Neurobiology of emotions: anatomy, neural circuits, and alexithymia

Ryan Smith, Harald Guendel, Richard D. Lane

<u>Summary of key statements</u>:

- **Emotion episodes can be broken up into three interacting processes: affect generation, affective response representation, and conscious access.**
- **Affect generation may involve interactions between ventral prefrontal cortices and subcortical nuclei**
- **Affect response representation may involve somatomotor and default network regions.**
- **Conscious access may involve frontoparietal network regions**
- **Individual differences in these processes, due to innate, developmental, or social learning-related factors, may account for psychosocial and brain-body interactions within the biopsychosocial model.**
- **Low emotional awareness (or high alexithymia) may result from a combination of deficits across these three processes and influence physical health outcomes via a biopsychosocial process involving a chronic failure to understand and regulate negative affect and its effect on peripheral physiology.**

Introduction

The project of trying to understand the neurobiology of emotions can be broken up into multiple steps. The first step involves decomposing the broad category of "emotions" into a set of more basic, interacting processes. The second step involves empirically mapping each of these more basic processes onto neural processes. The third step involves investigating how these neural processes interact. Finally, the fourth step involves determining whether the resulting neural model is able to 1) account for currently known aspects of emotion, and 2) predict new, previously unknown aspects of emotion. While described here in terms of a linear order, in practice this process is iterative, and progress on any given step is often capable of aiding in progress on each of the others. In this chapter we will briefly review current progress on each of these steps, and the neural model of emotion that results. With regard to the final step described above, we will also focus specifically on the trait variable of alexithymia (which includes "affective agnosia" or "very low emotional awareness"), as one important emotion-related variable that this neural model may be capable of accounting for.

Emotion-Related Processes

Affect Generation

One way of thinking about the category of emotion involves its decomposition into the following three processes: affect generation, affect representation, and selection for conscious access (See Figure 1)(Smith, Killgore, & Lane, 2018; Smith & Lane, 2015) (Smith, 2020). **Affect generation** is a process in which a characteristic set of modulatory influences are engaged, leading to changes in 1) the state of the body (e.g., heart rate, respiration, muscle tension, facial expression, circulating hormone levels, etc.; see Friedman, 2010; Kreibig, 2010), and 2) the state of other systems in the brain (e.g., modulatory influences on perception, attention, memory, motivation, decision-making, action selection, etc.; see Shiota & Kalat, 2012, Ch. 14). These changes are typically initiated in response to either 1) the detection of particular stimuli or 2) activation of a cognitive representation (e.g., a thought, memory, or interpretation of one's current situation). More specifically, an "appraisal" process is believed to occur, in which the meaning of such stimuli/representations is assessed along a range of dimensions, such as (for example) novelty, concern relevance, goal-congruence, compatibility with norms/values, and agency/control (e.g., see Scherer, 2009). Different appraisals across these dimensions then lead to the generation of different affective responses. For example, an appraisal of a stimulus as "novel," "concern-irrelevant," and "goal-congruent" might promote one type of affective response, whereas an appraisal of "concern-relevant," "goal-incongruent," and "out of my control" would lead to another. In either case, the initiated changes to the state of the brain and body would reflect mobilization of the predicted resources required to manage the situation at hand.

Affect Representation

Affect representation is a further process, which occurs in response to affect generation. More specifically, this process involves the subsequent perception/recognition of the changes in the brain/body that are initiated during affect generation. For example, when an increase in heart rate occurs following the visual detection of a snake, afferent signals from the body will then convey information about this change in heart rate – leading to an internal perceptual representation of that change (Khalsa, Rudrauf, Feinstein, & Tranel, 2009). At a more abstract level, and in conjunction with the modulation of other neurocognitive systems that occurs as part of an affective response, a cognitive representation of the conceptual meaning of that change will also be activated (e.g., "this increase in heart rate means that I am *afraid*"). The perceptual and conceptual representations arrived at during this process will also be influenced by other factors (Barrett, Mesquita, & Gendron, 2011; Reisenzein, 1983), such as learned expectations regarding what is most probable in a given context (e.g., one may be more likely to conclude they feel sad vs. afraid while at a funeral).

