Determinants of mid-life health behavior

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Planning Meeting on Socioeconomic Status and Increasing Mid-Life Mortality
Keck Center of the National Academies of Sciences, Engineering, and Medicine
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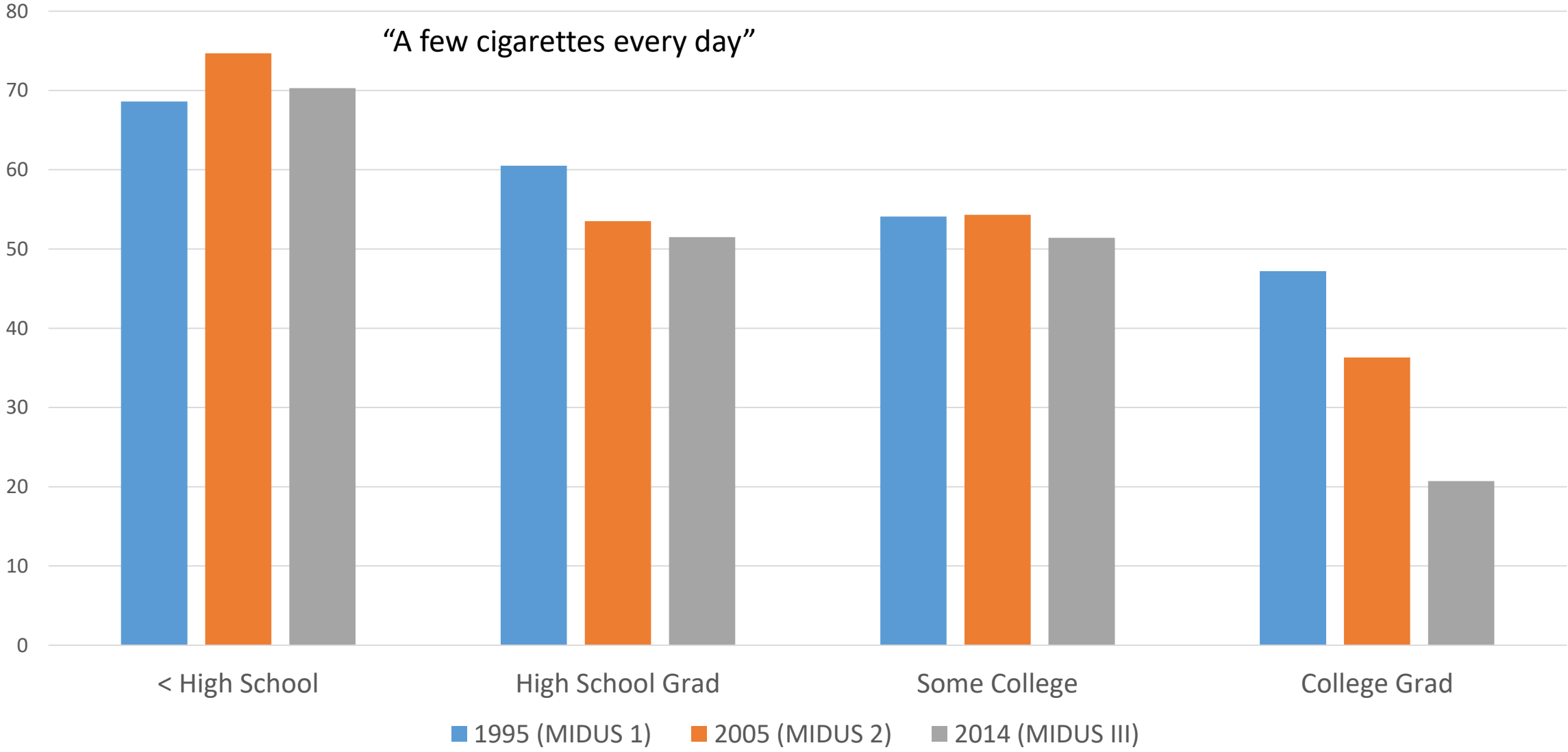
Socioeconomic Disparities in Health Behaviors

Fred C. Pampel,[1] Patrick M. Krueger,[2] and Justin T. Denney[3]

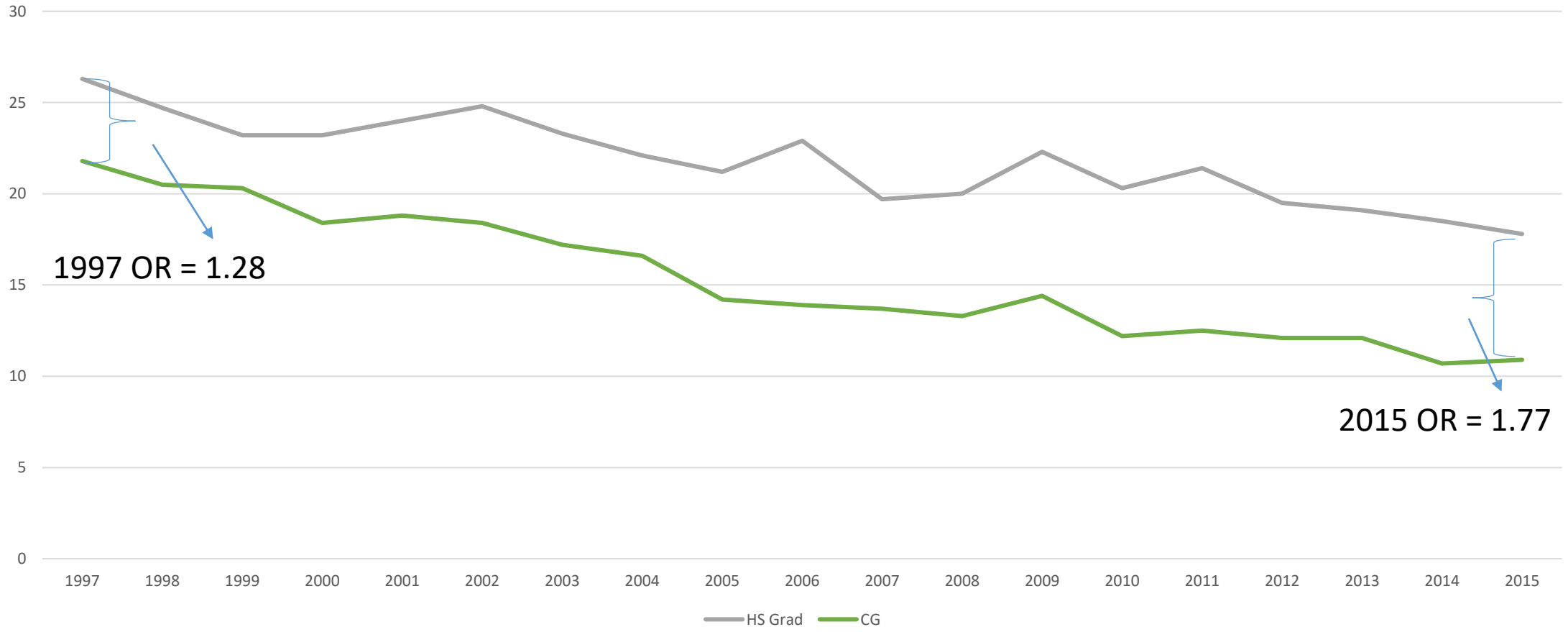
1. Most consistent SES factor is education
2. Strongest association is with smoking
3. Smoking, exercise, and BMI all cluster into a health lifestyle (Cockerham)
4. Changes in these associations over time is CRITICAL for understanding the mechanisms

