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The Global Epidemic of Childhood Obesity: Poverty, Urbanization, and the Nutrition Transition

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Introduction

Childhood obesity is increasing at rates of epidemic proportion across the world (1, 2). Previously a public health issue of primarily the modern, industrialized world, overweight now affects children of both developing and developed countries, and does so at an ever accelerating pace (1, 2). For example, over the last 20 years, the rates of childhood obesity have doubled in North America (1) and obesity is currently the most common health problem that US children face, with African-American, Hispanic children and children of low socio-economic status most severely affected (1, 3). In Thailand, a country in socioeconomic transition, childhood obesity reached 16 percent in 1993, up 4 percent from 1991 (1). Similar statistics have been documented in many other developing countries including China, Brazil, and South Africa, to name only a very few (2, 4). The increasing prevalence of obesity in children and adolescents even in the lowest income sectors is a relatively new and somewhat baffling phenomenon-it urgently begs the question of how is it that children living in poverty become obese? While the socio-economic, environmental, and biological factors involved in the growth of childhood obesity worldwide are complex, intricately interconnected, and varied both across countries and within them, there emerge several common forces that help elucidate the global trend of increasing obesity in many of the world's impoverished children.

Globalization, Urbanization and the Nutrition Transition

Many researchers seeking to explain the worldwide increases in childhood obesity emphasize similar basic dietary changes, called "nutrition transitions" that have occurred throughout the developing world in recent years as a result of rapid globalization and urbanization. Jefferey Sobal, one such researcher examining the relationship between nutrition transitions and global rises in obesity posits that Western post-industrialized food systems and consumer culture, now integrated into nearly every society across the world, are largely responsible for shifts in diet and physical activity levels that account for the increasing adiposity of the global population. Globalization has made cheap oils and fats widely available in all nations, causing increases in fat intake worldwide (2). Industrialized agro-food systems established by global corporations have successfully increased access to cheap calorie-dense foods across the world (5). Fast food chains and vending machines packed with lipid-rich foods as well as high calorie sodas can now be found in Santo Domingo, Dominican Republic and East Oakland, California alike.

Massive demographic shifts of people from rural to urban areas take place as industrialized agro-food systems replace local subsistence farming in rural areas (12). These urbanized areas worldwide are typically characterized by inexpensive public transportation and communication systems that discourage walking, bicycling, and other physical activity (2, 5). Furthermore, urban areas typically lack safe places for children to play or engage in recreational activities (2). In addition, as suburbs develop around large urban centers there is an increased dependence on mechanized transportation, as well as a disappearance of fields where ball-playing activities once took place (2). Children's diminishing access to sporting activities and other physical exercise, coupled with increased access to television, video games, and energy-saving appliances greatly

increases their susceptibility to obesity (2, 6). Sobal additionally makes the important assertion that children and adolescents tend to participate in global culture more rapidly than their parents and may more readily adopt food preferences and recreational activities that perhaps place them at higher risk than adults of becoming overweight (5).

Deprivation and Obesity: The Physiologic Intersection of Poverty and The New Global Diet

The precise mechanisms that interconnect socio-economic deprivation and childhood obesity remain unproven and controversial (7). Traditional theories tend to focus solely on reduced physical activity and increased consumption of lipid-rich foods, however the relevance of these factors for individuals in both the developed and developing world remains contested (8). Over the last 20 years, researchers have uncovered the increasing phenomenon of children who are both growth stunted and obese (9,10). Subsequent examination of this phenomenon suggests that undernutrition or malnutrition during pregnancy or infancy may play a role in promoting adult obesity (11, 4). Mechanisms theorized to explain this relationship include long-term effects of malnourishment in infancy or early childhood on metabolism, growth processes, gene expression, cell number, and clonal selection (13, 14). Barker et al. posits that infants' primary adaptation to undernourishment is a reduced growth rate and related alterations in fetal hormone products that result in long-term changes in production of insulin and growth hormone (15, 16).