Conscious Access

At any given time, it appears the brain represents a much larger amount of information than can simultaneously be present within consciousness. According to leading models (Dehaene, 2014), this problem is solved via a selection process, in which the many different activated representations compete for **conscious accessibility**. Therefore, a further aspect of emotion involves whether or not the various representations of an affective response (and associated appraisals) discussed above become consciously accessible. If such representations win this competition, they will be consciously experienced as percepts, thoughts, motivations, and so forth. If they do not win this competition, such representation will remain outside of awareness, but may still have influences on some aspects of behavior. Crucially, some represented aspects of an affective response could win this competition even if others do not. Thus, an individual might consciously recognize their strong motivation to punch someone, even if they don't consciously recognize that they are angry; or an individual might consciously perceive a change in heart rate, even if they don't consciously recognize that they are afraid. In principle, any combination of conscious/unconscious representations of the different aspects of an affective response is possible. In practice, gaining conscious access to some representations might facilitate gaining conscious access to others, and a range of factors appear to play a role in determining which representations are selected for conscious access in a given context. Some of these factors (often associated with "executive control") include the availability of attentional resources, the goal-relevance of the content of a given representation, and its estimated probability of accuracy based on previous experience (Dehaene, 2014).

Neural Processes

At present, the neural basis of affect generation appears to involve a distributed set of brain regions and interactive processes. First, states of neural activity within the brain's various perceptual processing systems (e.g., visual cortex) appear to play an important role in detecting sensory stimuli, and representing their various perceptual features (Gazzaniga, Ivry, & Mangun, 2014, Ch. 5-6). These same representational neural states – in conjunction with other temporal and frontal lobe structures – also appear to be re-used during the processes of remembering, imagining, or otherwise thinking about those same stimulus features (Barsalou, 2009; Danker & Anderson, 2010; Pobric, Jefferies, & Lambon Ralph, 2010). Once such representations are activated, they provide an internal description that can be evaluated along the various appraisal dimensions described above – most likely via interactions between perceptual processing systems and regions linked to more abstract cognitive processing. According to recent reviews (Brosch & Sander, 2013; Smith & Lane, 2015), some regions suggested to play a role in specific appraisals include the medial temporal lobe (novelty), the amygdala (novelty and concern-relevance), the dorsal anterior cingulate (goal-congruence), the nucleus accumbens (goal-congruence), lateral temporal cortex (norm/value compatibility), and sensorimotor cortices (agency/control). A distributed "limbic network" of orbitofrontal, ventromedial frontal, and anterior temporal cortical structures, as

well as related subcortical nuclei, has also been identified as playing an important role in generating and representing the visceromotor responses associated with emotions (Barrett & Satpute, 2013; Lindquist & Barrett, 2012; Yeo et al., 2011), and to allow memory systems to interact with affect generation (Gupta, Koscik, Bechara, & Tranel, 2011). However, while these regions appear to be involved, none of them should be understood as *specialized* for such appraisal functions. More generally, as many appraisals represent fairly abstract, conceptual evaluations, it is expected that distributed patterns of cortical activation across many brain regions will be engaged – forming connected webs of the perceptual, motor, and more abstract cognitive representational elements that ground the content of those conceptual evaluations (Kiefer & Barsalou, 2013; Wilson-Mendenhall, Barrett, Simmons, & Barsalou, 2011). As discussed further below, conceptual evaluation is also known to involve the brain's "default" network (Barrett & Satpute, 2013) (see Figure 2).

To then generate an affective response, the neural states that represent appraisals are expected to interact with multiple subcortical structures. This includes the amygdala, basal forebrain, hypothalamic nuclei, midbrain nuclei, and brainstem nuclei. These structures exhibit a pattern of connectivity with the autonomic nervous system, skeletomotor system, endocrine system, immune system, and with cortex – which allows them to simultaneously trigger broad changes in the state of brain and body (Pessoa, 2013, Ch. 9). This includes the changes in muscle tension, sympathetic tone, parasympathetic tone, and circulating hormone and cytokine levels that modulate bodily state. It also includes changes in the modulatory influence of several neuromodulators (e.g., dopamine, norepinephrine, serotonin, acetylcholine) over cortical processing, leading to emotion-related changes in motivation, vigilance, attention, memory, cognitive interpretation, decision-making, and so forth (e.g., see Cools, Nakamura, & Daw, 2011; Mather, Clewett, Sakaki, & Harley, 2015; Pessoa, 2013).

After the processes discussed above alter the state of the body, information about that altered state is conveyed back to the brain via afferent autonomic and spinal axonal pathways. When these afferent signals reach the brain, they appear to alter internally represented estimates at various locations and levels of abstraction (Smith & Lane, 2015; Smith, Thayer, et al., 2017). First, many brainstem nuclei appear to alter their activity levels in a manner that tracks changes in specific body state variables (e.g., changes in blood pressure). Via multiple pathways, such subcortical nuclei then convey such information for further cortical processing (Craig, 2002). Multiple networks of cortical regions, including areas of the insula, cingulate cortex, and postcentral gyrus (among others) – often referred to as the "somatomotor" and "ventral attention" (or "salience") networks - appear to subsequently represent more integrated estimates of the updated state of the body (Barrett & Satpute, 2013; Barrett & Simmons, 2015). A distinct network of regions (often referred to as the default network), including regions of the medial prefrontal cortex, cingulate gyrus, medial and lateral temporal lobe (among others), appears to represent higher-level conceptual interpretations of those updated body state estimates – including the application of emotion concept terms (Barrett & Satpute,

2013; Wilson-Mendenhall et al., 2011). Modulatory influences over basal ganglia, prefrontal, cingulate, and motor system regions may also strengthen representations of some potential actions over others – potentially leading to strong felt motivations to act in emotion-congruent ways (Berns & Sejnowski, 1996; Cisek, 2007; Niv, Daw, Joel, & Dayan, 2007; Pereira et al., 2010).