SES Variables	Currently smokes	No exercise	BMI obese
Education (years)			
0–11	2.9*	2.8*	1.5*
12	2.4*	2.1*	1.5*
13–15	2.1*	1.4*	1.5*
16+	1.0	1.0	1.0
Occupation			
Labor-Farm	1.2	1.7*	1.2
Protect-Service	1.1	1.2	1.4*
Admin-Sales	1.1	1.2	1.2
Prof-Manager	1.0	1.0	1.0
Income			
Low	1.5*	1.9*	1.2
Middle low	1.1	1.6*	1.0
Middle high	1.0	1.2	1.1
High	1.0	1.0	1.0
Unemployed			
Yes	1.6*	0.7*	1.0
No	1.0	1.0	1.0
Housing			
Rent	1.5*	1.1	0.9
Own	1.0	1.0	1.0

Lifetime regular smoker status by education level among midlife adults (45-55) across three recent decades



Ever smoked 100 cigarettes in your life (NHIS)45- 55 year olds by education



Fundamental Cause Theory (Link and Phelan 1995)

Point 1: Context, cohorts, and social epidemiology

- Social Epidemiology is characterized as “the branch of epidemiology that studies the social distribution and social determinants of states of health.” (Heymann 2000: 6).
  - Eco-Social domains (social, economic, institutional and built environment) that are multilevel in nature.
- Embodiment: “a concept to how we literally incorporate, biologically, the material and social world in which we live from conception to death” and “a biological expression for social relations” (Krieger 2001: 672)
- Pathways to embodiment (physical, **behavioral**, psychological)
- Important but too descriptive. Need for theory (Frohlich).

Collective health lifestyles

Pergamon

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EPIDEMIOLOGY AND THE WEB OF CAUSATION:
HAS ANYONE SEEN THE SPIDER?

NANCY KRIEGER

Sociology of Health & Illness Vol. 23 No. 6 2001 ISSN 0141–9889, pp. 776–797

A theoretical proposal for the relationship between context and disease

Katherine L. Frohlich,[1] Ellen Corin[2] and Louise Potvin[3]

Krieger → the spider-web analogy

Brought the eco-social environment in which the emphasis on individual-level factors was shown to have limitations.

The response was distinguishing between compositional and contextual effects but almost no concern with understanding HOW context actually affects health.

Frohlich points to the distinction of SPACE versus PLACE. Where the latter gives us a much better understanding the **mechanisms**.

Collective Lifestyles (Frohlich et al. in reading)

- The critical need is to study the "relationship between **agency** (the ability for people to deploy a range of causal powers), **practices** (the activities that make and transform the world we live in) and **social structure** (the rules and resources in society)."
- Collective lifestyles are defined as an expression of a shared way of relating and acting in a given environment and context is created by relationships between people
- In this manner, lifestyles are SOCIAL PRACTICES and should be theorized and measured accordingly.
- Health behaviors are not just individual actions, they express identity, they have a shared meaning that is linked to social context.

Collective lifestyle emphasizes PLACE but can easily be extended to COHORTS.

Lifestyle viewed as a collective attribute, or what we henceforth will call *collective lifestyles*, then becomes an analytic tool with which we could strive to understand how structure and practices influence disease outcomes. While we are conscious of the limits of the term lifestyle, and the connotations that the word carries, we re-appropriate it and offer a collective dimension. Collective lifestyles are defined here not just as the behaviours that people engage in, but rather, as the relationship between people's social conditions and their social practices. Social conditions are here defined as factors that involve an individual's relationship to other people. This includes positions occupied within the social and economic structures of society, such as one's race, SES, gender, etc. (Link and Phelan 1995).

This **midlife cohort** is experiencing **a new context** as they age

The significance of cohorts for midlife health behaviors (Ryder 1965)

- Ryder (1965). The cohort as a concept in the study of social change
- “Successive cohorts are differentiated by the changing content of formal education, by peer-group socialization, and by idiosyncratic historical experience.”(844)
- “A cohort has an age distribution of its person-years of exposure, provided by its successive sizes age by age. The age distribution varies from cohort to cohort because of mortality and migration. Thus a cohort experiences demographic transformation in ways that have no meaning at the individual-level of analysis because its composition is modified not only by status changes of the components but also selective changes of membership.” (845)
- “If age-specific norms, or the context within which they are being applied, change through time, cohort experiences will be differentiated” (846)

