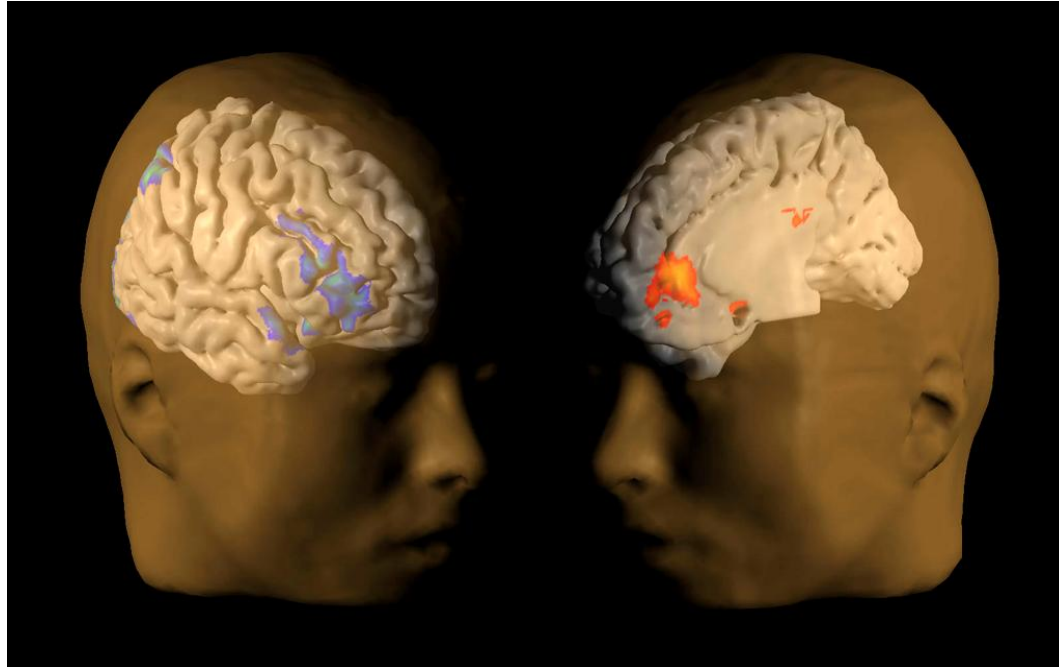


Biosocial Science



David Laibson
July 7, 2016

Outline:

- I. Biosocial science: definition**
- II. Neuroeconomics**
E.g., Multiple Systems Hypothesis
- III. Genoeconomics**

I. Biosocial science: definition.

Definition: Biosocial science is the study of the *biological microfoundations of economic cognition* and economic behavior.

- *Biological microfoundations* are neurochemical mechanisms and pathways, like brain systems, neurons, neurotransmitters, genetics, and epigenetics.
- *Economic cognition* is cognitive activity that is associated with economic perceptions, beliefs and decisions, including mental representations, emotions, expectations, learning, memory, preferences, and decision-making.

II: A neuroeconomics example

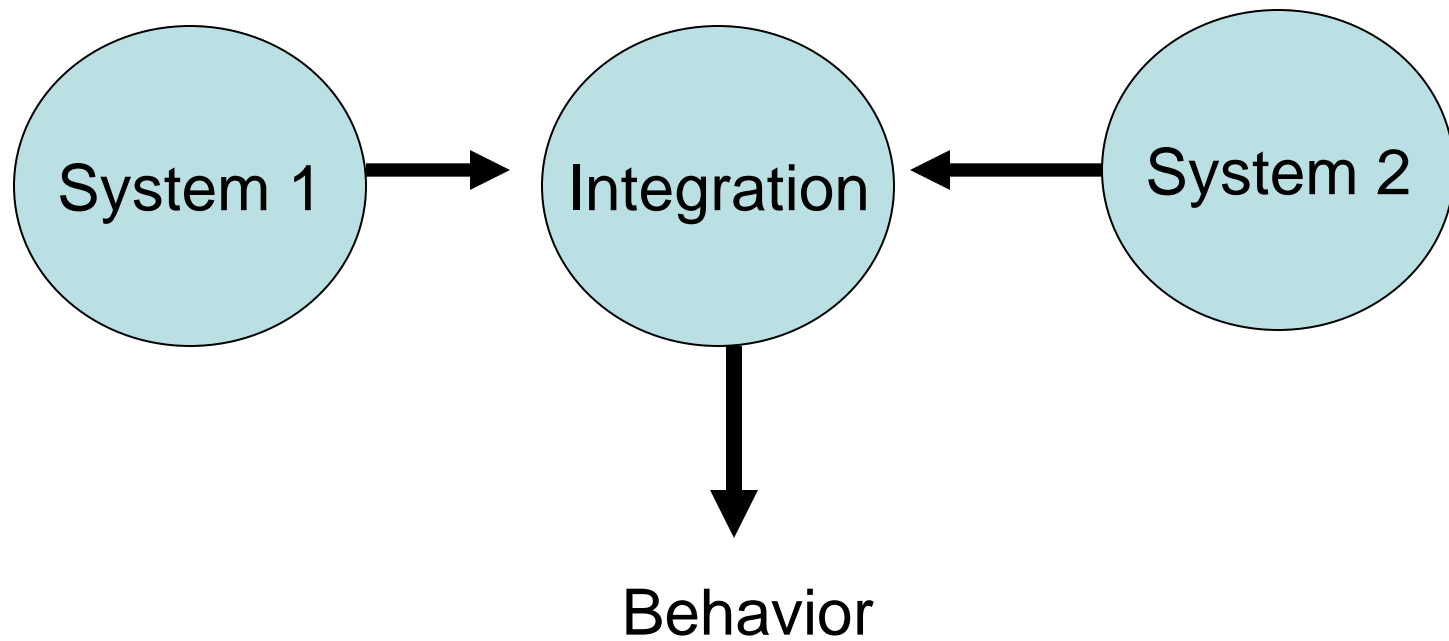
The Multiple Systems Hypothesis

- **Statement of Hypothesis**
- **Variations on a theme**
- **Caveats**
- **Neuroimaging**

Statement of Multiple Systems Hypothesis (MSH)

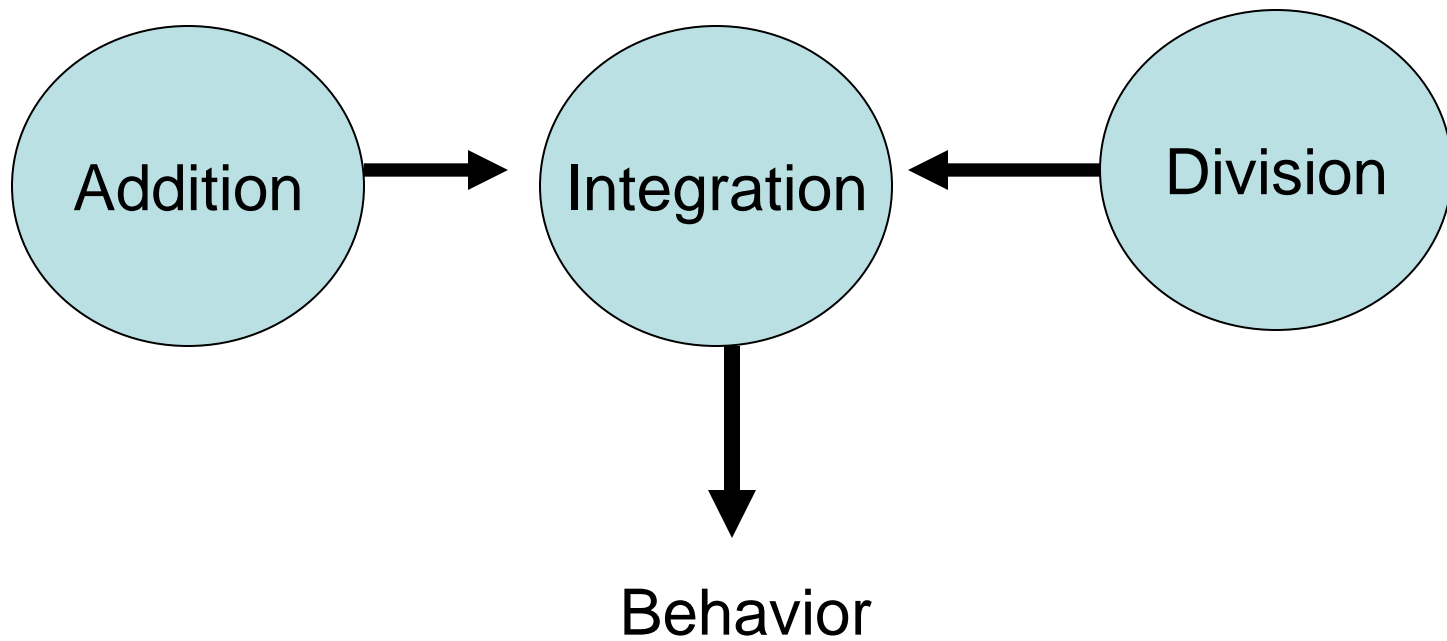
- The brain makes decisions (e.g. constructs value) by integrating signals from multiple systems
- These multiple systems process information in qualitatively different ways and in some cases differentially weight attributes of rewards (e.g., time delay)

