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Affective Neuronal Darwinism:
The Nature of the Primary Emotional Systems

Judith A. Toronchuk

Psychology and Biology Departments,

Trinity Western University

and

George F. R. Ellis

Mathematics Department,

University of Cape Town

Contact information:

Psychology Department, Trinity Western University

7600 Glover Road, Langley, B.C. V2Y 1Y1 Canada.

Phone: 604-888-7511 extension 3104

email address: toronchu@twu.ca

Abstract

Higher brain functions are sculpted on the basis of the mainly “hard-wired” primary emotions through the process of Affective Neural Darwinism. Here we make a tentative new proposal as to the nature of the primary emotional systems, based largely on a combination of the work of Panksepp on affective neuroscience, the work of Stevens, Price and others on evolutionary psychiatry, and data on animal behaviour and neuroanatomy. In particular we suggest that in addition to the systems identified by Panksepp, there is substantial evidence for a hard-wired affective system dealing with power, rank, dominance and subordination which instantiates competitive behaviour and guarding of mates and other resources and which is the evolutionary source of self-esteem in humans. In a previous paper we also proposed an emotional organising programme instantiating disgust reactions which originally functioned in ancient vertebrates to protect against infection and toxins.

Introduction

In a previous paper (Ellis & Toronchuk, 2005), we proposed that the secondary emotions and intellect are developed in each individual through the developmental influences of the primary emotions which act collectively as fitness criteria (called a ‘value system’ by Edelman) to guide further neuronal development. Neural plasticity as characterised by Edelman’s neural Darwinism (Edelman, 1989, 1992) is, we suggested, naturally complemented by Panksepp’s formulation of affective neuroscience (Panksepp, 1998, 2001). Important aspects of the mechanism by which valenced affective feeling states provide fundamental values for the guidance of behaviour might be explicated by linking basic emotions to neural Darwinism (hence “Affective Neural Darwinism”, AND). This provides a possible extension of the role of Hebbian connectionism in guiding brain development, and it also integrates understanding of brain structure with evolutionary psychiatry and animal behaviour.

This synthesis takes into consideration the phylogenetic origins of the emotional systems, which we believe is necessary to fully understanding the many functional explanations already provided in the literature. Our model is dependent on one or more forms of neural plasticity guided by a process of selection of the most effective pathways based on the valences provided by the primary emotions. Selection occurs, as suggested by Edelman, at the level of the neural circuit, but also occurs at the level of individual neurons and synapses especially during development. Further we believe consideration of the phylogenetic origins of these systems contributes to the many functional explanations already provided in the literature.

Neurotrophins such as brain derived neurotrophic factor (BDNF) and monoamines such as serotonin play critical roles in neural Darwinism by promoting neural plasticity and determining which neurons survive (e.g. Hua & Smith, 2004; Lauder, 1993; Poo, 2001). Neurotrophins promote activity dependent refinement of neural networks, including cooperative selection of simultaneously active neurons. Complex interactions between neurotrophins, monoamines, cytokines and information processing in the brain effect neuronal survival and plasticity, but under adverse

conditions may give rise to psychiatric conditions and neurodegenerative disorders (reviewed in Castrén, 2005; Duman & Monteggia, 2006; Goodyer, 2008). Because brainstem matures earlier than forebrain, we propose that early activity in emotional circuits influences the wiring of higher brain areas by altering neurotrophin and neurotransmitter levels. For example dopamine knockout mice have reduced BDNF in the frontal cortex which may lead to reduced plasticity (Fumagalli, Racagni, Colombo, & Rival, 2003). Serotonin, present in early embryogenesis, plays several critical roles during neural development which leave permanent effects (reviewed in Whitiker-Azmitia, 2001). Because levels of neurotrophins such as BDNF (Gordon, Burke, Akil, Watson & Panksepp, 2003), and nerve growth factor (Alfonso, Pollevick, van der Hart, Flügge, Fuchs, & Frasch, 2004) are modulated by activations of emotional systems, it is reasonable to propose that the wiring of brain circuitry will be widely influenced by the nature, timing and amount of activation in emotional circuits during early life. Furthermore both the endocrine (e.g. Fumagalli, Molteni, Racagni & Riva, 2007) and immune systems play important roles in this development. In our earlier paper we suggested the immune system plays a vital role in neural wiring and evidence continues to accumulate supporting an immune role in development, plasticity and behavioural disorders (e.g. Baharnoori, Brake & Srivastava, 2009; Bauer, Kerr & Patterson, 2007; Bilboa, Yirmiya, Amat, Paul, Watkins, & Maier, 2008; Kemeny, 2009; McAfoose & Baune, 2009; Miller, 2009). In addition it has become clear in recent years that prenatal stress can alter the developmental course of the nervous system producing long-term changes in neurotransmitter systems and thereby increasing the risk of various disorders (e.g. Ansorge, Hen & Gingerich, 2007). Early life trauma also has similar effects raising the incidence of depression and anxiety disorders (Nemeroff, Bremner, Foa, Mayberg, North, & Stein, 2006; Goodyer, 2008). Each monoamine system can be activated by several emotions, however the specific mixture and location of signalling molecules will be different for each. Furthermore similar effects will continue to act throughout life facilitating brain plasticity, with the detailed neuronal micro-connections being continually affected by similar processes through limbic and brain stem activity which is then conveyed to the neocortex.

Thus we proposed (Ellis & Toronchuk, 2005) that the primary emotions working together with the endocrine and immune systems provide the core nature of the selection criteria guiding the refinement of synaptic connections in interaction with the physical and social environment, and so provide the emotional palette that guides brain development. It follows that elucidation of the specific nature of these systems is crucial to understanding the way the brain functions and structures itself.

Panksepp has described seven “primitive emotional operating systems that exist in limbic and reptilian areas of the brain” (Panksepp, 1998 p.52). These hierarchically organized executive operating systems give rise to specific valenced affective states while interacting with several layers of non-specific perceptual, attentional, and cognitive processes. Panksepp stresses that the differences between primary or prototypical emotions and secondary emotions, such as shame and guilt, include instantiation in the phylogenetically ancient medial and ventral brainstem pathways which are richer in visceral innervation and utilize a variety of visceral neuropeptides (Panksepp, 1998, 2003a). We concur with Panksepp’s seminal work and further propose that valenced primary emotions should not only be capable of altering

evolutionary survival rates, but be effective at the ontological level in neuronal group selection.

In this paper we propose an integrative viewpoint including an emphasis on an *evolutionary view* informed by vertebrate (as opposed to solely mammalian) phylogeny for identification of primary systems, and a resulting *proposal for a complete set of primary emotions*, extending previous proposals. In our thinking a phylogenetic approach to emotions (e.g. as described by Lawrence and Calder, 2004) is appropriate, but can be better understood by consideration of the entire vertebrate lineage. Phylogenetic history should be one major criterion for identification of primary emotions.

Our list of primary emotions takes into account previous proposals by numerous authors (e.g. Darwin 1872, republished in Darwin, Ekman, & Prodger, 2002; Damasio, 1999; Ekman, 1972; Izard, 1992), however it is based primarily on the comprehensive studies of the neuroscience of emotions carried out by Panksepp (1998). Although Ekman (e.g. 1992) and others also invoke evolution, most criteria for basic emotions have emphasised human facial or semantic features and have assumed a primarily communicative role for emotions (see Sabini & Silver, 2005 for a critique of Ekman's criteria). Such approaches begin with human subjective experience and then search for *ad hoc* supporting data from other mammals. In contrast we are proposing a framework informed by wider vertebrate evolution.

Basic Insights from Evolutionary Psychiatry

Two key issues emerge from discussions in evolutionary psychiatry (Stevens & Price, 2002; also e.g. Gilbert, 1989; McGuire & Troisi, 1998; Price et al., 2007) .

1. *Evolutionary pressures during the course of phylogeny developed various psychological traits that are experienced by us as emotions and feelings and give rise to behaviour which enhanced survival in ancestral environments*; for example the need for reproductive effectiveness results in emotional states of desire and bonding that promote propagation of our genes. This is a clear statement of the causal efficacy of emotions in terms of affecting the evolutionary process.

2. *Many psychiatric disorders result from malfunctioning of these evolutionary adaptive mechanisms; hence the nature of such disorders is evidence of the nature of the underlying emotional mechanisms*. This means we can attempt to relate basic emotional systems to specific evolutionary adaptations using psychiatric data as supportive evidence.

Stevens and Price (2002) emphasise in particular the pathologies that result on the one hand from failures in the *attachment system*, and secondly the *rank system*. They state (page 50) that these archetypal systems underlying social adjustment can “function healthily when evoked in appropriate circumstances, but either can give rise to pathology when their goals are frustrated or when they are inappropriately activated.” Both systems arose in social species from the advantage obtained by operating in social groups. The attachment system promotes mutual support and group cohesion and facilitates infant survival, development and learning. The rank or power/

dominance system, by providing a structure to allocate resources, reduces group conflicts. The rank system is served by an algorithmic assessor strategy that “has been in existence since the evolution of fish and reptiles . . . it enables an individual to assess whether a rival is weaker or stronger and to produce the appropriate response” (Stevens & Price, p.75). Subordinate animals thereby survive with a chance to reproduce another day.

