Published in final edited form as:

Nat Rev Endocrinol. 2018 January ; 14(1): 5–6. doi:10.1038/nrendo.2017.157.

Trends in underweight and obesity: Scale of the problem

Jack A. Yanovski

Section on Growth and Obesity, Division of Intramural Research Eunice Kennedy Shriver National Institute of Child Health and Human Development, National Institutes of Health, 10 Center Dr, Room 1-3330 MSC 1103, Bethesda, MD 20892-1103

Standfirst

A recent study reports that the global prevalence of paediatric obesity increased to >5% between 1975–2016. This increase occurred even as the prevalence of paediatric underweight decreased slightly but remained >8%. These average prevalence changes blur the remarkable regional variations in how prevalence has changed. There is a pressing need to address both underweight and obesity worldwide.

A recent paper documents that worldwide, the prevalence of underweight remains high, even though the prevalence of obesity is increasing in many regions of the world¹. Childhood undernutrition is associated with sarcopenia, cardiac and renal dysfunction, and immunological defects and might cause as many as 3.1 million deaths annually (45% of all deaths of children worldwide)². Obesity during childhood increases the risk for abnormalities in cardiovascular function, glucose homeostasis, pulmonary function, and cognition during childhood³, as well as risk for obesity (and therefore mortality) in adulthood⁴.

The prevalence of obesity in the USA increased dramatically from the 1970s to the early 2000s⁵. A slower rate of increase has occurred since that time, such that in 2015–2016, the prevalence of obesity in the USA was an alarming 13.9% among preschool-aged children (2 to 5 years of age), 18.4% in school-aged children (6 to 11 years of age), 20.6% among adolescents (12 to 19 years of age), and an even more alarming 39.8% in adults⁶. From 1975 to 2014, the prevalence of underweight (BMI <5th percentile standard) in US children 2 to 19 year of age decreased from ~5.1% to ~3.8%⁷; the prevalence of underweight among US adults also decreased, from 3.6% to 1.4%⁸.

Worldwide data from 2416 population-based studies have now been pooled and analysed by investigators in the Non-communicable Disease Risk Factor Collaboration to produce the most complete global perspective of how underweight and overweight prevalence changed from 1975 to 2016¹. The authors carefully vetted the data — they only included data sets where actual (measured) heights and weight were collected using a probabilistic sampling

Refers to: NCD Risk Factor Collaboration (NCD-RisC). Worldwide trends in body-mass index, underweight, overweight, and obesity from 1975 to 2016: a pooled analysis of 2416 population-based measurement studies in 128.9 million children, adolescents, and adults. The Lancet [http://dx.doi.org/10.1016/S0140-6736\(17\)32129-3](http://dx.doi.org/10.1016/S0140-6736(17)32129-3) (2017)

Competing interests statement

The author declares no competing interests.

method during a defined sampling frame. The investigators also excluded participants with implausible BMIs and those recruited based on biased selection strategies (for example, people who had been included or excluded based on their health status or cardiovascular risk).

The total sample size in the present study was 128.9 million participants, among whom were 31.5 million children and adolescents. The investigators organized the data mostly according to geographical and national income regions, with the exception of a geographically dispersed group of high-income English-speaking countries (Australia, Canada, Ireland, New Zealand, UK, and USA) that seemed sufficiently similar in their trends to be combined. The group analysed the data using a model that allowed for regional differences in BMI within countries and modelled time trends for BMI with non-linear terms that would enable dissection of changes in the pace with which BMI had changed.

The results confirmed and extended prior global studies, finding evidence for increases in obesity prevalence and small decreases in underweight prevalence. Overall, the prevalence of paediatric obesity increased more than 5-fold to >5% for both boys and girls aged 5–19 years. In addition, the worldwide prevalence of paediatric underweight decreased in girls from 9.2% to 8.4% and in boys from 14.8% to 12.4%. The study also documented remarkable regional disparities in how BMI has changed in the last 40 years. In Eastern Europe, there seemed to be little increase in paediatric obesity prevalence since 1975. By contrast, in Northwestern Europe, the high-income English-speaking countries and a few other places, the rate of increase in BMI was high before 2000, but slowed more recently. In Micronesia and Polynesia the prevalence of paediatric obesity rose dramatically throughout the past 40 years, such that >30% of boys and girls in the Cook Islands, Nauru and Palau were obese last year. At the same time, underweight still affected more than 20% of girls and 30% of boys living in India.

The study showed that, worldwide, 124 million children had obesity and 192 million children were moderately or severely underweight in 2016. We must therefore confront a bidirectional crisis: how do we ameliorate underweight while preventing obesity at the same time? The proposed solutions, which address how best to manage both energy intake and energy expenditure, suggest action not only by individual families but also by governments and the food and beverage industries. The evident disparities in regional prevalence offer opportunities to test approaches and identify the most promising strategies⁹.

