Affective Neuronal Darwinism:

The Nature of the Primary Emotional Systems

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Abstract

Based on studies in affective neuroscience and evolutionary psychiatry, a tentative new proposal is made here as to the nature and identification of primary emotions. Our model stresses phylogenetic origins of emotional systems, which we believe is necessary for a full understanding of the functions of emotions and additionally suggests that emotional organising systems play a role in sculpting the brain during ontogeny. Emotions thus affect cognitive development. A second proposal concerns two additions to the affective systems identified by Panksepp. We suggest there is substantial evidence for a primary emotional organising programme dealing with power, rank, dominance and subordination which instantiates competitive and territorial behaviour and becomes the evolutionary source of self-esteem in humans. A programme underlying disgust reactions which originally functioned in ancient vertebrates to protect against infection and toxins is also suggested.

Introduction

Cognitive development of individuals, we have suggested, proceeds in part due to influences of primary emotional operating systems which act collectively as fitness criteria guiding further neuronal development (Ellis & Toronchuk, 2005). In short, Panksepp's (1998, 2001) formulation of affective neuroscience can be seen as a compliment to neural Darwinism as proposed by Edelman (1989, 1992). Important aspects of neural development could be explicated by linking emotional systems to neural Darwinism, a concept for which we have used the term Affective Neural Darwinism. It 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. 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 primary emotional systems. Although the notion of valence (usually as approach/avoidance or pleasure/pain)is common in theories of emotion, we use the term as referencing reward and punishment, probably reflecting internal states or markers (see discussions in Prinz, 2004; 2010; Rolls, 2005).

Neurotrophins, such as brain derived neurotrophic factor (BDNF), and monoamines such as serotonin, play critical roles in neural Darwinism by promoting neural plasticity, activity dependent refinement of neural networks, and selection of simultaneously active neurons for survival (e.g. Hua & Smith, 2004; Lauder, 1993; Poo, 2001). Under adverse conditions, however, complex interactions between neurotrophins, monoamines, cytokines and environmental influences may result in psychiatric conditions or neurodegenerative disorders (reviewed in Castrén, 2005; Dinan, 2009; Duman & Monteggia, 2006; Goodyer, 2008). Early activity in emotional circuits may thereby influence the wiring of later developing brain areas. For example dopamine knockout mice have reduced BDNF in the frontal cortex which may lead to reduced plasticity (Fumagalli, Racagni, Colombo, & Rival, 2003). Serotonin, perhaps the earliest transmitter present in embryogenesis, plays several critical roles during neural development which leave permanent effects (reviewed in Whitiker-Azmitia, 2001). Because BDNF (Gordon, Burke, Akil, Watson &. Panksepp, 2003), and nerve growth factor (Alfonso, Pollevick, van der Hart, Flügge, Fuchs, & Frasch, 2004) can be modulated by activations of emotional systems, the wiring of brain circuitry may be widely influenced by activation in emotional circuits during early life. The immune system is also implicated 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; Müller & Schwarz, 2007). Prenatal stress causing endocrine and immune changes in neurotransmitter systems can alter the developmental course of the brain increasing the risk of various disorders (e.g. Ansorge, Hen & Gingerich, 2007). Early childhood trauma has similar effects (Nemeroff, Bremner, Foa, Mayberg, North, & Stein, 2006; Goodyer, 2008). Both prenatal and postnatal activation of the monoamine, endocrine and immune systems may thereby alter brain plasticity, and although this may occur initially at lower brainstem levels, ultimately the developmental course of cortical areas may be altered.

Thus primary emotional systems, influenced by endocrine and immune functions, mediate the core nature of the selection criteria guiding the refinement of synaptic connections to provide the emotional palette that guides individual brain development (Ellis & Toronchuk, 2005). This mechanism ultimately influences learning, cognitive and social 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 which guide flexible behaviour while interacting with several layers of non-specific perceptual, attentional, and cognitive processes. Panksepp stresses that primary emotions, in contrast to secondary (discussed below), include instantiation in the phylogenetically ancient medial and ventral brainstem pathways rich in visceral innervation which utilize a variety of visceral neuropeptides (Panksepp, 1998, 2003a). While concurring with Panksepp, we propose that valenced primary emotions should

not only be capable of altering evolutionary survival rates of organisms, but be effective at the ontological level in determining which synapses survive in individual brains in accord with Edelman's neural Darwinism. We propose an integrative perspective emphasizing both individual ontogeny *and* a broad evolutionary narrative for identification of primary systems with a resulting *proposal for a more complete set of primary emotional systems*.

Our list of primary emotions takes into account previous proposals by authors from Darwin onwards (e.g. Damasio, 1999; Ekman, 1972; Izard, 1992), but is based primarily on the comprehensive studies of Panksepp. 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 primarily a 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 rather than starting with a framework informed by vertebrate evolution. The type of phylogenetic approach to emotions described by Lawrence and Calder (2004) is necessary, but should extend beyond the mammalian order.

Basic Insights from Evolutionary Psychiatry

Two key issues emerge from discussions in evolutionary psychiatry (Stevens & Price, 2002; also e.g. Gilbert, 1989; Price, Gardner, Wilson, Sloman, Rohde & Erickson, 2007) .

1. *Evolutionary pressures have developed various psychological traits that are experienced by us as emotions and which give rise to behaviourpatterns which originally enhanced survival*; 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 emotional systems 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 the pathologies that result from failures in the *attachment* and *rank systems* These archetypal systems 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" (Stevens & Price, p. 50). The rank or power/dominance "enables an individual to assess whether a rival is weaker or stronger and to produce the appropriate response" (p.75). This system has been well-studied in fish, reptiles (MacLean, 1990) and even crayfish (Panksepp & Huber, 2002) attesting to its ancient lineage. In line with these concepts from evolutionary psychiatry, we develop below a systematic proposal which incorporates a *rank/power/dominance system.*

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.