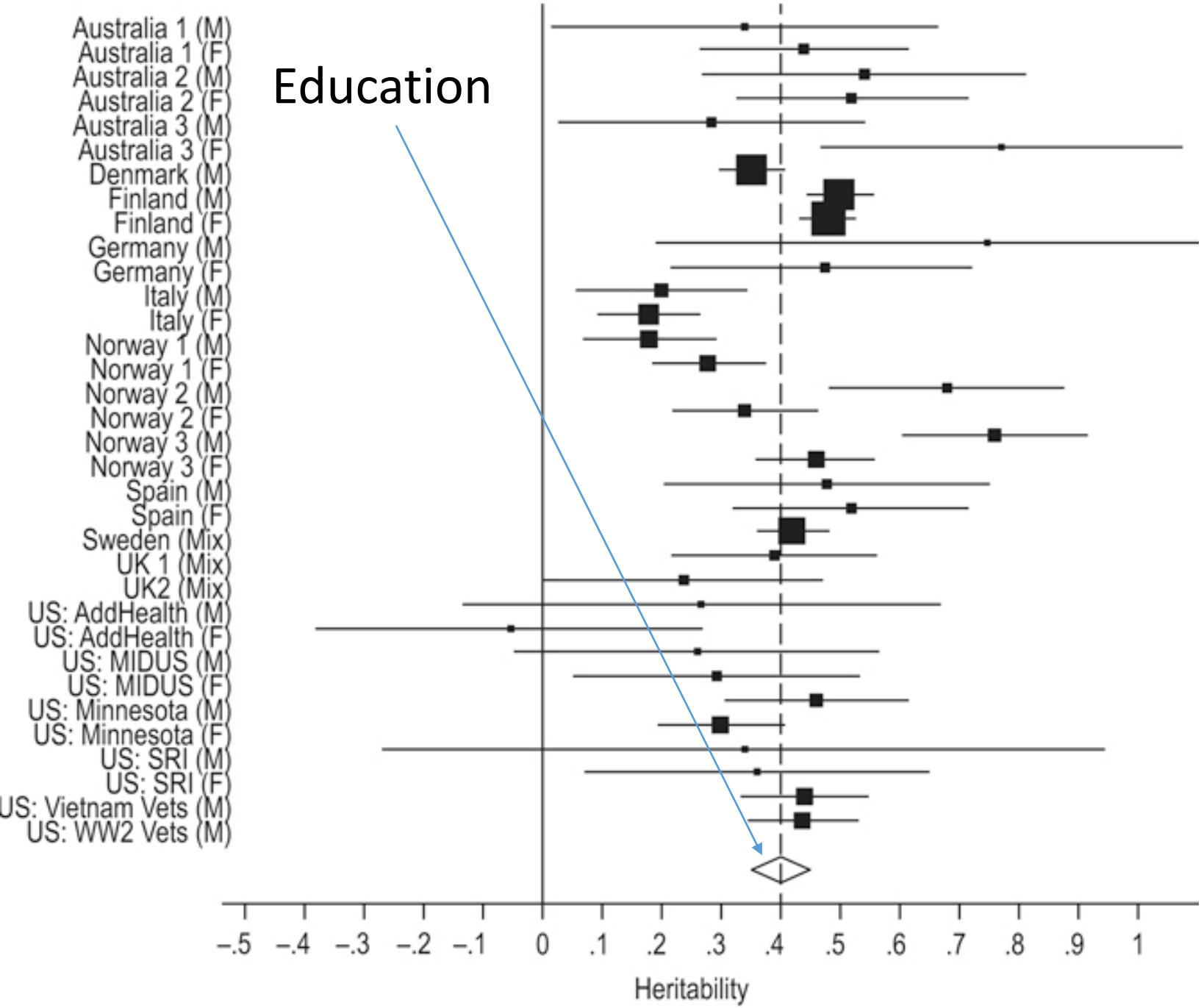
Ryder, Cohorts, and Education

- "The consequences of distinctive educational preparation prevail in the cohort's occupational flow chart." Accordingly, increasingly limited occupational selection for those with lower levels of education is UNIQUE to this cohort.
- When speaking about the possibility of social change, Ryder (1965) says that most people take two things for granted (1) that social change cannot occur without personality change; and (2) that personality change is only possible during childhood but it is then fixed. But he says that "the social system rather than the personality system belongs at the center of any model of social transformation." It is a social process in which cohort experiences and resources are the place that we start our interrogaion.
- Cohort uniqueness is fundamental to social change. How cohorts experience education and health behaviors may enable or limit social change.

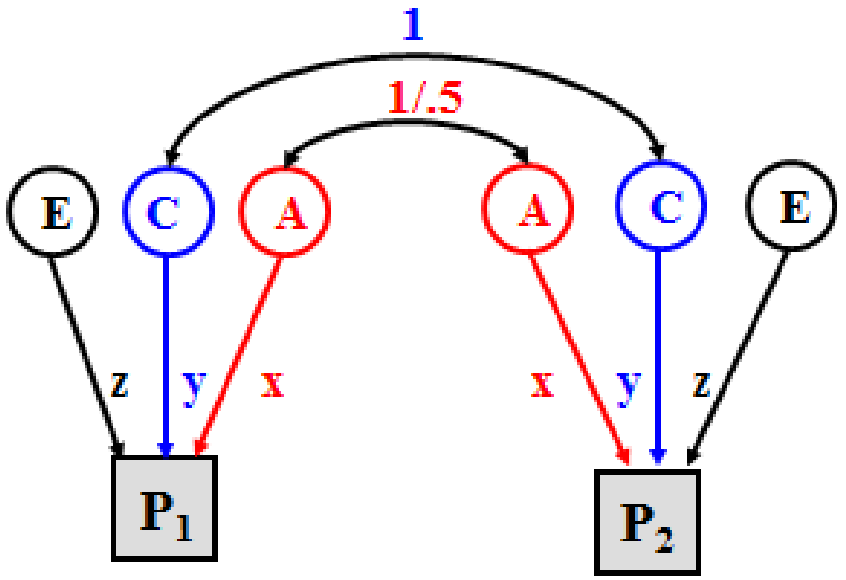
Cohorts and continuity across the lifecourse

- The two key components
  - 1) social change implies a transformation of the relative contributions to socialization made by the various agencies of socialization (e.g., k-12 and post secondary education).
  - 2) this transformation identifies a cohort as a social reality, reflecting and implementing the social change to which it owes its existence.
- More importantly he highlights that families are the central agency of socialization followed closely by schools as children age
  - Points of entry for both genes that link education and smoking
- These early life cohort effects define the “sources of continuity in individual lives.”