An (oversimplified) multiple systems model



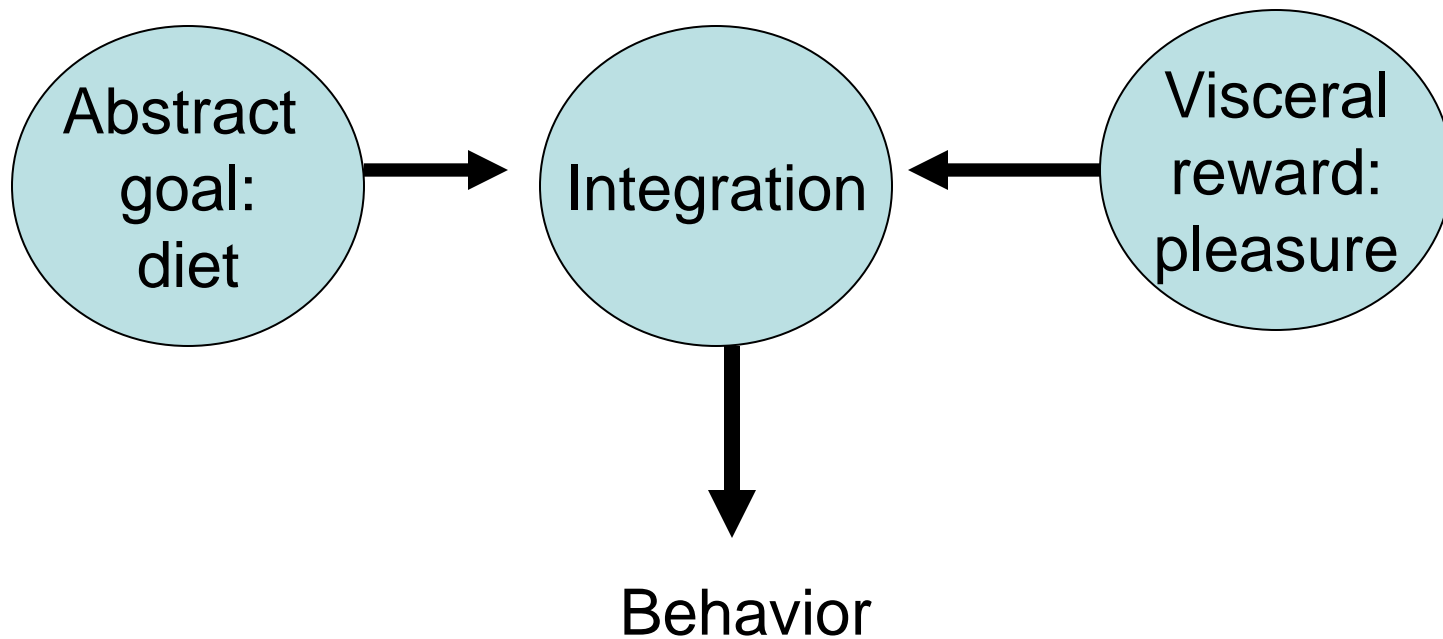
An uninteresting example

What is 6 divided by 3?



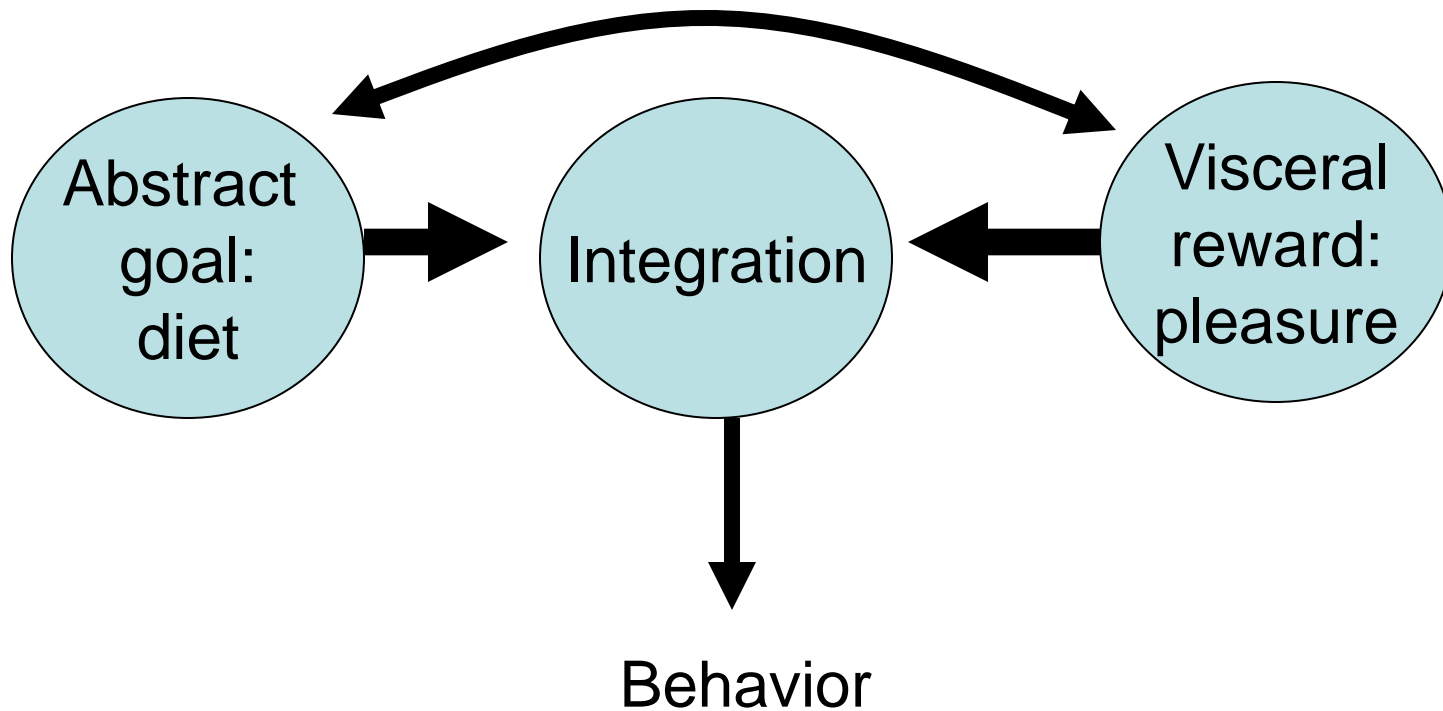
A more interesting example

Would you like a piece of chocolate?



A more interesting example

Would you like a piece of chocolate?



Variations on a theme

- Charioteer's two horses (Socrates/Plato, The Phaedrus, 370 BC):
“First the charioteer of the human soul drives a pair, and secondly one of the horses is noble and of noble breed, but the other quite the opposite in breed and character. Therefore in our case the driving is necessarily difficult and troublesome.”
- Interests vs passions (Smith)
- Superego vs Ego vs Id (Freud)
- Controlled vs Automatic (Schneider & Shiffrin, 1977; Benhabib & Bisin, 2004)
- Cold vs Hot (Metcalf and Mischel, 1979)
- System 2 vs System 1 (Frederick and Kahneman, 2002)
- Deliberative vs Impulsive (Frederick, 2002)
- Conscious vs Unconscious (Damasio, Bem)
- Effortful vs Effortless (Baumeister)
- Planner vs Doer (Shefrin and Thaler, 1981)
- Patient vs Myopic (Fudenburg and Levine, 2006)
- Abstract vs Visceral (Loewenstein & O'Donoghue 2006; Bernheim & Rangel, 2003)
- PFC & parietal cortex vs dopamine reward system (McClure et al, 2004)

Affective vs. Analytic Cognition

Frontal cortex

Frontal cortex
Caudate nucleus and putamen (striatum)

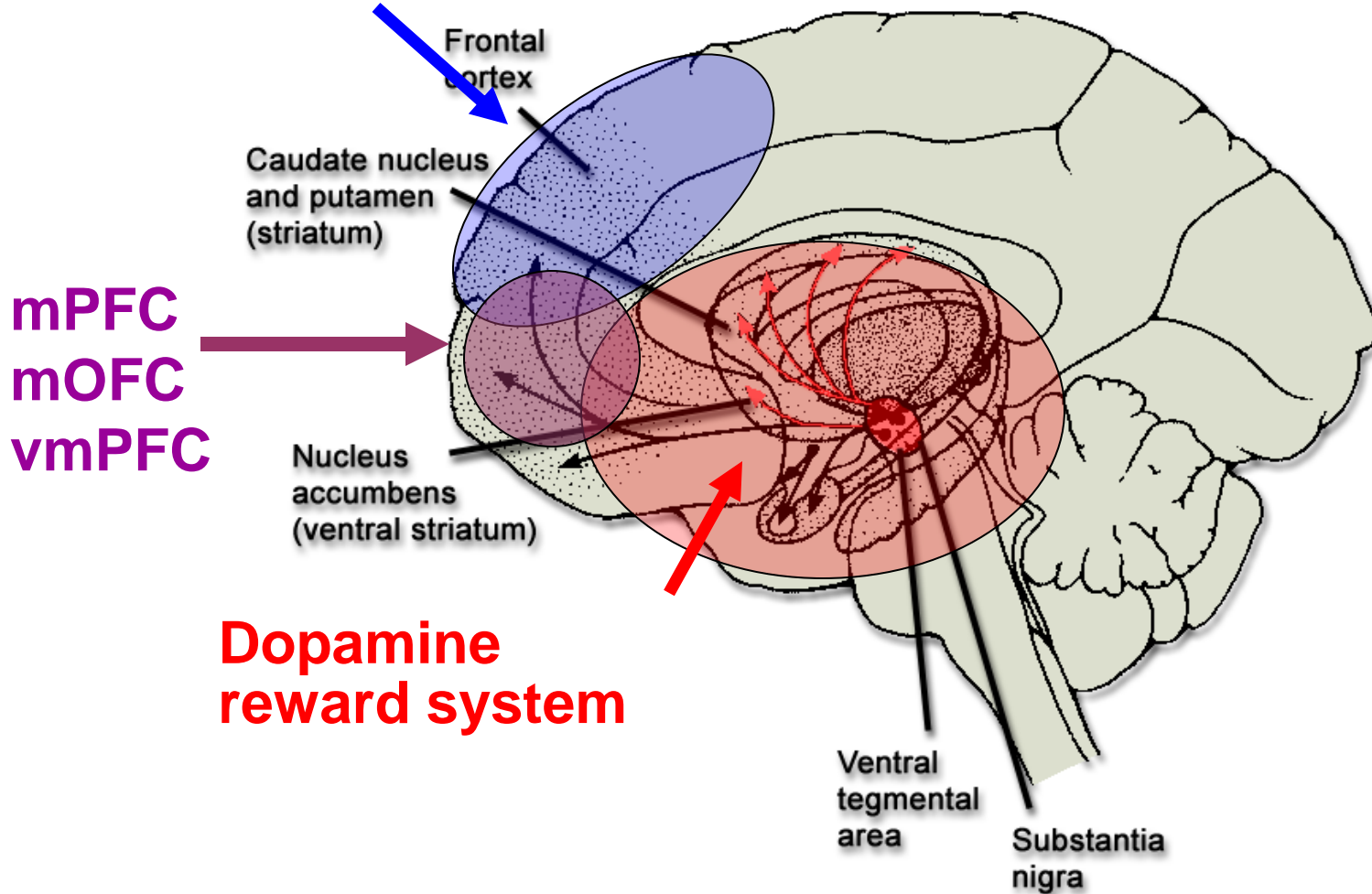
**mPFC
mOFC
vmPFC**

Nucleus accumbens (ventral striatum)