2. Emotional systems were selected for in terms of their enhancement of survival resulting in the evolution of primary emotional systems which then come to shape the development of both intellectual capacities and secondary emotions.

3. Survival of individuals may be enhanced by group membership that allows benefits in terms of food procurement, protection, learning, and eventually culture.

4. To make group membership effective, there must be both group cohesion mechanisms, and mechanisms for resolving conflict and resource allocation tensions. These necessitate some form of communication between members.

5. The attachment systems and the power/dominance system evolved to meet the needs of individuals living in groups; they supplement the basic systems for survival and learning.

6. Humans experience, particularly through subjective feelings thereby induced, emotional systems whose mechanisms lie beneath psychological and developmental events without being aware of their evolutionary origins and function.

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 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 a combination of 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 utilize overlapping pathways.

C3. *Function*: Each primary emotion system enlivens immediate affective functioning and because a unique combination of neurochemicals affects each primary system, each can functionally take part in neural Darwinism during ontogeny of the brain.

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

C5. *Origin*: On a phylogenetic level, each system 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*: Primary emotional systems occur 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 (2000, 2005) refers to as reflexive and sensory affects from true primary emotional systems. Primary emotions are action promoting valenced states with distinct 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 (e.g. startle) are closely time-locked to triggering stimuli. An emotion, therefore, is a *superordinate* program which orchestrates and integrates the activities of various functional subprograms including reflexive affects, perception, cognitive appraisals and feeling states (Cosmides & Tooby, 2000). Emotional systems organize complex extremely flexible reactions by activating or inhibiting autonomic, hormonal and/or somatic changes. The specific combination of behavioural components will depend on context, experience and eliciting stimulus.

A second key problem is to differentiate primary from 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 neocortex (Panksepp, 2000; Prinz, 2004, p.144-147) whereas primary emotions are largely dependent on subneocortical structures. 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. Mere *existence* of a speculative evolutionary explanation is not sufficient, but *its absence* is a strong mark against any proposal.

The Relation between Needs and Emotional Systems

The primary emotions identified by Panksepp (1998) are[1]:

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.

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 list, 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*. We propose that *these emotional systems, adapted for survival needs of individual organisms, embody selection criteria underlying neural thereby determining brain development*. Thus they provide affective valence (as reward/punishment markers) underlying cognitive and social development (as discussed in Ellis & Toronchuk, 2005). The first group of systems relate primarily to the functioning of individuals, and the second primarily to functioning of individuals in social groups. Although we have used this distinction as a broad classification, it is not essential to our proposal. (Compare e.g. Buck's 1999 grouping of selfish vs. social biological emotions corresponding to right vs. left hemisphere activation).

Basic Functioning

Panksepp's **SEEKING System** is the primary task-oriented pathway by which affective goals are met (Panksepp, 1998). This generalised system activated by primary biological needs characterised by homeostatic detection mechanisms, can also function in a non-specific manner. Other primary emotional systems **E3-E9,** each characterised by a genre of intention and intensity of desire, feed information to the

SEEKING system, as do secondary emotions thereby affecting overall motivational state [2.] Through inclusion of conscious volitional goals in **E1,** intentions and resulting purposive action gain emotive power: "I want that job", "I need that house", and so on, have affective components. This is ultimately the way that ethical choices and values (the basis on which we choose acceptable actions) become effective in guiding action, as they too have underlying affective components.

Evidence now suggests (e.g. Berridge & Kringelbach, 2008; Berridge, Robinson & Aldridge, 2009) that reward functions are parsed into two components--a motivational, appetitive system (corresponding to Panksepp's SEEKING/expectancy system) and a distinct hedonic appraisal or consummatory system. This "wanting" vs "liking" distinction is supported by the fact that addiction involves craving but not necessarily satisfaction (Robinson & Berridge, 2003). The relevant neural pathways normally function together, but can be behaviourally dissociated (e.g. Cannon & Besikri, 2004; Knutson, Fong, Adams, Varner & Hommer, 2001), even in fish (Spector, 2000) which suggests the division is of ancient origins.

The earliest protovertebrate ancestors would of necessity had consummatory appraisal responses even before appetitive seeking behaviours evolved, hence "liking" might be considered the primordial reward system. In mammals the lowest level for hedonic appraisal must reside in the brainstem as decerebrate rats and anencephalic infants also show hedonic responses (Steiner, Glaser, Hawilo & Berridge, 2001). Although taste, which relies on medullary nuclei, was probably the earliest effective stimulus, the ventral pallidum, nucleus accumbens and several other forebrain structures evolved 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 described by Panksepp (see e.g. Burgdorf & Panksepp, 2006; Kelley & Berridge, 2002).

The mesolimbic dopamine system, extending from midbrain ventral tegmental area (VTA), lateral hypothalamus, nucleus accumbens shell to orbitofrontal cortex, was traditionally implicated in the neural basis of reward; however it is now recognized that both "wanting" and "liking" mechanisms also utilize endogenous opioids (Levine & Billington, 2004; Pecinia, 2008) and the ventral pallidum is a necessary component of the pathway (Smith, Tindell, Aldridge, & Berridge, 2009). These systems play a fundamental role in learning, possibly by associating arousal with specific activities, thus attaching a positive affective value to them and then acting as a stimulus for repetition of these activities (see e.g. Rolls, 2005; Wise, 2004).

Following Panksepp's suggestion (1998) that the SEEKING system evolved to provide a common currency of reward, we suggest that facilitated by associated hedonic appraisals it 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 and pallidum (Kelley & Berridge, 2002; Smith et al., 2009). The ventral pallidum also responds to a wide variety of food, sexual and affiliative cues (e.g. Dillon, Holmes, Birk, Brooks, Lyon-Ruth, & Pizagalli, 2009; Shin, Dougherty, Alpert, Orr, Lasko, et al., 1999; reviewed in Smith et al, 2009).