Point 2: Genes and latent traits



By comparing the correlation of traits between identical and fraternal twins it is possible to infer the proportion of variation that is due to broad sense heritability



Heritability estimates ~ .40 (Branigan, McCallum, and Freese, 2013)

Genetics and health behaviors

Review

Nature Reviews Genetics **13**, 640-653 (September 2012) |

The continuing value of twin studies in the omics era

Jenny van Dongen, P. Eline Slagboom, Harmen H. M. Draisma, Nicholas G. Martin & Dorret I. Boomsma

Lifestyle and life events			
Exercise participation	0.48–0.71‡	37,051	89
Dietary patterns	0.41–0.48	3,262‖	90
Smoking initiation	M: 0.37; F: 0.55	Meta-analysis	147
Smoking persistence	M: 0.59; F: 0.46	Meta-analysis	147
Alcohol abuse or dependence	0.50–0.70	Review	148
Stressful life events	0.28	Meta-analysis	92

*Note that numbers refer to twin pairs unless stated otherwise, and most heritability estimates refer to the narrow-sense heritability (h^2; BOX 2). ‡Range of heritabilities from different countries or study samples. §Female twin pairs with child (offspring-of-twin design). ‖Only females. ¶The original paper reports estimates for various age categories from 3–71 years, separately for males and females. F, females; M, males.

Environmental moderation of genetic influences on smoking

State-Level Moderation of Genetic Tendencies to Smoke

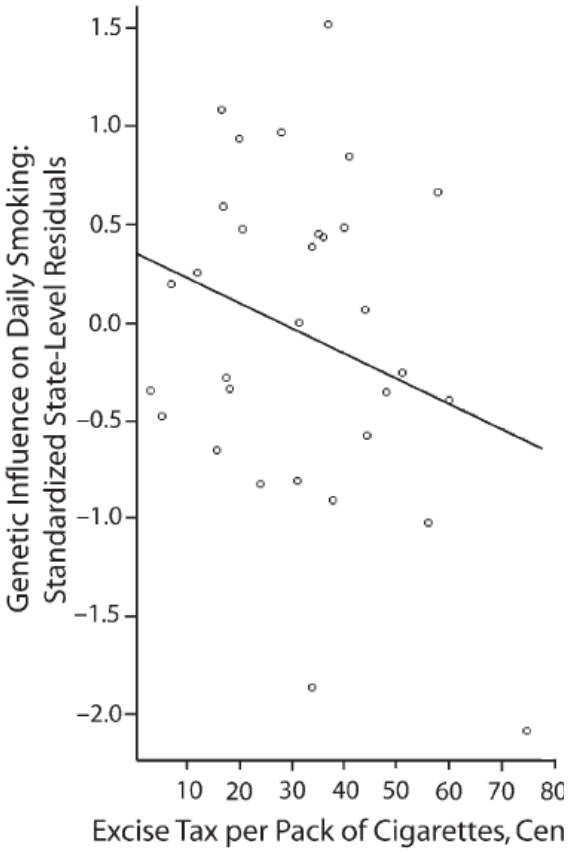
Jason D. Boardman, PhD

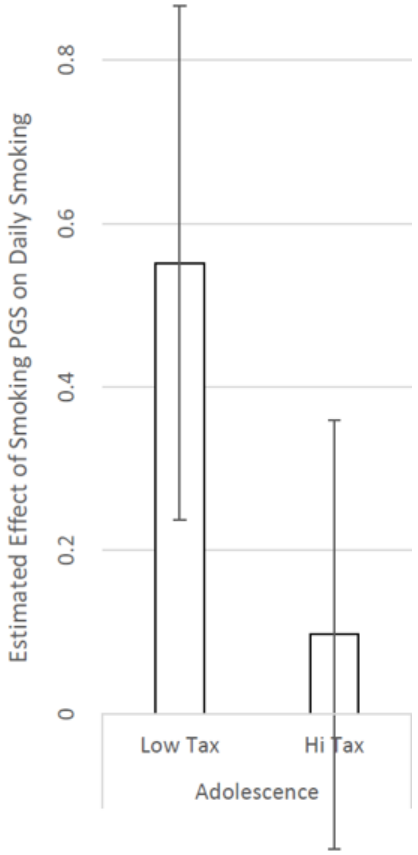
American Journal of Public Health | March 2009, Vol 99, No. 3

TABLE 2—Quantitative Genetic Parameter Estimates for Smoking Onset and Daily Smoking Status Among Twin and Sibling Pairs (N = 2060): National Longitudinal Study of Adolescent Health, Wave 2, September 1994–April 1995

	Smoking Onset, Variance (95% CI)	Daily Smoking, Variance (95% CI)
Heritability	0.42 (0.15, 0.66)	0.54 (0.29, 0.74)
Shared environment	0.21 (0.06, 0.36)	0.29 (0.14, 0.44)
Nonshared environment	0.37 (0.10, 0.19)	0.17 (0.09, 0.29)

Note. Heritability estimates and 95% confidence intervals (in parentheses) were calculated by using Mx version 1.7.03 (Medical College of Virginia, Richmond, VA). This freely available structural equation modeling package contains a number of standard procedures to decompose phenotypic variance into genetic and environmental components. A modified version of the script ctVCut2c.mx was used to estimate the parameters presented.



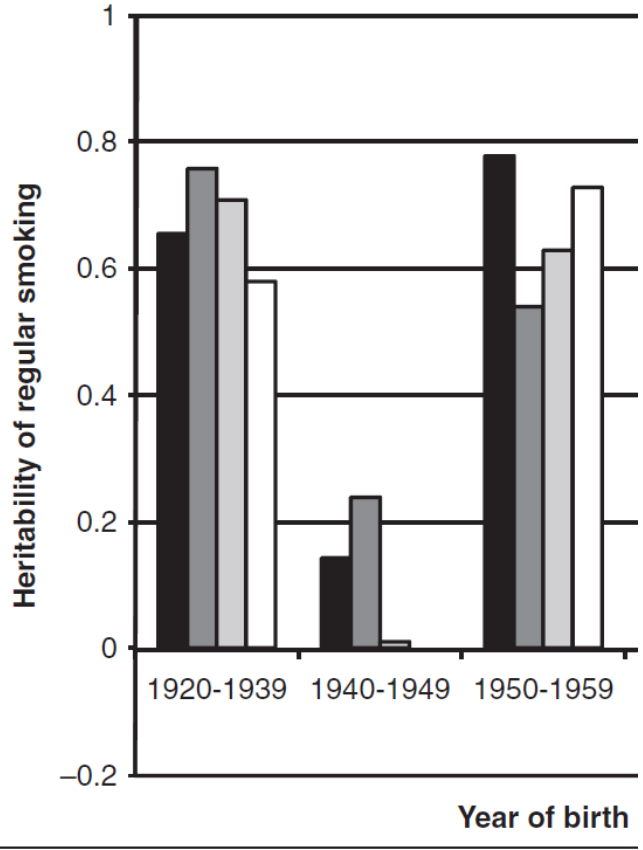


Point 3: Cohorts as environmental moderators of genes

Trends in the Genetic Influences on Smoking

Journal of Health and Social Behavior
51(1) 108–123
© American Sociological Association 2010
DOI: 10.1177/0022146509361195
http://jhsb.sagepub.com