Most recently published research claims that an additional physiologic mechanism may be at play that further explains the relationship between early nutritional insults and obesity. A study conducted by Hoffman et al. in 2000 with shantytown children in Sao Paulo, Brazil has shows that nutritionally stunted children have impaired fat oxidation when compared to non-stunted counterparts (8). Fat that is not oxidized must be stored, therefore children with impaired fat oxidation will tend to experience accelerated fat deposition over time (8). Utilizing evidence from prior findings of biological mechanisms Hoffman et. al postulates that altered hormone function underlies impaired fat oxidation. More specifically, the team explains that long-term undernutrition is accompanied by decreases in insulin-like growth factor I (IGF-1). Because IGF-I increases the activity of hormone-sensitive lipase to lipolytic hormones, reduced levels of IGF-I may result in impaired fat oxidation (17, 18, 19).

Physiologic explanations of the effects of early undernutrition on childhood obesity are essential in understanding how socio-economic deprivation may translate into childhood obesity. These explanations alone, however, do not explain why the phenomenon of childhood stunting and obesity has only recently begun to occur, as childhood stunting has occurred previously in history without the concomitant obesity (4). One must look again to the larger global forces at play that have, in relatively recent history, vastly altered the diets and activity levels of even individuals from the poorest segments of society. Prior to the nutrition transition and globalized urbanization, poor socioeconomic conditions did not allow for the manifestation of obesity in growth stunted children. It is then the new global diet rich in fats and calorie-dense foods and the increasingly

sedentary nature of urban life that provide the material conditions necessary for obesity to express itself in chronically nutrient-deprived children.

Taking a Closer Look at Poverty and Childhood Obesity in the US

The US is a nation considered to be almost completely globalized (5). Considering the now virtually stereotypical ramifications of globalization on diet and physical activity levels, it is conceivable that as many as 14 percent of children in the US are obese (3). Despite the almost universal penetration of global food and transportation systems throughout the US, all children are not impacted by obesity equally. Children of minority backgrounds and lower socio-economic status have been found in multiple studies to be more severely afflicted by obesity (1, 3). Although there is conflicting evidence as to whether particular ethnic minority status is an independent factor in increased risk for childhood obesity, multiple studies have found that race and ethnicity as factors in childhood obesity become insignificant when socioeconomic status is adjusted for (1, 20, 21, 22, 30).

Researchers describe a variety of mechanisms that help explain the how socioeconomic status impacts childhood obesity. Income, a major component of socioeconomic status is related to obesity primarily through access to resources (29). The ability to procure nutritious, low-fat foods, for oneself and one's family as well as the ability to participate in voluntary exercise is largely dependent on one's socioeconomic status (29). For example, Neumark-Sztainer et. al found that of adolescents in Minnesota, 28 percent had inadequate fruit intake and 38 percent had inadequate vegetable intake, but that as many as 40 percent of adolescents from low-socioeconomic backgrounds had both inadequate fruit and vegetable consumption (23). This data suggests that low socioeconomic status may decrease access to fruits and vegetables. Kinra et al points out that as a result of limited financial resources, it is likely that poor people are forced to buy more efficiently, purchasing foods richer in energy (particularly fats and sugars). Such high energy foods, are cheaper per unit of energy than low-fat foods such as fruits and vegetables (7). Additional studies have indicated that children from lower socioeconomic status backgrounds are less physically active and less fit than children from more affluent backgrounds (20, 21, 22). Such findings support the notion that impoverished children in the US, like children in the developing world, face barriers to participating in regular physical activity and as a result lead increasingly sedentary lives.

An additional factor previously mentioned that influences childhood obesity among impoverished US children is growth stunting. Reports indicate that growth stunting in the US is as high as 10 percent (24) and is correlated with low socioeconomic status (25). Like their counterparts in developing countries, economically deprived US children are at increased risk of chronic malnutrition leading to long term biological changes that increase susceptibility to obesity.

Conclusions

The rise in childhood obesity worldwide is of great concern as it poses a significant health risks for those affected both during their childhood and well into adulthood. Obese children are at increased risk of developing hypertension and hypercholesterolemia, atherosclerosis, and diabetes; conditions known to be predictive of coronary artery disease (2, 26, 27). The increased mortality and morbidity associated with childhood obesity has ramifications beyond individual disease status. Some studies suggest that adolescent and child obesity itself may cause low socio-economic status (28, 29). If such findings are accurate, childhood obesity may be an important factor contributing to widening income disparities worldwide. Furthermore, as childhood obesity increases we will begin to see increasing rates of chronic disease in the population as it ages. These epidemiological shifts will have a serious impact on demands for health care and support services for the chronically ill, potentially placing significant strains on international economies in the future (2).