This evidence from evolutionary psychiatry points to the existence of *a primary emotional system concerned with social rank or power/dominance as an addition to the SEEKING, FEAR, RAGE, LUST, PANIC, CARE and PLAY systems proposed by Panksepp*. Both the affiliative and dominance systems rely in part on phylogenetically ancient structures (Stevens & Price, p. 75). The evolution of lactation in mammals and then the period of prolonged primate infancy, reaching an apex in humans, necessitated an increase in parental care, which provided the basis for formation of increasingly stable social groups and highly developed attachment systems. In line with this we develop below a systematic proposal relating emotional systems and evolutionary survival needs incorporating both the power/dominance and attachment systems as important components.

The Underlying Propositions and Criteria

Our proposals are based on the following series of causal mechanisms:

1. Emotional systems emerged because they were causally effective in changing behavioural patterns and conscious emotional feelings.
2. Emotional systems were selected for in terms of their enhancement of survival capacity; this results in the evolution of relatively hard-wired primary emotional systems subject to limited fine-tuning by early life experiences, which then come to underlie the development of both intellectual capacities and more soft-wired secondary emotions.
3. Survival of some species is enhanced by group formation which allows cooperation, enhanced protection, group learning, and eventually evolving culture.
4. To make group membership effective, there must be both group cohesion mechanisms, and mechanisms for resolving conflict and resource allocation tensions.
5. The attachment system and the power/dominance system are two major emotional systems that evolved to meet these group needs; they supplement the basic systems for individual survival and learning, and include the systems for maternal care.
6. Individuals experience these as emotional systems underlying psychological and developmental events, particularly through subjective feelings thereby induced, without being aware of their evolutionary origins and function.

These mechanisms shared a common evolutionary imperative to meet developmental, needs in the ancestral environment, and continue to act in analogous ways today although in a new environmental context. We explore the implications in the next section.

Following on this, one can propose a clear set of criteria characterising primary emotional systems based on evolutionary aspects. We propose that a well-established primary emotional system should have *all* the following characteristics:

C1. *Concept:* It corresponds to a specific range of human affects and characteristic behaviours, associated with clear eliciting stimuli and with universal affective outcomes, expressed in specific bodily behaviour which may include facial expressions.

C2. *Structure:* It is effective through specific neural circuitry affected by various transmitters, neuromodulators, hormones and cytokines and will ultimately be traceable by neuroanatomical techniques. Each primary emotional circuit supervenes on a distinct pattern of neural pathways rather than on an exclusive set of structures. These circuits comprise distributed networks extending from brainstem to cortex; each is integrated with the pathways of other primary emotions which may use components of common brainstem pathways.

C3. *Function:* Each primary emotion system affects immediate affective functioning and because a particular combination of transmitters, neuromodulators, hormones and cytokines affects each primary system, each can functionally take part in neural Darwinism.

C4. *Development:* Development of these systems will be initiated by multiple genes and therefore susceptible to alteration by mutation or deletion. Environmental influences will further affect expression of these genes.

C5. *Origin:* It can be associated with adaptations expressed in cladistic homologous traits (Griffiths, 1997, p.213), and hence can be clearly related to an evolutionary process.

C6. *Occurrence:* It occurs universally in humans and can be associated with homologous systems and evolutionary precursors; this enables a correspondence of the features listed above (2-5) between humans and other vertebrates.

C7. *Outcome:* Dysfunctional aspects can be associated with behavioural or psychiatric disorders, whose nature is related to deletion of functions and/or disinhibition of lower level components of its circuitry, or to over-activation of these functions; and hence such disorders shed light on normal function.

One key problem is to separate what Panksepp refers to as reflexive affects such as the startle reflex, pain, hunger, thirst (2000), and sensory affects such as taste and smell (2005) from true primary emotional systems. Primary emotions are action promoting valenced states with distinct neural circuitry and neurochemistry, the initiation of which can precede or anticipate potential environmental events and the consequences of which can outlast the precipitating conditions. In contrast reflexive affects are closely time-locked to their triggering stimuli. An emotion is a *superordinate* program which orchestrates and integrates the activities of various functional subprograms including reflexive affects, but also subprograms governing perception, cognitive appraisals and feeling states (Cosmides & Tooby, 2000). Emotional systems thus organize complex but flexible reactions by activating or inhibiting autonomic, hormonal and/or somatic changes that were adaptive during evolutionary history. The specific combination of behavioural components will depend on context and eliciting stimulus.

A second key problem is to differentiate which of the human emotions should be considered primary and which secondary emotions. In principle items **C1**, **C2**, **C3** and **C4** should be universally consistent in each primary emotional system, but not necessarily consistent for each secondary emotion. Secondary emotions arise from interactions between primary emotional systems and cognitions instantiated in the more recently developed neocortex (Panksepp, 2000; Prinz, 2004, p.144-147). Therefore we do not expect secondary emotions to occur with as great universal consistency of structure and function nor do we expect widespread occurrence in other mammals.

Although there is no single criterion for discriminating primary from secondary emotions, our proposal is based on converging evidence from several methodologies (see Prinz, 2004, p.90). We suggest that a good proposal can be made for a primary emotional system when all of Items **C1-C3 and C5, C6** above occur; and is indisputable if the full set of items **C1-C7** have been established, with the cladistic link **C5** being especially important. *Existence* of an evolutionary explanation on its own is worth little because they are so easy to devise (Griffiths, 1997), but *its absence* is a strong mark against any proposal.

Emotional programmes are largely dependant on subneocortical structures, and thus primitive forms of consciousness may be present in other animals (Panksepp, 2005). Thus it is possible that the basic affective states of other animals, while not identical, may nevertheless correlate with those of humans and underlie what Damasio (1999, p.16) terms “core consciousness”.

The Relation between Needs and Emotional Systems

The primary emotions identified by Panksepp (1998) are¹:

E1: The SEEKING system: incentive motivation, seeking, expectancy.

E2: The RAGE system: rage/anger.

E3: The FEAR system: fear/anxiety.

E4: The LUST systems: lust/sexuality in the male and female.

E5: The CARE system: providing parental care/nurturance.

E6: The PANIC system: panic/separation, need of care.

E7: The PLAY system: rough-housing play/joy.

Table 1 summarises our proposed completion of Panksepp’s set of primary emotional systems, together with the functions and relation to evolutionary needs. *Each basic developmental need has been matched during evolution by a corresponding emotional system that has become genetically programmed in accord with the above theses.*

The basic emotional systems we identify are adaptations to survival needs of the individual in accord with the above theses. We propose that these emotional systems embody the selection criteria underlying neural Darwinism (functioning together to comprise Edelman’s value system) and thereby determining brain development. Thus they provide the affective valence underlying higher cognitive development (as proposed in Ellis & Toronchuk, 2005).

The first group of systems relate to the functioning of the individual, and the second to the functioning of individuals in social groups. Basic sensory-motor components involved in pattern-recognition, motor output and problem-solving underlie the

functioning of each emotional system in the same manner in that they make possible cognitive development and intellectual capacity. It is now acknowledged (e.g. Pessoa, 2008; Phelps, 2006) that cognition and emotion are entangled on all levels of processing and are not as easily separated as has been suggested in the past.

Basic Functioning of the Individual

Panksepp's **SEEKING System** is the primary task-oriented pathway by which various affective goals are met (Panksepp, 1998). The SEEKING system is generalised in its goals, able to be activated by any of the components of need but can also function in a non-specific manner. In Table 2 we provide examples of some hypothesized components of **E1**, which arouse, energize, and motivate the organism to engage with the environment. It is activated by primary biological needs (**D1-D6**), characterised by homeostatic detection mechanisms. Other primary emotional systems **E3-E9** also feed information to the SEEKING system, as do the secondary emotions **S1-S_N**, thereby each affecting the overall motivational state of the individual² **and** each characterised by a genre of intention and an intensity of desire ("I've got to drink first and then rest"). Furthermore it is through inclusion of conscious volitional goals **V₁-V_n** in **E1** that intentions and resulting purposive action gains its emotive power: "I want that job", "I need that house", and so on, have affective components. This is then the way that ethical choices and values (which are the basis on which we choose acceptable actions, for they are the topmost level of the hierarchy of goals) become effective in guiding action. They too have affective components underlying their effective power: "I want to be good", "I hate that evil", "I feel bad about poverty", "I must improve the situation", and so on. The subjective feeling associated with activation would be that of wanting or lacking something.

An accumulating body of evidence (e.g. Berridge, Robinson & Aldridge, 2009; Berridge & Kringelbach, 2008) suggests that reward functions may be parsed into two components--a motivational, appetitive system (corresponding to Panksepp's SEEKING/expectancy system) and an overlapping, but distinct hedonic appraisal or consummatory system.. Berridge refers to the former as "wanting" and the latter as "liking". The distinction is illustrated by the fact that addiction involves craving of substances or experiences but not necessarily experiencing satisfaction by them (Robinson & Berridge, 2003). Hence wanting and liking components involve separate neural pathways. While normally functioning together, these two systems can be behaviourally dissociated and function independently in rats (reviewed in Cannon & Besikri, 2004), humans (Knutson, Fong, Adams, Varner & Hommer, 2001) and even fish (see Spector, 2000), suggesting an evolutionary distinction between the two components.

