

## From “Oh, OK” to “Ah, yes” to “Aha!”: Hyper-systemizing and the rewards of insight

Chris Fields

Apdo 363-4013

Atenas 20501

Costa Rica

chrisfields38@gmail.com

### Abstract

Hyper-systemizers are individuals displaying an unusually strong bias toward systemizing, i.e. toward explaining events and solving problems by appeal to mechanisms that do not involve intentions or agency. Hyper-systemizing in combination with deficit mentalizing ability typically presents clinically as an autistic spectrum disorder; however, the development of hyper-systemizing in combination with normal-range mentalizing ability is not well characterized. A review of anecdotal reports, survey-based measurements, and experimental studies of systemizing suggests the hypothesis that hyper-systemizing in the presence of normal-range mentalizing develops as an addiction syndrome driven by the positive affect associated with insight solutions. A neurocognitive model of hypersystemizing as an outcome of insight addiction is constructed based on the incentive-sensitization model of addiction. If this model is correct, assaying subjects for bias on the systemizing – mentalizing spectrum would be expected to reveal significant activity differentials in temporal-parietal-frontal networks on cognitive tasks with systemizing or mentalizing components within the neurotypical population. Predictions of the model accessible to survey-based instruments and standard cognitive measures are outlined, and evidence pertaining to them considered.

**Keywords:** Systemizing, Mentalizing, Development, Addiction, Insight, Default network, Analogy

### 1. Introduction

The concept of “systemizing” was introduced by Baron-Cohen and colleagues to describe a problem-solving and explanatory strategy or style characterized by appeals to natural laws, physical mechanisms, algorithms or other concepts of causation that do not involve agency or intentions (Baron-Cohen, 2002; 2008). Systemizing or “mechanizing” (Crespi and Badcock, 2008) solutions and explanations are explicitly distinguished from “empathizing” or “mentalizing” solutions and explanations, which do appeal to causal agents and to actions taken to be guided by intentions, beliefs, desires, goals, fears, worries and other “folk psychological” attributes associated with agency by a theory of mind (ToM) system (Frith and Frith, 1999; 2003). Systemizing answers “how?” or “how did this come to be?” questions by tracing agency-independent causal mechanisms, without addressing why a particular mechanism is acting in any sense of “why?” that implies teleology or intention. As an

experimentally-accessible construct capturing defining characteristics of scientific, technological, engineering and mathematical thinking (Baron-Cohen, 2008), the notion of systemizing drives a wedge between activities commonly thought of as explanation (Gopnik, 2000; Lombrozo, 2006), and suggests that humans may possess an integrated and specialized cognitive-affective “system brain” analogous to the “social brain” employed in mentalizing (Adolphs, 1999; Dunbar, 2003; Saxe *et al.*, 2004; Frith, 2007).

Questionnaire-based studies as well as common experience indicate considerable variation in the inclination toward or use of systemizing across the human population, with some individuals biased toward systemizing and others biased against it, i. e. biased toward mentalizing (Baron-Cohen *et al.*, 2002; 2003). While both males and females considered as subpopulations exhibit the full range of biases for or against systemizing, males as a group show a greater tendency toward systemizing than females as a group (Baron-Cohen *et al.*, 2003; Goldenfeld *et al.*, 2006; Nettle, 2007); differences in testosterone exposure *in utero* have been advanced as a possible explanation of this difference (Baron-Cohen *et al.*, 2004). The distinction between systemizing and mentalizing arises early in development, initially in association with the distinction between animate and inanimate objects (Karmaloff-Smith, 1995; Subrahmanyam *et al.*, 2002). A robust ability to attribute agency develops in infancy (Johnson *et al.*, 2007; Saxe *et al.*, 2005; 2007); infant tendencies toward mentalizing predict later childhood tendencies toward mentalizing (Wellman *et al.*, 2004). An understanding that inanimate objects respond in predictable ways to external causes develops in early childhood (Gopnik and Schultz, 2004). While infants separately categorize self-propelled objects and attribute internal casual powers to them (Subrahmanyam *et al.*, 2002; Luo *et al.*, 2009), an understanding that objects may exhibit spontaneous behaviors due to hidden, internal causes – arguably the earliest indication of robust systemizing – can typically be inferred around age four (Sobel *et al.*, 2007). A competitive mechanism for switching between systemizing and mentalizing strategies for problem solving is suggested by classic experiments with animated geometric figures, which demonstrate transitions between systemizing and mentalizing, in both children and adults, driven by minor changes in the motions of simple geometric shapes (Scholl and Tremoulet, 2000); such switching may be implemented by mirror-system neurons attuned to biological motion patterns (Puce & Perrett, 2003; Engel *et al.*, 2007). A bias against systemizing, i. e. toward mentalizing correlates strongly with the Big Five personality factor Agreeableness; a bias toward systemizing correlates significantly with the Big Five factors Conscientiousness and Openness (Nettle, 2007), consistent with documented personality characteristics of scientists, and particularly of more creative scientists (Feist, 1998).

Individuals strongly biased toward systemizing are considered “hyper-systemizers” (Baron-Cohen *et al.*, 2002; Baron-Cohen, 2008). The notion of a hyper-systemizer is clearly somewhat culture-dependent: adopting a systemizing approach to human origins is unremarkable in contemporary western Europe, for example, but remarkable as a minority position in the United States (e.g. Paul, 2009); adopting a systemizing approach to human language understanding is commonplace in artificial intelligence circles, but is considered incoherent by many philosophers (e.g. Searle, 1980). Nonetheless, hyper-systemizing can be recognized as an unusually pervasive bias against mentalizing, e. g. as indicated by a Systemizing Quotient (SQ) score more than two standard deviations from the mean (Baron-Cohen *et al.*, 2002). The aversion to strong emotions, and to interpersonal conflict in particular among physicists noted by Feist and Gorman (1998) is consistent with hyper-systemizing. Hyper-systemizing in conjunction with deficit mentalizing ability presents in early development as an autistic spectrum disorder (ASD), with severity dependent on the severity of mentalizing deficit (Baron-Cohen *et al.*, 2001; 2002; 2003; Crespi and Badcock, 2008; Ring *et al.*, 2008); physical

sciences, engineering and mathematics are common career choices of high-functioning individuals diagnosed with Asperger Syndrome or high-functioning autism (Baron-Cohen *et al.*, 2001; Fitzgerald & O'Brien, 2007). It is not clear whether hyper-systemizing plays a causal role in the etiology of ASD, or is a developmental consequence of deficit mentalizing or other underlying deficits (Rejendran and Mitchell, 2006; Markram *et al.*, 2007). Despite the strong correlation between hypersystemizing and ASD (Baron-Cohen *et al.*, 2001; 2002; 2003; Goldenfeld *et al.*, 2006; Baron-Cohen, 2008), hypersystemizing is also observed in both males and females with normal-range mentalizing ability (Baron-Cohen *et al.*, 2003; Goldenfeld *et al.*, 2006), as would be expected given the existence of highly systemizing-oriented scientists, engineers and mathematicians of both sexes who are able to attribute mental states to others, predict behavior on the basis of inferred intentions, and function effectively in large organizations. The mechanisms driving development of hyper-systemizing in conjunction with normal-range mentalizing ability, and in particular the motivational components of such asymmetric development, are not well characterized.