Dopamine reward system

Ventral tegmental area

Substantia nigra



Relationship to quasi-hyperbolic model

- Hypothesize that the fronto-parietal system (PFC) is patient
- Hypothesize that dopamine reward system (DRS) is impatient.
- Then integrated preferences are quasi-hyperbolic

	now	t+1	t+2	t+3	
PFC	1	1	1	1	...
DRS	1	0	0	0	...
Total	2	1	1	1	...
Total normed	1	1/2	1/2	1/2	...

Relationship to quasi-hyperbolic model

- Hypothesize that the fronto-parietal system is patient
- Hypothesize that dopamine reward system is impatient.
- Here's one implementation of this idea:

$$\begin{aligned}U_t &= u_t + \beta [\delta u_{t+1} + \delta^2 u_{t+2} + \delta^3 u_{t+3} + \dots] \\(1/\beta)U_t &= (1/\beta)u_t + \delta u_{t+1} + \delta^2 u_{t+2} + \delta^3 u_{t+3} + \dots \\(1/\beta)U_t &= \underbrace{(1/\beta - 1)u_t}_{\text{DRS}} + \underbrace{[\delta^0 u_t + \delta^1 u_{t+1} + \delta^2 u_{t+2} + \delta^3 u_{t+3} + \dots]}_{\text{fronto-parietal cortex}}\end{aligned}$$

Commonalities between classification schemes

Affective system

- fast
- unconscious
- reflexive
- myopic

Analytic system

- Effortful
- slow
- conscious
- reflective
- forward-looking
- (but still prone to error: heuristics may be analytic)

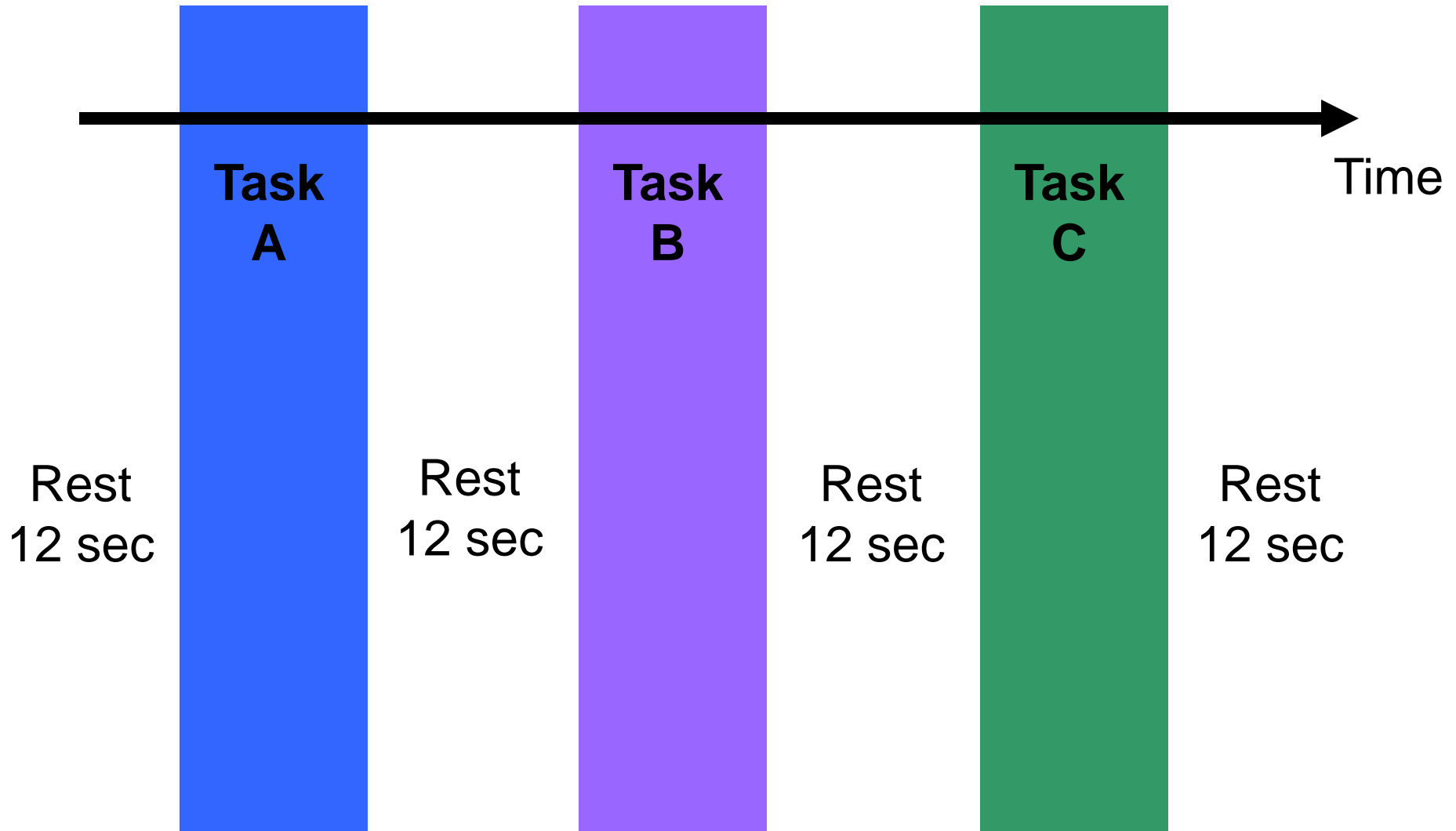
Functional Magnetic Resonance Imaging (fMRI)



Basic methodology

- Divide brain into 30,000+ voxels (cubes 2 mm on edge)
- Measure blood flow at the voxel level (BOLD signal)
- Relate blood flow to experimental task

Basic experimental design



Basic econometric methodology

- Run “regressions” (general linear model) relating BOLD signal to covariates:

$$BOLD_{v,i,t} = FE_i + \text{controls}_t + \text{task dummy}_t$$

- Indexes for voxel (v), subject (i), and time (t)
- Controls: time in scanner, lagged reward event, etc.
- Task dummy: decision, experience, event, etc.
- Analogous method: “contrast”

$$\text{Contrast at voxel } v = \sum_{i \in I} \left(BOLD_{v,i,t} - BOLD_{v,i,t'} \right)$$

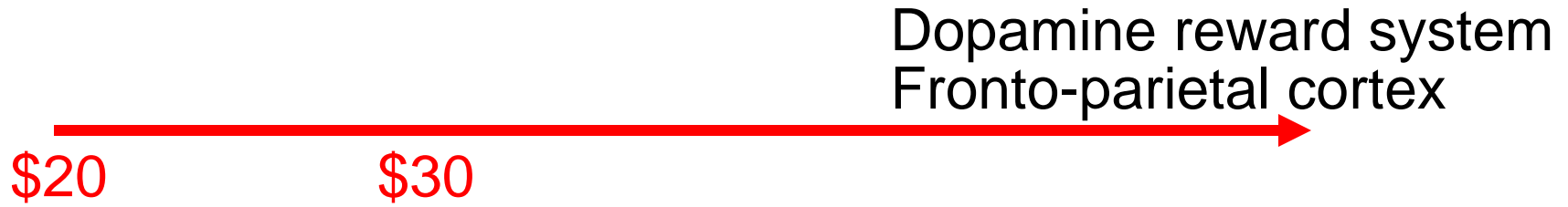
Multiple-testing problem (cf Vul et al 2010)

Don't worry:

