GENETICS

EVOLUTION AT WORK

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While I never found it difficult to test for bacterial micro-evolution (like in the already famous 2009 E coli paper) I have considerable problems to see this also in contemporary human populations. As an epidemiologist I am now attracted by a new PNAS paper that addresses this problem (for the first time?).

> Our aims were to demonstrate that natural selection is operating on contemporary humans … To do so, we measured the strength of selection, estimated genetic variation and covariation, and predicted the response to selection for women in the Framingham Heart Study … We found that natural selection is acting to cause slow, gradual evolutionary change. The descendants of these women are predicted to be on average slightly shorter and stouter, to have lower total cholesterol levels and systolic blood pressure, to have their first child earlier, and to reach menopause later than they would in the absence of evolution.

Athough the abstract is quite clear, I am hesitating to follow their analysis strategy

> The solution we chose was to calculate the response surface of each trait for age and time and to express the measurement of that trait for each individual as an average deviation from that surface. Thus, for several traits we asked whether through their adult years individuals tended to have higher or lower values than other individuals of the same age measured in the same year.

Following that procedure they regress the cholesterol residuals on lifetime reproductive success (LRS). Funny to see that all examined traits (height, weight, blood pressure) were significantly associated with LRS – a rather unlikely outcome.
Maybe we should note, that a new risk factor in the next generation (food) associated with an increase in an QTL (cholesterol) does not need to involve any selection. Even simple coupling to LRS does not provide any proof – LRS of just one generation is a complicated

thing influenced by hundreds of factors – like family economy (the authors admit that level of education is a strong cofactor!). So their table 4 is largely uninterpretable – I wonder also if the results are really for LRS or just for age at first birth as the legend says?
The question remains – is there any chance to do such an analysis? My attempt would be to model the interaction of LRS and cholesterol difference to parents against current cholesterol values. Maybe the inclusion of cholesterin gene variants (using a as Mendelian randomization approach) would be appropriate?