The Emergence and Persistence of Inequality: Introduction

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I. Emergence

1. Pre-holocene (24,000ya): humans lived in foraging-bands, not much economic differentiation.

2. Inequality emerged after domestication of plants and animals (class societies)

a. Keatly Creek fishers: sedentary population demonstrates intergenerational transfer of wealth (prime fishing sites)

3. Explanations of inequality emergence

a. climate change (Boyd et al 2001)

b. food storage (Kuijt 2008, Testart 1982)

c. control of circumscribed resources, such that cost of desertion are high (Boone 1992)

d. promotion of luxury consumption and ceremonial display (Hayden 2001)

II. Intergenerational Transmission of Wealth

1. Kinds of wealth- skills in subsistence, social connections, land, livestock, material possessions, somatic wealth (body weight)
   * + Material
     + Relational
     + Embodied
2. Steady state balance- children of both very rich and very poor are closer to the mean- then offsetting injection of new inequality due to shocks

3. Transmission Enhancing Mechanisms

* Wealth transmission is statistical association between offspring’s and parent’s wealth (% difference in offspring wealth associated with % difference in parental wealth: Beta)
* Positive Assortment: wealthy sharing with wealthy (limits regression to mean)
* Cumulative Advantage- holdings grow over time
* Wealth acquired in parent’s allows offspring to withhold others from it’s use (knowledge- i.e. tool making or where to find honey)
* \*\*disparities emerge due to differences in degree in transmission\*

III. Measuring Wealth Transmission, Importance and Inequality

1. Beta: probability that offspring whose parent is in top quintile, will also end up in top quintile
2. Alpha: “importance weighted” average

**Goodman et al. 2005 Social Inequality in Biomarkers of Cardiovascular Risk in Adolescence**

Objective: explore associations between socioeconomic status and a range of biomarkers reflective of cardiovascular risks, and a cumulative physiological risk score among adolescents.

Why Adolescence? (1) physiological changes associated with pubertal development that reflect alterations in central regularoty systems such as the HPA axis (rise in insulin resistance) (2) ability to think abstractly develops, which may lead to renewed sense of social surroundings and hyper-activated HPA stress response (3) evidence that asymptotic CVD begins early in life

Results: After adjusting for BMI, association btwn parent educ and insulin, ins- resistance, and waist circum became non-significant. However, relationship btwn parent educ and glucose, LDL and HDL remained signif

Mean cumulative risk score was one, but 1/5 did have cumulative risk of 3 or more.

Lower parenta educ was sign associated with higher cumulative risk score in ordinal logistic regression.

Discussion:

Parental education is assoc with multiple risks in adolscents: which suggests strong intergenerational transfer of education’s effect on cardiovascular health.

McDade, T. W., Tallman, P. S., Madimenos, F. C., Liebert, M. a, Cepon, T. J., Sugiyama, L. S., & Snodgrass, J. J. (2012). Analysis of variability of high sensitivity C-reactive protein in lowland Ecuador reveals no evidence of chronic low-grade inflammation. *American Journal of Human Biology : The Official Journal of the Human Biology Council*, *24*(5), 675–81. doi:10.1002/ajhb.22296