Once these many representations are activated, the selection process discussed above – that determines which representations become consciously accessible and which do not – is believed to draw heavily on another distributed cortical network (often called the "frontoparietal network" or "executive control network"), which includes regions of lateral prefrontal cortex, parietal cortex, and cingulate cortex (among others; see Barrett & Satpute, 2013; Dehaene, Charles, King, & Marti, 2014). When a given representational neural state is strengthened sufficiently to win the competition for selection, it is believed that a top-down signal emanating from the frontoparietal network is engaged (Dehaene, Changeux, Naccache, Sackur, & Sergent, 2006). This top-down signal both 1) amplifies/maintains the strength of the selected representation, allowing its content signal to be "globally broadcast" throughout the brain (and therefore become accessible to a broad range of neurocognitive systems), and 2) suppresses the strength of competing representations that would interfere with processing and/or which are goal-irrelevant. Any represented aspect of an affective response will only be consciously experienced if it is globally broadcast as a result of selective amplification by this top-down signal (Panksepp, Lane, Solms, & Smith, 2017).

Alexithymia

The first author to describe the clinically relevant phenomenon of alexithymia, Jürgen Ruesch, named it "infantile personality" and characterized it as follows (Ruesch, 1948): "In the psychoneuroses, we deal with a pathological development, while in psychosomatic conditions one meets primarily arrested development. Some of these infantile patients have persisted since childhood in expressing themselves in somatic terms." Ruesch spoke of a "somatic language"("They feel with their bodies") and an underrepresentation of symbolic functions such as vivid phantasies and dreams in these patients (Ruesch, 1957). In psychodynamic thinking, this "pensée opératoire" (Marty & de M'uzan, 1963) could mean that negative, especially aggressive, impulses would have "no way out" (de M'uzan, 1977)and could have direct negative effects on biological functions (Fain, 1966). Or, as Paul McLean hypothesized, "emotional feelings, instead of finding expression and discharge in the symbolic use of words and appropriate behavior, might be conceived as being translated into a kind of 'organ language'" (Maclean, 1949; MacLean, 1970).

The subsequently developed and related constructs of alexithymia, affective agnosia, and low emotional awareness (EA) attempt to capture this initial observation that some individuals appear to have limitations to varying degrees in their ability to generate, represent, and/or consciously experience various aspects of emotion (Lane, Weihs, Herring, Hishaw, & Smith, 2015). The theoretical construct

of affective agnosia has since been more thoroughly developed (for recent reviews and discussion, see (Lane & Smith, 2021; Lane et al., 2020; Lane et al., 2021)) and links have been proposed to chronic pain and other biopsychosocial processes (Lane et al., 2018; Smith, Gudleski, et al., 2020; Smith, Weihs, et al., 2019). Individuals with such limitations – which going forward we will refer to as having low EA – appear to be more at risk for various other physical and emotional health issues (i.e., in continuity with the first clinical impressions described above)(Kojima, 2012; Smith & Lane, 2016; Taylor, Bagby, & Parker, 1997). There is a history of intense discussions about whether low EA (or alexithymic traits) may be more due to personality development or (repetitive) traumatic experiences (e.g., Steffen, Fiess, Schmidt, & Rockstroh, 2015). Current knowledge also underscores the huge variety of, and at least partly unknown, contributing factors that may lead to the clinical phenomenon of alexithymia, including its more severe expression in affective agnosia.

A better understanding of low EA at the neural process level might therefore represent an important step in designing interventions to alleviate such risks. More generally, low EA represents one emotion-related individual difference variable that a successful neural model of emotions should be capable of accounting for. Toward this end, recent neurocomputational models of emotion concept learning and emotion-focused working memory have demonstrated multiple mechanisms in quantitative simulations that could explain various causes of low EA (Smith, Lane, et al., 2019; Smith, Parr, et al., 2019). This includes, for example, strong expectations that affective sensations indicate physical health concerns, poor emotion concept learning from impoverished early environments, prior expectations that information about emotions is unreliable or that emotional states are highly volatile, stress-induced reductions in working memory capacity, and reinforced patterns of attention that avoid cues to emotional information (e.g., because such information is considered of low value/relevance). Each of these mechanisms can in turn be linked to particular clinical assessments, measures, and therapeutic interventions (see Table 1 in (Smith, Lane, et al., 2019)).