This paper suggests that the development of hyper-systemizing in conjunction with normal-range mentalizing is productively conceptualized as driven by addiction to the affective rewards experienced following insight. In Section 2, differences between the affective experiences typically accompanying problem-solving by systemizing or mentalizing are reviewed. The affective spectrum from the “Oh, OK” of everyday solution-finding through the “Ah, yes” of mild surprise to the “Aha!” of insight is characterized in terms of increasing intensity of affective reward for current-state to goal-state conflict resolution. Historical and anecdotal evidence suggesting that hyper-systemizing individuals exhibit behavior patterns typical of addiction syndromes is then reviewed. Section 3, the neurocognitive consequences that would be expected in an individual addicted to insight experiences are examined, primarily in the context of the incentive-sensitization model of addiction (Robinson and Berridge, 1993; 2008). It is shown that neurocognitive processes common to addiction syndromes would be expected to increase the salience of cues previously associated with systemizing, and specifically to decrease default-network activity. Default network activity is typically experienced as mentalizing; decreases in default-network activity correlate with both goal-directed problem solving and the formation of long-distance semantic links and structural analogies (Buckner *et al.*, 2008; Kounios and Beeman, 2009). Hence systemizers in whom such an addictive process is active can be expected to become both more highly biased toward systemizing and more likely to form long-distance semantic links and structural analogies leading to insights. Section 4 proposes that both systemizing and mentalizing are productively viewed not as functions of parallel “system” and “social” brains, but as developmental specializations of the temporal-parietal-frontal network. If this is the case, correlations with subject SQ scores may resolve the inter-subject variation observed in many functional imaging studies into psychologically-meaningful evidence of neurocognitive diversity in the “neurotypical” population. Section 5 outlines predictions of the addiction model of the development of hyper-systemizing that are accessible to survey instruments and standard cognitive tests.

## **2. Affective experiences associated with systemizing**

Common experience as well as developmental and adult cognitive studies suggest that many if not most people enjoy solving problems; it has been suggested that the human drive to explain is universal and analogous to the human drive for sex (Gopnik, 2000). The pleasure associated with discovery and understanding is regarded as an intrinsic motivation toward learning, not only in academic environments (Gottfried, 1985), but also in curiosity-driven unstructured play (Gibson, 1988;

Karmaloff-Smith, 1995; Kaplan and Oudeyer, 2007). However, common experience also suggests that there are large individual differences in the extent to which the pleasure of learning is motivational, and desires that do not have pleasurable experience as primary components are often motivational in both academic (Covington, 2000) and general (Reiss, 2004a) environments. Consistent with such diversity, problem solutions or explanations do not uniformly induce pleasure; many religious explanations, for example, appear to be designed to induce fear, dread, or a social emotion such as affiliative solidarity (Reiss, 2004b). Hence, while a drive to explain may be universal, it appears to be a drive with multiple, separable components, and with multiple affective associations.

A diverse body of evidence indicates that feelings of pleasure are both more commonly and more likely to be associated with systemizing rather than mentalizing solutions or explanations. Mentalizing capacities develop in, and are generally regarded as having evolved in, small-group social contexts in which correct assessments of the intentions of others are critical for survival (Adolphs, 1999; 2003; Dunbar, 2003). Hence solutions to the most basic mentalizing problem – does this approaching person intend help or harm? - are naturally associated with primary emotions of affiliative bonding and fear. Solutions of more subtle mentalizing problems, such as determining whether a partner in a social exchange is cheating, are typically associated with social emotions such as, in this case, righteous anger or jealousy (Adolphs, 2003). Consistent with the strong emotional associations of mentalizing, expectation-reality conflicts in mentalizing often induce anxiety, and can induce pain comparable in character and intensity to physical pain (Eisenberger and Lieberman, 2004; Eisenberger, 2006). In contrast, the inanimate objects with which most systemizing is concerned tend not – with the exception of some products of technology – to be harmful or rewarding in and of themselves in the ways that animals and other humans are. Systemizing solutions and explanations can, therefore, be expected to lack the rich emotional tonality associated with mentalizing. Consistent with this expectation, the primary emotions associated with systemizing in self reports are pleasure and frustration (Shaw, 1999; Amabile *et al.*, 2005). The dynamic range of these emotions is quite large: prominent historical figures from Archimedes onward have reported or displayed emotions ranging from mild pleasure to ecstatic elation following discoveries, and intense frustration bordering on despair when solutions seemed unreachable (Fitzgerald and O'Brien, 2007). Technical workplace subjects report feelings of pleasure ranging from “relieved and happy” to “all hyped” and “wonderful” accompanying successful systemizing, and varying levels of frustration accompanying failures (Amabile *et al.*, 2005). Similarly, working scientists report frustration levels from “agitation” to “real bitter” in the face of seemingly intractable problems, and describe break-through insights as “really exciting” and “orgasm” (Shaw, 1999). The verbal richness of such reports indicate none of the alexithymia typically associated with ASD (Fitzgerald and Bellgrove, 2006); indeed, many subjects in these studies provide elaborate descriptions of “Aha!” contexts that clearly indicate competent mentalizing.

Personality characteristics and pathologies typical of hyper-systemizers and hypo-systemizers (i. e. hyper-mentalizers) provide supporting evidence that systemizing and mentalizing are associated with distinct affective spectra. Obsessive focus and social withdrawal are common to creative artists and scientists, but creative artists do not display the aversion to strong social emotions typical of creative scientists (Feist, 1998; 1999). Highly creative scientists report bouts of intense frustration, but clinical depression is more common in highly creative writers and artists (Feist, 1999; Nettle, 2001). Highly creative scientists may experience ecstatic pleasure following insights, but also report periods of uncertainty following the initial pleasure (Shaw, 1999) and demonstrate high levels of the Big Five factor Conscientiousness (Feist, 1998, Nettle, 2007). Highly creative artists demonstrate lower levels of Conscientiousness (Feist, 1998), and creative writers and artists are more prone to mania, and to

psychotic spectrum disorders (PSD) in general (Nettle, 2001). It is interesting in this regard that while many prominent scientists have been retrospectively diagnosed with ASD (Fitzgerald & O'Brien, 2007), many prominent religious figures have been retrospectively diagnosed with epilepsy (Saver and Rabin, 1997), consistent with the more general association of hyper-religiosity with PSD (Nettle, 2001; Previc, 2006; Crespi and Badcock, 2008).

While the ability of putative hyper-systemizers to experience intense pleasure following insightful discovery has been much celebrated, a second characteristic of this population is less often noted: many take and have taken substantial risks, and many have died, in pursuit of explanations. Much scientific work is inherently dangerous, and the dangers are often best understood by those who risk them. Marie Curie and Enrico Fermi, for example, are only the best known of the nuclear physicists who have died of radiation-associated cancers. Risky self-experimentation by naturalists and physicians has been commonplace throughout history. The pursuit of systemizing explanations has, moreover, been vigorously suppressed by governments and religious authorities until well into the 20<sup>th</sup> century, and the list of scientists and philosophers persecuted or executed for violating legal or socio-cultural sanctions against systemizing as a cognitive activity is long. It could be argued, on a case-by-case basis, that various forms of social status motivated this history of risk taking, but the alternative hypothesis of intrinsic motivation is more consistent with cases for which self-reports are available (e. g. Fitzgerald and O'Brien, 2007). An understanding of hyper-systemizing as a cognitive-affective style must provide a natural explanation for this extraordinary tolerance of mortal risk.

Behavior patterns characterized by intense frustration and pleasure, obsessive focus on reward-producing activities, and a high tolerance for significant risk in the pursuit of rewards are suggestive of addiction (Hyman *et al.*, 2006). The primary hypothesis of this paper is that the human drive to explain is implemented, in the case of systemizing but not of mentalizing, by the mesocorticolimbic pathway active in drug addictions. Hyper-systemizers, in other words, are addicted to the experience of insight. The next section shows that this hypothesis not only explains the distinctive affective and personality associations of hyper-systemizing, but also provides mechanistic bases for the divergence of systemizing from mentalizing and for the characteristic, structural-analogy based creativity of hyper-systemizers.

