

Investigating peer influence using Social/Indirect Genetic Effects

Amelie Baud^{1,2}, Francesco Paolo Casale¹, Oliver Stegle¹

1. European Bioinformatics Institute, Cambridge

2. Psychiatry department, University of California San Diego

Background



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The Contribution of Social Effects to Heritable Variation in Finishing Traits of Domestic Pigs (*Sus scrofa*)

R. Bergsma,^{*,†,1} E. Kanis,[†] E. F. Knol^{*} and P. Bijma[†]

^{*}IPG, Institute for Pig Genetics, 6640 AA Beuningen, The Netherlands and [†]Animal Breeding and Genomics Centre, Wageningen University, 6700 AH Wageningen, The Netherlands

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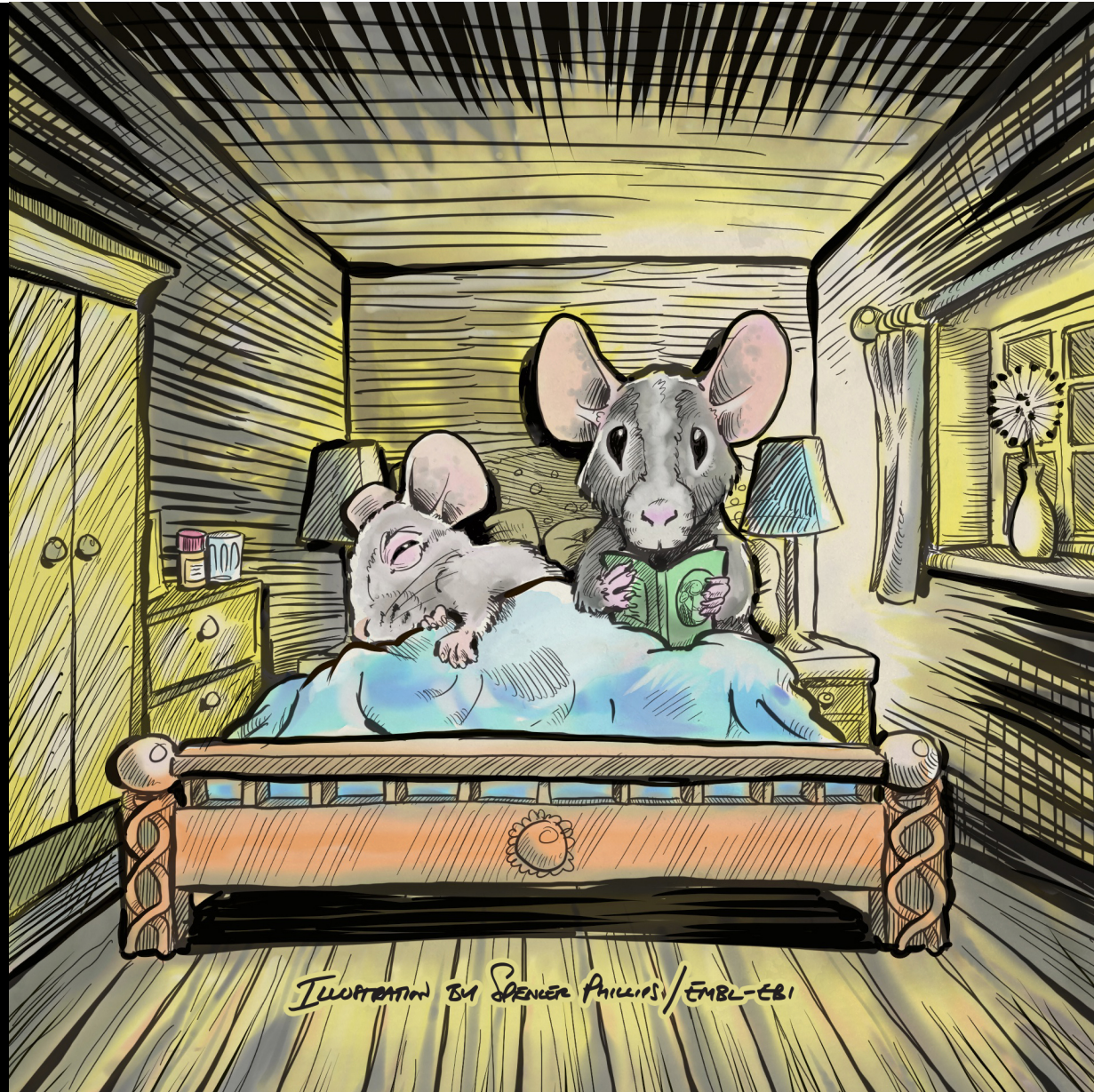


