Computations in extraversion

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Abstract: We make two suggestions with regard to Depue & Collins's
(D&C's) target article. First, regarding the functioning of MOC13, we pro-

vide data indicating that, contrary to D&C's apparent position, this structure is not necessary for instrumental conditioning. Second, we suggest that D&C's approach would be advanced by reference to formal computational theory, in particular the work of Grossberg. We suggest that an integration of Grossberg's and D&C's models can provide a more complete account of extraversion.

Depue & Collins's (D&C's) target article makes a substantial contribution to relating neurobiology and personality research. They have provided a detailed and convincing development of the notion that the incentive motivation system underpins individual differences in extraversion. D&C propose that incentive motivation information is encoded and integrated in a circuit involving Brodmann's posterior medial orbital prefrontal cortical area 13 (MOC13).

The link between neuroanatomy and extraversion is interesting, but the putative roles of the specific structures could be challenged. In particular, we would like to question the role of orbitofrontal cortex (MOC13). D&C claim that "MOC13 forms higher-level conditional representations of sensory events by associating them with existing or newly-developing response-reinforcement contingencies" (sect. 4.3.4). This implies that MOC13 is implicated in instrumental conditioning. Although the electrophysiological data provided by Thorpe et al. (1983) indicate that neurons are responsive to information about the reward or punishment associated with a stimulus, this does not imply that MOC13 is crucial for instrumental conditioning. Indeed, Thorpe et al. state that OFC represents whether particular stimuli continue to be associated with reinforcement, and it allows behaviour to be modified when it is no longer appropriate. In line with this, OFC lesions in monkeys and humans do not impair instrumental conditioning; they impair the ability to modify responses to stimuli that are no longer reinforced (e.g., Dias et al. 1996; Rolls et al. 1994).

D&C's detailed description of the neuroanatomy of incentive motivation is extremely interesting. They provide a valuable account of the circuitry involved in incentive motivation processes, from the encoding of incentive stimuli to the production of an incentive motivational state that triggers behaviour. However, we believe that their approach would be advanced by considering formal computational theory, such as the work of Armony et al. (1995), and in particular, Grossberg (e.g., Grossberg & Levine 1987). One problem with D&C's focus on the neuroanatomy is

that it fails to account for crucial aspects of incentive motivation; for example, the dissociation between instrumental learning and relearning, and the "persistence problem." In contrast, a computational approach such as Grossberg's adaptive resonance theory (ART) of classical and instrumental conditioning can.

D&C's account appears to predict an association between instrumental learning and relearning. In contrast, Grossberg's ART circuit predicts the observed dissociation. In Grossberg's model there are interactions between attentional and orienting subsystems (see Fig. 1). Incentive motivational learning is achieved by interactions between drive and sensory cue representations. Relearning occurs when mismatches between reinforcements and learned expectations of reinforcements activate the orienting subsystem, which resets the activation levels of the sensory representations. As D&C describe, the role of MOC13 in detecting unexpected reinforcements could suggest that this region is the neural locus of an orienting subsystem. This integration of Grossberg's and D&C's models makes possible an explanation of the specific relearning deficit seen in subjects with damage to MOC13.

Second, D&C's account cannot explain the persistence problem, also known as the "turkey-love fiasco," namely, how incentive motivation and appropriate behaviour are maintained during the parallel processing of several motivationally incompatible conditioned stimuli. To illustrate, "during an otherwise uneventful turkey dinner with one's lover, suppose that one alternately looks at lover and turkey, where lover is associated with sexual responses . . . and turkey is associated with eating responses. Why do we not come away from dinner wanting to eat our lover and have sex with turkeys?" (Grossberg & Levine 1987, pp. 5019–20). D&C argue that MOC13 is involved in updating reinforcement priorities but this is not sufficient to explain how, for example, the turkey-love fiasco could be resolved. In Grossberg's model, a sensory cue with incentive motivational properties can quickly augment attention to itself via self-generated incentive motivational feedback signals. In this way, erroneous conditioning from a CS to the wrong CR when more than one CS is present cannot occur. The sensory feedback signals occur independent of the orienting subsystem. If this subsystem is mediated by MOC13, then blocking, unblocking, and latent inhibition, for example, should all occur in MOC13 lesioned animals.

Thus, Grossberg's model could be usefully integrated with the anatomical claims made by D&C. Moreover, as detailed by D&C, the evidence that dopamine acts as a facilitator of incentive motivation is strong. It could be suggested that the nucleus accumbens shell, ventral pallidum, and ventral tegmental area are implicated in the incentive motivational learning pathways shown in Figure 1. Indeed, Grossberg (1982) speculates that dopamine is the neurotransmitter that subserves the gated dipoles in these pathways. As described by D&C, dopamine antagonists would reduce conditioned incentive motivation-governed behaviour but it would not affect unconditioned consummatory behaviour and information about stimulus-reinforcement associations (represented by the conditioned reinforcer learning pathway).

Variation in sensitivity to different classes of stimuli across individuals, as suggested by Gray (1973), could be represented as differing responsiveness of drive representations. For example, an individual highly responsive to positive social cues might be one whose drive representations for those cues have a low threshold for activation. This will manifest itself behaviourally as extraversion. In other words, Gray's suggestion that individual differences in extraversion follow from variation in sensitivity to different classes of stimuli can be fully realised at both the cognitive and neuroanatomical levels by an integration of Grossberg's and D&C's models. This also raises the question of whether there are individual differences in sensitivity to more specific classes of stimuli than just reward and punishment. For example, according to Blair's violence inhibition model, psychopaths suffer from a specific insensitivity to distress cues (e.g., Blair 1995); it seems more than plausible that there could be a continuum of sensitivity to distress cues and other types of stimuli in the normal population.
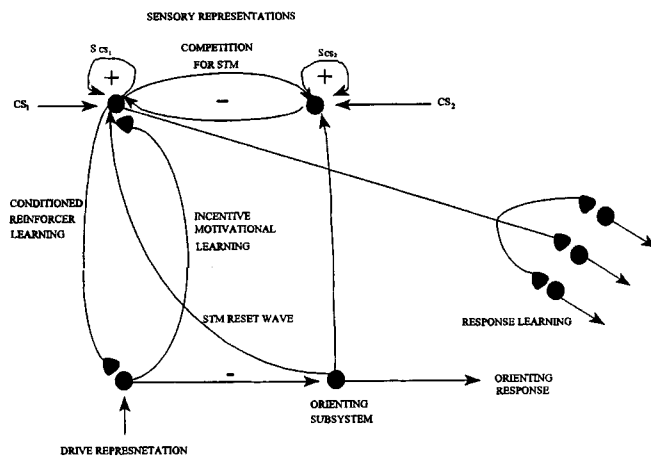

Figure 1 (Fine & Blair).   Grossberg's schematic conditioning circuit: conditioned stimuli (CS) activate sensory representations (S_{CSi}), which compete amongst themselves for limited short-term memory activation and storage. The activated S_{CSi} signals elicit conditionable signals to drive representations and motor command representations. Mismatches between learned expectations and drive input representations trigger the orienting subsystem, resetting STM activations of sensory representations. Adapted from Grossberg & Levine (1987; p. 5019).