### **3. Hyper-systemizing as addiction**

Over the past decade, the previous primarily hedonic concept of addiction has been replaced by an integrated-systems concept that emphasizes reward-related learning, and in particular unconscious increases in salience of reward-related stimuli (Kelley and Berridge, 2002; Hyman, 2005; Nestler, 2005; Hyman *et al.*, 2006; Grace *et al.*, 2007; Robinson and Berridge, 2008). The primary components of this model are that 1) a specialized mesocorticolimbic network encompassing nucleus accumbens (NAc), ventral tegmental area (VTA), amygdala, orbito-frontal cortex (OFC), dorsolateral prefrontal cortex (DLPFC), anterior cingulate cortex (ACC) and hippocampus mediates detection of, assignment of reward relevance and salience to, and learning and memory of cues associated with natural rewards including food and mating opportunities; 2) hedonic experience is generated in response to reward-related cues by activation of opiate and cannabinoid receptors in NAc and ventral pallidum, but is experienced in association with perceived cues and influences attentional focus via its representation in OFC and ACC (Berridge and Kringelbach, 2008; Kringelbach and Berridge, 2009; Smith *et al.*, 2009); 3) addictive drugs activate NAc and/or VTA directly, and induce both salience

enhancement and associative learning of drug-related cues and pleasurable drug-related experience via induced activity of the pathway as a whole. Both the increased salience and pleasurable associations of drug-related cues contribute to the craving and risk-tolerant reward-seeking behaviors typical of addictions.

The perceptual cues that contribute to representations of problems to be solved by mentalizing are in most cases, and arguably in all cases relevant to early development, associated with either natural rewards (e. g. smiling human faces) or natural dangers (e. g. angry human faces, snarling animals). In contrast, the perceptual cues that contribute to representations of problems to be solved by systemizing do not in general indicate natural rewards; in principle, *any* perceptual cue, however subtle, could contribute to the representation of a systemizing problem. The pleasure associated with systemizing solutions must therefore, like the pleasure associated with addictive drugs, result from cognitively-mediated, not direct perceptual, activation of the mesocorticolimbic reward system. That an internally-generated representation, not a percept, triggers the pleasure response associated with systemizing is further indicated by the fact that the sudden, intense pleasure of “Aha!” is associated not with the often-extended process of grappling with a problem, but rather with the recognition of a solution. Indeed, as discussed below, the pleasure response may be a component of solution recognition.

Multiple lines of evidence, all indirect, suggest that ACC is the locus of interaction between problem solving processes and the mesocorticolimbic reward pathway, and that an ACC-generated task-completion signal, in an appropriate context, is the internal representation that triggers an “Aha!” response. ACC monitors conflicts between cortical representations of current and goal states and evaluates the results of actions against the goals they are intended to achieve (Botvinick *et al.*, 2004; Botvinick, 2007; Carter and van Veen, 2007), signaling both positive and negative progress toward a goal in a context-dependent way (Kennerley *et al.*, 2006; Holroyd *et al.*, 2008; Quilodran *et al.*, 2008). Reciprocal interactions between ACC and DLPFC implement attentional control (Ridderinkhof *et al.*, 2004; Carter and van Veen, 2007); reciprocal interactions between ACC and OFC enable context-dependent re-evaluation of goals (Rushworth *et al.*, 2007; Rolls and Grabenhorst, 2008). ACC task-monitoring signals to the basal forebrain modulate the intensity of attentional focus; feedback of task-monitoring signals to OFC via NAc and VTA enables the experience of attentional focus combined with expectation of success as concentration, task difficulty, motivation to continue, or fatigue (Sarter *et al.*, 2006; Grace *et al.*, 2007; Boksem and Tops, 2008). Hence ACC measures progress, or lack thereof, toward a solution, and communicates this measurement to other components of the mesocorticolimbic reward pathway in a way that not only enables the use of such discrepancies to manage the problem-solving process, but also enables both the affective re-evaluation of goals and the affective experience of problem-solving progress or frustration.

Consistent with the role of ACC as an affect-mediating progress monitor, processes in which actual-state to goal-state conflicts are small are experienced as pleasurable and motivating, presumably via activation of dopamine-driven reward pathways (Kaplan and Oudeyer, 2007). Fluent performance of an effortful task requiring focused concentration can be intensely enjoyable, provided the performance remains fluent and conscious decision-making is not required (Csikszentmihalyi, 1996; Dietrich, 2004); the fact that the effortful activity itself, not just its successful outcome, is experienced as pleasurable indicates that in such cases process monitoring is directly coupled to affective experience. The specific dissociation of executive control and the feeling of effort in a patient with left ACC and OFC damage (Naccache *et al.*, 2005) confirms this direct link from ACC to the conscious representation of task progress. Positive affect correlates with ease of performance, and hence presumably with small actual-

state to goal-state conflict signals from ACC, in tasks in which performance difficulty is varied without a subject's knowledge (Winkielman and Cacioppo, 2001; Winkielman *et al.*, 2003), and processing fluency may explain preferences for prototypes in cases ranging from facial features to abstract designs (Winkielman *et al.*, 2006). These observations all suggest that positive affect increases as ACC-signaled discrepancy from a problem-solving goal decreases. If this is correct, the sudden convergence of a problem-solving process to its goal would be expected to induce a burst of positive affect, an “Aha!” experience.

If it is assumed that an ACC-generated task-completion signal indeed couples problem solving to the mesocorticolimbic reward pathway, the hypothesis that hyper-systemizing reflects addiction to insight becomes the hypothesis that, in at least some individuals with a pre-existing bias toward systemizing, ACC-generated task-completion signals can induce sufficiently strong responses in VTA and NAc to trigger enhanced positive valence assignment to and enhanced salience of cues associated with systemizing. On this model, routine solutions to routine problems would be expected to produce mild positive affect, but insightful solutions of hard problems, in particular, would be expected to provide sudden and strong task-completion responses and hence sudden and intense pleasure. One would expect, therefore, enhanced positive valence and enhanced salience of cues associated, in particular, with hard problems similar to ones previously solved with insight. Typical of drug addictions is the overvaluing of drug-related cues, to the extent that they supplant even cues for natural rewards such as food and sex, with a resulting narrowing of focus of behavior (Hyman *et al.*, 2006). If insight addiction proceeds by a similar pathway, similar overvaluing of insight-problem related cues would be expected in this case. Hence by analogy with drug addictions, one would expect overvaluing and enhanced salience not only of specific problem-identifying cues, but also of contextual and interoceptive cues associated with solving hard problems by insight.

Three aspects of context and interoceptively-accessible cognitive “set” are consistent across anecdotal reports, survey-based measurements, and experimental studies of systemizing resulting in insight: moderate social withdrawal (Feist, 1998; Fitzgerald and O'Brien, 2008), positive affect (Shaw, 1999; Fredrickson, 2004; Amabile *et al.*, 2005, Fitzgerald and O'Brien, 2008; Kounios and Beeman, 2009), and an attentional focus correlated with decreased default-network activation (Buckner *et al.*, 2008; Kounios and Beeman, 2009). These effects are not uncorrelated. The default network links temporal-parietal junction (TPJ) areas implementing ToM to attention-control areas of medial prefrontal cortex including ACC (Raichle and Snyder, 2007; Buckner *et al.*, 2008). Default network activity is experienced as self-conscious and self-relevant reminiscence and future-oriented planning, typically in the modality of inner speech and accompanied by content-relevant emotions, typically social emotions (Northoff *et al.*, 2006; Raichle and Snyder, 2007; Buckner *et al.*, 2008; Schilbach *et al.*, 2008). Default network activity is often obsessive, and high levels of default activity correlate with both major depression (Sheline *et al.*, 2009) and schizophrenia (Kim *et al.*, 2009). Artists often find inspiration in such emotionally-colored reflections (Nettle, 2001), but scientists tend toward discomfort with social emotions, particularly those associated with inter-personal conflict (Feist, 1998), and employ social withdrawal both to escape such emotions and to avoid distractions. Scientists are often happiest when “lost in their work,” a flow-like state characterized by attentional focus on non-self-oriented elements of a task, and hence correlated with low default network activation (Buckner *et al.*, 2008). It seems reasonable, therefore, to propose that decreased default network activity, experienced as a pleasant absence of uncomfortable and distracting self-oriented social emotions, is a highly-salient, over-learned cue associated with insight by hyper-systemizers.