The common ancestor of tunicates, lancelet and vertebrates was probably a filter feeder with adult behaviour limited to ingesting or rejecting particles that flowed through its oral cavity. Thus consummatory appraisal responses must have been present before appetitive seeking behaviours. The neural circuitry evaluating the hedonic value of oral stimuli is distributed from the medulla through the hypothalamus, ventral pallidum, nucleus accumbens and amygdala to orbitofrontal, insular and cingulate cortex. Decerebrate rats and anencephalic infants (lacking nucleus accumbens and other forebrain structures) show both hedonic responses and consummatory swallowing (reviewed in Steiner, Glaser, Hawilo & Berridge, 2001)

demonstrating that the lowest level of control for hedonic appraisal resides in the brainstem. Although taste was probably the earliest effective stimulus, the ventral pallidum, nucleus accumbens and several other forebrain structures developed to process and respond to pleasurable stimuli in many modalities. Thus the multi-modal nature of hedonic appraisal parallels that of the general purpose SEEKING system (see e.g. Burgdorf & Panksepp, 2006; Kelley & Berridge, 2002).

The mesolimbic dopamine system extending from the midbrain ventral tegmental area (VTA) through the lateral hypothalamus to the nucleus accumbens shell and orbitofrontal cortex has been widely implicated in the neural basis of reward; however both the wanting and liking components of the reward system also utilize endogenous opioids (Levine & Billington, 2004; Pecunia, 2008). Opioids act directly on VTA dopaminergic cells, suggesting that the pleasure and seeking components are intertwined. The mesolimbic dopamine system plays a fundamental role in learning, probably by associating arousal with specific activities, thus attaching a positive affective value to these activities which then acts as a stimulus for their repetition (see e.g. Wise, 2004).

Following Panksepp's suggestion (1998) that the SEEKING system evolved to provide a common currency of reward, we suggest that the SEEKING (**E1**) system facilitated by associated hedonic appraisals provides general coordination of affective responses. The dopamine system is activated not only by food, drugs, sex, electrical stimulation and monetary reward, but also by aversive stimuli with response segregation according to positive or negative valence occurring in separate regions of the nucleus accumbens (reviewed in Kelley & Berridge, 2002). The ventral pallidum implicated in the hedonic appraisal system also responds to a variety of stimuli; for example, the adjoining ventral globus pallidum of human males is activated both during sexual and competitive arousal (Rauch, Shin, Dougherty, Alpert, Orr, Lasko, et al., 1999) implying that the hedonic appraisal system also gives rise to pleasurable feelings associated with social dominance, as discussed further below.

Psychological illnesses associated with malfunctioning of the SEEKING system include most, if not all, types of addictions and cravings as well as eating disorders and possibly schizophrenia (see Panksepp, 1998; Panksepp & Harro, 2004). Panksepp (2002) also considers obsessive compulsive behaviours as malfunctions of this system. We will consider below the possibility that obsessive compulsive symptoms involving washing may arise instead from malfunctions of the DISGUST system while symptoms of checking and hoarding may arise from the POWER/dominance system.

Basic Survival of the Individual

In previous papers (Toronchuk & Ellis, 2007a, b) we argued for the inclusion in the primary emotional systems of one which instantiate behaviour opposite to that of SEEKING, but in like manner can be activated by many sensory modalities as well as ideational components. We chose to designate this as the **DISGUST system** rather than AVOIDANCE or AVERSION system because of the previous inclusion of disgust in various lists of basic emotions from Darwin onwards. Together the SEEKING and DISGUST systems would have been the primal organismal operating systems, the latter evolving from primitive chemosensory mechanisms adapted to

eject pathogens and their toxins from the gut. We propose this occurred in conjunction with the development of interaction between the immune and nervous systems (c.f. the discussion in Ellis & Toronchuk, 2005; Rubio-Godoy, Aunger & Curtis, 2007), as the primitive innate immune cells were probably phylogenetically developed first to deal with invasion of the internal milieu. Further adaptation led to learned avoidance of toxic or infected material before ingestion, as opposed to vomiting afterwards, and eventually led to the subjective human experience of disgust as an anticipatory mechanism for avoidance of harm before it occurs.

The DISGUST system meets the criteria we enumerate above as well as Panksepp's criteria for primary emotional systems (Panksepp, 1998, p.48ff; Panksepp, 2000; Toronchuk & Ellis, 2007a, b). In his discussion of our proposal for a disgust system Panksepp (2007) raises the concern that *distaste* may instead be merely a sensory affect; however we are using the term *disgust* in a broader general sense which parallels appetitive SEEKING. It is now widely accepted that disgust is more than avoiding bad taste and hinges on avoidance of contamination (Haidt, Rozin, McCauley & Imada, 1997; Rozin & Fallon, 1987). Our proposal is that nutritional-, sexual-, and socially-related stimuli plus ideational components are all able to activate either the SEEKING or DISGUST systems in analogous ways. Unlike the sensory affect *distaste*, disgust is elicited by olfactory, gustatory, auditory, tactile or visual cues (see Curtis & Biran, 2001; Curtis, Aunger & Rabie, 2004).

A taste need not have negative hedonic value to produce disgust, as shown by the ease with which conditioned taste aversions (CTA) can be elicited to sweet tastes previously paired with illness (e.g. Garcia, Hankins & Rusiniak, 1974; Parker, Rana & Limebeer, 2008). This implies that the disgust response of humans did not arise merely as a reaction to bad taste, but due to association with increased likelihood of illness, as proposed by Curtis. Conditioned immune responses may also form from a single pairing of a novel taste with antigens (Pacheco-Lopez, Niemi, Kou, Harting, Del Rey, Besedovsky, & Schedlowski, 2004), which together with the immune system's triggering of sickness behaviours might implicate an evolutionary role of the immune system in disgust.

Rather than being a brainstem reflex, formation of a CTA requires an intact amygdala and insula; and acquisition of a CTA in rats activates both insula and amygdala (Ferreira, Ferry, Meurisse, & Levy, 2006). Although both structures are active in certain types of emotional evaluations, the insula has a unique role in comparing incoming and stored tastes (Koh & Bernstein, 2005) and the amygdala may be responsible to cue danger but not the illness component in the CTA (Parker et al., 2008). Rats with insular lesions fail to learn anticipatory discrimination although they remain capable of hedonic responses (Kesner & Gilbert, 2007). Decerebrate rats and anencephalic human neonates (reviewed in Steiner et al., 2001) also show appropriate facial expressions to normally preferred and aversive tastes, but the insula is necessary for complex disgust responses.

In humans the anterior insula (AI) is activated during experience, observation and imagination of disgust (Jabbi, Bastiaansen & Keysers, 2008). Human disgust sensitivity is correlated with the size of AI (Kipps, Duggins, McCusker & Calder, 2007) and insular responses to aversive tastes vary according to expectations (Nitschke, Dixon, Sarinopoulos, Short, Cohen, Smith, 2006). Although most fMRI

studies report AI activation associated with disgust, some do not perhaps because of differences in stimulus delivery (e.g. Borg, Lieberman, & Kiehl, 2008) or due to subjects' individual differences in disgust sensitivity (Beaver, Lawrence, van Ditzhuijzen, Davis, Woods, & Calder, 2006; Calder, Beaver, Davis, van Ditzhuijzen, Keane & Lawrence, 2007). The allocortical AI contains the gustatory cortex, but is also necessary for human disgust because of its role in self-awareness (reviewed in Toronchuk & Ellis, 2007). Many behaviours integrating higher cognitive processing with "gut level" feelings also activate AI (see Saper, 2002). Human AI, rather than providing a mere module for disgust, participates together with other areas in the instantiation of a variety of other aspects of interoceptive bodily and conscious feeling states (Toronchuk & Ellis, 2007a).

In primates instead of the amygdala and striatopallidum remaining the major focus for valuation of gustatory stimuli and passing this information on to AI as in rats, the insula receives direct thalamocortical taste and visceral input which allows increased cortical integration of body states (Craig, 2005). A further adaptation of the disgust response occurred as emotional contagion or "resonance" allowed activation after observing disgust responses in conspecifics (Wicker, Keysers, Plailly, Royet, Gallese, & Rizzolatti, 2003). Higher cortical processing unique in humans allowed blending of primary DISGUST with social learning giving rise to secondary emotions involving social status and morality. The appearance of von Economo neurons in human AI and adjacent orbitofrontal area is perhaps associated with the development in humans of theory of mind, and moral reasoning. This development might then be considered a preadaptation influenced by the role of the insula in awareness of disgust in self and others. We are thus suggesting that the primitive emotive circuit which originally functioned to defend the self by regulating consummatory behaviours gave rise to a primary emotional system which facilitates evaluation of reinforcers (Toronchuk & Ellis, 2007). A role in social cognition for the insula has been proposed as a visceromotor centre which simulates the activity of the other person in a manner similar to the "mirror" neurons previously described in monkeys (Gallese, Keysers and Rizzolatti, 2004; Keysers & Gazzola, 2007).

Glutamate and acetylcholine are released in the insula during conditioned taste aversion with glutamate being necessary for acquisition (Berman, Hazvi, Neduva, & Dudai, 2000). In addition the use of serotonin by the gut, vagus nerve, and brainstem mechanisms for disgust, as well as for immune signalling suggests a defensive continuum of immune & disgust systems linked by serotonin (Rubio-Godoy et al., 2007). Serotonin release is essential for development of disgust reactions and is opposed by the cannabinoid system (see Parker et al., 2007).