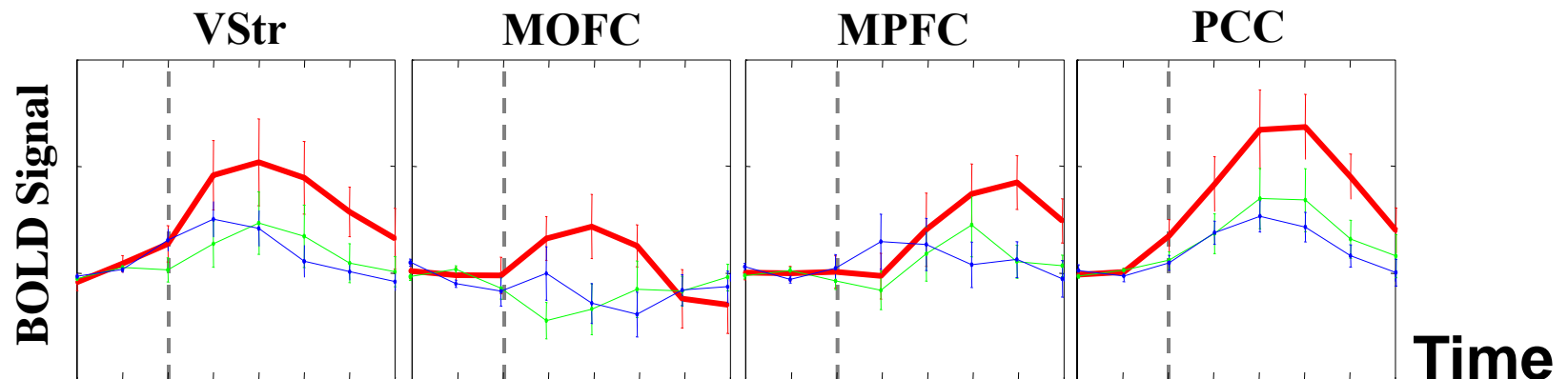
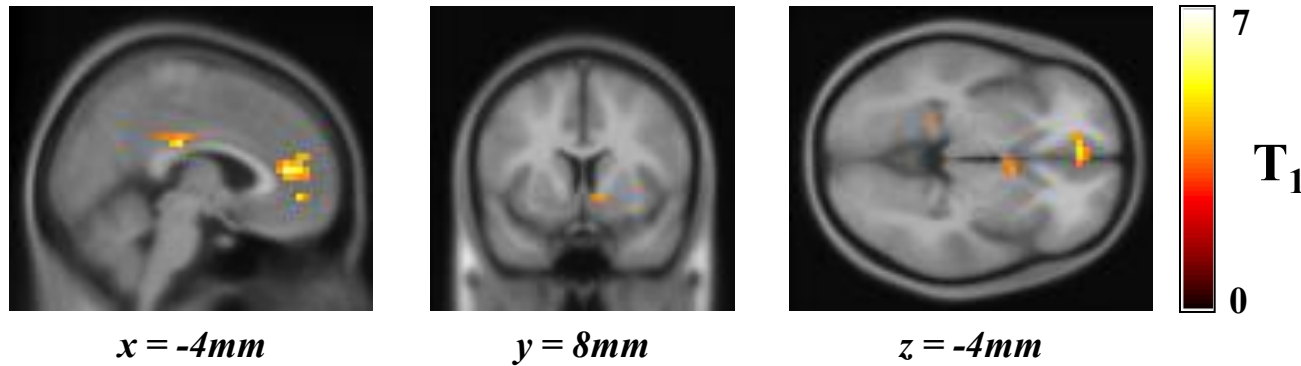
- Strict thresholds ($\alpha = 0.001$ and 5-voxel contiguity)
- Pre-specification of hypothesis (ROI)
- Replication
- Converging lines of evidence (fMRI, single neuron measurement, knock-outs, lesions, rTMS)
- This is the same multiple testing problem that hangs over all empirical research

Worry:

- Are all significant voxels reported?
- Were the specification searches reported?
- Are all GLM's (regressions) reported?
- Is the neuroscientific explanation of the data post-hoc?
- Shouldn't the effect size be adjusted for multiple testing



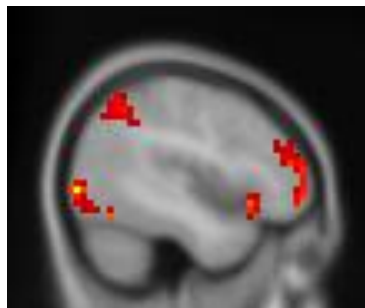
Regions that respond “only” to immediate rewards



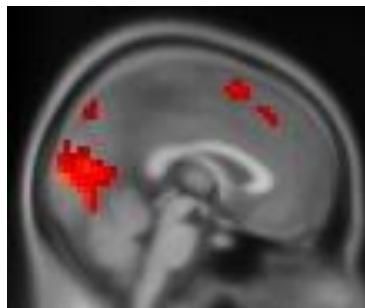
- Delay to earliest reward = Today**
- Delay to earliest reward = 2 weeks**
- Delay to earliest reward = 1 month**

0.2%
2 sec

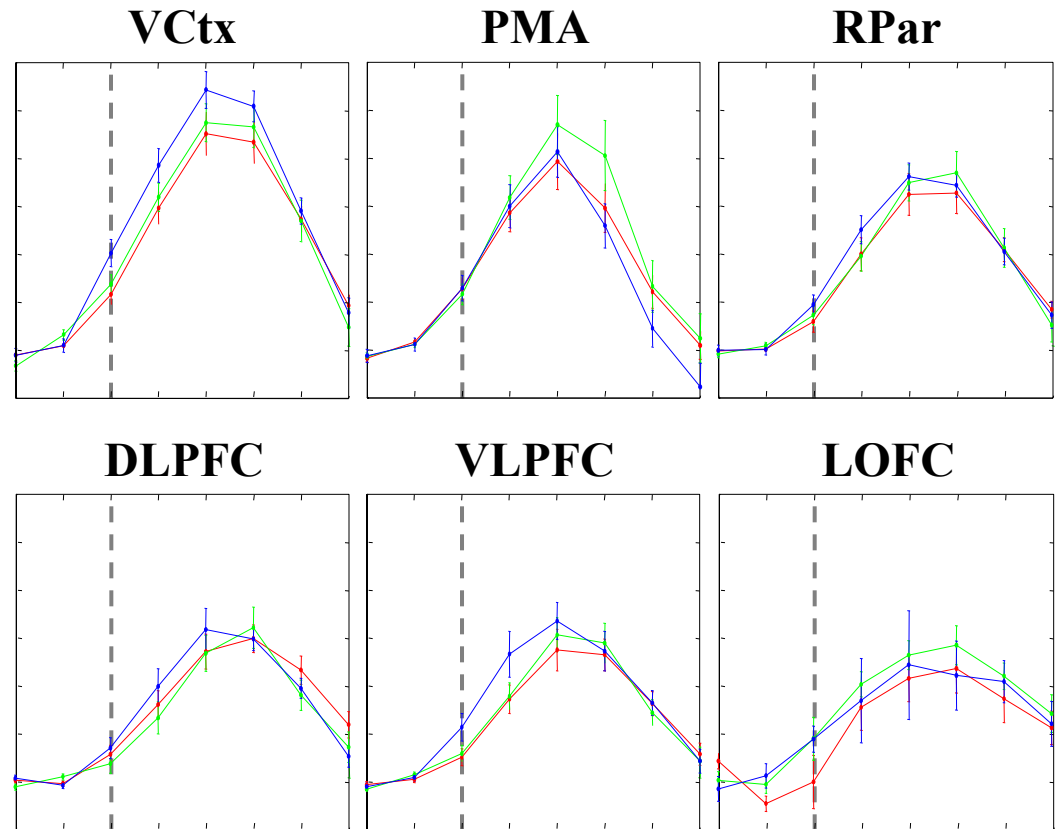
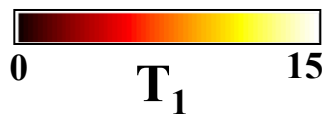
Regions that respond “equally” to all rewards



$x = 44mm$



$x = 0mm$



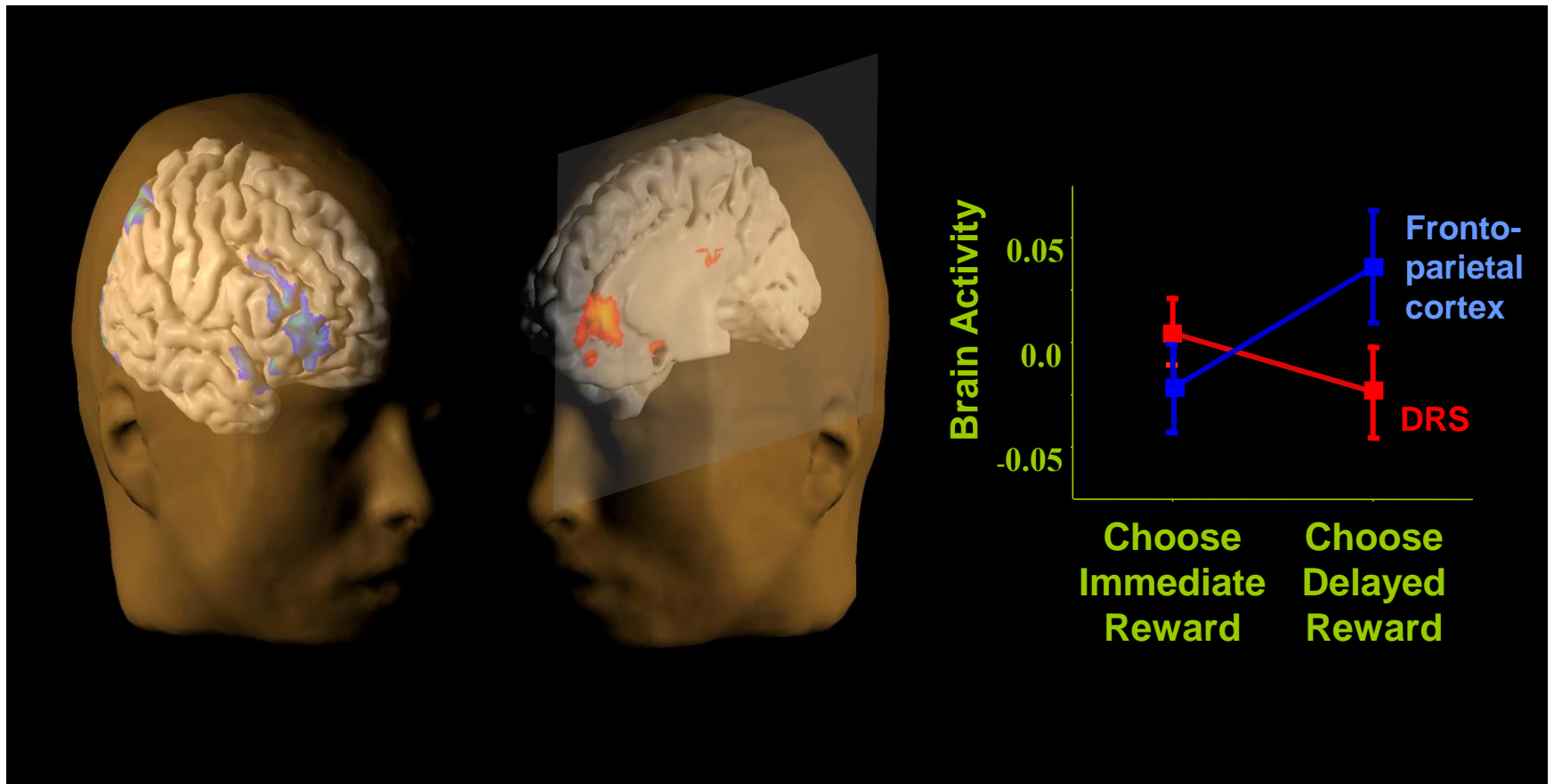
— Delay to earliest reward = Today

— Delay to earliest reward = 2 weeks

— Delay to earliest reward = 1 month

Brain activity in the frontoparietal system and dopamine reward system predict behavior