Psychological illnesses associated with malfunctioning of the SEEKING system include addictions and cravings, eating disorders and possibly schizophrenia (see Panksepp, 1998; Panksepp & Harro, 2004). Panksepp (2002) considers obsessive compulsive behaviours to be malfunctions of this system, however, we suggest below

that washing symptoms may arise instead from malfunctions of the DISGUST system, while symptoms of checking and hoarding may arise from the POWER/dominance system.

Basic Survival

In previous papers (Toronchuk & Ellis, 2007a, b) we described a primary emotional system which like SEEKING can be activated by many sensory modalities and ideational components, but with opposite functions. We designate this as the **DISGUST system** rather than AVOIDANCE or AVERSION because of the previous inclusion of disgust in various lists of basic emotions from Darwin onwards. Together SEEKING and DISGUST would have been the primal operating systems. DISGUST evolved from primitive chemosensory mechanisms adapted to both avoid pathogens and their toxins and eject them from the gut if necessary. We propose this occurred in conjunction with the development of interaction between the immune and nervous systems (c.f. discussion in Ellis & Toronchuk, 2005; Rubio-Godoy, Aunger & Curtis, 2007) because non-specific (innate) immune cells are found in almost all multicelled organisms. Evolutionary adaptation led to learned avoidance of toxic or infected material *before* ingestion, as opposed to vomiting afterwards, ultimately giving rise to the subjective human experience of disgust as an anticipatory mechanism for avoidance.

The DISGUST system meets the criteria we enumerate above for primary emotional systems (Toronchuk & Ellis, 2007a, b, but see Panksepp, 2007). This system is intended as a parallel mechanism to appetitive SEEKING.. Broader than the sensory affect *distaste*, disgust is elicited by olfactory, gustatory, auditory, tactile or visual cues (see Curtis & Biran, 2001; Curtis, Aunger & Rabie, 2004). Therefore it is not restricted to avoiding bad taste, but hinges on avoidance of contamination (Haidt,

Rozin, McCauley & Imada, 1997; Rozin & Fallon, 1987; Rozin, Haidt, McCauley, 2008). 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.

We suggest that the disgust response did not arise merely as a reaction to bad taste, but due to association with increased likelihood of illness, as proposed by Curtis. Serotonin release is essential for development of disgust reactions and its use by the gut, vagus nerve, brainstem disgust mechanisms and immune signaling suggests a defensive continuum of immune and disgust systems linked by serotonin (Rubio-Godoy et al., 2007). Negative hedonic value is not necessary for disgust as conditioned taste aversions (CTA) can be elicited to sweet tastes paired with illness (e.g. Garcia, Hankins & Rusiniak, 1974; Parker, Rana & Limebeer, 2008). Conditioned immune responses may form from single pairings of novel taste with antigens (Pacheco-Lopez, Niemi, Kou, Harting, Del Rey, Besedovsky, & Schedlowski, 2004) further suggesting to us an evolutionary role of the immune system in disgust. Formation of a CTA requires activation of the insula (Ferreira, Ferry, Meurisse, & Levy, 2006) a structure which compares incoming and stored tastes (Koh & Bernstein, 2005). Rats with insular lesions fail to learn anticipatory discrimination although they remain capable of hedonic responses (Kesner & Gilbert, 2007).

Human AI, containing the gustatory cortex, also plays a role in self-awareness (reviewed in Toronchuk & Ellis, 2007a). It is activated during experience, observation and imagination of disgust (Jabbi, Bastiaansen & Keysers, 2008. Insular responses to aversive tastes vary according to expectations (Nitschke, Dixon, Sarinopoulos, Short, Cohen, Smith, 2006) and disgust sensitivity (Calder, Beaver, Davis, van Ditzhuizen,

Keane & Lawrence, 2007) as predicted for a primary emotional system. Although many behaviours integrating higher cognitive processing with "gut level" feelings also activate AI (see Saper, 2002), it does not provide a simple module for disgust, but functions together with other areas to instantiate a variety of interoceptive bodily and conscious feeling states. The adjacent orbitofrontal cortex also plays a major role in taste, smell and visceral responses and in assigning reward values to stimuli (Rolls, 2005; Rolls & Grabenhorst, 2008).

Increased cortical integration of body states occurs in primates because the insula receives direct thalamocortical taste and visceral input which in rodents comes via the amygdala (Craig, 2005). Emotional contagion or "resonance" is a further adaptation promoting disgust activation when observing socially relevant disgust responses in conspecifics (von dem Hagen, Beaver, Ewbank, Keane, Passamonti, Lawrence et al., 2009; Wicker, Keysers, Plailly, Royet, Gallese, & Rizzolatti, 2003). The insula may play a role in social evaluation cognition as a viscero-motor centre which simulates the activity of others in a manner similar to "mirror" neurons previously described in monkeys (Gallese, Keysers and Rizzolatti, 2004; Keysers & Gazzola, 2007).Cognitive processing allows blending of primary DISGUST with social learning to produce secondary emotions incorporating social status and morality. Activation patterns of facial muscles in response to unpleasant tastes, contaminated objects, and unfair treatment are consistent with the suggestion of shared neural evaluative mechanisms for distaste, olfactory disgust and moral disgust (Chapman, Kim Susskind and Anderson, 2009). The role of the insula in awareness of disgust in self and others facilitated by the emergence of von Economo neurons in AI and adjacent orbitofrontal area may be a preadaptation setting in motion the development of theory of mind and moral reasoning. We are thus suggesting that the

primitive emotive circuit which originally functioned to defend the self by regulating consummatory behaviours contributes to an emotional system which facilitates some of the most highly developed human capacities of social evaluation (Toronchuk & Ellis, 2007).