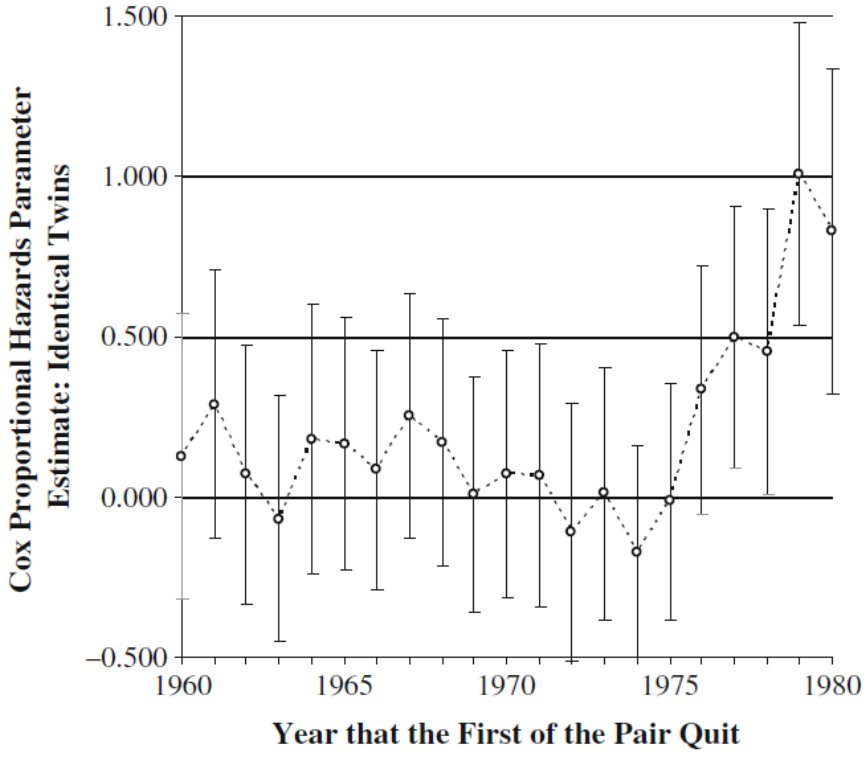
Jason D. Boardman[1], Casey L. Blalock[1], and Fred C. Pampel[1]



Demography (2011) 48:1517–1533
DOI 10.1007/s13524-011-0057-9

Population Composition, Public Policy, and the Genetics of Smoking

Jason D. Boardman · Casey L. Blalock · Fred C. Pampel · Peter K. Hatemi · Andrew C. Heath · Lindon J. Eaves

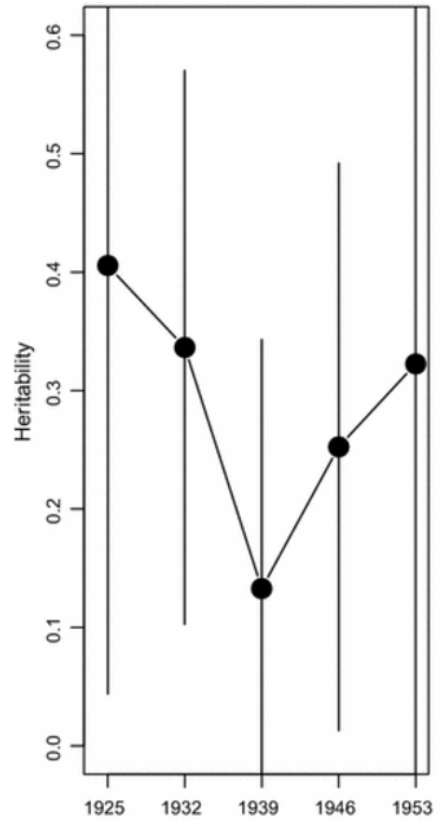


Behav Genet (2016) 46:31–42
DOI 10.1007/s10519-015-9731-9

ORIGINAL RESEARCH

Cohort Effects in the Genetic Influence on Smoking

Benjamin W. Domingue[1] · Dalton Conley[2] · Jason Fletcher[3] · Jason D. Boardman[4]



Point 4: rGxE (3 observations and 2 questions)

