



COMPUTATIONAL UNHAPPINESS

By Quentin J. M. Huys and Peter Dayan

Psychiatry has made great strides over the past few decades, with large-scale studies yielding an inventory of robust psychiatric constructs. Yet, while the nomenclature of diseases has been progressively refined, their definitions remain syndromal and incompletely tied to neuroscientific foundations. Qualitative concepts, such as uncontrollability and anhedonia, lack the precision to dissect multifactorial conditions, such as depression, which involve dysfunction among complex interacting systems (Hasler et al., 2004).

A new approach¹ is to seek a firmer foundation of the science of affective decision making. Making choices in the face of rewards and punishments is supported by multiple, partially separate, neural systems.

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Detailed computational models of these offer the hope of a specific and quantitative anatomy of normal and abnormal function, along with the prospect of rigorous tests for each underlying defect. We point towards three examples associated with depression (Huys, 2007).

Anhedonia is the inability to enjoy previously enjoyable things, often operationalized as a reduced willingness to choose apparently worthier actions in simple learning paradigms (Pizzagalli et al., 2005). Anhedonia can arise from several or all of: a flatter mapping of objective to subjective worth (reducing the difference in relative worth), an increase in the stochasticity of choice (reducing the effect of a given difference on the propensity to choose better actions), or a change in the rate at which affectively-charged information is accumulated (reducing measurable differences in short experiments). Computational models parameterize these factors, and tasks can be designed to infer normal and abnormal parameter values.

¹ Also pioneered by Todd Braver <http://ccpweb.wustl.edu/>; Jonathan Cohen <http://www.csmbm.princeton.edu/ncc/>; Michael Frank <http://ski.cog.brown.edu/>; Shitij Kapur <http://www.iop.kcl.ac.uk/departments/?locator=986>; Read Montague <http://www.cpu.bcm.edu/>; Diego Pizzagalli <http://www.wjh.harvard.edu/~daplab/>; Klaas Stephan <http://www.fil.ion.ucl.ac.uk/~kstephan/>; Douglas Steele http://www.dundee.ac.uk/cmdn/staff/douglas_steele and Jonathan Williams.

Learned helplessness, hopelessness and chronic mild stress are models of depression that concentrate on how subjects generalize across environments beliefs about the extent to which reinforcers are under their behavioral control. One computational account has subjects balancing beliefs, acquired from prior learning, with new observations, to decide what a fresh environment affords. If desirable outcomes have previously been unachievable, or undesirable outcomes rife, then subjects will a priori come to expect little controllable reinforcement in future environments. This can have critical consequences - if there is likely not to be a beneficial action, then there is no point in exploring in the first place. Controllability can interact with anhedonia and other symptoms, again, a model-based task design can unpick these.

Finally, consider serotonin, which has been suggested to control the inhibition of courses of action leading to potentially deleterious consequences. Such inhibition offers a crutch for other control systems; the consequences of knocking away that crutch are both immediate (resembling a form of impulsivity) and longer term (possibly analogous to aspects of chronic mild stress). Systemic interactions of this sort make for rich, complex and confusing patterns of empirical findings.

Computational models are not a panacea; the discipline of being quantitatively crisp and concrete reveals much about the huge gaps in our understanding; and subjective phenomena, such as mood, are much less accessible than objective aspects of choice. We can nevertheless look to modeling to integrate diverse psychological and neuroscientific empirical data, and to help loosen some of the field's conceptual shackles. 

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