Decreased default-network activity, and hence decreased mentalizing, self-referential thinking, and experienced social emotions (Buckner *et al.*, 2008) appears to be not only a correlate of problem solving by insight, but an enabler and possibly a prerequisite of problem solving by insight. Experimental studies of insight have focused on the formation of distant semantic connections (Bowdon *et al.*, 2005), a precursor of the formation of the structural analogies typical of insightful solutions of real-world problems (Gentner, 2003; 2005; Leech *et al.*, 2008). Formation of distant semantic connections requires activity in predominantly right-hemisphere association areas (Jung-Beeman *et al.*, 2004; Kounios *et al.*, 2007; Sandkuhler and Bhattacharya, 2008) that overlap strongly with the default network (Bar, 2008). Recruiting these resources for systemizing would, therefore, require suppressing attention to default network activity. Interactions between ACC and rostral PFC appear to implement this suppression of default network activity in insight (Kounios *et al.*, 2006; Subramaniam *et al.*, 2009), analogy (Green *et al.*, 2006), and integration of solution components from different subtasks (De Pisapia and Braver, 2008). ACC-driven suppression of default network activity correlates with and may implement the facilitation of insight by positive affect (Subramaniam *et al.*, 2009; Kounios and Beeman, 2009). The involvement of rostral PFC in suppression of default network activity is consistent with its general role in attention switching and multi-tasking (Gilbert *et al.*, 2005; Dreher *et al.*, 2008).

The foregoing considerations show that if the mesocorticolimbic addiction pathway is activated by the experience of insightful solutions to systemizing problems, the activity of this pathway can be expected to 1) increase any pre-existing bias toward systemizing; 2) decrease the frequency and intensity of mentalizing, self-referential thinking, and self-relevant emotional experience; and 3) increase the probability of recognizing distant semantic connections and analogies, all by the common mechanism of over-learned suppression of default-network activity. The hypothesis that hyper-systemizing is insight addiction thus provides a mechanism by which hyper-systemizing can develop in individuals with normal-range mentalizing ability, and explains both an increasing bias against mentalizing and increased creativity in solving systemizing problems in such individuals. Moreover, it explains common personality characteristics of hyper-systemizers, including their tendencies toward social withdrawal and their aversion to social conflict and social distractions. It is significant, moreover, that an addiction-driven mechanism would not be expected to develop hyper-mentalizing from a pre-existing mentalizing bias. As outlined above, mentalizing is associated with a broad spectrum of social emotions, many of them unpleasant; hence mentalizing cannot be expected to consistently activate the reward pathway.

#### **4. Hyper-systemizers as “different minds”**

Since the suggestion of Happé (1999) that autism be conceptualized as a cognitive style, it has become increasingly clear that individuals with Asperger's Syndrome and high-functioning ASD think differently from others, often in ways that may be beneficial to their societies (Fitzgerald and O'Brien, 2008; Spikins, 2009). The systemizing concept extends this idea into the general population. What has been shown here is that systemizing can develop into hyper-systemizing through the action of the relatively well-understood mesocorticolimbic reward pathway; hence hyper-systemizing would be expected to appear in a human population even in the absence of deficit mentalizing, provided that a distribution of biases toward systemizing exists in the population, and that some individuals experience systemizing with sufficient intensity to trigger the reward pathway. If this is correct, hyper-systemizers are “different minds” with functional characteristics similar to but less pronounced than those

diagnostic of Asperger's Syndrome, produced by a more gradual developmental trajectory from an initial state that would not be considered deficit.

The default network is a neurofunctional locus of the cognitive differences observed in hyper-systemizing and ASD. Default network activity is significantly lowered in ASD, as is default-network deactivation in attention-switching tasks (Kennedy *et al.*, 2006). It has been suggested that disruption of the default network, or of executive control systems regulating its activation, may underlie the cognitive deficits observed in ASD (Buckner *et al.*, 2008). A broader review of functions associated with TPJ, however, suggests that differences in default-network activity in both ASD and hyper-systemizing may indicate broader differences in functional specialization of the temporal-parietal-frontal midline network. TPJ has long been associated with the “social brain” (Saxe *et al.*, 2004; Frith, 2007). However, it is increasingly clear that regions of TPJ associated with ToM activity overlap with regions implicated in the ventral attention network (Decety and Lamm, 2007; Corbetta *et al.*, 2008; Mitchell, 2008), as well as with association areas involved in planning, semantic association, and analogy (Bar, 2008). No study of TPJ to date has classified neurotypical or control subjects by their biases on the systemizing – mentalizing axis. The intriguing possibility that TPJ and surrounding areas of temporal and parietal cortex are employed differently by systemizers as compared to mentalizers is supported by the observation that mirror-system neurons, distributed throughout a frontal-parietal network but concentrated in the superior temporal sulcus (STS), are responsive to non-biological as well as biological motion patterns (Vingerhoets *et al.*, 2002; Schubotz and van Cramon, 2004; Engel *et al.*, 2007), change their specificities in response to usage patterns (Catmur *et al.*, 2007; 2008), and appear to be employed for such typical systemizing tasks as estimating forces in physical interactions (Hegarty, 2004; Wolff, 2007) and solving mathematical problems (Qin *et al.*, 2004; Cantlon *et al.*, 2006). Daydreaming involves co-activation of the default network, ACC, and rostral PFC (Christoff *et al.*, 2009), but systemizers may daydream about physics or math problems; default network activity during daydreams with these contents has not been specifically investigated. The default deactivation anomalies (Kennedy *et al.*, 2006), low level of self-referential thinking (Lombardo *et al.*, 2007), and differences in medial-frontal connection patterns observed in ASD (Gilbert *et al.*, 2009), as well as the attentional control differences observed in hyper-systemizers (Billington *et al.*, 2008), may all reflect differences in resource allocation across the temporal-parietal-frontal midline network, not deficits, disorganization or dysfunction. Even such seemingly hard-wired structures as the fusiform face area (FFA) may implement non-social functions in some individuals (Grill-Spector *et al.*, 2006/2007). The “social brain,” in other words, may be a developmental outcome, not an anatomical category. Such a finding would vindicate the proposal, made over a decade ago by Karmaloff-Smith (1995), that functional modularization results from developmental processes acting within relatively broad genetic constraints.

If systemizing and mentalizing are in fact different specialized uses of the same temporal-parietal-frontal networks, inter-subject activation differences observed in imaging studies of a variety of cognitive tasks can be expected to resolve into meaningful subpopulation differences if correlated with subject SQ scores. Subjects with high SQ scores would, in particular, be expected to display lower face specificity in FFA, lower biological-motion specificity in the mirror system, and lower contrasts network-wide between systemizing and mentalizing tasks. Confirmation of such differences in subjects with high SQ scores but normal-range mentalizing ability would provide direct support for functionally-significant neurocognitive diversity in the “neurotypical” population.

## 5. Predictions of the addiction model of hypersystemizing

The model of hyper-systemizing developed here does not attempt to explain the origin of a bias toward systemizing in some individuals; such a bias may be innate, or may develop in response to differential experience with objects during infancy (Rakison and Yermolayeva, 2010). The current model does propose an explanation of the development of hyper-systemizing in individuals with normal-range mentalizing, and hence with no clinically-significant deficits in mentalizing that may associate with neurofunctional deficits. The explanation proposed here is that hyper-systemizing is a consequence of decreases in default network activity driven by the mesocorticolimbic reward pathway in response to process-monitoring and task-completion signals generated by ACC. In particular, the current model proposes that hyper-systemizing is driven by addiction to insight: to the experience of solving problems, not to the practical or social outcomes of successful problem solving.