Observations

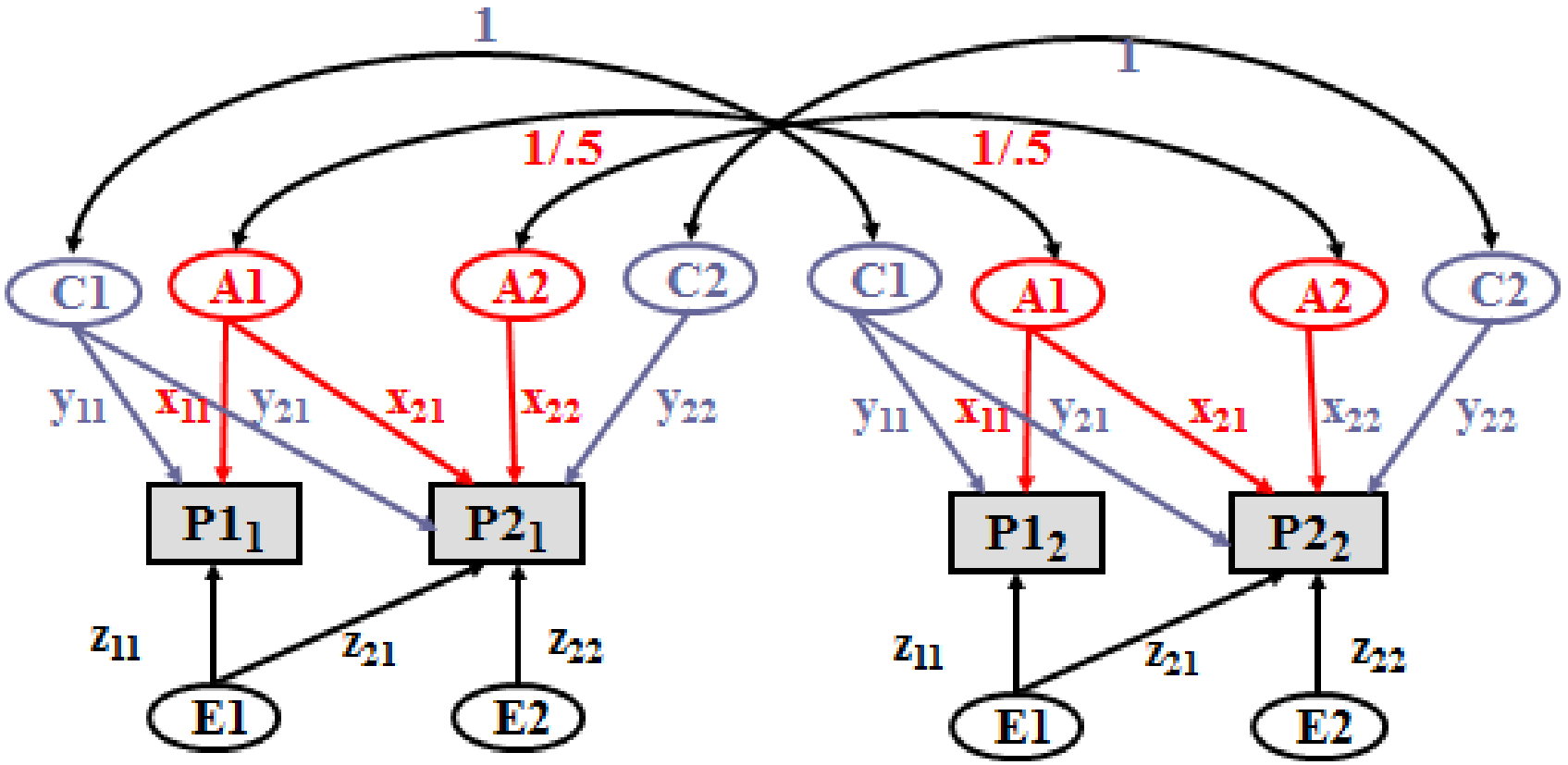
1. The heritability of regular smoking is roughly 40-50% and it has been increasing across birth cohorts
2. The heritability of education has remained at roughly 40% across birth cohorts
3. The correlation between education and smoking (negative) has increased across birth cohorts.

Questions

1. Are the genes related to education and smoking the same (genetic correlation)? [rG]
2. Has the genetic correlation between smoking and education increased across birth cohorts? [rGxE]

Using twins to evaluate the genetic covariance between education and smoking (Bivariate Cholesky models).

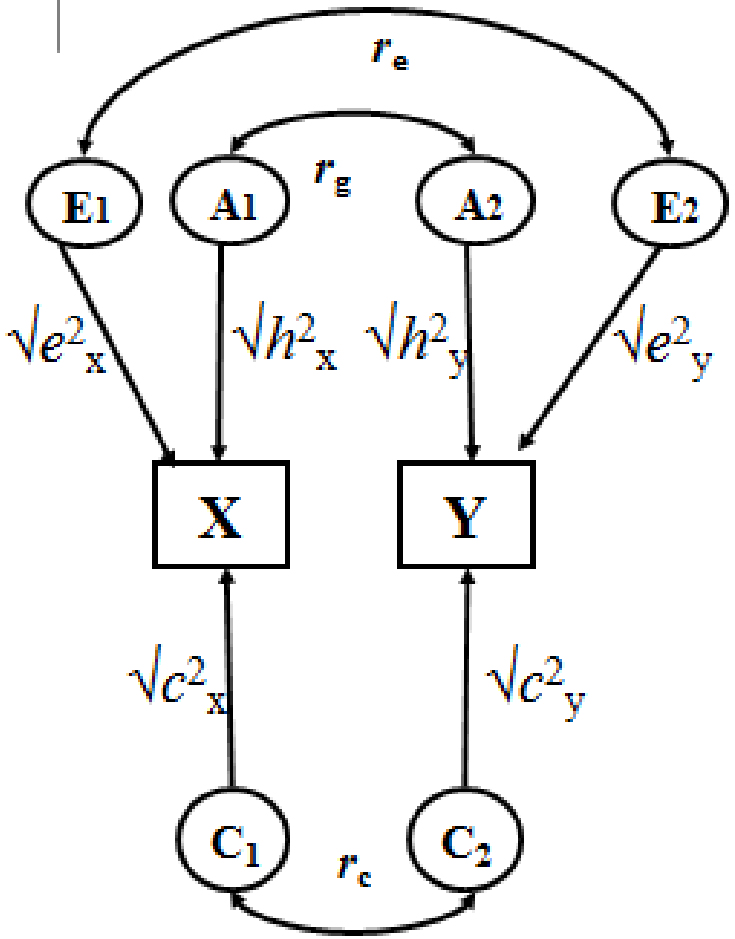
Compare the cross-twin cross-trait covariance.



If I know the education level of twin 1, can I predict the smoking status of twin 2 better among MZ twins than DZ twins.

Why is this useful?