There is some evidence that individual disgust sensitivity has a genetic basis (Olatunji & Broman-Fulks, 2007). In addition disgust sensitivity is inversely correlated with sensation seeking (Haidt et al., 1997) suggesting to us that DISGUST and SEEKING represent opposing emotional systems, both of which are reflected in personality traits and psychiatric disorders. Genetic influence is consistent with the finding that pre-symptomatic genetic carriers of Huntington's disease show selective deficits in recognition of disgust and reduced activity in AI (Hennenlotter, Schroeder, Erhard, Haslinger, Stahl, Weindl, et al., 2004) and insular size in these patients is correlated with disgust recognition (Calder et al., 2007). On the other hand increased activation of the insula in OCD patients is associated with contamination related symptoms (Stein, Liu, Shapira & Goodman, 2001). Furthermore Phillips and Mataix-Cols (2004)

suggest that patterns of brain activation in OCD patients to disgust or anxiety-producing objects vary according to the patient's major symptom type with increased insular activity associated specifically with washing-related symptoms (Shapira, Liu, He, Bradley, Lessig, James, et al., 2003). The activation of disgust stimuli occurs independently from the role of anxiety in these symptoms (Husted, Shapira & Goodman, 2006; Lawrence, An, Mataix-Cols, Ruths, Speckens, & Phillips, 2006). We suggest therefore that psychological illness associated with the malfunctioning of the DISGUST system can be seen in the OCD variant typified by contamination/washing related symptoms.

In addition to internal threat, organisms must protect themselves from the dangers posed by external threats. In this case, safety is facilitated by the "fight/flight" pair of defence systems: the **RAGE system E3** which may emerge from a basic response to resist restraint and the **FEAR System E4**, engaged when the individual perceives retreat to be the best option. These ancient systems are included in Panksepp's list (1998) and will only be briefly discussed here. Forerunners can be found throughout the animal kingdom (Darwin et al., 2002/1872). Which of the two solutions to threat is implemented in any particular situation is the outcome of an evaluative process based on previous experience and assessment of the present circumstances. The neural circuit for fear utilizes the amygdala and the hypothalamus, as well as the output of the PAG (see Calder, Lawrence & Young, 2001). As with DISGUST certain stimuli are predisposed to easily activate FEAR (Öhman, & Mineka, 2001) and some of the same structures activated during the production of fear and anger are also activated during recognition of fear and anger in others, suggesting the existence of a neural simulation circuit (see Goldman & Sripada, 2005). According to Panksepp (2002), psychological illnesses associated with the malfunctioning of RAGE are aggression, psychopathic tendencies and personality disorders; and those associated with FEAR are anxiety disorders, phobias and PTSD variants.

Reproduction

Sexual reproduction, of obvious importance in the mechanism of evolutionary selection is the outcome of the **Sexual LUST system E5** which could be further differentiated into "wanting" and "liking" subsystems as discussed above for the SEEKING system. The appetitive and consummatory components of LUST can function independently and their behaviours can be dissociated in the same manner as those of the SEEKING system (Cantor, Binik & Pfaus, 1999; Kippin, Sotiropoulos, Badih & Pfaus, 2004; Pfaus, 1996). The associated affects are lust/sexual desire in the appetitive stage, and sexual satisfaction at the consummation stage. Although it is clearly a phylogenetically ancient system, the nature of the LUST system has changed considerably in the transition to mammals and then to humans. Indeed, the evidence summarized in Panksepp's text (1998) is that evolution took sexual choice mechanisms and tweaked them to produce attachment. Thus the LUST system is related to both the PANIC/attachment and CARE systems, In social animals reproductive needs play a considerable role in social bonding (but also in social tensions). Because of the differential reproductive strategies of the two sexes, however, the relationship between LUST, PANIC and CARE varies in human males and females, which may lead to different adult attachment styles (see Del Giudice, 2009; Taylor, 2002, 2006).

The social emotional systems are mediated by hormones, synaptic signalling and other biochemical signals as employed by other elements of the value system. They can be causally effective in terms of contributing to neural Darwinism because these signals have also been shown to affect brain plasticity. Indeed because hormones are employed (including leutenizing hormone-releasing hormone, gonadal steroids, oxytocin and vasopressin) their influence is pervasive throughout the whole body, rather than just the organs innervated by emotional centres. Oxytocin and vasopressin in particular function as neuromodulators in the value systems associated with both adult social bonds and parental behaviour (reviewed in Carter, 2003, 1998). For example during mating vasopressin is released in the ventral pallidum and nucleus accumbens (areas also associated with **SEEKING**) of male prairie voles; and blockade of vasopressin in the ventral pallidum prevents pair bond formation (Lim & Young, 2004). Vasopressin also plays a role in sexual competition (see e.g. Sewards & Sewards, 2003a).

The LUST system also parallels the "wanting" and "liking" distinction in that dopamine is associated more with the appetitive phases and endogenous opioids more with the consummatory phases (Van Ree et al., 2000). Dopamine secretion increases as sexual arousal increases, but at consummation dopamine decreases while secretion of oxytocin and opioids increase (van Furth, Wolterink, & van Ree, 1995; Pfaus, 1996). Psychological illnesses associated with the malfunctioning of this system are fetishes and sexual addictions as noted by Panksepp (2002), and we would also add disorders of desire and orgasm which reflect either over-activation or under-activation of the system.

Primary emotions of social bonding

The emotional systems promoting social bonding are CARE, PANIC, PLAY, and as we suggest a **POWER** or RANK system. According to MacLean (1990, p.247) the differentiation of mammals from reptiles involved 1) lactation and associated maternal care, 2) vocal communication to maintain mother-infant contact, and 3) playful behaviour facilitating social learning. Because lactation and maternal care are essential for the survival of all mammals, significant selection pressure would act on the neural mechanisms controlling these behaviours. In mammals social bonding and group cohesion is initially effected primarily by the **PANIC/attachment (E6)** or separation distress system in the young, which triggers emotional panic during separation, but which also provides contentment during closeness. This necessitated the tandem development of a complementary **CARE (E7)** system, through which parents respond to the young. Panksepp (1998) includes PANIC as a separation distress system in his list; however, we suggest that distress is only one mode of operation of a PANIC system, which also operates in a positive fashion when the infant's needs are satisfied. Subjective feelings in the human infant would include panic/distress when separation occurs, and contentment/comfort when in contact with the caregiver (**E6**); and in the care-giver, tenderness/ affection, carrying over to reciprocal distress when the infant is perceived as in distress (**E7**). These two social systems likely give rise to both positive and negative emotions with the positive feelings instantiated, at least in part, by the opioid pleasure system. Both the PANIC and CARE systems are crucial in the social and cognitive development of those animals that depend on their parents for survival (Schore, 1994; de Waal, 1996, Chapter 2).

Panksepp (1998, 2003b) notes that the biological origins of human sadness are rooted in the extended brain system involving the cingulate gyrus that mediates separation distress in infant animals, although this neurological substrate has an even longer evolutionary history in that it was used earlier in phylogeny for perception of physical pain. This is further suggested by the role of endogenous opioids in pain reduction as well as reward. This may be another example, akin to distaste and DISGUST, in which a phylogenetically ancient sensory affect (pain) gives rise to a basic emotional system (panic) over the course of vertebrate evolution. Separation from loved ones is thus perceived in humans as so very similar to pain and panic because of these evolutionary origins.

In reviewing his many years of work with primates, Mason concludes that the psychoneuroendocrine core “consisting of the limbic system, the hypothalamic-pituitary-adrenal axis, the autonomic nervous system and the immune system” provides the basis for both infant and adult attachments (Mason, 2008). Studies in humans and other mammals (e.g. Carter, 1998, 2003; Insel & Young, 2001; Lim & Young, 2004; Taylor, 2006) have identified oxytocin as mediating both maternal and adult pair-bonding. Estrogen enhances the effects of oxytocin, perhaps providing a basis for the gender differences that exist in attachment styles (see del Giudice, 2009, Taylor, 2006). The widespread commonalities of the endocrine mechanisms of social relationships suggests that infant/adult bonding came to be supported by the more ancient circuitry of the sexual attraction system.

Compared with other mammals, hominid infants have a relatively long period of helplessness combined with subsequent need for training in foraging techniques and social behaviours.. This is true of all hominids but two additional factors discussed by Falk (2004) contributed to the extended helpless period of human infants. The first was the trend toward a narrow pelvis associated with bipedalism, and the second was the expansion of the human brain. Together these factors would have selected for human infants that were delivered at an increasingly immature stage of development. The absolute necessity of nurture for immature offspring during this increasingly extended infancy would have put considerable selection pressure on the neural circuitry for emotional attachment between hominid mother and infant. This circuitry would be expected to be most highly developed in humans.

Infant chimps lack the ability to cling to their mothers' fur in the first two months and so must be supported by the mother on her ventral surface (see Falk, 2004). Mothers of older infants use body language and gestural signals to encourage climbing on the mother's back when it is time to move on. The mother's use of gestures and facial expressions play a key role in communication with infants, who then develop an intense interest in their mother's face. Chimp mothers also teach which foods can be eaten and perhaps even tool use (see Falk, 2004; Goodall, 1986). Chimp infants signal various types of distress to the mother through whisper, hoo, and scream vocalizations. Hominid evolution therefore involved tandem evolution of emotional circuitry in adults to provide not merely food, but also emotional nurturance and instruction, and the parallel circuitry in the young to seek and respond to others. This entailed the increasing use of gesture, facial expression, and tactile and vocal communication. The increasingly extended period during which hominin young were dependent on caregivers necessitated the development of even more skilled caretaking and the ability of adults to provide instruction. Although the role of early emotion in

producing adult human emotional and social behaviour has been well studied, the evolutionary role of infant emotion as a selection factor in cognitive development has been less well researched. According to the theory we suggest, mother-infant communication likely provided the emotional motivation for the initial development of language, and its use in adult coalitions was likely a more secondary development.