The model proposed here makes predictions in three areas. First, it predicts a developmental trajectory from early childhood to a hyper-systemizing adult that is consistent with an addiction process. Such a trajectory would be expected to include a childhood interest in parts and mechanisms, precocious specialization in one or more areas of endeavor that involve challenging problems demanding systemizing solutions, social self-isolation and decreased self-relevant emotion, active seeking for harder and harder problems, and intense frustration in situations in which “interesting” problems are unavailable or unsolvable. While anecdotal evidence suggests that such trajectories are not uncommon among scientists, longitudinal studies that incorporate the SQ or a similar instrument together with specific observations at multiple ages would be needed to test this prediction. The observation that narrow intense interests at pre-school ages often involve functional objects with discernible parts and occur most frequently in males (DeLoach *et al.*, 2007) may indicate a productive starting point for characterizing the developmental trajectory of hyper-systemizing.

The present model also predicts distinctive associations between affect and task in hypersystemizers. Individuals oriented toward systemizing and hence scoring high on SQ would be expected merely on the basis of the instrument design to enjoy solving problems demanding systemizing more than would individuals with low orientations to systemizing. The present model predicts, however, that high-SQ individuals will report greater enjoyment when solving hard but tractable problems than when solving easy problems. It predicts that individuals with high SQ scores will display greater intolerance of interruption or social interactions during problem solving than low- or moderate-SQ individuals, even if the social interactions are positive in tone. It also predicts that high-SQ individuals, but not low- or moderate-SQ individuals, will neglect otherwise-attractive stimuli, including stimuli indicating natural rewards, when engaged in a problem-solving session.

Finally, the present model predicts that although SQ is a measure of orientation toward or preference for systemizing as a problem-solving strategy, individuals with high SQ scores will also exhibit better performance than individuals with “balanced” minds and hence moderate SQ scores when presented with systemizing tasks. The recent observation that high SQ correlates with high mental rotation ability (Cook and Saucier, 2010) is consistent with this prediction. The decreased default network activation invoked as mechanism by the model predicts, in particular, that high-SQ individuals will display enhanced analogical reasoning abilities. High-SQ individuals would be expected to perform better than “balanced” controls of matched verbal ability on semantic-association tests as employed in insight studies (Jung-Beeman *et al.*, 2004; Kounios *et al.*, 2007; Sandkuhler and Bhattacharya, 2008) and on standard verbal analogies (Gentner, 2003; Holyoak, 2005). High-SQ individuals would also be

expected to perform better than “balanced” controls of matched visuo-spatial ability on tool-improvisation analogies, which comparative and neuro-functional evidence suggests are implemented primarily by the perceptual binding and pre-motor systems (Fields, 2010).

## **6. Conclusion**

A minority of the human population exhibits a strong bias toward systemizing, a problem-solving and explanatory style that relies on hypothesized physical mechanisms, not on intentional agency. The phenomenology of hyper-systemizing is suggestive of an addiction syndrome. The hypothesis that hyper-systemizing is driven by the mesocorticolimbic reward pathway active in drug addictions explains not only the phenomenology typical of hyper-systemizing, but also the bias against mentalizing displayed by hyper-systemizers and their tendency toward creativity, particularly in the generation of novel structural analogies. It further suggests that systemizing and mentalizing are different developmental specializations of a common temporal-parietal-frontal network. If this hypothesis is correct, significant differences in activity patterns in multiple regions would be expected if “neurotypical” subject populations were tested for systemizing bias. Hyper-systemizers would be expected, moreover, to exhibit a higher than average probability of insight solutions to standard word problems when controlled for vocabulary size and verbal abilities, a correlation between positive affect and low default-network activity, and similar, higher levels of ventral attention-system activity in both externally-oriented and imaginative systemizing tasks compared to either externally-oriented or imaginative mentalizing tasks.

The model of hyper-systemizing outlined here implies that hyper-systemizing is an instance of developmental specialization producing functionally-significant neurocognitive diversity. Spikins (2009; 2010) has suggested, based in part on the culturally-significant abilities associated with Asperger's Syndrome, that neurocognitive diversity may be a major driver of human evolution. For this to be the case, culturally-significant neurocognitive diversity must be both heritable and culturally tolerated. If the conclusions reached here are correct, hyper-systemizing will appear in human populations provided small biases toward systemizing occur in the population, and these biases are amplifiable by the mesocorticolimbic reward pathway. In this case, the requirement for heritability is reduced to a requirement for a population-level distribution in systemizing bias, and the requirement for tolerance is reduced to a requirement for tolerance of small biases toward systemizing. Hyper-systemizing could appear, in such a population, without assortative mating between individuals biased toward systemizing, and even without cultural tolerance for hyper-systemizing. Similar mechanisms capable of amplifying small differences in network activity into culturally-significant neurocognitive diversity may be active in other areas as well.

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## **Conflict of Interest Statement**

The author declares that he has no conflicts of interest relevant to the work presented in this paper.

## References

- Adolphs, R. (1999). Social cognition and the human brain. *Trends in Cognitive Sciences* 3(12), 469-479.
- Adolphs, R. (2003). Cognitive neuroscience of human social behavior. *Nature Reviews Neuroscience* 4, 165-178.
- Amabile, T. M., Barsade, S. G., Mueller, J. S. & Staw, B. M. (2005). Affect and creativity at work. *Administrative Science Quarterly* 50, 367-403.
- Bar, M. (2008). The proactive brain: Using analogies and associations to derive predictions. *Trends in Cognitive Sciences* 11(7), 280-289.
- Baron-Cohen, S. (2002). The extreme male brain theory of autism. *Trends in Cognitive Sciences* 2(2), 248-254.
- Baron-Cohen, S. (2008). Autism, hypersystemizing, and truth. *The Quarterly Journal of Experimental Psychology* 61(1), 64-75.
- Baron-Cohen, S., Wheelwright, S., Skinner, R., Martin, J. & Clubley, E. (2001). The Autism-Spectrum Quotient (AQ): Evidence from Asperger Syndrome/high-functioning autism, males and females, scientists and mathematicians. *Journal of Autism and Developmental Disorders* 31, 5-17.
- Baron-Cohen, S., Wheelwright, S., Lawson, J., Griffin, R. & Hill, J. (2002). The exact mind: Empathizing and systemizing in autism spectrum conditions. In U. Goswami (Ed.) *Handbook of Cognitive Development*. Oxford: Blackwell.  
([http://www.autismresearchcentre.com/docs/papers/2002\\_BCetal\\_Goswami.pdf](http://www.autismresearchcentre.com/docs/papers/2002_BCetal_Goswami.pdf))
- Baron-Cohen, S., Richler, J., Bisarya, D., Gurunathan, N. & Wheelwright, S. (2003). The systemizing quotient: An investigation of adults with Asperger syndrome or high-functioning autism, and normal sex differences. *Philosophical Transactions of the Royal Society of London B* 358, 361-374.
- Baron-Cohen, S., Knickmeyer, R. C. & Belmonte, M. K. (2004) Sex differences in the brain: Implications for explaining autism. *Science* 310, 819-823.
- Berridge, K. E. & Kringelbach, M. L. (2008). Affective neuroscience of pleasure: reward in humans and animals. *Psychopharmacology* 199, 457-480.
- Billington, J., Baron-Cohen, S. & Bor, D. (2008). Systemizing influences attentional processes during the Navon task: An fMRI study. *Neuropsychologia* 46, 511-520.
- Boksem, M. A. S. & Tops, M. (2008). Mental fatigue: Costs and benefits. *Brain Research Reviews* 59, 125-139.