The genetic correlation (rG) can be used in conjunction with the heritability of each trait and their corresponding bivariate correlation to assess the extent to which the two traits are correlated because of common genetic influences. One could say “25% of the association between education and health is due to common genetic influences”



Example: McCafferty et al. (2008): rg = -0.30

$$h^2_{x,y} = \sqrt{h^2_x} * r_g * \sqrt{h^2_y}$$

$$h^2_{x,y} = \sqrt{.29} * -.27 * \sqrt{.49} = -.10$$

$$\%G = -.10 / -.30 = .33$$

Genome wide Bivariate results-GCTA Using HRS

Variance-Covariance Estimates	BMI	Depression	SRH
Genetic Variance -- Health	10.489	0.010	0.210
	(2.170)	(.003)	(.059)
Genetic Variance -- Education	1.812	1.833	1.808
	(.518)	(.517)	(.517)
Genetic Covariance	-0.688	-0.071	-0.360
	(.750)	(.029)	(.129)
Environmental Variance -- Health	16.673	0.030	0.535
	(1.872)	(.003)	(.052)
Environmental Variance -- Education	4.772	4.753	4.775
	(.453)	(.453)	(.453)
Environmental Covariance	-0.149	-0.035	-0.336
	(.652)	(.026)	(.113)
Total Variance -- Health	27.162	0.040	0.745
	(.591)	(.001)	(.016)
Total Variance -- Education	6.583	6.586	6.583
	(.141)	(.141)	(.141)
Heritability -- Health	0.386	0.256	0.282
	(.075)	(.077)	(.076)
Heritability -- Education	0.275	0.278	0.275
	(.075)	(.075)	(.075)
Genetic Correlation (rG)	-0.158	-0.520	-0.585
	(.172)	(.198)	(.174)

Contents lists available at ScienceDirect

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journal homepage: www.elsevier.com/locate/socscimed

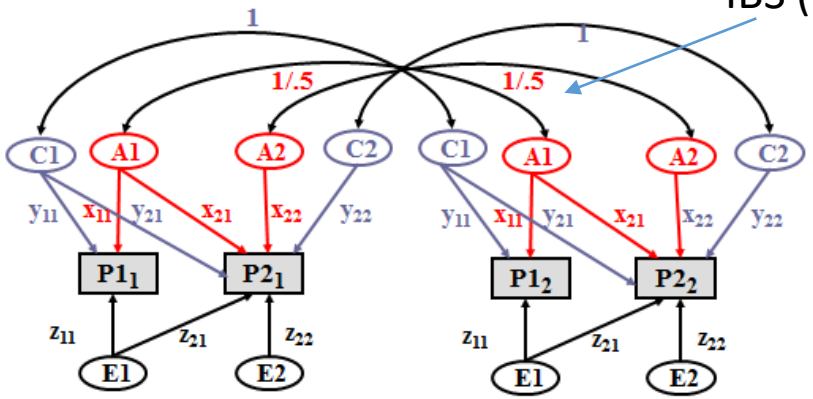
What can genes tell us about the relationship between education and health?☆

Jason D. Boardman [a, *], Benjamin W. Domingue [a], Jonathan Daw [b]

[a] University of Colorado, Boulder, United States
[b] University of Alabama, Birmingham, United States

	h2	rG	r(x, educ)	G	%G
Depression	0.26	-0.52	-0.38	-0.14	0.37
SRH	0.28	-0.59	-0.31	-0.16	0.53

IBS (pi_hat)



rGxE and the existing rG framework: cohorts?

- rG (smoking, education). Is this value increasing or decreasing across cohorts?
- What are the mechanisms?

Biological: r(ed, sm) changing. No.

Parental effects: constant h2 for education. No

Assortative mating: very small magnitude and little evidence that it has changed over time. No.

Mediated pleitropy

- FCT G->Ed->Sm (maybe)
- G -> Sm -> Ed (possible)
- G -> EP -> Ed & Sm (likely)

