Genes and Choice

Andrew Caplin, David Cesarini, Magnus Johannesson and Kevin Thom

May 1 2012

- Caffeine metabolized by CYP1A2 enzyme.
- Encoded by *CYP1A2* gene.
- Genetic variation determines rapid vs. slow metabolizer
- 0.2 cups coffee/day per allele (Sulem *et al.*, 2011, Cornelis et al., 2011, *CYP1A2*):
- *CYP1A* linked to blood pressure (Levy et al., 2009).
- Possible link to heart attack (Cornelis, 2006)

- Cigarettes (The Tobacco and Genetics Consortium, 2010, *CHRNA3*): nicotinic receptor gene
  - 1.03 cigarettes/day per allele.
- Alcohol (Li et al., 2011, *ADH1B*): alcohol metabolism
- BMI (Frayling et al., *FTO*)

- **Subjective Biological Production Function:**
  $F : X \rightarrow \Delta(B)$
  - Commodity space X dynamic consumption path
  - B holistic) subjective state (incl. biology)
  - Mapping biological/neurological/belief-based
  - Taste immediate, predictable
  - Future production uncertain
    - health effects & addiction lagged, uncertain
    - knowledge/ beliefs matter: diet
    - signals valuable

- **Biological Types:** $\gamma \in \Gamma$ informs $F^{\gamma} : X \rightarrow \Delta(B)$:
  - Genes!
  - Taste: bitterness
  - Body: caffeine, alcohol, nicotine - metabolism
  - Health state: vulnerabilities
  - Drug interactions
  - Habit build up/ extinction (dopaminergic)
- Subjective differences vs. expert knowledge

- Expected Utility $U : B \longrightarrow \mathbb{R}$
  - Preferences over dynamic holistic lotteries
  - Taste: Bitter
  - Health: lotteries
  - Mental states:
    - stimulated not caffeinated
    - anxious not subjected to trauma
  - Habit: dynamic
- Choice reveals only composition $U \circ F^{\gamma}$
  - Hypothesis: U independent of biological type

- Coffee: Genes and Choice
  - Life cycle approach
  - Genetic product design (pharmaceuticals)
  - Possible health connection
- Alcohol and Tobacco: Don't Start if you Can't Stop
  - Early information on cessation genes
  - Learning model for identification
  - Highly policy relevant

- *n=9,617* Swedish twins born 1926- 1958.
- Illumina HumanOmniExpress BeadChip ~600,000 genetic markers.
- SALT survey in 2000: coffee, alcohol, smoking, BMI, health
- Roughly half similar survey in 1973.
- Potential for directed re-survey/ field test
- SNP not gene unit of observation

- Each additional T-allele on rs2472297
  - Located near *CYP1A2*
  - *0.38* more cups of coffee per day ($p = 10^{-18}$).
  - Increase of 0.2 cups per day ($p = 10^{-4}$) more growth 1973 - 2000.
  - Life cycle perspective
- Cups per day in table

rs2472297	0	1	2
Q73	3.68 (2.50)	3.97 (2.67)	4.12 (2.96)
N	2500	2015	385
SALT	3.62 (2.41)	4.02 (2.60)	4.35 (2.94)
N	4962	3956	758
Δ	-.06 (2.54)	0.09 (2.81)	0.37 (2.79)
N	2487	2008	384

Note: All highly significant

- In spirit of Becker and Murphy (1988). Period utility:

$$U_t(C_t, A_t, \epsilon_t, H_t) = (\alpha_1 + \epsilon_t)\left(\frac{C_t}{1+A_t}\right) + \alpha_2\left(\frac{C_t}{1+A_t}\right)^2 - H_t$$

- **Addiction stock** evolves according to:

$$A_{t+1} = (1-\delta_1)A_t + \delta_2 C_t$$

- **Health shock**: H_t takes the value h with probability $\frac{\exp(\phi_1+\phi_2 C_t)}{1+\exp(\phi_1+\phi_2 C_t)}$, and the value 0 otherwise
- **Taste shocks**: ϵ_t may be serially correlated.
- **Addiction formation / extinction**: δ_1 governs depreciation, δ_2 affects formation
- No pricing special to coffee

- Standard estimation of gene-dependent parameters from consumption data
  - Incorporate health data: beliefs?
- Interpretations:
  - Taste: α_1, α_2
  - Consumption/health risk interaction: h, ϕ_2
  - Growth of addiction: δ_2
  - Difficulty with cessation: δ_2: add asymmetry to model?
  - May be general: dopamine

- Reminder: Cessation/health important many cases
- Potentially coffee
- Cigarettes (The Tobacco and Genetics Consortium, 2010, *CHRNA3*): nicotinic receptor gene
    - 1.03 cigarettes/day per allele.
- Alcohol (Li et al., 2011, *ADH1B*): alcohol metabolism
- BMI (Frayling et al., *FTO*)

- Health interaction requires structural model
- Traditional regression:

$$Y = \beta_0 + \beta_1 \cdot SNP_S + PC \cdot \beta_2 + X \cdot \beta_3 + \varepsilon,$$

- Limitations:
  - Ignores endogenous response to genotype
  - Ignores other moments
  - Specific functional form
  - Silent on mechanisms
  - Counter-factuals and policy

- If high risk gene more likely to get prior risk signal...
  - Can have impact on variance as much as expected value
- Let $G \in \{0, 1\}$ represent an individual's genetic type.
- Genotype-specific habituation: $\delta_2 = \underline{\delta_2}$ if $G = 0$, and $\delta_2 = \overline{\delta_2}$ if $G = 1$.
- Individuals receive informative signal (belief about the probability of being type $G = 1$):
- Simple parametrization.

- If the signal is correlated with type:

	G=1	G=0	Diff or Ratio
Mean	0.65	0.62	0.03
Variance	0.36	0.29	1.27**
Simulations	1000	1000	

- But if the signal is not correlated with type:

	G=1	G=0	Diff or Ratio
Mean	0.67	0.62	0.05*
Variance	0.37	0.29	1.26**
Simulations	1000	1000	

- Excellent data for dynamic structural model in SALT
- Understanding of cessation
  - Change in smoking
  - Information on need to stop (direct health/pregnancy etc)
  - Efforts to stop
  - Twins for information flow
  - Snuff/chewing tobacco substitution

- Promise in identifying biological cessation pathway
  - One SNP in gene DBH significantly associated with smoking cessation (The Tobacco and Genetics Consortium, 2010).
    - *DBH* catalyzes conversion of dopamine to norepinephrine,
    - Across addictive goods given dopamine?
  - Many other hints in literature

- Policy impact
  - Genetic elasticity of demand
  - Early warning to change demand
  - Gene specific cessation treatments
- Alcohol identical path