Disgust sensitivity is inversely correlated with sensation seeking (Haidt et al., 1997) further suggesting that DISGUST and SEEKING represent opposing emotional systems which are then reflected in personality traits and psychiatric disorders. Genetic influence in disgust sensitivity (Kang, Kim, Nankoong & An, 2010; Olatunji & Broman-Fulks, 2007) is consistent with the finding that even pre-symptomatic genetic carriers of Huntington's disease show selective deficits in recognition of disgust (Hennenlotter, Schroeder, Erhard, Haslinger, Stahl, Weindl, et al., 2004) and insular size in presymptomatic patients is correlated with disgust recognition (Kipps, Duggins, McCusker & Calder, 2007). In contrast increased activation of the insula in OCD patients is associated with contamination specifically washing-related symptoms (Shapira, Liu, He, Bradley, Lessig, James, et al., 2003; Stein, Liu, Shapira & Goodman, 2001). Phillips and Mataix-Cols (2004) find that patterns of brain activation in OCD patients to disgust or anxiety-producing objects vary according to the patient's major symptom type. Brain activation to disgust stimuli occurs independently of anxiety supporting the view that OCD typified by contamination/washing symptoms represents an underlying malfunction of the DISGUST system (Husted, Shapira & Goodman, 2006; Lawrence, An, Mataix-Cols, Ruths, Speckens, & Phillips, 2006).

In contrast to internal threat, protection from external sources is provided by the **RAGE E3** and **FEAR Systems E4** described in detail by Panksepp (1998). Previous experience and assessment of the present circumstances determines which

solution to threat will be implicated. As with DISGUST certain stimuli are predisposed to easily activate FEAR (Öhman, & Mineka, 2001) and as with DISGUST the same structures activated during the production of fear and anger are also activated during recognition of fear and anger in others (see discussion in Goldman & Sripada, 2005). 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 (Panksepp, 2002).

Reproduction

Sexual reproduction**,** an obvious necessity for evolutionary selection, is the outcome of the **LUST system E5** which also infers "wanting" and "liking" components. As with the SEEKING system the appetitive and consummatory aspects can function independently with corresponding behavioural dissociation (Kippin, Sotiropoulos, Badih & Pfaus, 2004; Pfaus,1996). The transition to mammals brought about modifications in this ancient system to produce attachment necessary for lactation (Panksepp, 1998). Thus the LUST system is related to both the PANIC/attachment (or separation) and CARE systems. Due to different mating strategies, the complex relationship between LUST, PANIC and CARE likely differs in males and females giving rise to different attachment styles in adult humans (see Del Giudice, 2009; Taylor, 2006).

All the social emotional systems are mediated by hormones, synaptic signalling and other biochemical signals as employed by other elements of the value system. Social emotions can be therefore causally effective in terms of contributing to neural Darwinism because these signals affect brain plasticity. 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 associated with **SEEKING)** of male prairie voles; and its blockade prevents pair bond formation (Lim & Young, 2004). In addition, vasopressin plays a role in sexual competition (see e.g. Sewards & Sewards, 2003a).

In parallel with the SEEKING system, dopamine is associated more with the appetitive phase of LUST and endogenous opioids more with the consummatory phase. Dopamine secretion increases with sexual arousal, but at consummation dopamine decreases while secretion of oxytocin and opioids increase (Pfaus, 1996; Van Ree, Niesink, Van Wolfswinkel, Ramsey, Kornet, & Van Furth, et al., 2000). Psychological illnesses associated with the malfunctioning of this system are fetishes and sexual addictions (Panksepp, 2002), and disorders of desire and orgasm reflecting either over-activation or under-activation of the subcomponents.

Primary emotional systems of social bonding

CARE, PANIC, PLAY are also important emotions of social behaviour discussed in detail by Panksepp (1998, 2002) Here we discuss primarily their crucial roles in sculpting mammalian social and cognitive development. 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 mammalian survival, significant selection pressure would act on the neural mechanisms controlling these behaviours. In mammals social bonding is initially effected primarily by the **PANIC/attachment (E6)** system in the young, which triggers emotional panic during separation, but signals contentment during closeness. This necessitated the tandem development of a complementary

CARE (E7) system, through which parents respond to the young. Panksepp's (1998) PANIC is basically a separation distress system; however, we suggest that distress is only one mode because the system also operates in a positive fashion when needs are satisfied. Subjective feelings in the human infant include panic/distress during separation, and contentment/comfort during contact (**E6**); and in the care-giver, tenderness/ affection, carrying over to reciprocal distress when the infant is perceived as in distress (**E7**).

The biological origins of human sadness are rooted in an extended system involving the cingulate gyrus that mediates separation distress in infant mammals, although this neurological substrate was used earlier in phylogeny for perception of physical pain (Panksepp,1998; 2003b). An origin in pain perception is supported by the role of endogenous opioids in both pain reduction and the positive feelings associated with contact between individuals 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/attachment) over the course of vertebrate evolution. Separation from loved ones is thus perceived in humans as similar to pain and panic because of these evolutionary origins. The underlying commonalities in endocrine mechanisms of social relationships suggest that infant/adult bonding is also supported by the more ancient circuitry of the sexual attraction system. Oxytocin is a mediator in both maternal and adult pair-bonding (Carter, 1998, 2003; Insel & Young, 2001; Lim & Young, 2004; Taylor, 2006). Estrogen further enhances the effects of oxytocin, perhaps providing a basis for gender differences in human attachment styles (see del Giudice, 2009, Taylor, 2006).