Research and Policy Recommendations

Global interventions must be enacted in order to address the global epidemic of childhood obesity and prevent both the associated increasing rates chronic disease as well as the reverberations this epidemiologic shift will have on international economies and widening income disparities. As Jeffrey Sobal asserts, designing effective global policies to address the problem of childhood overweight, will first require a better understanding of how collective social, economic, and political structures, as well as cultural changes influence childhood obesity (5). It is not enough for epidemiologists to focus simply on individual physiology and personal characteristics, nor is it sufficient to consider childhood obesity only in the context of individual, local, or even national cases. Researchers must begin to ask questions that probe the wide-sweeping effects of globalization and urbanization on childhood obesity. National surveys should include questions regarding consumption of processed foods, television viewing, affects of media advertisements for sodas and fast food on childhood and adolescent dietary preferences and practices.

National dietary recommendations, as well as culturally and class sensitive nutritional education programs must be made national and international priorities. Perhaps advertisements for sodas and fast food should be eliminated from television programming targeted to children and adolescents, or eliminated from television all together as was done with advertisements for cigarette smoking in the US. It is not, however, enough to simply distribute information without changing access to resources. Perhaps countries should consider issuing a tax on foods such as fast food, high-fat snack foods, and high calorie sodas that have been identified to contribute to childhood obesity. Funds garnered from such taxation, along with additional national and international monies could then be invested in efforts to produce and deliver cheap, fresh fruits and vegetables to poor, urbanized populations. In addition, public exercise programs and free sporting activities should be made widely available to children and adolescents in poor, urbanized areas. Only by addressing the global forces that affect childhood obesity, and improving impoverished people's access to healthy food and physical activity can we solve the

worldwide problem of childhood obesity and prevent its imminent social, economic, and epidemiologic consequences.

References

1. Wang, Youfa. Cross-national comparison of childhood obesity: the epidemic and the relationship between obesity and socioeconomic status. *International Journal of Epidemiology*. 2001; 30: 1129-1136.
2. Schneider, Dona. International trends in adolescent nutrition. *Social Science and Medicine*. 2000; 15: 955-967
3. Strauss, Richard, S., Pollack, Harold, A. Epidemic increase in childhood overweight 1986-1998. *JAMA*. 2001; 286: 2845-2848.
4. Popkin, Barry, M, Richards, Marie, K., Montiero, Carlos, A. Stunting is associated with overweight in children in four nations that are undergoing the nutrition transition. *Journal of Nutrition*. 1996; 126: 3009-3016.
5. Sobal, Jefferey. Commentary: Globalization and the epidemiology of obesity. . *International Journal of Epidemiology*. 2001; 30: 1136-1137.
6. O'Loughlin, J., Paradis, G., Renaud, L., Meshefedjian, G., Gray-Donald, K. One- and two year predictors of excess weight gain among elementary school children in multiethnic, low-income, inner-city neighborhoods. *American Journal of Epidemiology*. 2000; 152: 739-745.
7. Kinra, Sanjay, Nelder, Robert, P., Lewendon, Gill, J. Deprivation and childhood obesity: a cross sectional study of 20,973 children in Plymouth, United Kingdom. *Journal of Epidemiology and Community Health*. 2000; 54: 456-460.
8. Hoffman, David, J., Sawaya, Ana, L., Verreschi, Ieda, Tucker, Katherine, L., Roberts, Susan, B. Why are nutritionally stunted children at increased risk of obesity? Studies of metabolic rate and fat oxidation in shantytown children from Sao Paulo, Brazil. *American Journal of Clinical Nutrition*. 2000; 72: 702-707.
9. Adriezen, B.T., Baertl, J.M., Graham, G.G. Growth of children from extremely poor families. *American Journal of Clinical Nutrition*. 1973; 26: 926-930.
10. Forrester, T.E., Wilks, R.J., Bennett, F.I., Simeon, D., Osmond, C., Allen, M., Chung, A.P., Scott, P. Fetal growth and cardiovascular risk factors in Jamaican school children. *British Medical Journal*. 1996;312: 156-160.
11. Sawaya, A.L., Grillo, L.P., Verreschi, I., Carlos da Silva, A. Roberts, S.B. Mild stunting is associated with higher susceptibility to the effects of high-fat diets:

- studies in a shantytown population in Sao Paulo, Brazil. *Journal of Nutrition*. 1997; 128 (suppl): 415S-420S.
12. Mosse, Julia, Cleves. *Half the World, Half a Chance: An Introduction to Gender and Development*. Oxford: Oxfam, 1993. pp 115-151.
 13. Lucas, A. Programming in early nutrition in man. In: Bock GR, Whelan J, eds. *The childhood environment and adult disease*. London: John Wiley and Sons, 1991: 38-49
 14. Waterland, R.A., Garza, C. Potential mechanisms of metabolic imprinting that lead to chronic disease. *American Journal of Clinical Nutrition*. 1999; 69: 179-197.
 15. Barker, D.J.P., *Fetal and Infant Origins of Adult Disease*. London: British Medical Journal Publishing, 1992
 16. Barker, D.J.P., *Mothers, Babies, and Disease Later in Life*. London: British Medical Journal Publishing, 1994
 17. Marcus, C., Bolme, P., Mich Johnson, G., Margery V., Bronegard, M. Growth hormone increases lipolytic sensitivity for catecholamines in adipocytes from healthy adults. *Life Science*. 1994; 54: 1335-1341.
 18. Bjorntorp, P. Hormonal control of regional fat distribution. *Human Reproduction*. 1997; 12 (suppl): 21-25.
 19. Hussain, M.A., Schmitz, O, Mengel, A., et. al. Comparison of the effects of growth hormone and insulin-like growth factor I on substrate oxidation and on insulin sensitivity in growth hormone-deficient humans. *Journal of Clinical Investigation*. 1994; 94: 1126-1133.
 20. Kromhohlz, H. Physical performance in relation to age, sex, social, class, and sports activities in kindergarten and elementary school. *Perceptual Motor Skills* 1997; 84: 1168-1170.
 21. Gottlieb, N.H., Chen. M.S., Sociocultural correlates of childhood sporting activities; their implications for heart health. *Social Science and Medicine*. 1985; 21: 533-539.
 22. Allison, K.R., Predictors of inactivity: an analysis of Ontario Health Survey. *Canadian Journal of Public Health*. 1996; 87: 354-358.
 23. Neumark-Sztainer, D., Story, M., Resnick, M.D., Blum, R.W. Correlates of inadequate fruit and vegetable consumption among adolescents. *Preventive Medicine*. 1996; 25: 497-505.

24. Interagency Board for Nutrition Monitoring and Related Research. Third Report on nutrition monitoring in the U.S. Washington DC: US Government Printing Office, 1995.
25. Brunner, E., Marmot, M., Nanchahal, K., Sansfeld, S., Juneja, M., Alberti, K. Social inequality in coronary risk: central obesity and the metabolic syndrome. Evidence from the Whitehall II study. *Diabetologia* 1997; 40: 1341-1349.
26. Berenson, G.S., Srinivasan, S.R., Nicklas, T.A. Atherosclerosis: A nutritional disease of childhood. *American Journal of Cardiology*. 1998; 26: 22T-29T
27. Mokdad, Ali, H., Bowman, Barbara, A., Ford, Earl, S., Victor, Frank, Koplan, Jefferey, P. The continuing epidemics of obesity and diabetes in the United States. *JAMA*. 2001; 286: 1195.
28. Gortmaker, SL, Must, A., Perrin, J.M., Sobol, A.M., Dietz, W.H. Social and economic consequences of over-weight in adolescence and young adulthood. *New England Journal of Medicine*. 1993; 329: 1008-1012.
29. Sobal, Jefferey. Obesity and socioeconomic status: a framework for examining relationships between physical and social variables. *Medical Anthropology*. 1991; 13: 231-247.
30. Lindquist, Christine, H., Reynolds, Kim, D., Goran, Michael, I. Sociocultural determinants of physical activity among children. *Preventive Medicine*. 1999; 29: 305-312.