
Response to Richard L. Atkinson

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We thank Dr Atkinson for his interesting comments in response to our article on the genetics of body mass index (BMI) stability (Franz et al., 2007). We agree that human adenovirus-36 (Ad-36) could be another contributor to the current obesity epidemic. Of course, further research is needed given that the evidence is cross-sectional and based on relatively small samples, that Ad+ twins in his study were only in the low overweight range (26.1; Atkinson et al., 2005), and that there is a lack of evidence that heavier individuals experience greater exposure to viruses. It also remains to be seen how much of the variance in obesity can be accounted for by Ad-36.

Atkinson also stated that ‘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.’ These factors may contribute to the current obesity epidemic, but we did not suggest that they are sufficient. Also, the purpose of our model testing is to elucidate factors that contribute to both twin similarities and differences, and we do not think that these psychosocial factors necessarily lead to mostly similar behavior. Indeed, a recent large-scale study found that even the adiposity of one’s social networks can predict changes in BMI across 32 years (Christakis &

Fowler, 2007). The risk of becoming obese was substantially increased by having a close friend or spouse become obese. Geographic differences did not modify risk, suggesting that common environmental exposure was not an adequate explanation for the ‘spread’ of obesity. Clearly more longitudinal behavior genetic research is needed to understand this important public health issue, and we agree that viral exposure is one of several environmental factors that is worthy of further study.

References

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