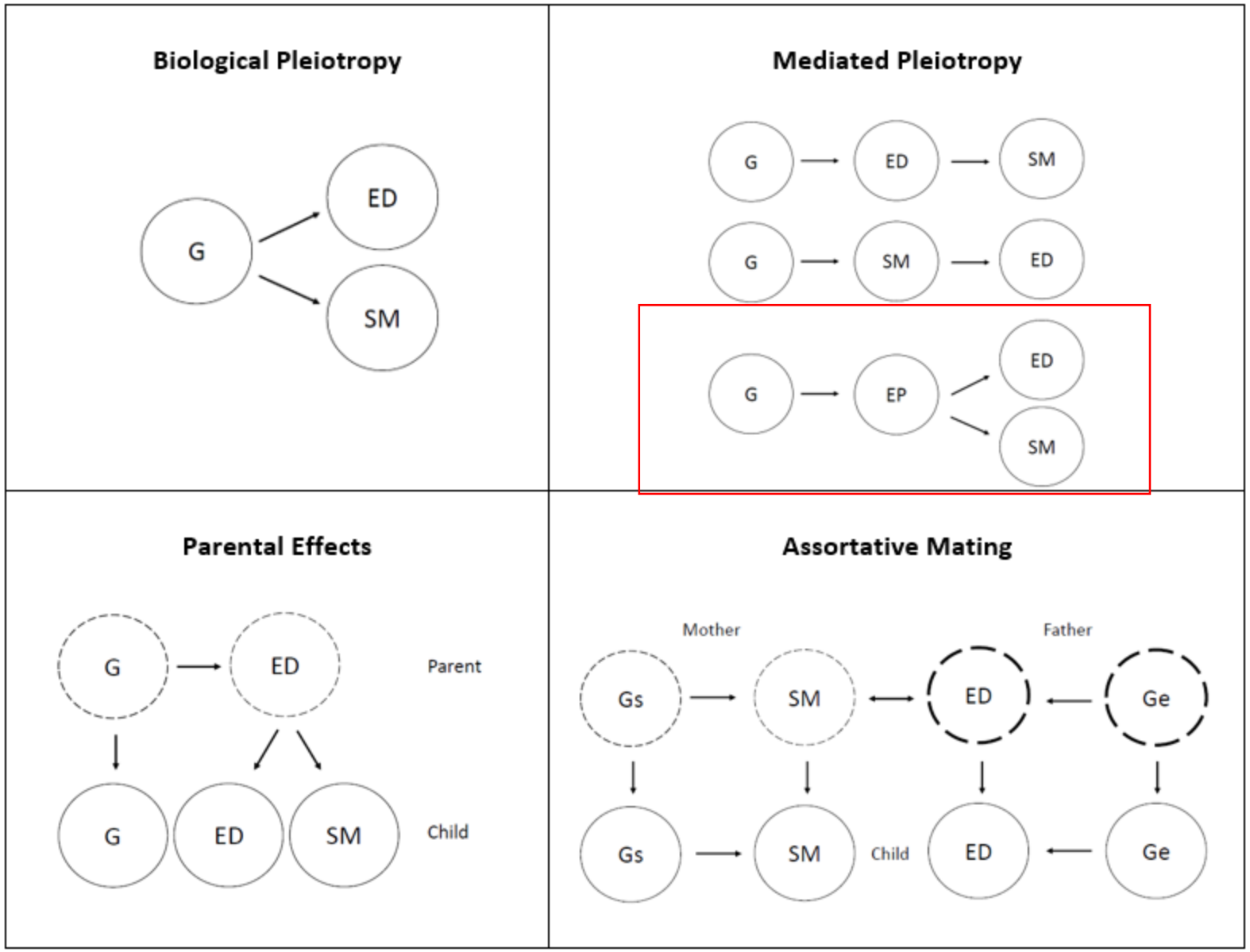
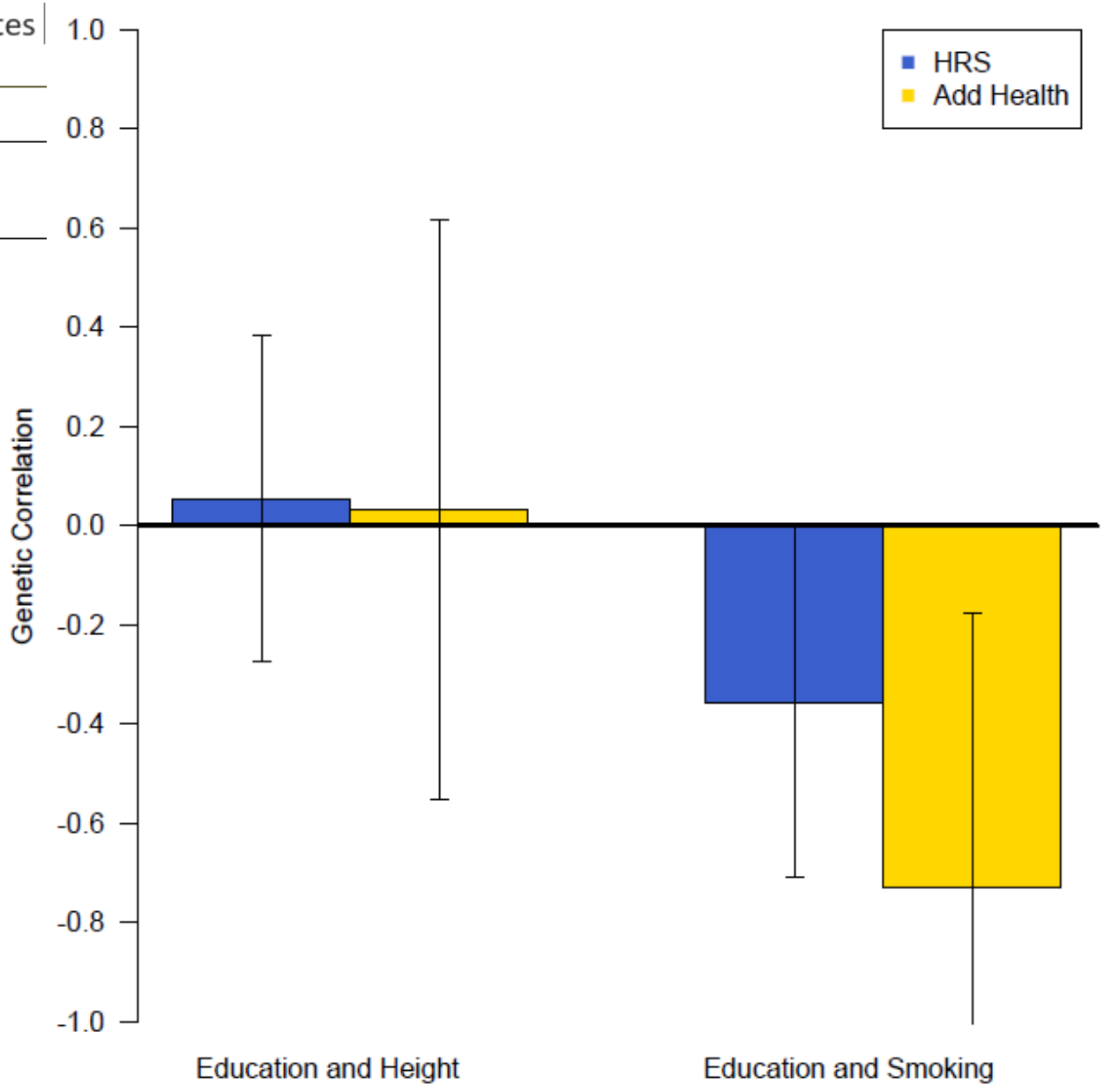


Figure 2. LDSC genetic correlations between education and height and between education and smoking in the HRS and Add Health, with 95% confidence intervals

LDSC estimates of pairwise genetic correlations and heritability estimates

COHORT	*rG*	*p*-value	h^2 Education	h^2 Smoking
HRS	-0.357	**0.047**	0.116	0.129
Add Health	-0.729	**0.010**	0.197	0.242



Same results with twins and a different data source

Cohort	h^2 Education	h^2 Smoking	rG	r(x,y)
All MIDUS (1920-1970)	0.40	0.53	-0.39	-0.22
MIDUS C1 (1920- 1945)	0.47	0.19	-0.17	-0.18
MIDUS C2 (1946- 1970)	0.27	0.55	-0.48	-0.25
Add Health (1975-1983)	0.37	0.57	-0.52	-0.25

Conclusions

- The relationship between education and smoking is increasing significantly over recent cohorts
- This association is primarily among college educated individuals who are reducing smoking at a far greater rate than other individuals.
- Each trait is moderately heritable
  - Education has been consistent over time
  - Smoking has increased over time
- Genetic correlation has increased dramatically in most recent cohort
  - HRS: rG explains roughly 14% of the association between ed -> smoking
  - Add Health: rG explains roughly 27% of the ed -> smoking association

Significance

- This cohort and possibly the next is experiencing a classification component (evocative rG) that is placing people in tracks that increase both low levels of education and high levels of smoking. This selection is increasingly due to genetic selection.
- Theories of SES and Health need to consider this selection pattern and what it means about the reproduction of inequality. Genes will be correlated with both traits but the significant component of the causal model is the institutional experience that is unique to each cohort.
- Nearly all of the ways that people emphasize aging with a sensitivity to cohort is the environmental influences. However, the non-random selection of genetically similar people into education and health lifestyle patterns which will complicate the role of COHORT as a purely environmental effect.
  - Consider, for example increasing role of genetically assortative mating