Hominid infants have a relatively long period of helplessness combined with a need for training in foraging and social behaviours. Two additional factors discussed

by Falk (2004) that may have contributed to the extended helpless period of human infants was the trend toward a narrow pelvis associated with bipedalism, and the expansion of the human brain. Together these factors possibly selected for infants that were delivered at an increasingly immature stage of development. The absolute necessity of nurture during an increasingly extended infancy likely put considerable selection pressure on the neural circuitry for emotional attachment between mother and infant with this circuitry predicted to be most highly developed in humans.

Neonatal chimps lack the ability to cling and must be initially supported by the mother (see Falk, 2004). Mothers of older infants use body language and gestural signals to encourage climbing on the mother's back for transport. The mother's use of gestures and facial expressions plays a key role in communication with infants, who correspondingly develop an intense interest in the mother's face. Chimp mothers also teach which foods are edible, and perhaps even tool use (Falk, 2004; Goodall, 1986). Chimp infants communicate various types of distress through specific vocalizations. Hominid evolution therefore involved tandem evolution of emotional circuitry in adults to provide not merely food, but also emotional nurturance and instruction, and parallel circuitry in the young to seek and respond to others. This entailed increasing use of gesture, facial expression, tactile and vocal communication. The lengthening period of dependency in early humans necessitated in turn the development in adults of even more skilled caretaking and teaching ability. Although the role of early emotion in producing individual adult 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 could have provided the emotional motivation for the development of language, with language use in adult coalitions being a more

secondary development. Learning in infants is critically enabled by reciprocal interaction with the care-giver during early life (e.g. Schore, 1994). The ability for shared attention between infant and care-giver is necessary for the development of a theory of other minds and 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 care-giver.

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 a negative feedback mechanism. Early tactile stimulation of female rodent regulates the expression of estrogen receptors in the medial pre-optic area of the hypothalamus, resulting in adulthood an increase in oxytocin receptor binding leading to increased licking, grooming and nursing. 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. Long term effects are illustrated by the finding that adults who experienced childhood adversity both rate reward cues less positively and show less response in the reward-evaluating pallidum (Dillon, et al., 2009). These effects possibly illustrate how the PANIC and CARE systems may work in tandem to influence emotional and cognitive behaviours throughout life.

Gender differences also exist in the endocrine and neural responses to stress (e.g. Dalla, Antoniou, Kokras, Drossopoulou, Papathanasiou, Bekris, et al., 2008). 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 some other early response to maternal behaviour), thus perhaps underlying differences in attachment styles. Specifically human males show more avoidant attachment and females more ambivalent attachment from middle childhood through adulthood (Del Giudice, 2009). Fight and flight behaviour is more typical of males under stress, while oxytocin facilitates tend and befriend behaviour in females (Taylor, 2006).

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). Influenced by Harlow's work with macaques, Bowlby then, established that infant separation had long term impact on human social development. According to Panksepp (2002), malfunctioning of the PANIC and CARE systems can result in panic attacks, pathological grief, depression, agoraphobia, social phobias, dependency, attachment disorders, and may also be a contributing factor to autism. The risk for post-traumatic stress disorder may also be increased (Nemeroff et al., 2006). Thus, the PANIC and CARE systems provided the basis for evolution of human social bonding, altruistic behaviour, and possibly even language evolution (see Toronchuk & Ellis, 2009).

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 preparation for adult food procurement and social roles, suggests that play should be considered a basic emotional program necessary for the normal cognitive development of humans. It is facilitated evolutionarily by the enlargement of the cerebral cortex and the prolonged infant/maternal interaction necessitated by lactation (MacLean, 1990). The

developmental role of play 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) and functions to allow assessment of social commitments. Playful teasing allows the exploring of social boundaries and dominance interactions.

Mother infant interaction in chimps and especially bonobos are characterised by extensive play periods involving facial gestures, vocalizations and often laughter (reviewed in Falk, 2004a). It is possible that optimal cortical plasticity in mammals may depend on the rewarding effects provided by play. Hence 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 an important role in human learning the underlying neurophysiological mechanisms are not yet defined.

We agree with Panksepp's (1998) inclusion of "rough and tumble play" in the list of primary emotions, while suggesting that in humans the system has been extended to include representational and intellectually imaginative play. There is also evidence that both captive and wild apes may 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" to the capacity for representational play facilitated the evolution of language by enabling a mechanism which allows one concept to represent another. Ability to understand and empathise with others is another critical part of imaginative play likely necessary for language evolution. Play remains particularly important in the ontogenetic processes of language development (see Bruner, 1983; Paley, 2004; Zigler, Singer & Bishop-Josef, 2004).

Many aspects of human culture such as play-acting may ultimately depend on extensions of this system (Frost, Wortham & Reifel, 2001). It is an essential component of the performing arts, ceremonial and celebratory behaviour, as well as being an important source of creativity (even in science). The associated feeling is joy/fun. Humour retains a basic tie to rough and tumble activity, including the often embedded dominance paradigms in which we "make fun" of others, "reducing" them in a comical way. The social aspects of play such as turn taking and feinting behaviors may also contribute to development of altruistic behaviour.

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

Group Function: Regulating Conflict and Competition

A main point of the present paper is to 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 many invertebrates, and vertebrates alike revealing their ancient origins. Group living may enhance individual survival through cooperative food procurement, protection, and learning , but also entails competition for resources. Selection pressure likely favoured the development of mechanisms which communicated the dominant status of some individuals, while allowing survival of those subordinates who participate in cooperative group activities. These latter may themselves have a chance to reproduce at a later time. Panksepp (1998, 2002, 2007) describes social dominance as arising from interactions between the childhood PLAY system and the adult FEAR and RAGE systems. Although

ontogenetic development of dominance in individuals is often enabled by play, it can be pointed out that this emotional system is phylogenetically more ancient than play. In terms of evolution POWER predates the emergence of both mammals and PLAY.