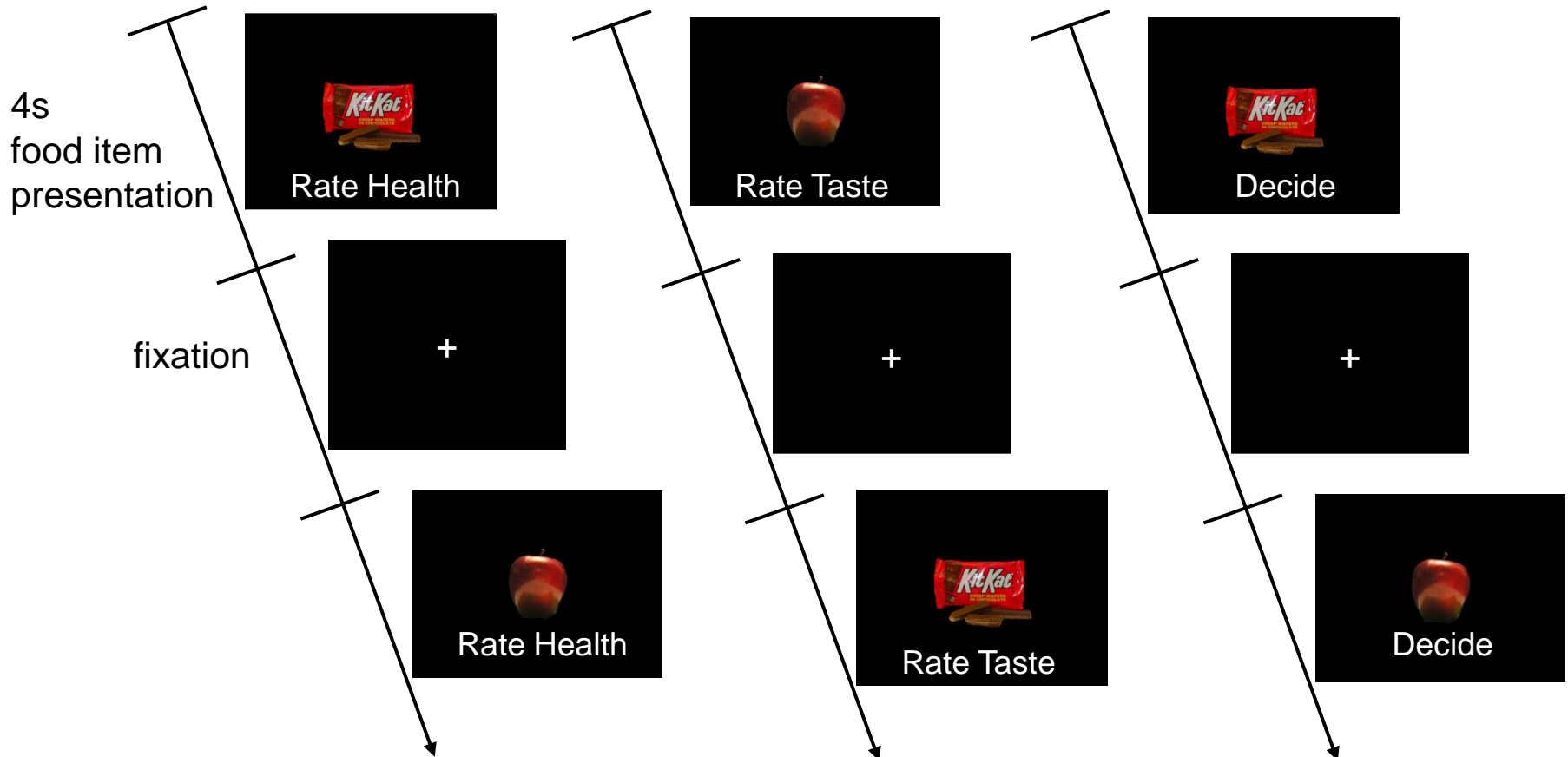
(Data for choices with an immediate option.)



Hare, Camerer, and Rangel (2009)

Health Session ↔ Taste Session

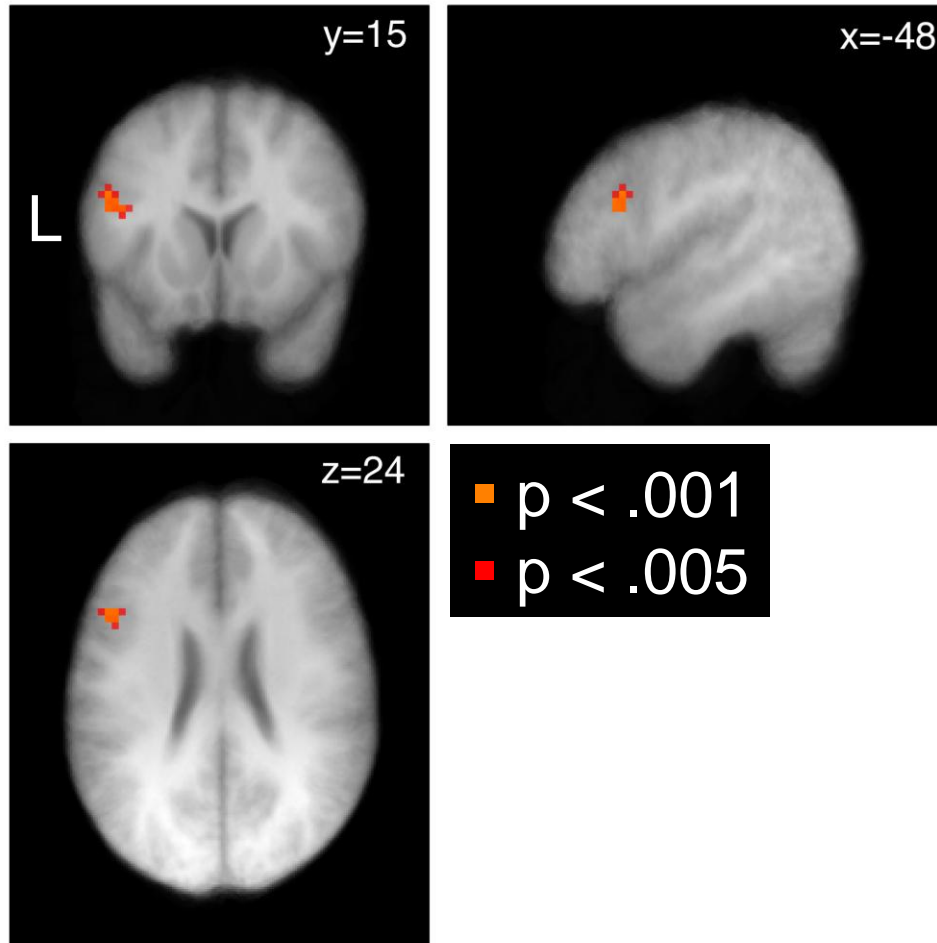
Decision Session



Details

- Taste and health ratings made on five point scale:
-2,-1,0,1,2
- Decisions also reported on a five point scale:
SN,N,0,Y,SY
“strong no” to “strong yes”
- Subject choices sometimes reflect **self control**
 - Rejection of an unhealthy, good tasting food, OR
 - Consumption of a healthy, bad tasting food

More activity in DLPFC in successful self control trials than in failed self control trials



Figner, Knoch, Johnson, Krosch, Lisanby, Fehr and Weber (2010)

- Disruption of function of left, but not right, lateral prefrontal cortex (LPFC) with low-frequency repetitive transcranial magnetic stimulation (rTMS) increased choices of immediate rewards over larger delayed rewards.
- rTMS did not change choices involving only delayed rewards and did not change valuation *judgments* of immediate and delayed rewards.
- Causal evidence for a neural lateral-prefrontal cortex–based self-control mechanism in intertemporal choice.

Part II:

Genoeconomics

Heritabilities for Economic Outcomes

(estimated by comparing concordance between MZ & DZ twins)

- Psychological traits: 0.20 - 0.80. (Plomin et al., 2008)
- Economic preferences/outcomes: roughly similar, but less reliably measured. Examples:
 - Years of schooling: ~0.40
 - Taubman (1976), Behrman and Taubman (1976), Ashenfelter and Krueger (1994), Miller, Mulvey, and Martin (1995), Rouse (1999).
 - Income: ~0.40
 - Ibid.
 - Or higher, if income is better measured.

When Taubman (1976) found that income has heritability ~40%, Hans Eysenck was quoted in the *Times of London*:

These results “really tell the [Royal] Commission [on the Distribution of Income and Wealth] that they might as well pack up.”

Arthur Goldberger (1979) wrote
(with heavy sarcasm):

“A powerful intellect was at work. In the same vein, if it were shown that a large proportion of the variance in eyesight were due to genetic causes, then the Royal Commission on the Distribution of Eyeglasses might as well pack up. And if it were shown that most of the variation in rainfall is due to natural causes, then the Royal Commission on the Distribution of Umbrellas could pack up too.”

What Heritability Does *Not* Imply

Major Fallacy: Over the years, high heritability often (mis)interpreted as indicating little scope for policy to affect the outcome.

