Commentary: Haldanes and trends in phenotypic change in humans

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Accepted 20 December 2012

There has been growing recent interest in applying evolutionary theory to issues in human health and disease.1 Although it has taken some time to develop acceptance,2 as ideas and methodology have historically remained largely separated between these fields of research, (see Fig. 1, Nesse and Stearns3), evolutionary theory is proving extremely useful for helping further understand origins of human diseases. In some cases it even provides a context for aiding design and development of direct clinical treatments (for recent review, see Stearns1). A new study by Krieger and colleagues4 in this issue of International Journal of Epidemiology demonstrates how a metric designed by evolutionary biologists—the haldane—to estimate standardized rates of phenotypic change within a generational framework can extend upon currently available methods in public health research, which are rates or proportional changes in phenotypes per year or decade. Furthermore, Krieger et al.’s findings help strengthen the growing recognition that humans continue to change and evolve despite modern medicine, with rates of phenotypic change similar in magnitude to those from plant and animal populations experiencing rapid environmental changes, and that the direction of phenotypic change for some human traits is strikingly similar between recent studies.
Why is the haldane so useful?

J.B.S Haldane first proposed such a measure in 1949. This was later termed the ‘haldane’ by Gingerich. Statistically, it is calculated on log-transformed traits as the change in standard deviations of a trait per generation where log-transformation provides standardization necessary to make phenotypic measurements comparable. Thus, the haldane is a convenient way to compare rates of phenotypic change across studies, species, traits and time periods. This is demonstrated in reviews that have compared thousands of haldane estimates directly, for example to assess what would be considered rapid phenotypic change and whether human activity induces rapid phenotypic change in the wild.

What is the difference between ‘phenotypic’ versus ‘genetic’ haldanes?

Before discussing rates of phenotypic change, it is useful to clarify that haldanes should be qualified as either phenotypic or genetic. As is also the case for many other studies that have estimated trait changes in haldanes (see Table 1, Hendry and Kinnison), the haldane estimates in Krieger et al. are phenotypic in that it is unknown what proportion of that phenotypic change is due to phenotypic plasticity (i.e. gene-by-environment effects) versus genetic change. The latter requires specialized methods (e.g. common environment, pedigree analysis with restricted maximum likelihood) to disentangle these effects.

As the potential for genetic change is often slower than phenotypic change (for an exception, see Carroll et al.), haldane rates accounting for genetic effects may be smaller. An example of this possible difference in the context of modern human populations is the comparison of the haldane rates found in Krieger et al. (up to 0.3 haldanes) with those estimated from another US population, which were much smaller (up to 0.03 haldanes). The likely reason they were much smaller in Byars et al. is that trait changes were measured through their association with lifetime reproductive success and genetic covariance among traits, which may constrain estimated phenotypic responses if correlations between traits are high and opposing. This should be taken into consideration when comparing haldane estimates across studies, species or different time periods.

How rapidly are modern humans changing?

The rates of phenotypic change estimated by Krieger et al. were based on US-born White and Black non-Hispanic individuals aged 20–44 years from the longitudinal NHES/NHANES datasets. These spanned 1959–2008 and are comparable to fast rates of phenotypic change estimated for plant and animal populations in the wild (i.e. typically ranging between 0.1 and 0.3 standard deviations per generation). They provide support for the growing recognition that modern human populations continue to change and evolve in response to changing environmental and cultural conditions.

What are recent studies telling us about how we are changing?

An interesting observation arises from recent studies, including Krieger et al., which utilized health records of contemporary human populations. This is the consistency in the direction of phenotypic change for certain traits of medical significance such as weight and cholesterol. Over a few generations defined by Krieger et al., weight and body mass index increased, which is similar to other studies examining this trait in other contemporary human populations measured across similar time periods (see Table 2, Stearns et al.). Cholesterol also shows a consistent decline in two different US studies suggesting the health implications of these changes may also be similar across populations. Other traits such as height do not show such consistent phenotypic shifts.

In summary, the study by Krieger et al. demonstrates that the haldane has the potential to be extremely useful in public health research—standardizing phenotypic change into the historical context of a haldane will make future comparisons across studies and different time periods easy and meaningful. This is especially important now that many long-term health studies are beginning to produce data spanning several generations. Given the known complex interactions between quantitative human traits of medical importance, partitioning the various genetic and non-genetic sources of variation remains an important challenge. It promises to provide better resolution of how generational phenotypic shifts relate to changing patterns of modern disease epidemics such as obesity and cardiovascular disease.

Funding

S.G.B. is supported by Marie Curie International Fellowship FP7-PEOPLE-2010-IIF-276565 and Copenhagen University.

Conflict of interest: None declared.

References