ILLUSTRATION BY SPENCER PHILLIPS / EMBL-ERI

Genetic Variation in the Social Environment Contributes to Health and Disease

Amelie Baud^{1*}, Megan K. Mulligan², Francesco Paolo Casale¹, Jesse F. Ingels², Casey J. Boh², Jacques Callebert³, Jean-Marie Launay³, Jon Krohn⁴, Andres Legarra⁵, Robert W. Williams², Oliver Stegle^{1*}

PLOS Genetics, 2017

PERSPECTIVE

The social genome: Current findings and implications for the study of human genetics

Benjamin W. Domingue^{1*}, Daniel W. Belsky^{2,3*}

PLOS Genetics, 2017

The social genome of friends and schoolmates in the National Longitudinal Study of Adolescent to Adult Health

Benjamin W. Domingue^{a,1}, Daniel W. Belsky^{b,c}, Jason M. Fletcher^{d,e,f}, Dalton Conley^g, Jason D. Boardman^{h,i},
and Kathleen Mullan Harris^{j,k,1}

PNAS, 2018

REVIEW

Deconstructing the sources of genotype-phenotype associations in humans

Alexander I. Young^{1,2,*}, Stefania Benonisdottir¹, Molly Przeworski^{3,4,*}, Augustine Kong^{1,*}

+ See all authors and affiliations

Science 27 Sep 2019:
Vol. 365, Issue 6460, pp. 1396-1400
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Genetic Nature or Genetic Nurture? Quantifying Bias in Analyses Using Polygenic Scores

Sam Trejo, Benjamin W. Domingue

doi: <https://doi.org/10.1101/524850>

This article is a preprint and has not been certified by peer review [what does this mean?].

Key questions for SGE research

- 1) quantify the impact of SGE on an outcome of interest
- 2) understand how SGE arise

Approach taken by others

Domingue et al. 2018; Kong et al. 2018, Bates et al. 2018,...

- in cohort 1: estimate the $\hat{\beta}$ for phenotype A
- in cohort 2: calculate PRS for social partners (friends or parents) and correlate with phenotype A' of focal individuals

Only looking at SGE mediated by phenotype A

Approach I propose: SGE GREML

$$y_f = \underbrace{X_f \underline{b}}_{\text{Phenotype of interest}} + \underbrace{a_{D,f}}_{\text{Aggregate DGE}} + e_{D,f} + \underbrace{W_f \underline{c} + \underbrace{Z_f a_S}_{\text{Aggregate SGE}}}_{\text{Aggregate SGE}} + \underbrace{Z_f \underline{e_S}}_{\text{Aggregate SGE}}$$

Advantages:

- makes no assumption as to the traits of social partners mediating SGE
- no prior knowledge required & no PRS required so can be used on *any* phenotype of interest