- Botvinick, M., Cohen, J. & Carter, C. (2004). Conflict monitoring and anterior cingulate cortex: An update. *Trends in Cognitive Sciences* 8(12), 539-546.
- Botvinick, M. (2007). Conflict monitoring and decision making: Reconciling two perspectives on anterior cingulate function. *Cognitive, Affective, & Behavioral Neuroscience* 7(4), 356-366.
- Bowden, E. M., Jung-Beeman, M., Fleck, J. & Kounios, J. (2005). New approaches to demystifying insight. *Trends in Cognitive Sciences* 9(7), 322-328.
- Buckner R, Andrews-Hanna J & Schacter, D (2008). The brain's default network: Anatomy, function, and relevance to disease. *Annals of the New York Academy of Sciences* 1124, 1-38.
- Burgess, P. W., Simons, J., Dumontheil, I. & Gilbert, S. (2007). The gateway hypothesis of rostral prefrontal cortex (area 10) function. In J. Duncan, L. Phillips & P. McLeod (Eds.) *Measuring the Mind: Speed, Control, and Age* (pp. 217-248). Oxford University Press.
- Cantlon, J., Brannon, E., Carter, E. & Pelphrey, K. (2006). Functional imaging of numerical processing in adults and 4-y-old children. *PLOS Biology* 4(5) 0844-0854.
- Carter, C. S. & van Veen, V. (2007). Anterior cingulate cortex and conflict detection: An update of theory and data. *Cognitive, Affective, & Behavioral Neuroscience* 7(4), 367-379.
- Catmur, C., Walsh, V. & Heyes, C. (2007). Sensorimotor learning configures the human mirror system. *Current Biology* 17, 1527-1531.
- Catmur, C., Gillmeister, H., Bird, G., Liepelt, R., Brass, M. & Heyes, C. (2008). Through the looking glass: Counter-mirror activation following incompatible sensorimotor learning. *European Journal of Neuroscience* 28, 1208-1215.
- Christoff, K., Gordon, A. M., Smallwood, J., Smith, R. & Schooler, J. W. (2009). Experience sampling during fMRI reveals default network and executive system contributions to mind wandering. *Proceedings of the National Academy of Sciences USA* 106(21), 8719-8724.
- Cook, C. M. & Saucier, D. M. (2010). Mental rotation, targeting ability and Baron-Cohen's empathizing-systemizing theory of sex differences. *Personality and Individual Differences* (in press).
- Corbetta, M., Patel, G. & Shulman, G. L. (2008). The reorienting system of the human brain: From environment to theory of mind. *Neuron* 58(3), 306-324.
- Covington, M. V. (2000). Goal theory, motivation, and school achievement: An integrative review. *Annual Review of Psychology* 51, 171-200.
- Crespi, B. & Badcock, C. (2008). Psychosis and autism as diametrical disorders of the social brain. *Behavioral and Brain Sciences* 31, 241-320.
- Csikszentmihalyi, M. (1996). *Creativity*. New York: Harper Perennial.

- De Pisapia, N. & Braver, T. S. (2008). Preparation for integration: The role of anterior prefrontal cortex in working memory. *NeuroReport* 19(1), 15-19.
- Decety, J. & Lamm, C. (2007). The role of right temporoparietal junction in social cognition: How low-level computational processes contribute to meta-cognition. *The Neuroscientist* 13(6), 580-593.
- DeLoach, J. S., Simcock, G. & Macari, S. (2007). Planes, trains, automobiles – and tea sets: extremely intense interests in very young children. *Developmental Psychology* 43(6), 1579-1586.
- den Ouden, H. E. M., Frith, U., Frith, C. & Blakemore, S.-J. (2005). Thinking about intentions. *NeuroImage* 28, 787-796.
- Dichter, G. & Belger, A. (2007). Social stimuli interfere with cognitive control in autism. *Neuroimage* 35(3) 1219-1230.
- Dietrich, A. (2004). Neurocognitive mechanisms underlying the experience of flow. *Consciousness and Cognition* 13, 746-761.
- Dreher, J.-C., Koechlin, E., Tierney, M. & Grafman, J. (2008). Damage to the fronto-polar cortex is associated with impaired multitasking. *PLOS One* 3(9), e3227.
- Dunbar, R. (2003). The social brain: Mind, language, and society in evolutionary perspective. *Annual Reviews of Anthropology* 32, 163-181.
- Eisenberger, N. I. & Lieberman, N. D. (2004). Why rejection hurts: a common neural alarm system for physical and social pain. *Trends in Cognitive Sciences* 8(7), 294-300.
- Eisenberger, N. I. (2006). Identifying the neural correlates underlying social pain: Implications for developmental processes. *Human Development* 49, 273-293.
- Engel, A., Burke, M., Fiehler, K., Bien, S. & Rosler, F. (2007). How moving objects become animated: The human mirror system assimilates non-biological movement patterns. *Social Neuroscience* 3, 368-387.
- Feist, G. J. (1998). A meta-analysis of personality in scientific and artistic creativity. *Personality and Social Psychology Review* 2(4), 290-309.
- Feist, G. J. (1999). Affect in artistic and scientific creativity. In S. W. Russ (Ed) *Affect, Creative Experience and Psychological Adjustment* (pp. 93-109). London: Taylor & Francis.
- Feist, G. J. & Gorman, M. E. (1998). The psychology of science: Review and integration of a nascent discipline. *Review of General Psychology* 2(1), 3-47.
- Fields, C. (2010). Implementation of structure mapping by event-file binding and action planning: A model of tool improvisation analogies. *Psychological Research* (in press).

- Fitzgerald, M. & Bellgrove, M. A. (2006). The overlap between alexithymia and Asperger's Syndrome. *Journal of Autism and Developmental Disorders* 36(4) 573-576.
- Fitzgerald, M. & O'Brien, B. (2007). *Genius Genes: How Asperger Talents Changed the World*. Shawnee Mission, KS: Autism Asperger Publishing.
- Fredrickson, B. L. (2004). The broaden-and-build theory of positive emotions. *Philosophical Transactions of the Royal Society of London B* 359, 1367-1377.
- Frith, C. (2007). The social brain? *Philosophical Transactions of the Royal Society of London B* 362, 671-678.
- Frith, U. and Frith, C. (1999). Interacting minds – A biological perspective. *Science* 286, 1692-1695.
- Frith, U. & Frith, C. (2003). Development and neurophysiology of mentalizing. *Philosophical Transactions of the Royal Society of London B* 358, 459-473.
- Gentner, D. (2003). Why we're so smart. In: D. Gentner & S. Goldin-Meadow (Eds.) *Language and Mind: Advances in the Study of Language and Thought*. Cambridge, MA: MIT Press. 195-235.
- Gentner, D. (2005). The development of relational category knowledge. In: L. Gershkoff-Stowe & D. Rakison (Eds) *Building object categories in developmental time* (pp. 245-275). Hillsdale, NJ: Erlbaum.
- Gibson, E. J. (1988). Exploratory behavior in the development of perceiving, acting, and the acquiring of knowledge. *Annual Review of Psychology* 39, 3-41.
- Gilbert, S., Frith, C. & Burgess, P. (2005). Involvement of rostral prefrontal cortex in selection between stimulus-oriented and stimulus-independent thought. *European Journal of Neuroscience* 21, 1423-1431.
- Gilbert, S. J., Meuwese, J. D. I., Towgood, K. J., Frith, C. D. & Burgess, P. W. (2009). Abnormal functional specialization within medial prefrontal cortex in high-functioning autism: a multi-voxel similarity analysis. *Brain* 132, 869-878.
- Goldenfeld, N., Baron-Cohen, S. & Wheelwright, S. (2006). Empathizing and systemizing in males, females, and autism. *Clinical Neuropsychiatry* 2, 338-345.
- Gopnik, A. (2000) Explanation as orgasm and the drive for understanding: The evolution, function and phenomenology of the theory-formation system. In F. Kiel & R. Wilson (Eds.) *Cognition and Explanation* (pp. 299-323). Cambridge, MA: MIT Press.
- Gopnik, A. & Schulz, L. (2004). Mechanisms of theory formation in young children. *Trends in Cognitive Sciences* 8(8) 371-377.
- Gottfried, A. E. (1985). Academic intrinsic motivation in elementary and junior high school students. *Journal of Educational Psychology* 77(6), 631-645.