Learning in human infants has been shown in numerous studies to be critically enabled by reciprocal interaction with the primary care-giver in the early stages of life (e.g. Schore, 1994). The ability for shared attention between infant and mother is critical for the development of a theory of other minds and influences the development of language. Thus social emotions provide the valenced state necessary for infant learning, initially taking place in relation to predicting and responding to the actions and emotions of the primary carer. These systems may also facilitate altruistic behaviour among social mammals such as primates, cetaceans and canids which have well developed memories for social interactions.

Tactile stimulation in infancy mediates upregulation of glucocorticoid receptors in the hippocampus through DNA methylation, an effect which in rodents persist throughout life (Kauffman & Meaney, 2007). This increase in receptors decreases the base-line level of reactivity of the hypothalamic-pituitary-adrenal (HPA) axis by decreasing input to hypothalamic neurons which control activation of the HPA axis. Early tactile stimulation also regulates the expression of estrogen receptors in the medial pre-optic area of the hypothalamus, resulting in a downstream increase in oxytocin receptor binding accompanied by increased licking, grooming and arched-back nursing in rodent females. Maternal care by nurturing mothers also enhances learning in offspring by enhancing NMDA receptor activity in the hippocampus. Kaffman and Meaney suggest that these mechanisms are conserved from rodents to humans. These effects may explain how the PANIC and CARE systems work in tandem to alter emotional and cognitive behaviours throughout life.

Evidence now suggests there are differences in the endocrine and neural responses to stress of males and females (e.g. Dalla et al). It seems worth investigating whether these differences might be related to demethylation of estrogen receptors in early life as described by Kauffman and Meaney (or to some other early differentiation in brain responsiveness to maternal behavior), underlying the differences in human attachment styles discussed above. Specifically Del Giudice (2009) suggests that males show more avoidant attachment and females more clinging ambivalent attachment from middle childhood to adulthood. Taylor (2002) has described the differential effects of oxytocin in producing tend and befriend behaviour in females under stress, rather the fight and flight behaviour more typical of males.

In the 1940s Rene Spitz described how separation of infant and caretaker leads to the serious physical and emotional stunting of hospitalisation syndrome (van der Horst & an der Veer, 2008). Later Bowlby, influenced by Harlow's work with macques, established that infant separation had long term impact on social development. The outcome of the quality of maternal care on adult emotions and social cognition is now well established (e.g. Schore, 1994). According to Panksepp (2002), psychological illnesses associated with the malfunctioning of these systems are panic attacks, pathological grief, depression, agoraphobia, social phobias, dependency and

attachment disorders, and may also be a contributing factor to autism. A tendency to post-traumatic stress disorder may also develop (Nemeroff et al., 2006).

Thus, the PANIC and CARE systems became a primary influence on the evolution of human social bonding, sympathy and altruistic behaviour (see Toronchuk & Ellis, in press). The associated human feelings are communality/inclusion and loneliness/exclusion.

Learning and Development

Although learning is enabled by the SEEKING system (see Berridge et al., 2009) it is ontologically dependent in mammals on the **PLAY System E8**. The tendency for young mammals to be involved in play as part of their preparation for both food procurement and adult social roles, suggests that play should also be considered a basic emotional program in the human ancestral lineage and necessary for the normal cognitive development of children. It is facilitated evolutionarily by the enlargement of the cerebral cortex and the prolonged infant/maternal interaction necessitated by lactation (MacLean, 1990). The role of play in development of the young is influenced by the reciprocal relationship of the CARE and PANIC/attachment systems in adult and infant. Play also involves learning social roles and social behaviours (e.g. Bekoff & Byers, 1998; Brosnan, 2006; Keltner, 2006).

Within social groups play may function to involuntarily reveal social commitment which can then be assessed by others. Playful teasing allows the exploring of social boundaries and dominance interactions.

Chimp and bonobo mothers in particular engage in extensive nuzzling, tickling, play-biting, chasing and other forms of play with their infants and play periods are accompanied by facial gestures and vocalizations often including laughter (reviewed in Falk, 2004a). It is a real possibility that optimal cortical plasticity may depend on the activation of play, affection, and other rewards of close attachment. Allowing juvenile rats 30 minutes of rough and tumble play results in increased BDNF transcription in the amygdala and dorsolateral frontal cortex (Gordon et al., 2003). Although much anecdotal information suggests this may also be true in humans the underlying neurophysiological mechanisms are still poorly defined.

We agree with Panksepp's (1998) inclusion of "rough and tumble play" in the list of primary emotions, but believe that in humans the nature of this system has been extended to include representational and intellectually imaginative play. There is evidence that both captive and wild apes may also engage in representational play, in which one object comes to stand for another (Lyn, Greenfield and Savage-Rumbaugh, 2006). We speculate that the phylogenetic transition from rough and tumble play alone to the capacity for representational play facilitated the development of language by enabling the process of allowing one concept to represent another. Another factor contributing to language evolution would have been the necessity of understanding and empathising with others that is a critical part of imaginative play. Play remains particularly important in the ontogenetic process of language development (see Bruner, 1983, Chapter 3; Owocki, 1999; Paley, 2004; Zigler, Singer & Bishop-Josef, 2004).

In humans the nature of this system has been extended to include play-acting (Frost et al., 2001) and ultimately allows the development of many aspects of human culture. It

is also an essential component of much of the performing arts on the one hand, and ceremonial and celebratory behaviour on the other, as well as being an important source of creativity (and so is significant also in the development of science). The associated feeling is joy/fun. Humour retains this basic tie to rough and tumble activity, including the dominance paradigms often invisibly embedded in it, in that we "make fun" of the other, "reducing" them in a comical way. The social aspects of play such as turn taking and feinting may also provide one of the biological bases for the evolution of altruistic behaviour

A detailed assessment of the play system, and the role of low levels of opioids in its activation, has been given by Panksepp (1998, 2002). He has suggested that attention-deficit hyperactivity disorder (ADHD), mania, and perhaps impulse control disorders may be associated with the malfunctioning of the PLAY system.

Group Function: Regulating Conflict

We propose that a genetically determined emotional system, **the POWER/dominance System**, concerned with territoriality, dominance and subordination should be added to the list of basic emotional circuits. Dominance displays are found in all vertebrate orders and even in many invertebrates (e.g. Panksepp, J. B. & Huber, 2002). For many species, group living is crucial for survival advantage, both in terms of finding food and protection against predators as well as in enabling learning (e.g. Manning & Dawkins, 1998, pp.372-401; Slater, 1999, pp.188-203) However this inevitably entails a competition for resources that needs regulation to minimize damage to individuals as well as to group cohesion. Selection pressure likely favoured the evolutionary development of affective means to enable social dominance in a way serving the interests of the group as a whole, and even the best interests of low ranking individuals by allowing them to survive long enough to reproduce another time.

Allocation of rank occurs in animals and humans alike by various competitive processes, leading to agonistic behaviour that regulates competition in a socially non-destructive way (e.g. de Waal, 1996, Chapter 3; Stevens and Price 2002, pp.49-52). Competition takes place in relation to the control of "territory" regarded in the widest sense, relating not merely to material resources (such as food, possessions, and geographical areas) but also for example to social control, sexual mates, status symbols, and ultimately intellectual turf (Ardrey, 1966). Personal identity of humans, therefore, comes to be closely influenced by basic emotional circuitry for territory.³ Based on data from both human psychiatry and animal behaviour, we propose that this ancient system is the phylogenetic precursor to competition for status, and the need to excel and obtain social approval. With a few modifications it is similar to the "power dominance" drive described by Sowards and Sowards (2003b) as underlying Nietzsche's will to power and Winter's (1973) implicit power motive.

Panksepp (1998, 2002, 2007) has held that social dominance probably arises from interactions between the childhood PLAY system and the FEAR and RAGE systems. Although ontogenetic development of individuals certainly depends on play, we prefer the alternative view that this system is evolutionarily more ancient than play because dominance displays, usually related to territory and/or access to females, are found in all vertebrate orders and therefore likely predated the PLAY system. Indeed this system involving the dominance and submission subroutines, precedes the

evolution of mammals with their dependence on the PLAY, NEED and CARING systems.

John Price and his colleagues (reviewed in Price, Gardner, Wilson, Sloman, Rohde & Erickson, 2007) have convincingly suggested that elevated mood facilitates a rise in rank enabling coping with leadership, while depressed mood allows lower ranking individuals to accept their status and forgo reward. The desire for higher rank is characterised by *Resource Holding Power*, or RHP associated with feelings of pride/high self-esteem (for high perceived RHP) during success and shame/low self-esteem (for low perceived RHP) during defeat.⁴ These are represented in humans by feelings of self-confidence and depression respectively. They place this model in the context of MacLean's triune brain locating the instinctual aspects of depressed mood as largely a function of the reptilian brain. According to MacLean (1990), the striatal complex (including basal ganglia) was responsible for instinctual behaviour patterns in ancestral vertebrates with additional limbic structures being recruited in mammals. We will hypothesise further on possible circuitry and neurotransmitters below and also suggest links with depression and OCD.

