22296744]
7. Health Effects Institute. Traffic-Related Air Pollution: A Critical Review of the Literature on Emissions, Exposure, and Health Effects (Special Report 17). 2009
8. Jerrett M, McConnell R, Wolch J, et al. Traffic-related air pollution and obesity formation in children: a longitudinal, multilevel analysis. *Environ Health*. 2014; 13(1):49. [PubMed: 24913018]
9. McConnell R, Shen E, Gilliland F, et al. A Longitudinal Cohort Study of Body Mass Index and Childhood Exposure to Secondhand Tobacco Smoke and Air Pollution: The Southern California Children's Health Study. *Env Health Perspect*. 2014
10. Rundle A, Hoepner L, Hassoun A, et al. Association of childhood obesity with maternal exposure to ambient air polycyclic aromatic hydrocarbons during pregnancy. *Am J Epidemiol*. 2012; 175(11):1163–72. [PubMed: 22505764]
11. Bolton JL, Smith SH, Huff NC, et al. Prenatal air pollution exposure induces neuroinflammation and predisposes offspring to weight gain in adulthood in a sex-specific manner. *Faseb J*. 2012; 26(11):4743–54. [PubMed: 22815382]
12. Irigaray P, Ogier V, Jacquet S, et al. Benzo[a]pyrene impairs beta-adrenergic stimulation of adipose tissue lipolysis and causes weight gain in mice. A novel molecular mechanism of toxicity for a common food pollutant. *FEBS J*. 2006; 273(7):1362–72. [PubMed: 16689925]
13. Xu Z, Xu X, Zhong M, et al. Ambient particulate air pollution induces oxidative stress and alterations of mitochondria and gene expression in brown and white adipose tissues. *Part Fibre Toxicol*. 2011; 8:20. [PubMed: 21745393]
14. Xu X, Liu C, Xu Z, et al. Long-term exposure to ambient fine particulate pollution induces insulin resistance and mitochondrial alteration in adipose tissue. *Toxicol Sci*. 2011; 124(1):88–98. [PubMed: 21873646]
15. Sun Q, Yue P, Deiluiis JA, et al. Ambient air pollution exaggerates adipose inflammation and insulin resistance in a mouse model of diet-induced obesity. *Circulation*. 2009; 119(4):538–46. [PubMed: 19153269]
16. Yan Z, Zhang H, Maher C, et al. Prenatal Polycyclic Aromatic Hydrocarbon, Adiposity, Peroxisome Proliferator-Activated Receptor (PPAR) gamma Methylation in Offspring, Grand-Offspring Mice. *PLoS One*. 2014; 9(10):e110706. [PubMed: 25347678]
17. Wenger D, Gerecke AC, Heeb NV, et al. In vitro estrogenicity of ambient particulate matter: contribution of hydroxylated polycyclic aromatic hydrocarbons. *Journal of Applied Toxicology*. 2009; 29(3):223–32. [PubMed: 19021152]
18. Newbold RR, Padilla-Banks E, Snyder RJ, Jefferson WN. Perinatal exposure to environmental estrogens and the development of obesity. *Molecular nutrition & food research*. 2007; 51(7):912–7. [PubMed: 17604389]
19. Jacobson SH, King DM, Yuan R. A note on the relationship between obesity and driving. *Transport Policy*. 2011; 18(5):772–6.