- Grace, A. A., Floresco, S. B., Goto, Y. & Lodge, D. J. (2007). Regulation of firing of dopaminergic neurons and control of goal-directed behavior. *Trends in Neurosciences* 30(5), 220-227.
- Green, A., Fugelsang, J., Kraemer, D., Shamosh, N. & Dunbar, K. (2006). Frontopolar cortex mediates abstract integration in analogy. *Brain Research* 1096, 125-137.
- Grill-Spector, K., Sayres, R. & Ress, D. (2006/2007). High-resolution imaging reveals highly selective nonface clusters in the fusiform face area. *Nature Neuroscience* 9, 1177-1185. Corrigendum. *Nature Neuroscience* 10, 133.
- Grimm, S., Ernst, J., Boesiger, P., Schuepbach, D., Hell, D., Boeker, H. & Northoff, G. (2009). Increased self-focus in major depressive disorder is related to neural abnormalities in subcortical-cortical midline structures. *Human Brain Mapping* 30(8), 2617-2627.
- Happé, F. (1999). Autism: Cognitive deficit or cognitive style? *Trends in Cognitive Sciences* 3, 216-222.
- Hegarty, M. (2004). Mechanical reasoning by mental simulation. *Trends in Cognitive Sciences* 8 (6) 280-285.
- Holroyd, C. B. & Coles, M. G. H. (2008). Dorsal anterior cingulate cortex integrates reinforcement history to guide voluntary behavior. *Cortex* 44, 548-559.
- Holyoak, K. (2005). Analogy. In: K. Holyoak and R. Morrison (Eds) *The Cambridge Handbook of Thinking and Reasoning*. (pp. 117-142) Cambridge: Cambridge University Press.
- Hyman, S. E. (2005). Addiction: A disease of learning and memory. *American Journal of Psychiatry* 162, 1414-1422.
- Hyman, S. E., Malenka, R. C. & Nestler, E. J. (2006). Neural mechanisms of addiction: The role of reward-related learning and memory. *Annual Review of Neuroscience* 29, 565-598.
- Johnson, S. C., Shimizu, Y. A. & Ok, S.-J. (2007). Actors and Actions: The role of agent behavior in infants' attributions of goals. *Cognitive Development* 22(3), 310-322.
- Jung-Beeman, M., Bowden, E. M., Haberman, J., Frymiare, J. L., Arumbel-Liu, S., Greenblatt, R., Reber, P. J. & Kounios, J. (2004). Neural activity when people solve verbal problems with insight. *PLOS Biology* 2(4), 0500-0510.
- Kaplan, F. & Oudeyer, P.-Y. (2007). In search of the neural circuits of intrinsic motivation. *Frontiers in Neuroscience* 1(1), 225-236.
- Karmaloff-Smith, A. (1995). *Beyond Modularity: A Developmental Perspective on Cognitive Science*. Cambridge, MA: MIT Press.
- Kelley, A. E. & Berridge, K. C. (2002). The neuroscience of natural rewards: Relevance to addictive

drugs. *Journal of Neuroscience* 22(9), 3306-3311.

Kennedy, D., Redcay, E. & Courchesne, E. (2006). Failing to deactivate: Resting functional abnormalities in autism. *Proceedings of the National Academy of Sciences USA* 103(21) 8275-8280.

Kennerley, S. W., Walton M. E., Behrens, T. E. J., Buckley M. J. & Rushworth M. F. S. (2006) . Optimal decision making and the anterior cingulate cortex. *Nature Neurosciences* 9(7), 940-947.

Kim, D. I., Manoach, D. S., Mathalon, D. H., Turner, J. A., Mannell, M., Brown, G. G., Ford, J. M., Gollub, R. L., White, T., Wible, C., Belger, A., Bockholt, H. J., Clark, V. P., Lauriello, J., O'Leary, D., Mueller, B. A., Lim, K. O., Andreasen, N., Potkin, S. G. & Calhoun, V. D. (2009). Dysregulation of working memory and default-mode networks in schizophrenia using independent component analysis, an fBIRN and MCIC study. *Human Brain Mapping* 30(11), 3795-3811.

Kounios, J., Frymiare, J. L., Bowden, E. M., Fleck, J. I., Subramaniam, K., Parrish, T. B. & Jung-Beeman, M. (2006). The prepared mind: Neural activity prior to problem presentation predicts subsequent solution by sudden insight. *Psychological Science* 17, 882-890.

Kounios, J., Fleck, J. I., Green, D. L., Payne, L., Stevenson, J. L., Bowden, E. M. & Jung-Beeman, M. (2007). The origins of insight in resting-state brain activity. *Neuropsychologia* 46, 281-291.

Kounios, J. & Beeman, M. (2009). The "Aha!" moment: The cognitive neuroscience of insight. *Current Directions in Psychological Science* 18(4), 210-216.

Kringelbach, M. L. & Berridge, K. E. (2009). Toward a functional neuroanatomy of pleasure and happiness. *Trends in Cognitive Sciences* 13(11), 479-487.

Leech, R., Mareshal, D. & Cooper, R. (2008) Analogy as relational priming: A developmental and computational perspective on the origins of a complex cognitive skill. *Behavioral and Brain Sciences* 31, 357-378.

Lombardo, M., Barnes, J., Wheelwright, S. & Baron-Cohen, S. (2007). Self-referential cognition and empathy in autism. *PLOS One* 9 (e883) 1-11.

Lombrozo, T. (2006). The structure and function of explanations. *Trends in Cognitive Sciences* 10(10), 464-470.

Luo, Y., Kaufman, L. & Baillargeon, R. (2009). Young infants' reasoning about physical events involving inert and self-propelled objects. *Cognitive Psychology* 58, 441-486.

Markram, H., Rinaldi, T. & Markram, K. (2007). The Intense World Syndrome – an alternative hypothesis for autism. *Frontiers in Neuroscience* 1, 77-96.

Mitchell, J. P. (2008). Activity in right temporo-parietal junction is not selective for theory-of-mind. *Cerebral Cortex* 18, 262-271.

Naccache, L., Dehaene, S., Cohen, L., Habert, M.-O., Guichart-Gomez, E., Galanaud, D. & Willer, J.-