Allocation of rank leads to agonic behaviour which regulates competition while minimizing tension (e.g. de Waal, 1996, pp. 89-125; Stevens & Price 2002, pp.49-52). Human competition takes place for "territory" in the widest sense, including material resources, social control, sexual mates, status symbols, and intellectual turf. Personal identity, therefore, comes to be closely influenced by the basic emotional circuitry for territory.[3] Based on data from human psychiatry and animal behaviour, we propose that the ancient system of dominance and submissive subroutines is the phylogenetic precursor to competition for status, including the need to excel and obtain social approval. With a few modifications it is similar to the "power dominance" drive described by Sewards and Sewards (2003b) as underlying Nietzsche's will to power and Winter's (1973) implicit power motive.

Price and co-workers (Price et al., 2007) point out that elevated mood facilitates a rise in rank which enables coping with leadership, while depressed mood allows lower ranking individuals to communicate acceptance of their status and forgo reward. The desire for higher rank is associated with feelings of pride/high self-esteem during success and shame/low self-esteem or depression during defeat.[4] They locate the instinctual aspects of depression as largely a function of the striatal complex of MacLean's reptilian brain. This area according to the triune brain theory, was responsible for ancient instinctual behaviour patterns involved in display behaviours while limbic structures were only recruited after the evolution of mammals (MacLean, 1990).

The striatal complex, particularly the basal ganglia, plays a major role in vertebrate dominance behaviours. Its activation occurs during social/territorial displays among male lizards along with release of serotonin and dopamine in different patterns correlated with dominance and subordination (Baxter, 2001; MacLean, 1990). The localization of serotonin and dopamine is similar in the lizard and primate basal ganglia inferring similar mechanisms (Baxter, 2003). Lesions in globus pallidus of the mammalian basal ganglia disrupt dominance and courtship displays given by male monkeys (MacLean, 1990; Newman, 2003) while non-sexual competitive arousal activates the human ventral pallidum, an area associated with reward (Rauch, et al.,1999). The basal ganglia, as Price suggests, may thus mediate components of depression which act as appeasement displays after loss of agonistic encounters. The role of the ventral pallidum in reward assessment (Smith et al., 2009) may thus be relevant to this type of depression.

In humans the ventromedial prefrontal and anterior cingulate cortices (ACC) are also involved in the POWER system. The prefrontal cortex, including orbitofrontal area, plays a role in perception of status (Karafin, Tranel, & Adolphs, 2004; Marsh, Blair, Jones, Soliman & Blair, 2009) and also in OCD (Baxter, 2003) as elaborated below. ACC is activated during competitive arousal (Rauch et al.,1999); and low activity here is implicated in the pathogenesis of depression with different subregions playing differential roles (see Davidson et al., 2002). Sewards & Sewards (2003b) describe an area of ACC (Brodman's 32 ventral to the fear representational area) as participating in the "power dominance" drive and equate this area with the infralimbic area identified in hamsters as responsible for dominance (Kollack-Walker, Don, Watson & Akil, 1999). They propose the major contribution of ACC is to the learned, voluntary components of the power dominance drive rather than the

involuntary components which are mediated elsewhere. In Price's model, following MacLean, these involuntary components would be controlled by the basal ganglia. Activation of submissive circuitry may lead to depression (see Gilbert, 1992; Price Gardner & Erickson, 2004; Price, et al., 2007; Stevens & Price, 2002), a possible human counterpart of social defeat (e.g. Kroes, Burgdorf, Otto, Panksepp, & Moskal, 2007; Sloman,Gilbert & Hasey, 2003; Sloman & Gilbert 2000). Low status is a major risk factor for depressive behaviour (e.g. Gilbert, 1989; Panksepp, Moskal, Panksepp & Kroes, 2002) and adverse health effects in humans and other primates (Sapolsky, 2005) and according to MacLean even reptiles.

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 influence one another in both vertebrates and invertebrates (e.g. crayfish, Panksepp & Huber, 2002). An example from *Anolis* lizards is that manipulation of dominance relationships 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 dominance behaviour in human males (Moskowitz, Pinard, Zuroff, Annable & Young, 2004) while decreasing serotonin reuptake reduces submissive behaviours.

Several hormones and neuropeptides 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 the effects in both sexes are probably correlated more with status behaviours than aggression (Eisenegger, Naef, Snozzi, Heinrichs & Fehr, 2010). Although testosterone may increase competitiveness and dominance, high testosterone accompanied by low serotonin may lead to impulsivity and therefore pathological aggression (Birger, Swartz, Cohen, Alesh, Grishpan, & Kotelr, 2003). Social defeat in rodents (Panksepp, Burgdorf, Beinfeld, Kroes & Moskal, 2007) results in widespread decreases in cholecystokinin (CCK) while play produces increased levels of CCK (Burgdorf et al., 2006). In these rats social defeat also increased levels of corticotrophin releasing hormone (CRH), a change otherwise associated with depression (see Brown, Varghese & McEwen, 2004). Another biochemical similarity between animal social defeat and human depression is the finding that interleukin 18, overexpressed in depressed humans, is also increased following social defeat in rats (Panksepp et al., 2007).

Vasopressin, and its nonmammalian homologue vasotocin, facilitates aggressive and dominance related behaviours in many species and is itself modulated by testosterone and serotonin. Increased vasopressin is also 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). Sewards and Sewards (2003a, b) propose that vasopressin is the primary generator of the "power dominance" drive. Given experimentally to men and women , however, it has a differential effect, producing to increased agonistic behaviour in men and increased affiliative behaviour in women (Thompson, George, Walton, Orr, & Benson, 2000). Social subjugation of hamsters decreases levels of vasopressin in the anterior hypothalamus (Delville, Melloni & Ferris, 1998). In contrast microinjection into this

area triggers stereotypical behaviours (grooming and scent marking) thought to model human OCD but these behaviours are suppressed by drugs used in its treatment (Ferris, Rasmussen, Messenger & Koppel, 2001).