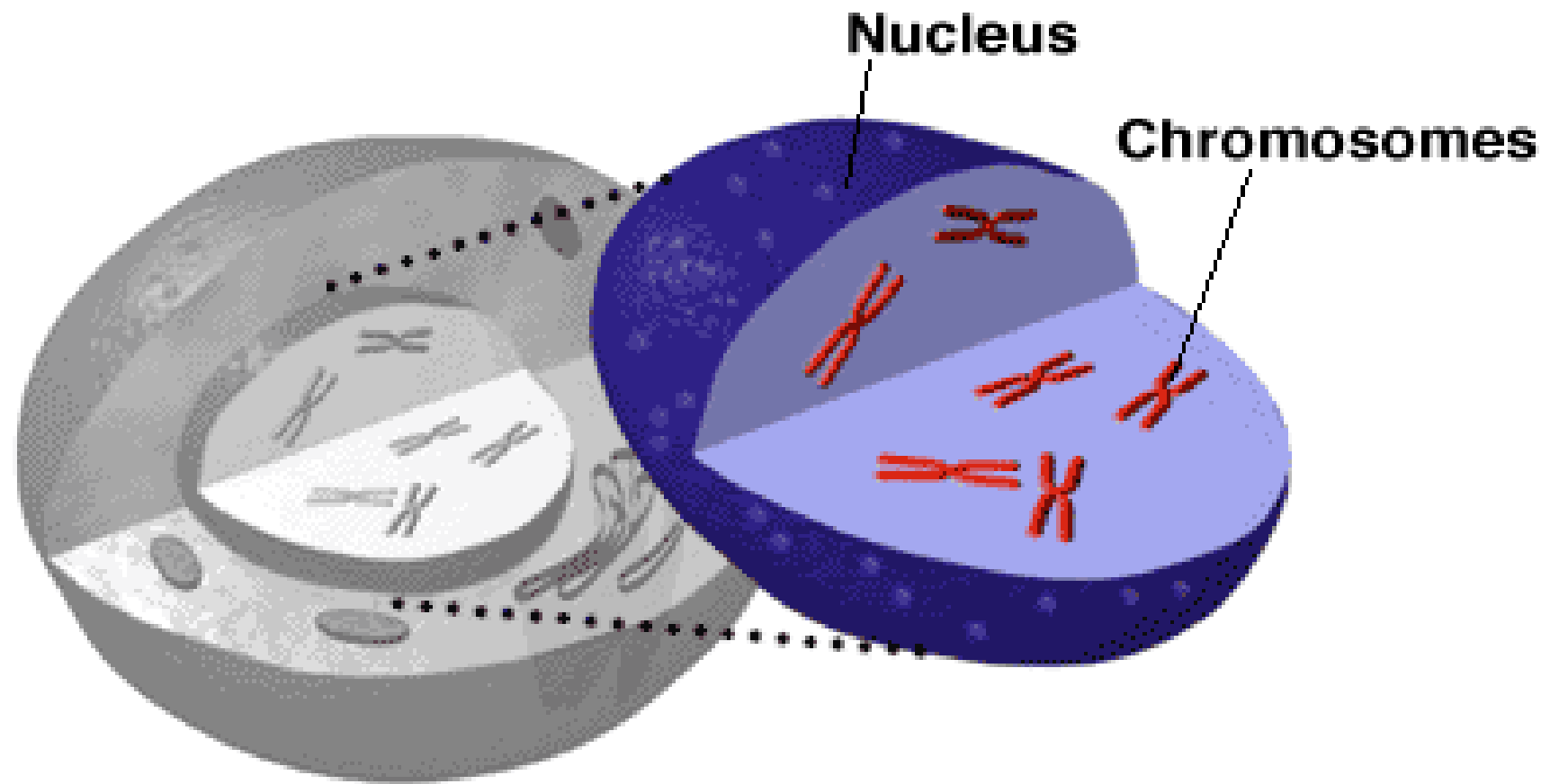
What Heritability *Does* Imply

1. There exist genes that are predictive of behavior, and thus these genes could be identified.
2. A variable constructed from genetic data could, in principle, have non-trivial predictive power (up to the level of heritability).

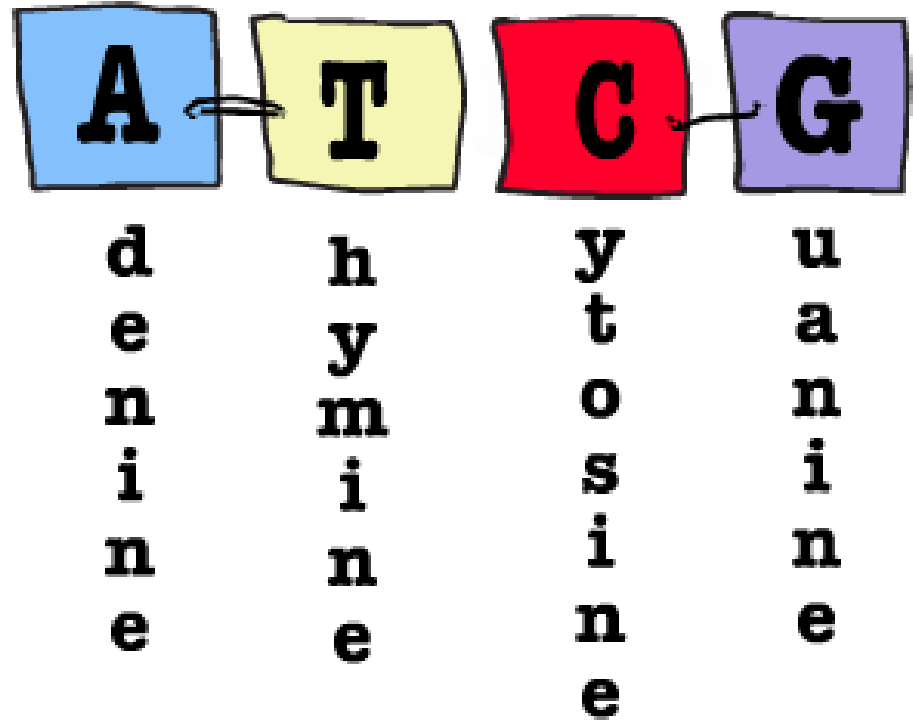
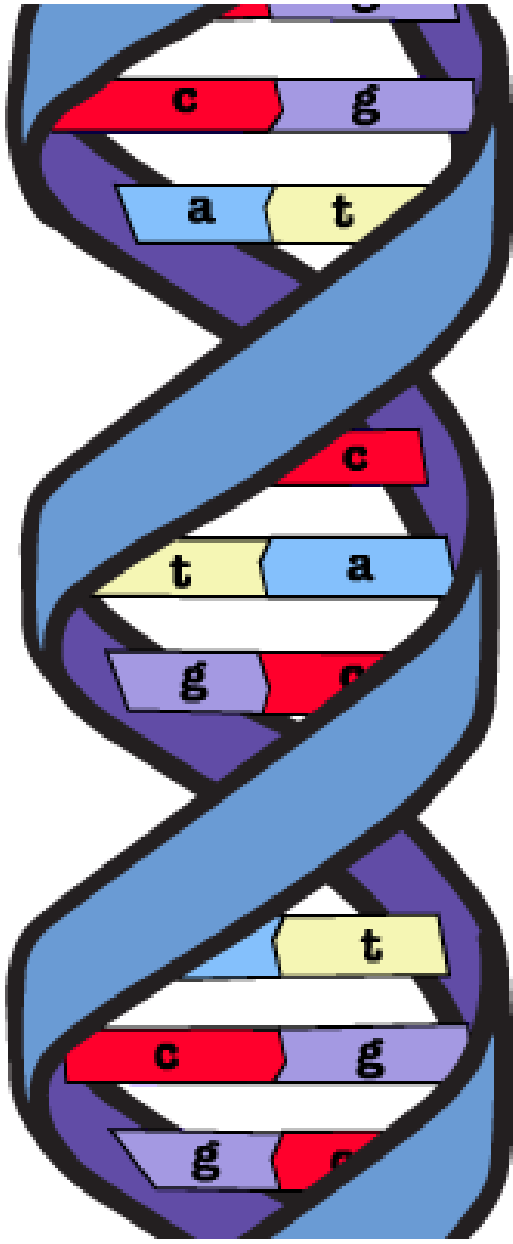
Identifying genes and constructing “polygenic scores” are central goals of **geneconomics**.

Outline

- 1. Conceptual Framework***
- 2. GWAS and Educational Attainment**







Genetics Primer

- Human DNA is a sequence of ~3 billion nucleotide molecules (spread across 23 chromosomes).
- This human genome has 20,000-25,000 subsequences called **genes**.
- Genes provide instructions for building proteins that in turn affect body function.
- At the vast majority of locations, there is no variation in nucleotides across individuals.

- ***Single-nucleotide polymorphisms (SNPs)***:
Nucleotides where individuals differ (a small % of all nucleotides).
(There are also other types of variation.)
- At vast majority of SNP locations, there are only 2 possible nucleotides:
 - ***major allele*** (more common)
 - ***minor allele*** (less common).
- From each parent, may inherit either allele; SNP unaffected by which received from whom.
- ***Genotype*** for each SNP: #minor alleles (0,1,2).

Outline

1. Conceptual Framework
- 2. *GWAS and Educational Attainment***

Genetic Effects

- Let i index individuals; j index SNP.
- Let y_i denote some outcome of interest.
- Linear approximation to true model:

$$y_i = \mu + \beta_j x_{ij} + \epsilon_i.$$

μ : population mean of the outcome.

x_{ij} : genotype $\in \{0,1,2\}$ of person i for SNP j .

β_j : effect of SNP j .

ϵ_i : effect of residual factors.

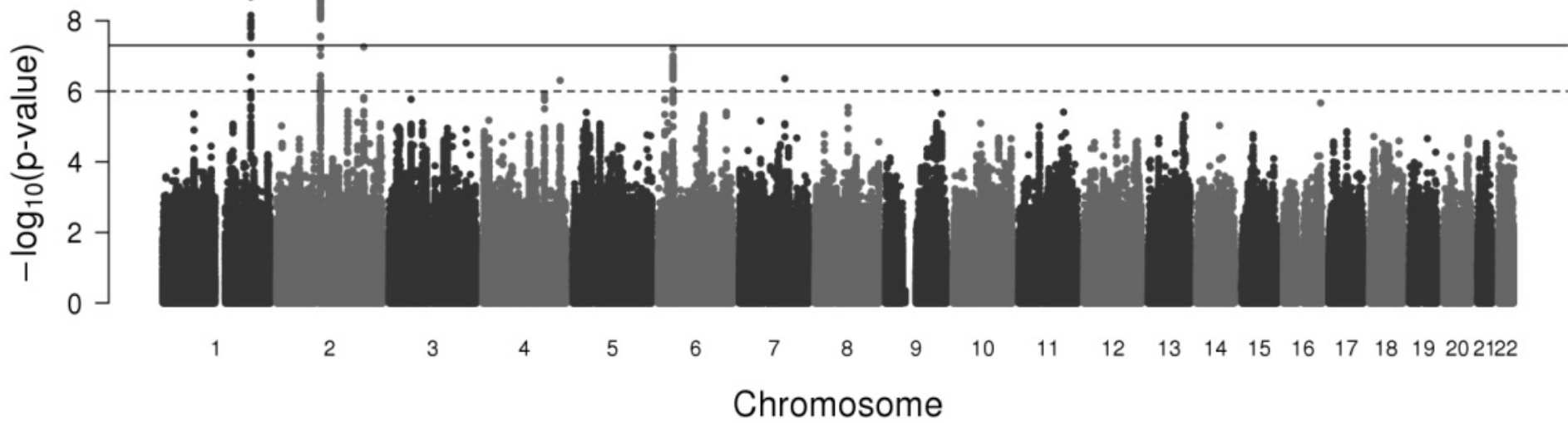
Genome-Wide Association Study (GWAS)

- Atheoretical testing of all SNPs measured on the chip (typically 0.5-5 million).
- Set significance threshold $\alpha = 5 \times 10^{-8}$ (since ≈ 1 million independent SNPs in genome).

GWAS of 126,559 Individuals Identifies Genetic Variants Associated with Educational Attainment

Cornelius A. Rietveld *et al.*
Science **340**, 1467 (2013);
DOI: 10.1126/science.1235488

Social Science Genetics Association Consortium:
Dan Benjamin, David Cesarini, Philipp Koellinger



Replicability and Robustness of Genome-Wide-Association Studies for Behavioral Traits

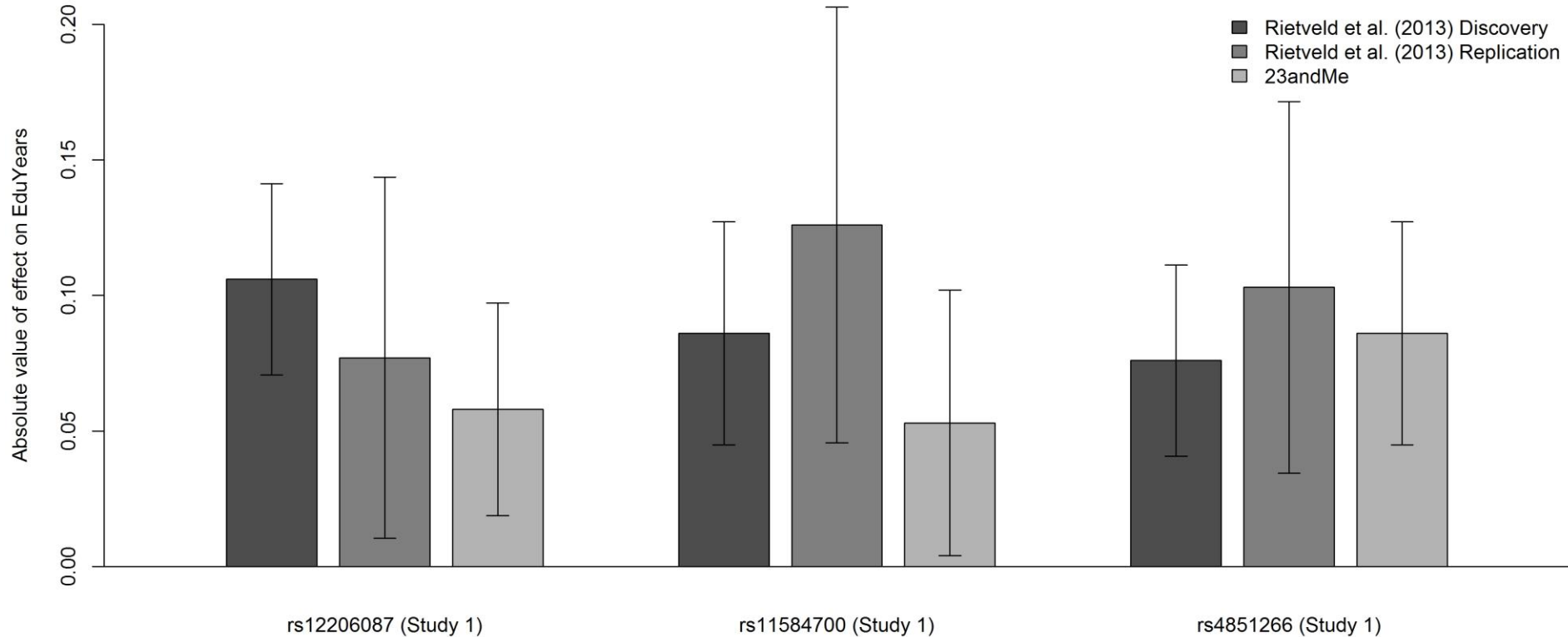
Psychological Science
1–12

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**Cornelius A. Rietveld^{1,2}, Dalton Conley³, Nicholas Eriksson⁴,
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Patrik K. E. Magnusson¹⁴, Joanna L. Mountain⁴, Sven Oskarsson¹⁵,
Olga Rostapshova¹³, Alexander Teumer¹⁶, Joyce Y. Tung⁴,
Peter M. Visscher^{7,17}, Daniel J. Benjamin¹⁸, David Cesarini^{19,20},
Philipp D. Koellinger^{1,2,21}, and the Social Science Genetics
Association Consortium**

Panel A: Individual SNPs, effect of an increase in one reference allele

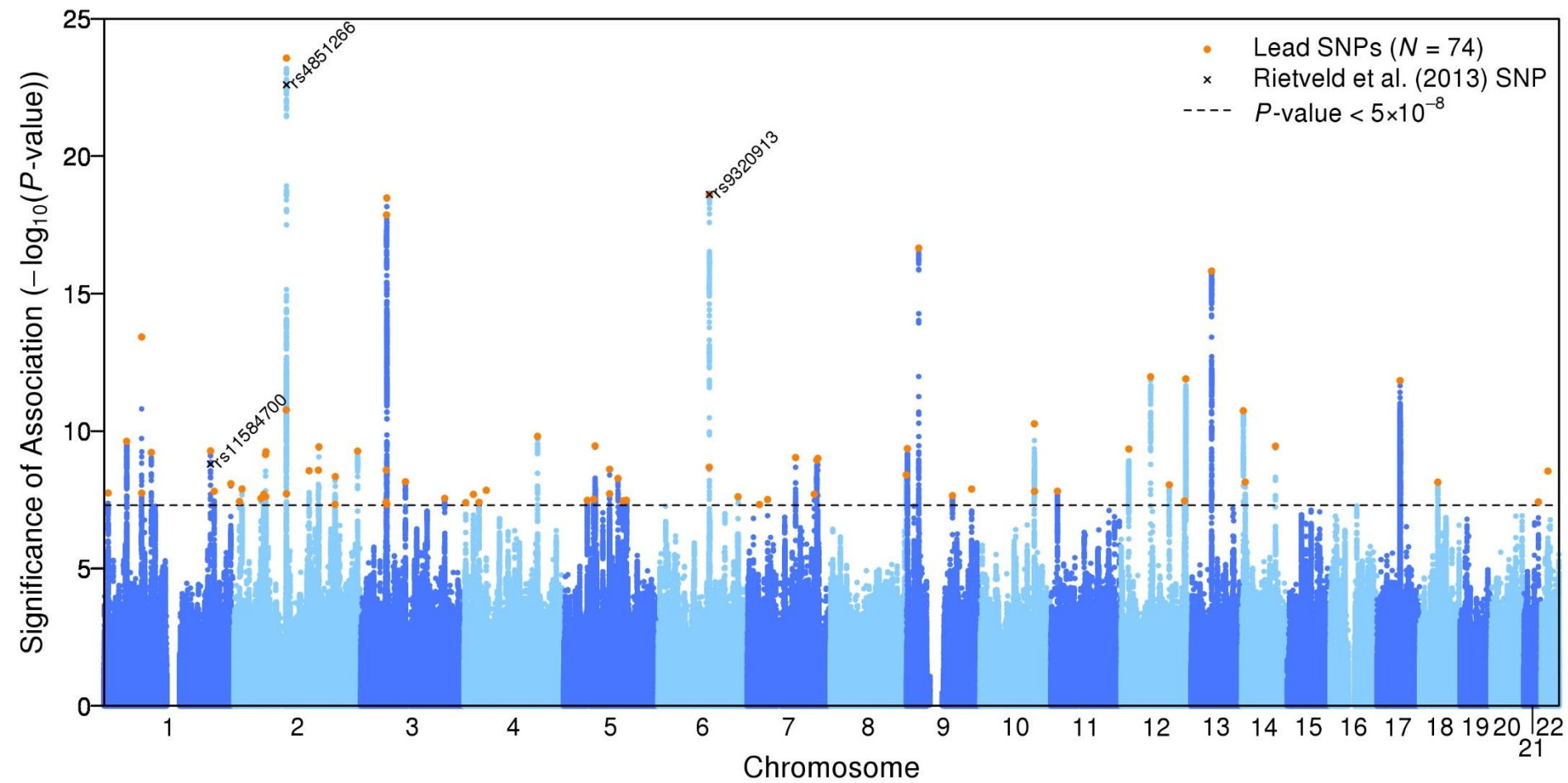


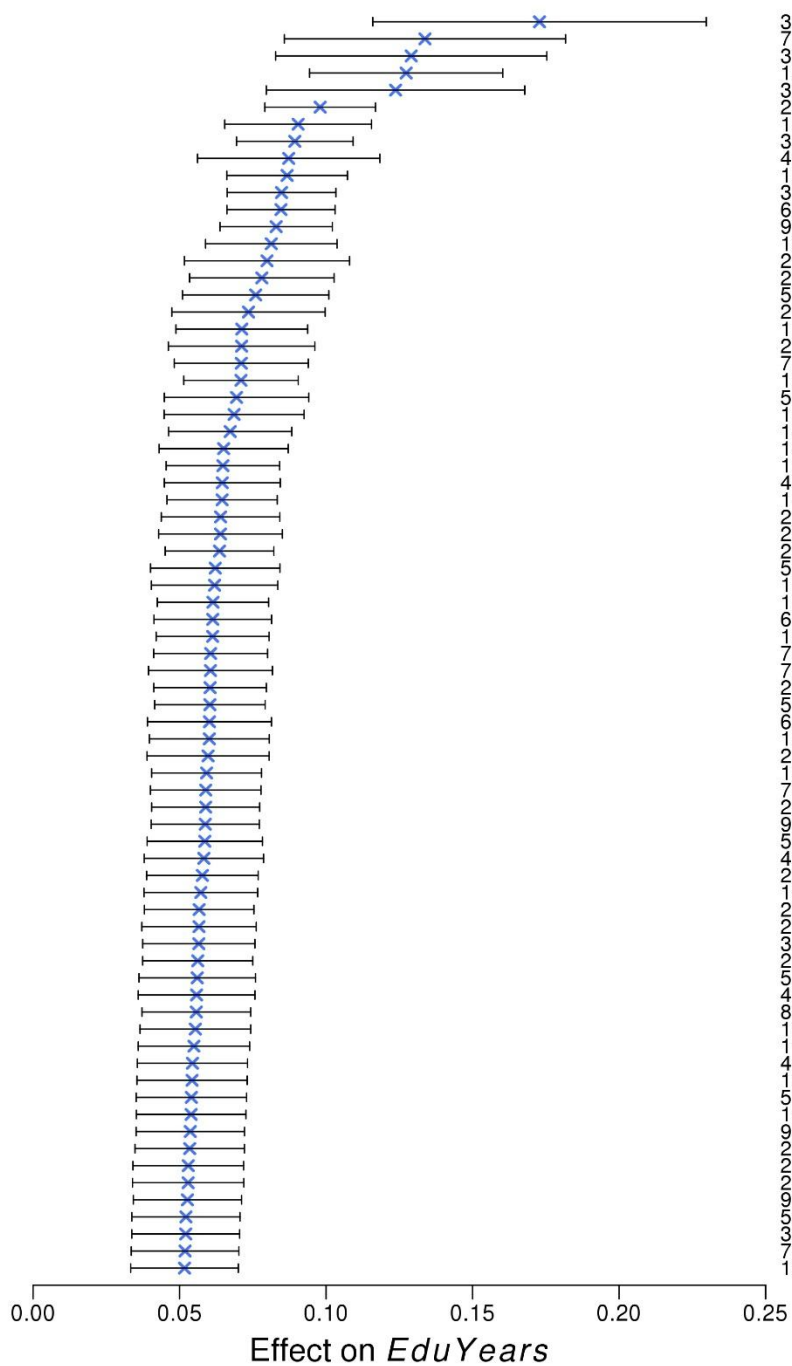
Effect size $R^2 \approx 0.02\%$ an *order of magnitude* smaller than for complex physical / medical traits.

EA2.0: Okbay et al (*Nature* 2016)

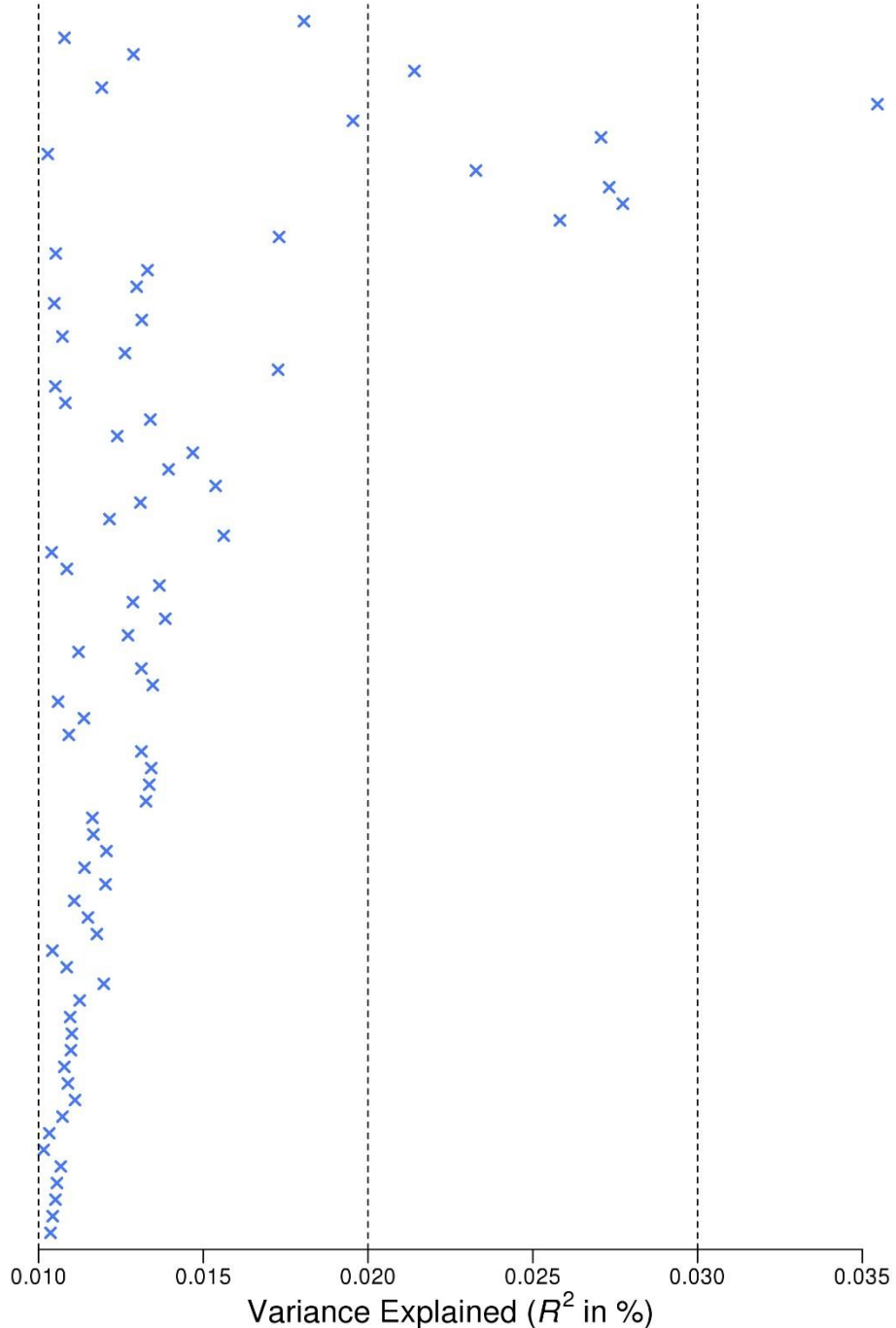
Social Science Genetics Association Consortium

- 63 datasets with sample size of $N = 293,723$.
- Similar analysis plan as EA1.0, except:
 - Newer reference panel (1000G instead of HapMap2).
 - Controlled for 10 PCs (rather than 4).
 - No replication phase (as in recent very large GWAS: Wood et al., 2014; Ripke et al., 2014; Locke et al., 2015).
 - Focus on years of schooling.
- Found 74 approximately uncorrelated genome-wide significant SNPs.





3:48939052
 7:23402104
 3:50075494
 1:72733610
 3:48623124
 2:100821548
 17:43991515
 3:49406708
 4:28801221
 13:58402771
 3:49914397
 6:98584733
 9:23358875
 12:123767929
 2:51873599
 2:237056854
 5:87934707
 2:10977585
 1:204587047
 2:60976384
 7:133302345
 12:56416928
 5:45188024
 1:8490603
 14:84913111
 14:27098611
 10:103802408
 4:140764124
 14:23373986
 2:61482261
 22:29880773
 2:100333377
 5:113987898
 1:72762169
 1:43982527
 6:98187291
 12:14653667
 7:135227513
 7:128402782
 2:60757419
 5:60111579
 6:153367613
 1:243503764
 2:100753490
 1:91189731
 7:92654365
 2:162818621
 9:1746016
 5:103947968
 4:18037231
 2:161920884
 18:35186122
 2:144152539
 2:15621917
 3:85674790
 2:194296294
 5:120102028
 4:3249828
 8:145712860
 12:92159557
 11:12748819
 4:42649935
 10:104082688
 5:87896602
 1:211613114
 9:124644562
 2:57387094
 21:42620520
 2:193731929
 9:88003668
 5:57535206
 3:160847801
 7:39090698
 12:121279083



Future Possibilities

- Personalized medicine.
- Planning for one's own cognitive and health trajectory?
 - Should some genetic data *not* be revealed? Huntington's? APOE status?
- Planning when to have children based on genetically predicted fertility?
- Parents targeting learning environment and activities to children's genes?
- Differential taxes?

Outline:

- **Neuroeconomics: definition**
- **Multiple Systems Hypothesis**
- **Genoeconomics**