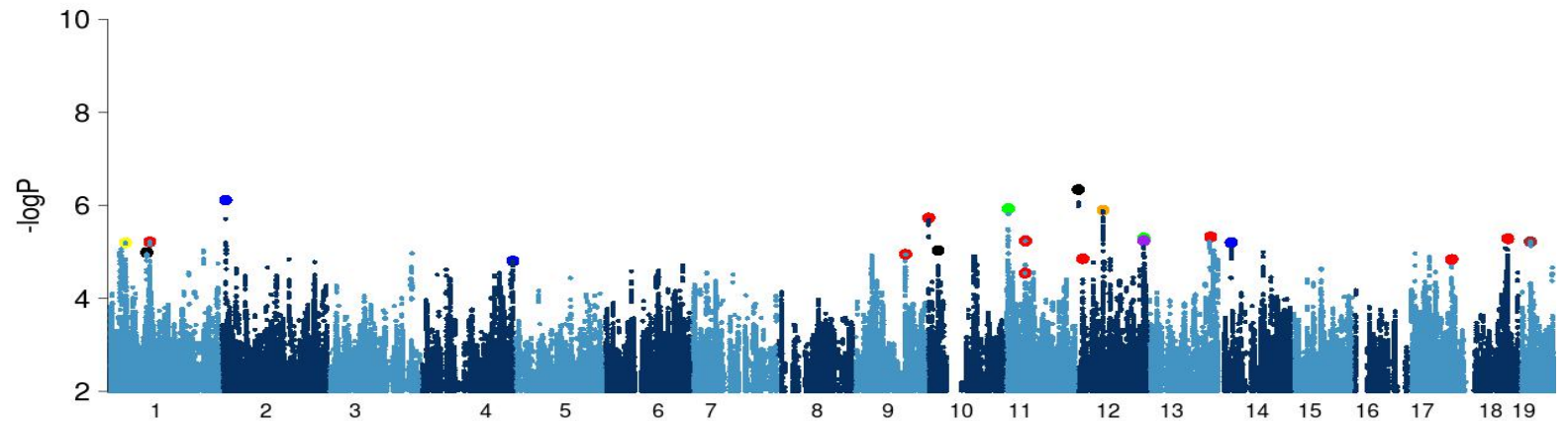
Results *in mice*

- SGE between cage mates affect a broad range of phenotypes including anxiety, immune system activation, body weight, **lung function**, **blood biochemistry** (e.g. LDL levels), and **rate of wound healing**
- SGE can be substantial: up to 29% of variance in lymphocytes proportions and SGE > DGE for 8 / 100 phenotypes
- correlation between DGE and SGE acting on the same phenotype is generally positive
- failing to account for SGE leads to biased estimates of DGE heritability

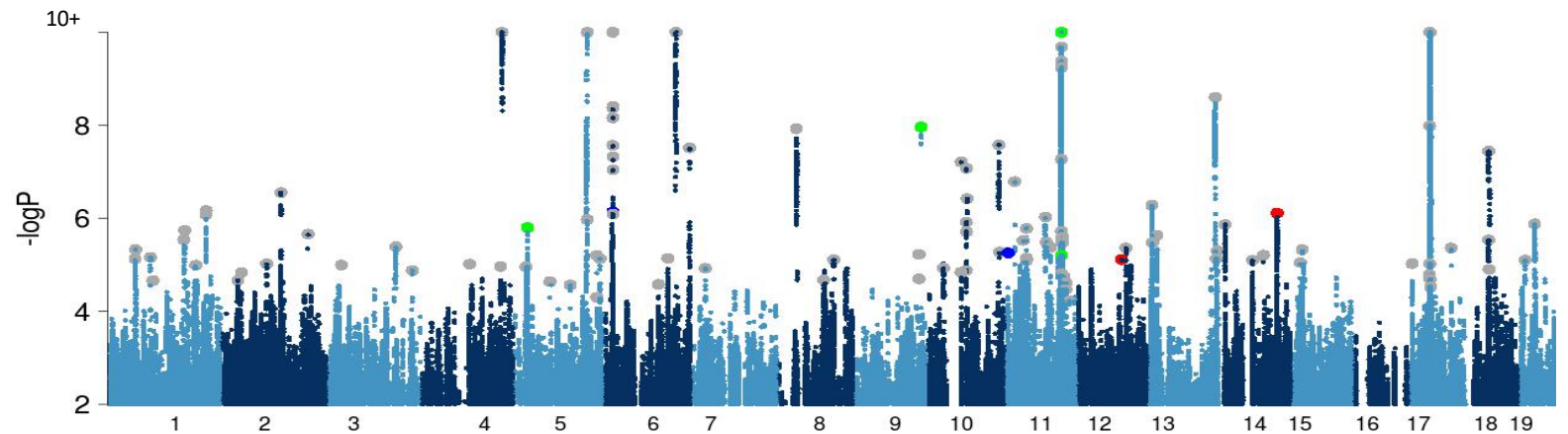
Baud *et al.*, *Genetic variation in the social environment contributes to health and disease*, PLOS Genetics (2017);

Once step further: SGE GWAS to understand how SGE arise

170 SGE GWAS:
21 SGE loci (FDR < 10%)



170 DGE GWAS:
118 DGE loci (FDR < 10%)



Baud *et al.*, bioRxiv (2019); <http://github.com/limix/SGE>