- C. (2005). Effortless control: executive control and conscious feeling of mental effort are dissociable. *Neurophychologia* 43(9), 1318-1328.
- Nestler, E. J. (2005). Is there a common molecular pathway for addiction? *Nature Neuroscience* 8(11), 1445- 1449.
- Nettle, D. (2001). *Strong Imagination: Madness, Creativity and Human Nature*. New York: Oxford University Press.
- Nettle, D. (2007). Empathizing and systemizing: What are they, and what do they contribute to our understanding of psychological sex differences? *British Journal of Psychology* 98, 237-255.
- Northoff, G., Heinzel, A., de Greck, R., Bernpohl, F., Dobrowolny, H. & Panksepp, J. (2006). Self-referential processing in our brain – A meta-analysis of imaging studies on the self. *NeuroImage* 31, 440-457.
- Paul G. (2009). The chronic dependence of popular religiosity upon dysfunctional social conditions. *Evolutionary Psychology* 7(3), 398-441.
- Previc, F. H. (2006). The role of extrapersonal brain systems in religious activity. *Consciousness and Cognition* 15(3), 500-539.
- Puce, A. & Perrett, D. (2003). Electrophysiology and brain imaging of biological motion. *Proceedings of the Royal Society of London B* 358, 435-445.
- Qin, Y., Carter, C., Silk, E., Stenger, V. A., Fissell, K., Goode, A. & Anderson, J. R. (2004). The change in brain activation patterns as children learn algebra equation solving. *Proceedings of the National Academy of Sciences USA* 101(15) 5686-5691.
- Quilodran, R., Rothe, M. & Procyk, E. (2008). Behavioral shifts and action validation in the anterior cingulate cortex. *Neuron* 57, 314-325.
- Raichle, M. E. & Snyder, A. Z. (2007). A default mode of brain function: A brief history of an evolving idea. *NeuroImage* 37, 1083-1099.
- Rakison, D. H. & Yermolayeva, Y. (2010). Infant categorization. *Wiley Interdisciplinary Reviews: Cognitive Science* 1 (in press).
- Reiss, S. (2004a). Multifaceted nature of intrinsic motivation: The theory of 16 basic desires. *Review of General Psychology* 8(3), 179-193.
- Reiss, S. (2004b). The sixteen strivings for God. *Zygon* 39(2), 303-320.
- Rejendran, G. & Mitchell, P. (2006). Cognitive theories of autism. *Developmental Review* 27, 224-260.
- Ridderinkhof, K. R., van der Wildenberg, W. P. M., Segalowitz, S. J. & Carter, C. S. (2004).

Neurocognitive mechanisms of cognitive control: The role of prefrontal cortex in action selection, response inhibition, performance monitoring, and reward-based learning. *Brain and Cognition* 56, 129-140.

Ring, H., Woodbury-Smith, M., Watson, P., Wheelwright, S. & Baron-Cohen, S. (2008). Clinical heterogeneity among people with high-functioning autism spectrum conditions: Evidence favoring a continuous severity gradient. *Behavioral and Brain Functions* 4(11) PMC2265729.

Robinson, T. E. & Berridge, K. E. (1993). The neural basis of drug craving: an incentive-sensitization theory of addiction. *Brain Research Reviews* 18, 247-291.

Robinson, T. E. & Berridge, K. E. (2008). The incentive sensitization theory of addiction: Some current issues. *Philosophical Transactions of the Royal Society B363*, 3137-3146.

Rolls, E. T. & Grabenhorst, F. (2008). The orbitofrontal cortex and beyond: From affect to decision-making. *Progress in Neurobiology* 86, 216-244.

Rushworth, M. F., Behrens, T. E., Rudebeck, P. H. & Walton, M. E. (2007). Contrasting roles for cingulate and orbitofrontal cortex in decisions and social behavior. *Trends in Cognitive Science* 11, 168-176.

Sandkuhler, S. & Bhattacharya, J. (2008). Deconstructing insight: EEG correlates of insightful problem solving. *PLOS One* 3(1), e1459.

Sarter, M., Gehring, W. J. & Kozac, R. (2006). More attention must be paid: The neurobiology of attentional effort. *Brain Research Reviews* 51, 145-160.

Saxe, R., Carey, S. & Kanwisher, N. (2004). Understanding other minds: Linking developmental psychology and functional neuroimaging. *Annual Review of Psychology* 55, 87-124.

Saxe, R., Tenenbaum, J. & Carey, S. (2005). Secret agents: Inferences about hidden causes by 10- and 12-month-old infants. *Psychological Science* 16, 995-1001.

Saxe, R., Tzelnic, T. & Carey, S. (2007). Knowing who dunnit: Infants identify the causal agent in an unseen causal interaction. *Developmental Psychology* 43, 149-158.

Schilbach, L., Eickhoff, S. B., Rotarska-Jagiela, A., Fink, G. R. & Vogeley, K. (2008). Minds at rest? Social cognition as the default mode of cognizing and its putative relationship to the “default system” of the brain. *Consciousness and Cognition* 17, 457-467.

Scholl, B. & Tremoulet, P. (2000). Perceptual causality and animacy. *Trends in Cognitive Sciences* 4 (8) 299-309.

Schubotz, R. & van Cramon, D. Y. (2004). Sequences of abstract nonbiological stimuli share ventral premotor cortex with action observations and imagery. *The Journal of Neuroscience* 24 (24) 5467-5474.

- Searle, J. R. (1980). Minds, brains and programs. *Behavioral and Brain Sciences* 3, 417-424.
- Shaw, M. P. (1999). On the role of affect in scientific discovery. In S. W. Russ (Ed) *Affect, Creative Experience and Psychological Adjustment* (pp. 147-162). London: Taylor & Francis.
- Sheline, Y. I., Barch, D. M., Price, J. L., Rundle, M. M., Vaishnavi, S. N., Snyder, A. Z., Mintun, M. A., Wang, S., Coalson, R. S. & Raichle, M. E. (2009). The default mode network and self-referential processes in depression. *Proceeding of the National Academy of Sciences USA* 106(6), 1942-1947.
- Smith, K. S., Mahler, S. V., 2010Pecina, S. & Berridge, K. C. (2009). Hedonic hotspots: Generating sensory pleasure in the brain. In M. L. Kringelbach & K. E. Berridge (Eds.) *Pleasures of the Brain* (pp. 27-49). Oxford: Oxford University Press.
- Sobel, D., Yoachim, C., Gopnik, A., Meltzoff, A. & Blumenthal, E. (2007). The blicket within: Preschooler's inferences about insides and causes. *Journal of Cognitive Development* 8(2) 159-182.
- Somerville, L., Heatherton, T. & Kelley, W. (2006). Anterior cingulated cortex responds differentially to expectancy violation and social rejection. *Nature Neuroscience* 9, 1007-1008.
- Spikins, P. (2009). Autism, the integrations of 'difference' and the origins of modern human behavior. *Cambridge Archaeological Journal* 19(2), 179-201.
- Spikins, P. (2010). 'The world don't move to the beat of just one drum': Autism and the challenge of 'different minds' for the social sciences. Preprint, University of York.
- Subrahmanyam, K., Gelman, R. & LaFosse, A. (2002). Animate and other separately-movable objects. In E. Fordes & G. Humphreys (Eds.), *Category-specificity in brain and mind* (pp. 341-373). London, England: Psychology Press.
- Subramaniam, K., Kounios, J., Parrish, T. B. & Jung-Beeman, M. (2009). A brain mechanism for facilitation of insight by positive affect. *Journal of Cognitive Neuroscience* 21, 415-432.
- Vingerhoets, G., de Lange, F., Vandemaele, P. Deblaere, K. & Achten, E. (2002) Motor imagery in mental rotation: An FMRI study. *NeuroImage* 17, 1623-1633.
- Wellman, H. M., Phillips, A. T., Dunphy-Lelii, S. & LaLonde, N. (2004). Infant social attention predicts preschool social cognition. *Developmental Science* 7(3), 283-288.
- Winkielman, P. & Cacioppo, J. T. (2001). Mind at ease puts a smile on the face: Psychophysiological evidence that processing facilitation elicits positive affect. *Journal of Personality and Social Psychology* 81(6), 989-1000.
- Winkielman, P., Schwarz, N., Fazendeiro, T. A. & Reber, R. (2003). The hedonic marking of processing fluency: Implications for evaluative judgement. In J. Musch & K. C. Klauer (Eds) *The Psychology of Evaluation: Affective Processes in Cognition and Emotion*. Mahwah, NJ: Lawrence Erlbaum.

Winkielman, P., Halberstadt, J., Fazendeiro, T. & Catty, S. (2006). Prototypes are attractive because they are easy on the mind. *Psychological Science* 17(9), 799-806.

Wolff, P. (2007). Representing causation. *Journal of Experimental Psychology, General* 136, 82-111.