The striatal complex, in particular the basal ganglia, plays a major role in dominance behaviours in many vertebrates (MacLean, 1990). Social/territorial displays among male anolis lizards are associated with activation of a comparable striatal area in which serotonin and dopamine are released in differing patterns in dominant and submissive animals (Baxter, 2001a, b; MacLean, 1990). The localization of serotonin and dopamine in the basal ganglia of these lizards is similar to that in primates, suggesting similar mechanisms across phylogenetic lines (reviewed in Baxter, 2003). In squirrel monkeys, lesions in the globus pallidus (one of the basal ganglia) disrupt the thigh-spread social display given by males during both dominance and courtship displays (MacLean, 1990; Newman, 2003). Competitive arousal produced by imagery in human males also activates the globus pallidus (Rauch et al., 1999). The basal ganglia therefore may be responsible for the components of depression which act as appeasement displays in individuals who have lost agonistic encounters (Price et al., 2007).

Additional areas come to be involved in the POWER/rank system in mammals, in particular the prefrontal and cingulate cortices, with the left prefrontal activated in positive mood and the right in depression (Davidson, Pizzagalli, Nitschke, & Putnam, 2002). The orbitofrontal cortex, which plays a role in both dominance (see Allman, 2000, p.25) and OCD (Baxter, 2003), is part of this system. The anterior cingulate cortex has been shown to be activated on the right during competitive arousal (Rauch et al., 1999); and low activity in ACC is implicated in the pathogenesis of human depression with different subregions of ACC playing differential roles (reviewed in Davidson et al., 2002). Swards & Swards (2003b) describe an area of ACC (Brodman's 32, but ventral to the fear representational area) as participating in the "power dominance" drive and equate this area with the infralimbic area previously identified as responsible for dominance in hamsters (Kollack-Walker, Don, Watson & Akil, 1999). Further they suggest the major contribution of the ACC is to the learned, voluntary components of the power dominance drive rather than involuntary components which are mediated by lower levels. In Price's model the involuntary components would be provided by the basal ganglia. While Swards and Swards consider anger to be an extreme form of

dominance, we reject this view on the basis that RAGE is a separate emotional system as described by Panksepp (1998). Furthermore the area described by Rauch and colleagues (1999) as activated by competition is medial and dorsal to the area described by Sowards and Sowards (2002) as having to do with anger.

Activation of the submissive circuitry can lead to depression (see Gilbert, 1992; Price Gardner & Erickson, 2004; Price, et al., 2007; Stevens & Price, 2002), a disorder that evolutionary psychiatrists see as the human counterpart of social defeat (e.g. Kroes, Burgdorf, Otto, Panksepp, & Moskal, 2007; Sloman et al., 2004; Sloman & Gilbert 2000). There is ample evidence (e.g. Gilbert, 1989; Panksepp, Moskal, Panksepp & Kroes, 2002) that low status is a major risk factor for depressive behaviour in mammals (and also reptiles, according to MacLean) and adverse health effects in humans and other primates (Sapolsky, 2005). The development of neocortex in humans allowed for further complex integration with cognition resulting in the emergence of secondary emotions such as guilt, shame and jealousy.

Biochemical activation of this system occurs in part by serotonin, which plays a general background role in numerous emotional systems, and may increase dominance by decreasing impulsive, species-typical behaviours. Low serotonin levels are reported in numerous psychiatric disorders including depression, suicide, anxiety, aggression, addiction, and OCD. Dominance behaviours and serotonin levels are related and can be separately manipulated in vertebrates and even invertebrates (e.g. crayfish, Panksepp & Huber, 2002). For example, manipulation of dominance relationships in *Anolis* lizards leads to changes in serotonin levels in several brain areas including basal ganglia (Korzan & Summers, 2004). Serotonin levels also correlate with rank order in vervet monkeys; and social standing among individual monkeys has been manipulated by altering its level (Raleigh, McGuire, Brammer, & Yuwiler, 1991). Increasing serotonin by means of oral tryptophan increases dominant behaviour in human males (Moskowitz, Pinard, Zuroff, Annable & Young, 2004) and antidepressants are well known to decrease submissive behaviours.

In addition to serotonin, several neuropeptides (particularly vasopressin as discussed below) and testosterone also play direct roles in the ranking system. In humans (reviewed by Archer, 2006) and macaques (Rilling, Winslow & Kilts, 2004) testosterone increases dominance behaviours, although it can in turn be modulated by performance of dominance behaviours. It has been hypothesized that while high testosterone levels lead to increased competitiveness and dominance, when accompanied by low serotonin levels high testosterone leads to impulsivity and pathological aggression (Birger, Swartz, Cohen, Alesh, Grishpan, & Kotelr, 2003). Social defeat in rodents (Panksepp, Burgdorf, Beinfeld, Kroes & Moskal, 2007) results in widespread decreases in CCK, although less stressful encounters during play produce increased levels of CCK (Burgdorf et al., 2006). Because CCK injections produce panic in rats, the combined data suggest increased utilization of CCK during social defeat (Panksepp et al., 2007). Although CCK modulation was the most prominent and robust change, social defeat also increased CRF levels, a factor otherwise associated with depression and changes in hippocampal size (see Brown, Varghese & McEwen, 2004). Another link between social defeat and depression is provided by the finding that gene expression of interleukin 18, which is found to be overexpressed in depressed humans, is increased following social defeat in rats (Panksepp et al., 2007).

Vasopressin, and its nonmammalian homologue vasotocin, also facilitates aggressive and dominance related behaviours in many species and is itself modulated by both testosterone and serotonin. Intranasal vasopressin has a differential effect on the behaviour of men and women, leading to increased agonistic behaviour in men and increased affiliative behaviour in women (Thompson, George, Walton, Orr, & Benson, 2006.) Swards and Swards (2003a, b) propose that vasopressin is the primary generator of the “power dominance” drive. Social subjugation of hamsters decreases levels of vasopressin in the anterior hypothalamus (Delville, Melloni & Ferris, 1998). A complementary finding is that microinjection of vasopressin into the anterior hypothalamus of hamsters triggers stereotypical grooming and scent marking behaviours which are suggested to provide an animal model for OCD (Ferris, Rasmussen, Messenger & Koppel, 2001), and these behaviours are suppressed by drugs used to treat OCD in humans. Increased vasopressin is linked to human OCD (Altemus, Pigott, Kalogeras, Demitrack, Dubbert, Murphy, et al., 1992; and may play a role in some forms of stress-related depression (Landgraf, 2006)

The combined involvement of the basal ganglia, vasopressin and serotonin in both social dominance and OCD is consistent with Stevens and Price’s (2002) view that OCD may result from a disorder of POWER/dominance. MacLean originally proposed that some forms of OCD might be the result of inappropriate release of territorial and defensive motor programme fragments which suggest checking and perhaps hoarding, but not washing-type symptoms of OCD. Another influence of the POWER system on human emotion is suggested by the hypothesis that depression might result from triggering the involuntary subordination response of animals losing competitive encounters, i.e. some forms of depression may also represent disorders of rank (e.g. Gilbert 1989, 1992; Sloman & Gilbert, 2000). In connection with this idea, it has been further suggested that the involuntary subordination response may have originated phylogenetically from the response to separation distress as both are characterised by negative mood, loss of self-confidence, and loss of resources, and both result in decreased activity (Sloman, Gilbert & Hasey, 2003). The latter point seems to neglect, however, the actual evolutionary ordering of events, because the dominance and subordination system, unlike the separation distress system, is present throughout the vertebrate phylum.

Following on Beck’s differentiation of sociotropy and autonomy, Weisfeld and Wendorf (2000) propose that depression assumes two forms. Depression which results from loss of status and often involves pride, guilt, and/or shame should be differentiated from depression associated with grief or loneliness. Sociotropy is a personality characteristic related to attachment and the need to please others, while autonomy is related to independence and attainment of goals. These two characteristics appear to be expressed in different symptoms (Robins, Block & Peselow, 1989). Autonomous traits have been found to be associated with a successful response to antidepressant medication, while sociotropic traits are not (Peselow, Sanfilippo, Robins, Block & Fieve, 1992). Also in the non-clinical population low moods are associated with different behaviours depending on whether they are precipitated by social losses or failure to reach a goal (Keller & Nesse, 2005).

Consistent with the notion of two forms of depression, the physiology and developmental course of the disorder can be seen to differ according to whether it is

precipitated by separation distress or by loss of status. Oxytocin reduces separation distress, but not low rank; separation anxiety involves the cingulate, but dominance behaviours in primates and pride and shame in humans involve the orbitofrontal cortex (Weisfeld & Wendorf, 2000; see also Allman, 2000; Panksepp, 1998). Another study suggesting two forms of depression, differentiated by social components, reported increased left frontal activation in depressive patients scoring high in reassurance-seeking, whereas relative right frontal activation was found in patients scoring low in reassurance-seeking (Minnix, Kline, Blackhart, Pettit, Perez, & Joiner, 2004). Weisfeld and Wendorf have noted the similarity between subordination displays and the behaviour of individuals whose depression is specifically linked to shame, guilt and failure e.g. averted gaze, slumped posture and slowed responses.

