



# Is the onset of obesity the same as aging?

The article by Belsky et al. (1) presents two summary measures of health as measures of “aging,” one cross-sectional and the other longitudinal. Their analysis is fundamentally flawed by a failure to recognize the biological meaning of the measures that are used in the summary index. Looking at the changes over time in the individual measures, it is quite clear that the cohort was gaining weight between ages 26 and 38 y. Many of the measures used, such as waist-to-hip ratio, blood pressure, cholesterol, triglycerides, and HgbA1C, are markers of declining physical activity and obesity. Weight gain would explain the increases in cardiovascular risk that are occurring at the same time. Such a pattern at this life stage was clearly described in the Framingham Study 20 y ago (2). The decline in physical functioning is likely a consequence of weight gain, as well as a decline in physical activity. Heavier individuals can appear older than their stated age and thinner individuals are viewed as more youthful in appearance. Thus, I believe that what is being

described in this index is largely related to weight gain in early midlife. To call this “aging” is to say that the measures are characterizing the fundamental aging process. These processes, including epigenetic changes, stem cell regenerative capacity, macromolecular damage, altered proteostasis and control of inflammation, and cellular senescence are thought to be intrinsic to the aging of the organism and may link aging to chronic-disease risk (3). At the whole-person level, several physiologic measures can be used that appear to be intrinsically related to aging. These measures include maximal oxygen consumption with exercise (4) and muscle strength (5). The rate of aging should not be confused with the development of preventable chronic-disease risk factors. One might think of weight gain and declining activity as accelerants of the aging process; indeed, caloric restriction is well known to slow aging in animal models. These external drivers of aging are not likely to be amendable by

antiaging therapies, which target intrinsic aging. Approaches are needed that assess age-related changes that are more closely related to the underlying biology of the aging process.

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