The goal of providing a nutritious and healthy diet that keeps most children at a healthy BMI has so far proven elusive. As we have reduced societal undernutrition, obesity has become more prevalent — this result is unfortunately not surprising. During the evolutionary past, proto-humans and their predecessors were likely chronically undernourished. Humans did not learn to preserve most foodstuffs until recent times, so the discovery of a food source necessitated immediate consumption. Those who could ‘feast’ to a greater degree might have had a reproductive advantage, leading to: the overrepresentation of genetic variants that promoted more rapid eating; tolerance of abdominal distention; and the efficient storage of excess energy in adipose tissue.

Evidence strongly supports the theory that a substantial portion of the variance in a child's body weight, perhaps ~80%¹⁰, can be attributed to inherited factors that are genetic or epigenetic. Considerable data also indicate the existence of multiple, redundant mechanisms to help ensure a rapid physiologic response to conserve energy when weight is too low. These systems are still essential for the survival of the many people who live in regions where undernutrition is common. As regions become more developed, however, the ready availability of inexpensive, energy-dense foods and the reduction of obligate energy expenditures for survival that make severe undernutrition rare, offer a perfect opportunity for genetic predispositions for obesity to become manifest. The past 40 years have thus provided an unparalleled opportunity to observe the interaction of genetic factors with the environment. Our as-yet unmet societal challenge remains how to alleviate the immediate and devastating consequences of undernutrition while avoiding our predispositions for overnutrition.

Acknowledgments

JAY is a Commissioned Officer in the United States Public Health Service (PHS). The opinions and assertions expressed herein are those of the author and are not to be construed as reflecting the views of the PHS or the Department of Health and Human Services. JAY receives research support from the NICHD Intramural Research Program ZIA-HD-00641, from Zafgen Inc. for a clinical trial of pharmacotherapy to treat obesity and hyperphagia in patients with the Prader-Willi syndrome and from Rhythm Pharmaceuticals Inc. for support of sequencing of genes upstream of the melanocortin 4 receptor in the leptin signalling pathway and for a clinical trial of pharmacotherapy to treat obesity due to proximal leptin signalling pathway defects.

References

1. N. C. D. Risk Factor Collaboration. Worldwide trends in body-mass index, underweight, overweight, and obesity from 1975 to 2016: a pooled analysis of 2416 population-based measurement studies in 128.9 million children, adolescents, and adults. *Lancet*. 2017
2. Black RE, et al. Maternal and child undernutrition and overweight in low-income and middle-income countries. *Lancet*. 2013; 382:427–451. DOI: 10.1016/S0140-6736(13)60937-X [PubMed: 23746772]
3. Yanovski JA. Pediatric obesity. An introduction. *Appetite*. 2015; 93:3–12. DOI: 10.1016/j.appet.2015.03.028 [PubMed: 25836737]
4. Collaborators, G. B. D. O. et al. Health Effects of Overweight and Obesity in 195 Countries over 25 Years. *N Engl J Med*. 2017; 377:13–27. DOI: 10.1056/NEJMoa1614362 [PubMed: 28604169]
5. Ogden CL, Carroll MD, Flegal KM. Epidemiologic trends in overweight and obesity. *Endocrinol Metab Clin North Am*. 2003; 32:741–760. vii. [PubMed: 14711060]
6. Hales, CM., Carroll, MD., Fryar, CD., Ogden, CL. Vol. 288. U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Health Statistics; Hyattsville, MD: 2017. p. 1-8.
7. Fryar, CD., Carroll, MD., Ogden, CL. Prevalence of Underweight Among Children and Adolescents Aged 2–19 Years: United States, 1963–1965 Through 2013–2014. Jul 18. 2016 <https://www.cdc.gov/nchs/data/hestat/underweight_child_13_14/underweight_child_13_14.htm>
8. Fryar, CD., Carroll, MD., Ogden, CL. Prevalence of Underweight Among Adults Aged 20 and Over: United States, 1960–1962 Through 2013–2014. Jul 18. 2016 <https://www.cdc.gov/nchs/data/hestat/underweight_adult_13_14/underweight_adult_13_14.htm>
9. Dietz WH. Double-duty solutions for the double burden of malnutrition. *Lancet*. 2017
10. Wardle J, Carnell S, Haworth CM, Plomin R. Evidence for a strong genetic influence on childhood adiposity despite the force of the obesogenic environment. *Am J Clin Nutr*. 2008; 87:398–404. [PubMed: 18258631]

Biography

Dr. Jack Yanovski is a Paediatric Endocrinologist and Chief of the Section on Growth and Obesity, in the Eunice Kennedy Shriver National Institute of Child Health and Human Development, NIH, Bethesda, USA. Dr. Yanovski holds MD and PhD degrees from the University of Pennsylvania, USA. Dr. Yanovski has conducted many clinical studies on evaluation and treatment of paediatric obesity as well as laboratory investigations of molecular etiologies for obesity. Dr. Yanovski has published over 250 papers and was a member of the expert panel that developed the 2017 Endocrine Society Clinical Practice Guideline on Pediatric Obesity Assessment, Treatment, and Prevention.

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript