Biosocial Science

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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 epigentics.
- *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

System 1 → Integration ← System 2

Behavior
An uninteresting example

What is 6 divided by 3?
A more interesting example

Would you like a piece of chocolate?

Abstract goal: diet → Integration ← Visceral reward: pleasure

Behavior
A more interesting example

Would you like a piece of chocolate?

Abstract goal: diet → Integration → Visceral reward: pleasure

Behavior
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)
Dopamine reward system

Frontal cortex

- Caudate nucleus and putamen (striatum)
- Nucleus accumbens (ventral striatum)
- Ventral tegmental area
- Substantia nigra

Affective vs. Analytic Cognition

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

### Relationship to quasi-hyperbolic model

<table>
<thead>
<tr>
<th></th>
<th>now</th>
<th>t+1</th>
<th>t+2</th>
<th>t+3</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>PFC</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>...</td>
</tr>
<tr>
<td>DRS</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>...</td>
</tr>
<tr>
<td>Total</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>...</td>
</tr>
<tr>
<td>Total normed</td>
<td>1</td>
<td>1/2</td>
<td>1/2</td>
<td>1/2</td>
<td>...</td>
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</tbody>
</table>
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:

\[
U_t = u_t + \beta [\delta u_{t+1} + \delta^2 u_{t+2} + \delta^3 u_{t+3} + ...] \\
(1/\beta)U_t = (1/\beta)u_t + \delta u_{t+1} + \delta^2 u_{t+2} + \delta^3 u_{t+3} + ... \\
(1/\beta)U_t = (1/\beta - 1)u_t + [\delta^0 u_t + \delta^1 u_{t+1} + \delta^2 u_{t+2} + \delta^3 u_{t+3} + ...]
\]

DRS  \hspace{1cm} \text{fronto-parietal cortex}
### Commonalities between classification schemes

<table>
<thead>
<tr>
<th>Affective system</th>
<th>Analytic system</th>
</tr>
</thead>
<tbody>
<tr>
<td>• fast</td>
<td>• Effortful</td>
</tr>
<tr>
<td>• unconscious</td>
<td>• slow</td>
</tr>
<tr>
<td>• reflexive</td>
<td>• conscious</td>
</tr>
<tr>
<td>• myopic</td>
<td>• reflective</td>
</tr>
<tr>
<td></td>
<td>• forward-looking</td>
</tr>
<tr>
<td></td>
<td>• (but still prone to error: heuristics may be analytic)</td>
</tr>
</tbody>
</table>
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

\[ \text{Contrast at voxel } = \sum_{i=1}^{n} BOLD_{v,i,t} \]

Indexes for voxel \((v)\), subject \((i)\), and time \((t)\)

• Controls: time in scanner, lagged reward event, etc.
• Event dummy: decision, experience, event, etc.
• Analogous method: “contrast”

\[ \text{Contrast at voxel } = \sum_{i=1}^{n} BOLD_{v,i,t} \]
Basic experimental design

Task A

Rest 12 sec

Task B

Rest 12 sec

Task C

Rest 12 sec

Time
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, legions, 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

- Intertemporal choice with time-dated Amazon gift certificates.
- Subjects make binary choices:
  - $20 now or $30 in two weeks
  - $20 in two weeks or $30 in four weeks
  - $20 in four weeks or $30 in six weeks
Dopamine reward system
Fronto-parietal cortex

Fronto-parietal cortex

Fronto-parietal cortex

$20$ $30$

$20$ $30$

$20$ $30$
Regions that respond “only” to immediate rewards

Delay to earliest reward = Today
Delay to earliest reward = 2 weeks
Delay to earliest reward = 1 month
Regions that respond “equally” to all rewards

- VCtx
- PMA
- RPar
- DLPFC
- VLPFC
- LOFC

- 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

4s food item presentation

fixation

Rate Health

Rate Taste

Decide

Rate Health

Rate Taste

Decide
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

- $p < .001$
- $p < .005$
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
  - 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 genoeconomics.
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).
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:

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

$\mu$ : population mean of the outcome.

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

$\beta_j$ : effect of SNP $j$.

$\epsilon_i$ : 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 $\approx$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

Cornelius A. Rietveld¹,², Dalton Conley³, Nicholas Eriksson⁴, Tönuf Esko⁵, Sarah E. Medland⁶, Anna A. E. Vinkhuyzen⁷, Jian Yang⁷, Jason D. Boardman⁸,⁹, Christopher F. Chabris¹⁰, Christopher T. Dawes¹¹, Benjamin W. Domingue⁸, David A. Hinds⁴, Magnus Johannesson¹², Amy K. Kiefer⁴, David Laibson¹³, Patrik K. E. Magnusson¹⁴, Joanna L. Mountain⁴, Sven Oskarsson¹⁵, Olga Rostapshova¹³, Alexander Teumer¹⁶, Joyce Y. Tung⁴, Peter M. Visscher⁷,¹⁷, Daniel J. Benjamin¹⁸, David Cesarini¹⁹,²⁰, Philipp D. Koellinger¹,²,²¹, and the Social Science Genetics Association Consortium
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.
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?
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• Genoeconomics