We suggest therefore that malfunctioning of the POWER/dominance system may take two forms. Depression may occur as a result of overactivation of subordination programmes; while OCD with symptoms of checking, ordering and hoarding may be related to overactivation of dominance programmes. OCD with washing symptoms would be related to malfunction in the DISGUST system as discussed above, rather than POWER/dominance; and depression with sociotropic symptoms would be related to the NEED/attachment (PANIC or Separation Distress system of Panksepp)

In summary, we propose that in addition to the systems listed by Panksepp (1998), there is a genetically determined emotional system concerned with dominance and subordination which has instinctual motor components based in the basal ganglia and emotional components based in limbic structures.

Conclusion

The importance of correctly determining the nature of the primary emotional systems, characterised according to the criteria given in section 3 above, is that according to the proposals of Affective Neuronal Darwinism, these then determine not only the nature of immediate emotional reactions but also the details of higher level brain functions such as cognitive modules and secondary emotions. It is important to have a *complete* list of the primary emotions, for then we know the *necessary and sufficient* variables underlying higher brain development (see Ellis & Toronchuk, 2005).

The proposals we make are summarised in Table 3. The main changes proposed here from the characterisation of primary emotional systems given in AND (based on Panksepp, 1998) are the proposals for (a) inclusion of the **DISGUST system E2** and (b) inclusion of the **POWER/dominance system E9**, implied by evolutionary psychiatry (Price, 1967) and tentatively suggested by Panksepp (1998).

The list of basic emotions has varied only slightly from Darwin to the present time (e.g. Damasio, 2003; Ekman, 1972, 1992; Izard, 1992; Plutchik, 2003; Tomkins, 1962). Virtually all lists include **Happiness**, **Sadness**, **Fear**, and **Anger**, and most also include **Disgust** and/or **Contempt**. Some include **Surprise** and **Interest**, but **Guilt** and **Shame** are less frequently included. As Panksepp (2000) notes, the major difficulty is in reaching agreement on the status of **disgust**, **surprise**, **interest**, **guilt** and **shame**.

Sadness, as we note above, seems to correspond to Panksepp's PANIC executive system (Panksepp, 2003b) which is concerned with need and the pain of separation. Interest corresponds to Panksepp's generalised SEEKING system, as does happiness, although it may be useful to follow Berridge (Berridge & Robinson, 2003; Berridge et al., 2009) in parsing SEEKING into incentive salience ("wanting") and hedonic appraisal ("liking"). These components may be separately activated by other emotions. Panksepp suggests the PLAY executive system is associated with joy or happiness, but while this is likely the case, we feel that happiness is also more broadly based and may correspond to a hedonic appraisal response activated by other social emotions. On the other hand we feel there is enough evidence to include DISGUST as a primary emotional system having a long phylogenetic history.

In contrast, we do not feel it is correct to label **Surprise** as a primary emotion, for despite its venerable heritage in terms of study by Darwin, Ekman, and others, it does not have the same nature as the other affect programs (Griffiths, 1997, p.241), is not necessarily valenced (Ortony & Turner, 1990; Ekman, 1992; Prinz, 2004, p.163), and additionally gives no specific action guidance related to survival value. However it, like the startle reflex, may well serve to urgently activate SEEKING and in that sense have a similar function. Should it ever be shown to have the same kind of neural correlates and transmitter systems as the other primary emotional systems, we would include it as a subsystem of SEEKING.

Contempt, embarrassment, shame, and guilt are not included in our list because firstly they are uniquely human emotions; and secondly they rely largely on neocortical functions and so are more plausibly secondary emotions. Shame and guilt may, however, emerge from cognitive interactions with the power/dominance or rank system as suggested by Gilbert (1998). We suggest this emergence provides another rationale for including dominance as a basic emotional system. Sabini and Silver (2005) argue for the inclusion of love and jealousy as basic emotions on the basis of their evolutionary past, and while we concur in part we prefer the terms CARE and POWER/dominance or rank as more consistent with their evolutionary origins.

Although some have argued that the concept of basic emotions is not useful (e.g. Ortony & Turner, 1990), there are three advantages from our perspective. First we see them playing a key role in brain evolution and development (as discussed in AND) which may generate future research on evolutionary and developmental processes. Secondly, focusing attention on the biological functions of these systems in the evolutionary past has led psychology to a greater awareness of biological complexities of human emotions. This has led and will continue to lead to new treatments and diagnostic techniques for various psychiatric disorders. Finally, along with the concept of neural Darwinism, it may lead to new ideas on the promotion of healthy emotional development in infancy. These causal links are supported by the suggestions we make above for psychiatric disorders associated with each of the proposed primary emotional systems. Developing and validating those proposals will be an important part of the further development of the ideas presented in this paper.

To complete the task of this paper, we need to check to what degree each of the above criteria C1 – C7 for a primary emotional system is satisfied by the proposed systems, and then try to fill in the gaps where there is a criterion that has not yet been met. To do this fully will be a lengthy task; Table 4 shows our view on the current state of

confirmation of these criteria and which still need to be investigated and hopefully confirmed. It will be clear from that table that while there is good overall support for our proposals, the area where most needs to be done is in terms of the relation of genetics to our proposed classification. Elucidation of this link will be an important task for the future, in spite of the difficulties engendered by the polygenic nature of the common heritable mental disorders and the relative rarity in the population of each of a very large number of specific mutations which contribute to each disorder (Keller & Miller, 2006).

Implications

As pointed out in Ellis and Toronchuk (2005), the nature of the primary emotions has consequences in many areas of human behaviour (for example affecting economics, politics, and education in crucial ways; see e.g. Ellis 2008 for a comment in relation to educational issues). The overall point is that given the assumptions of affective neural Darwinism, we have a much more nuanced version of motivational theory than simple conditioning theory. Each major need is related to one or more of the specific primary emotions characterised in a complete list of primary emotions, either directly, or indirectly through the way that they lead to development of secondary emotions; hence we can analyse psychological and psychiatric issues in terms of their relation to these primary emotional systems. These issues will be pursued in further papers in this series.

Further steps

Determination of the nature of the primary emotional systems is crucial to understanding the functioning of the brain, for these systems underlie higher-level emotional and cognitive development of individuals. The proposals here of the nature of those primary emotions are a small step towards developing the psychological implications of AND. Clearly they need careful scrutiny. They form the basis for further development, for example considering how universally experienced secondary emotions develop on the basis of the primary emotions. Thus key further steps are,

1. Validating the *list of primary emotions* proposed here (summarised in Tables 1 and 3) for correctness and completeness: is the case for their inclusion adequate (cf. Table 4)? Can they all be correctly characterised in cladistic terms? Are there further primary emotions omitted from this list?

2. Using the resulting complete list of primary emotions, modified where necessary in response to the questions above, to help determine and classify the *nature of secondary emotions*, arising out of these primary emotions via the processes of Affective Neural Darwinism.

3. Further elucidation of the effects of primary emotions on the cognitive development of individuals.

This is part of the broader scheme of clarifying the issue of *psychological universals*. Human commonalities and differences develop in the context of societies that have universal functional needs and physical environments with commonalities based on universal underlying physical laws.⁵ In examining the structuring and function of the human mind, the emotional systems must be taken in conjunction on the one hand with the constellation of systems for perception, pattern recognition, and memory, and on the other the mechanisms of volition that balance rationality with the unconscious, emotion, and value systems. Understanding the interactions between these systems leads to an enhanced understanding of the evolutionary and developmental basis of emotional disorders (Stevens & Price, 2002; see also

Panksepp, 2002). The proposals made here may help in clarifying this issue of human universals in a way that takes cognisance of present day neuroscience discoveries and current psychiatric knowledge, as well as data from animal behaviour and neurology. The acid test of this set of ideas would be to elucidate the many links between the prototype states and neuroplasticity molecules on the one hand, and genes on the other. Some workers are starting to do this (e.g. Kroes, Panksepp, Burgdorf, Otto & Moskal, 2006).

Finally, the question arises as to what difference there is between what is proposed here (Affective Neural Darwinism, or AND), and Hebbian processes of neural refinement on the one hand and Skinnerian conditioning on the other. A summary of the differences is shown in Table 5. Note that affective neural Darwinism includes Skinnerian conditioning as a special case; but is more flexible and nuanced. It also works in concert with Hebbian processes, but gives neuronal connection refinement a valenced or value-based direction that pure Hebbian processes lack. The overall key issue is why one needs a multi-dimensional affective assessment of the situation as proposed in AND and elucidated here, rather than a one-dimensional system as given by Skinnerian-type conditioning processes, based simply on positive (reinforcement) and negative (aversion) responses. The basic answer is that this makes survival more likely in a complex ecological and social environment that has been sampled numerous times over the course of evolutionary history, with the resultant lessons encapsulated in swift emotional reactions, relating to various survival problems as set out in Table 1. These also then shape the nature of higher brain functions both during early development and over the entire life span. In brief: it enables us to benefit directly from the survival lessons of life as experienced by our evolutionary ancestors, without needing to have the relevant experience ourselves. In this sense it is a precursor of cultural evolution. Further development of this proposal will need to demonstrate in detail how this leads to enhanced survival prospects, particularly in early life when an animal or human has not yet accumulated much experience of the world, as compared to a situation where only Skinnerian conditioning was operational.

