Response to Franz et al. (2007), Genetics of Body Mass Stability and Risk for Chronic Disease: A 28-Year Longitudinal Study

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The article by Franz et al. (2007) regarding the stability of body weight over 28 years attributed the variation in BMIs of twin pairs to nonshared environmental influences, but had little discussion of what these factors might be. Twins tend to have the same behaviors, so attributing the greater variation in BMI between modern twins and World War 2 twins to differences in physical activity, diet, partners, habits, or access to health care does not seem sufficient. The authors did not cite evidence regarding one factor that seems much more likely to be part of the nonshared environment, that of infectious disease. We reported that in a small number of twin pairs discordant for infection with human adenovirus-36 (Ad-36), the infected twins were heavier and fatter than the non-infected (Atkinson et al., 2005). A number of papers have been reported that demonstrate that Ad-36 is capable of producing obesity in several animal models, and two human studies suggested a correlation of infection and obesity (Atkinson, in press). Rogers et al. (in press) showed that the mechanism of Ad-36 is a direct effect on adipocytes via the viral E4orf1 gene to increase production of lipogenic enzymes in the cells. Additional studies in twin registries such as that of Franz et al. (2007) where serum has been stored over time would be the best evidence to demonstrate that Ad-36 infection has contributed to the epidemic of obesity that started in most countries of the world in the early 1980s.

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