Taken together the involvement of the basal ganglia, vasopressin and serotonin in social dominance and OCD may be consistent with the view of Stevens and Price (2002) that OCD is 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 underlying checking and perhaps hoarding, but not washing-type symptoms of OCD. A second hypothesis regarding POWER is 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). Some suggest further that the involuntary subordination response may have phylogenetic origins in separation distress because both are characterised by negative mood, loss of self-confidence, and result in decreased activity (Sloman et al, 2003) however, it should be pointed out that the ancient dominance and subordination system is found in reptiles with no known attachment systems.

Weisfeld and Wendorf (2000), building on Beck's differentiation of sociotropy and autonomy, propose that depression assumes two forms. Sociotropy is a personality characteristic related to attachment and the need to please others, while autonomy is related to independence and attainment of goals. 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. These two characteristics are expressed in different symptoms (Robins, Block & Peselow, 1989) with autonomous traits responding better to antidepressant medication than

sociotropic traits (Peselow, Sanfilipo, Robins, Block & Fieve, 1992). Also in the non-clinical population low moods are associated with different behaviours depending on whether such moods are precipitated by social losses or failure to reach a goal (Keller & Nesse, 2005).

Consistent with the notion of two forms of depression discussed above, the physiology and developmental course of depression appears to differ according to whether it is precipitated by separation distress or by loss of status. In line with this notion, oxytocin reduces separation distress, but not low rank; separation anxiety involves the cingulate cortex, while in humans, dominance perception, pride and shame involve the orbitofrontal/ prefrontal cortex (Weisfeld & Wendorf, 2000; Karafin et al., 2004; Marsh et al., 2009; Panksepp, 1998). Another study suggesting two forms of depression differentiated by social components, reported left frontal activation in depressives scoring high in reassurance-seeking, but relative right frontal activation in those low in reassurance-seeking (Minnix, Kline, Blackhart, Pettit, Perez, & Joiner, 2004. Although not necessary for our theory, it may be possible that differential laterality relates to Gray's (1987, 1990) BAS/BIS distinction or Buck's (1999) selfish vs. social division. Weisfeld and Wendorf note the similarity between subordination displays and the behaviour of individuals whose depression is associated with shame, guilt and failure e.g. averted gaze, slumped posture and slowed responses.

In summary, we propose that in addition to the systems listed by Panksepp, 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. The development of neocortex allowed for integration with cognition and the emergence of human secondary

emotions such as guilt, shame and jealousy. Malfunctioning of this POWER/dominance system might take two forms: overactivation of subordination programmes might result in depression; overactivation of dominance programmes might be related to checking, ordering and hoarding symptoms of OCD. In contrast OCD with washing symptoms more likely arises from malfunction in the DISGUST system; while depression with sociotropic symptoms is mediated by the PANIC /attachment system.

Conclusion

The importance of correctly characterising the primary emotional systems is that according to the proposals of Affective Neuronal Darwinism, it is these systems that ultimately determine the details of higher brain functions such as cognition and secondary emotions. We think it therefore useful to use the criteria C1 to C7 above as assessment tools in analysis of the *necessary and sufficient* variables underlying later brain development (Ellis & Toronchuk, 2005). A summary of the systems we include is given in Table 3. On the basis of evolutionary we have added (a) the **DISGUST system E2** and (b) the **POWER/dominance system E9** to those included in our previous paper (based on Panksepp, 1998).

The list of basic emotions has varied only slightly from Darwin to the present (e.g. Damasio, 2003; Ekman, 1972, 1992; Izard, 1992; Plutchik, 2003; Tomkins, 1962). Virtually all include **Happiness**, **Sadness**, **Fear**, and **Anger**, and most include **Disgust** and/or **Contempt.** Some suggest **Surprise** and **Interest**, or less frequently **Guilt** or **Shame**. Sadness, in our model, corresponds to Panksepp's PANIC executive system (Panksepp, 2003b) mediating attachment and pain of separation. As Panksepp (2000) states, the major differences concern the status of **disgust**, **surprise**, **interest**, **guilt** and **shame**. Interest corresponds to Panksepp's generalised SEEKING system,

as does happiness, although it appears useful to parse SEEKING into incentive salience and hedonic appraisal (Berridge & Robinson, 2003; Berridge et al., 2009),. two components which may be separately accessed by other emotions. While the PLAY executive system is certainly associated with joy or happiness (Panksepp, 1998), we suggest happiness is more broadly based and corresponds to generalized hedonic appraisal based in the "liking" subsystem.

In contrast, we do not consider **Surprise** to be a primary emotion, for despite its inclusion in many lists, it does not have the same nature as other affect programs (Griffiths, 1997, p.241), is not necessarily valenced (Ortony & Turner, 1990; Ekman, 1992; Prinz, 2004, p.163), and gives no specific action guidance for survival. However, like the startle reflex, it may serve to activate SEEKING. Contempt, embarrassment, shame, and guilt are not included in our list because firstly they are uniquely human; and secondly they rely largely on neocortical functions and so are more plausibly secondary emotions. Shame and guilt are likely secondary emotions emerging from cognitive interactions with the POWER/dominance system (see Gilbert, 1998). This underlying function for POWER would be consistent with its inclusion 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, however the terms CARE and POWER/ dominance is more consistent with the concept of basic emotions as *emotional operating systems* reflecting developmental and 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 these operating systems playing a key role in brain evolution and development (affective neural Darwinism), a proposal which may generate future research on

evolutionary and developmental processes. Secondly, focusing attention on the functions of these systems in the evolutionary past has led psychology to a greater awareness of the biological complexities of human emotions. This promotes research on new treatments and diagnostic techniques. Finally, along with the concept of neural Darwinism, that of basic emotions may lead to new ideas on the promotion of healthy emotional development in infancy. Causal links are supported by the suggestions made throughout this paper 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 here. The extent to which the proposed emotional systems satisfy the criteria **C1 – C7** above, must be confirmed and remaining gaps filled. Table 3 shows our view on the current state of confirmation. Elucidation of genetic links will be important, although this task is hampered by the polygenic nature of the common heritable mental disorders and the relative rarity of each of a very large number of specific mutations contributing to each disorder (Keller & Miller, 2006).