Overall, the project undertaken here is supported by the growing number of studies from both the neurological and psychological sides emphasizing the role of emotions in human development. As an example, Greenspan and Shanker (2004, p.1) state "*We have found that the capacity to create symbols and to think stems from what was often thought of by philosophers as the 'enemy' of reason and logic: our passions and emotions ... we will show how emotions actually give birth to our very ability to create symbols and to think*". This statement, based in detailed evidence on the nature of development, is fully in accord with our view on how higher cognition develops.

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Footnotes

¹ The numbering system **E1** to **E7** is introduced in Ellis & Toronchuk (2005) although we have changed the ordering in this paper. Panksepp uses capitalization to denote emotional organising systems rather than emotions per se.

² It could be possible for each goal to have a separate seeking and pleasure system, however, this would result in a combinatorial increase of complexity. It is much more economical to have many systems utilising the same superordinate seeking and pleasure systems. The dopamine and opiate pathways seem to be generalised and responsive to many stimuli (see Figure 2 of Berridge, 2004).

³ We thank Ian MacCallum for pointing this out.

⁴ Note that *guilt* is quite different, though it is often paired to shame, as it relates to failure to live up to social or individual behavioural expectations; thus it is probably a secondary emotion related to ethical behaviour and so to ethical standards, rather than to ranking (see the discussion below).

Nature of the Primary Emotional Systems

Table 1

Evolutionary needs, and the emotional systems that have evolved to meet them. E1 is a generalised system providing incentive for the others and this dependence is noted only once. The systems are renumbered in contrasted with Ellis and Toronchuk (2005), in order to better suit the present scheme. From here on in this document, this new numbering system will be used instead of the old system (presented in the text above).

EVOLUTIONARY NEEDS MET	PRIMARY EMOTIONAL SYSTEM	Works With:	FUNCTIONS
INDIVIDUAL NEEDS			
Basic Functioning	E1: SEEKING system	E2-9	Situation Evaluation, provides arousal/ excitement; incentive salience, hedonic appraisal, and facilitates learning
Basic Survival	E2: DISGUST system (repulsion)		Avoiding harmful foods, substances, environments
	E3: RAGE system	E4,E9	Defence: aggression, protection of resources, and con-specifics, limiting of restraint on movement
	E4: FEAR System	E3,E9	Defence: flight, limiting of tissue damage
SOCIAL NEEDS			
Reproduction	E5: LUST system (sexual desire, satiation)	E6,E7	Ensuring procreation, enhancement of bonding
Group cohesion: Development	E6: PANIC/attachment (affiliation, separation distress)	E7	Creates bonding through need for others
	E7: CARE system	E6	Caring for others, particularly offspring
	E8: PLAY system *	E6,E7	Bonding with con-specifics, development of basic adaptive and social skills, creativity
Group function: Regulating conflict	E9: POWER/dominance system (rank, status, submission)	E3,E4	Controlling aggression in society, allocating resources, esp. sexual ones.

*Panksepp describes this system as "rough and tumble play", which may describe the major component in many mammals, but in humans it also encompasses many other forms of play which facilitate learning and creativity.

Table 2

The components of the motivational subsystems of the SEEKING system, which respond to specific goals. The overall evaluation **E1** summarises the combined motivational nature of all these components.

Physical Components: <i>Biological Distress</i>	D1	Hunger
	D2	Thirst
	D3	Tiredness
	D4	Hot/Cold
	D5	Pain
	D6	Sickness
Emotional Components <i>Happiness/sadness aspects</i>	E3-E9, S1-S_N	Emotional state
Volitional Components <i>Dimensions of Desires</i>	V₁-V_n	Chosen Goals

Table 3

The proposed basic emotional systems together with their associated brain areas and key neuromodulators. The non-specific effects of serotonin and norepinephrine, are omitted, as are higher cortical areas. Based on Panksepp and Harro (2004), Watt (1999) and other sources. Key: CCK = cholecystokinin, CRH = corticotrophin releasing hormone, DA = dopamine, DBI = diazepam binding inhibitor, LH-RH = leutenizing hormone releasing hormone MSH = melanocyte stimulating hormone, NPY = neuropeptide Y; PAG = periaqueductal gray BNST = bed nucleus of the stria terminalis, NTS = nucleus tractus solitarius, POA = preoptic area, VMH = ventromedial hypothalamus, VTA = ventral tegmental area.

Nature of the Primary Emotional Systems

Table 3

EVOLUTIONARY NEEDS MET	PRIMARY EMOTIONAL SYSTEM	Neuromodulators/ Neuropeptides	Key Components of Neural Networks
INDIVIDUAL NEEDS			
Basic Functioning	E0: PLEASURE System (satiating, satisfaction)	Endorphins, GABA (+,-) enkephalins, DA(?)	Nucleus accumbens ventral pallidum, VTA, brainstem nuclei
	E1 SEEKING System	DA, glutamate, Ach, CCK (+,-), neurotensin	Nucleus accumbens, lateral hypothalamus and VTA to PAG
Basic Survival	E2: DISGUST System (repulsion)	Glutamate, Ach?, substance P (+)? cannabinoids (-)?	Anterior insula, putamen, lower brainstem (area postrema and NTS)
	E3: RAGE System	substance P (+), Ach (+), glutamate (+)	Medial amygdala, BNST, medial and perifornical hypothalamus, dorsal PAG
	E4: FEAR System	Glutamate(+), CRH, CCK, α -MSH, NPY	DBI, Lateral & central amygdala, medial & anterior hypothalamus to dorsal PAG and pontine nuclei
Learning	E5: PLAY System	Opioids (+,-) Ach	Dorso-medial diencephalon (thalamic nuclei) to ventral PAG
SOCIAL NEEDS			
Reproduction	E6: LUST System E6A: Sexual desire	Steroids, Vasopressin, Oxytocin, LHRH (also DA, CCK?)	Basal forebrain, amygdala, BNST, medial preoptic and VMH to ventral PAG
	E6B: Sexual satisfaction	Opioids, Oxytocin	Septum, medial preoptic (VMH in ♂?), VTA to PAG
Group cohesion: Social Bonding	E7: NEED/ATTACHMENT (separation distress)	Opioids(-,+), oxytocin (-,+), prolactin (-/+), CRH	Anterior cingulate, BNST, POA, VTA, to PAG
	E8: CARE/nurturance System	oxytocin (+), prolactin (+), dopamine, opioids(+/-)	Anterior cingulate, BNST, preoptic hypothalamus, to VTA and PAG
Group function: Regulating conflict	E9: POWER/dominance (rank, status, submission)	Serotonin (-), testosterone (+) vasopressin (+)	Basal ganglia, hypothalamic nuclei to PAG

Table 4

Satisfaction of criteria C1-C7 for Basic Emotional Systems by the proposed primary emotional systems E0-E7, as we understand them on the basis of data presently available. The criteria are **C1** = Concept (see Tables 1-3), **C2** = Structure (neuroanatomy, see Table 4), **C3** = function (neurotransmitters, see Table 4), **C4** = development (genetics), **C5** = Evolutionary Origin (see Table 1), **C6** = Occurrence (homologues, see main text), **C7** = Outcome (psychiatric outcomes, see main text).

PRIMARY EMOTIONAL SYSTEM	Criteria for Basic Emotional System satisfied? [Criteria Numbered as in Section 3]						
	C1	C2	C3	C4	C5	C6	C7
E1: SEEKING system (incentive salience)	Yes	Yes	Yes	Partly	Yes	Yes	Yes
E2: DISGUST system (repulsion)	Yes	Yes	Partly	Not yet	Yes	Yes	Yes
E3: RAGE system	Yes	Yes	Yes	Not yet	Yes	Yes	Yes
E4: FEAR System	Yes	Yes	Yes	Partly	Yes	Yes	Yes
E5: LUST system	Yes	Yes	Yes	Not yet	Yes	Yes	Yes
E6: PANIC/attachment (affiliation, separation distress)‡	Yes	Yes	Yes	Partly	Yes	Yes	Yes
E7: CARE system	Yes	Yes	Yes	Not yet	Yes	Yes	Yes
E8: PLAY system *	Yes	Partly	Partly	Not yet	Yes	Yes	Yes
E9: POWER/dominance system (rank, status, submission)	Yes	Partly	Partly	partly	Yes	Yes	Yes

Table 5

Comparison of Hebbian and Skinnerian processes with Affective Neural Darwinism. The Overall Affective State (Column 3) is just a 1-dimensional assessment of the overall state of the system (positive or negative; pain or pleasure). The further genetically-based Value System Dimensions (Column 4) in the case of AND incorporate nuanced survival information learned through the course of evolutionary history and then transmitted to later generations genetically. Thus they encode specific alternative inbuilt behaviour tendencies that are appropriate in different circumstances and are available without prior learning. The Overall Valence/Value System operating in the case of Affective Neural Darwinism (Column 5) incorporates emotional/affective evaluations, and so relates to the importance of emotional systems in behaviour, survival, and hence in evolution. These effects are not present in the cases of either simple Hebbian processes or Skinnerian conditioning.

<i>Neural Processes</i>	<i>Activity Dependent Neural Refinement</i>	<i>Response to Overall Affective State</i>	<i>Further Genetically-Based Value System Dimensions</i>	<i>Overall Valence/Value System</i>
<i>Hebbian processes</i>	Yes	No	No	None
<i>Skinnerian conditioning</i>	Yes	Yes	No	1-dimensional
<i>Affective Neural Darwinism</i>	Yes	Yes	Yes	9-dimensional (see Table 1)