Implications

The nature of the primary emotions has consequences in many areas of human behaviour e.g. economics, politics, and education (Ellis 2008). Given the assumptions of Affective Neural Darwinism, we have a much more nuanced version of motivational theory than simple conditioning theory provides. Each major need is related to one or more of the specific primary emotions either directly, or indirectly through secondary emotions; hence we can analyse psychological and psychiatric issues in terms of their relation to these primary emotional systems.

Further steps

The proposals here represent a small step towards developing the psychological implications of Affective Neural Darwinism. Further key steps include

1. Validating the *list of primary emotions* (summarised in Tables 1 and 3) for correctness and completeness. Is each case for inclusion adequate (cf. Table 3)?

2. Using the resulting list of primary emotions 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.

These steps relate to the broader task 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. 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; Panksepp, 2002).The proposals made here may help to clarify the issue of human universals in a way that takes cognisance of 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, as some research programmes are currently attempting (e.g. Kroes, Panksepp, Burgdorf, Otto & Moskal, 2006), the many links between the prototype states, neuroplasticity molecules and genes.

Finally, the question arises as to what difference there is between our proposal of Affective Neural Darwinism, and the Hebbian processes of neural refinement on one hand and Skinnerian conditioning on the other. A summary of the differences is shown in Table 4. 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 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 survival is enhanced 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 to various survival problems as set out in Table 1. These also then shape the nature of ongoing brain functions 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. In this sense it can be conceptualized as 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 experience of the world is not yet extensive, as compared to a situation operating only on Skinnerian conditioning.

Overall, our proposal is supported by a growing number of neurological and psychological studies emphasizing the role of emotions in human development. Cognition and emotion are clearly inseparably entangled on all levels of processing (e.g. see Gray, 1990; Pessoa, 2008; Phelps, 2006; Rolls, 2005). 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 on the nature of development, aptly encapsulates our approach.

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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 and we retain this designation.

[2]While it is possible for each goal to have separate seeking and pleasure systems, resulting in a combinatorial increase of complexity, it is more economical to have many systems utilising the same superordinate seeking and pleasure systems. The dopamine and opiate pathways are both generalised and responsive to many stimuli (see Berridge, 2004; Smith et al., 2009).

[3]We thank Ian MacCallum for pointing this out.

[4]Guilt, however, relates to failure to live up to expectations of self or others; thus it is a secondary emotion related to ethical behaviour rather than to ranking (see discussion below).

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 from Ellis and Toronchuk (2005), in line with our present scheme. The new numbering system will be retained in this paper .

EVOLUTIONARY NEEDS MET	***PRIMARY EMOTIONAL SYSTEM***	***Works With:***	***FUNCTIONS***
INDIVIDUAL NEEDS			
Basic Functioning	**E1: SEEKING system**	**E2-9**	Situation evaluation, incentive salience, hedonic appraisal, facilitates learning
Basic Survival	**E2: DISGUST system** (repulsion)		Avoiding harmful foods, substances, environments
	E3: RAGE system	**E4,E9**	Defence: protection of organism, 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: Bonding & Development	**E6: PANIC/attachment** (affiliation, separation distress)	**E5,E7**	Protection of vulnerable individuals; creates bonding through need for others
	E7: CARE system	**E5,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,E5**	Limiting aggression in social groups: allocating resources, esp. sexual ones.

Table 2

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 sources referenced throughout the text. 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.

Table 2

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

Table 3

Satisfaction of criteria C1-C7 for Basic Emotional Systems by the proposed primary emotional systems E1-E9, as we understand them on the basis of data presently available. The criteria are **C1** = Concept (see Tables 1-2), **C2** = Structure (neuroanatomy, see Table 2), **C3** = Function (neurotransmitters, see Table 2), **C4** = Development (genetics), **C5** = Evolutionary Origin (see Table 1), **C6** = Occurrence (homologues, see main text), **C7** = Outcome (psychiatric outcomes, see main text).

	Criteria for Basic System substantially satisfied? [Criteria Numbered as in Section 3**]**						
PRIMARY EMOTIONAL SYSTEM	***C1***	***C2***	***C3***	***C4***	***C5***	***C6***	***C7***
E0/E1: Pleasure and SEEKING (satisfaction and incentive salience)	**Yes**	**Yes**	**Yes**	**Partly**	**Yes**	**Yes**	**Yes**
E2: DISGUST system (repulsion)	**Yes**	**Yes**	**Partly**	**Partly**	**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 4

Comparison of Hebbian and Skinnerian processes with Affective Neural Darwinism. The Overall Affective State (Column 3) provides a 1-dimensional assessment of the organism's state (positive or negative; pain or pleasure). The genetically-based Value System Dimensions (Column 4) in the case of AND incorporate nuanced survival information selected during evolutionary history and transmitted genetically. Thus these encode specific inbuilt behaviour tendencies that are appropriate in different circumstances and are available without prior learning. The Overall Valence/Value System (Column 5) operating in the case of Affective Neural Darwinism 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.

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