In considering the neural processes discussed above, it appears that low EA could result from any combination of the three factors (i.e., possibly also resulting in different clinical manifestations). First, low EA could arise from individual differences in the affect generation process – where few emotions are experienced because few affective responses are generated. This might stem from individual differences in subcortical circuitry; alternatively it might stem from impoverished appraisals (e.g., overly "black and white" thinking), such that a low number of distinct affective responses could follow from a low number of the distinct appraisal that would initiate them. Second, low EA could arise from individual differences in the affect representation process – where few emotions are experienced because affective responses are categorized/represented in an impoverished manner. For example, if all unpleasant affective responses are represented as belonging to a single category (e.g., "bad"), then reported experiences may fail to discriminate between different unpleasant emotions (e.g., anger, fear, sadness, disgust, etc.).

Alternatively, affective responses may be inappropriately categorized in non-emotional terms (e.g., an intense increase in heart rate might be mistaken as a sign of an impending heart attack). Third, low EA could arise from individual differences in conscious access – where few emotions are experienced because representations of an affective response rarely win the competition for global broadcasting. This might occur, for example, if an individual has not learned to value or attend to emotion across a wide range of contexts.

Currently, studies of the neural basis of low EA (based on performance measures) provide the most support for the second possibility discussed above – that low EA follows from individual differences in affect representation processes (Lane et al., 2015). For example, two different task-based functional neuroimaging studies have demonstrated that lower EA scores are associated with lower activity in the dorsal anterior cingulate cortex (Lane et al., 1998; McRae, Reiman, Fort, Chen, & Lane, 2008), suggesting a link between EA and either the representation or use of body state information. Another study has also shown that, during recall of life-threatening experiences, lower EA scores were associated with lower activity in medial prefrontal cortex and rostral anterior cingulate (Frewen et al., 2008); as these regions are part of the default network, this suggests a link between EA and concept representation. More recent neuroimaging studies also bolster this conclusion by showing greater functional connectivity between default network regions in those with high EA (Smith, Alkozei, et al., 2017; Smith, Sanova, et al., 2018), and that individuals with higher EA show greater medial prefrontal activation when holding emotions vs. bodily sensations in working memory (Smith, Lane, Sanova, et al., 2018); also see (Smith, Lane, Alkozei, et al., 2018).
, Other studies point to the relevance of the first and third processes. A recent study found that lower EA predicted lower lateral prefrontal and insula activation during an emotion-focused working memory task, which suggests that differences in top-down amplification/maintenance processes (associated with global broadcasting and conscious access) may also be involved (Smith et al., 2017). This is also consistent with recent theoretical and empirical work linking EA to domain-general reflective cognitive processes (Smith, Persich, et al., 2022; Smith, Steklis, et al., 2022; Smith, Steklis, et al., 2020). It is also worth highlighting that each of the cortical regions linked to EA discussed above could also contribute to cognitive appraisal processes, and therefore play a role in the affect generation process as well. Consistent with this, one study found that individuals with higher EA showed greater cortical thickness in ventral prefrontal regions linked to visceromotor control and autonomic response generation (Smith, Bajaj, et al., 2018), while an in-depth case study of a woman with affective agnosia found absent or abnormal peripheral physiological responses to emotion-provoking images (Smith, Kaszniak, et al., 2019). Both of these studies therefore link low EA to abnormal affective response generation.

Based on these findings, the three-process framework, and the findings from our neurocomputational models, it has been proposed that alexithymia is best conceptualized as a phenotype that can arise from any combination of deficits

across the processes described above. As such, it was recently suggested that we have now entered a new, third era of alexithymia research in which the alexithymia phenotype is understood to arise from brain-body interactions mediating impairments in these processes (Lane, 2020). According to this perspective the first era began in 1948 with Ruesch's initial description and ended in 1976 when a consensus definition of alexithymia was reached at a conference in Heidelberg. The second era spanned 1976 to the present during which alexithymia was defined and measured by instruments such as the 20-item Toronto Alexithymia Scale, which were based on the Heidelberg definition. Just as edema is a phenotype with many different etiologies requiring different therapeutic interventions, the same may be true for alexithymia as a spectrum disorder with variations in etiology and severity. An implication of this perspective is that clinicians are encouraged to separately evaluate these different processes in individual patients thought to have alexithymia and adjust their interventions accordingly (see Table 1 in (Smith, Lane, et al., 2019)).

Conclusion

In summary, current progress on understanding the neurobiology of emotions suggests that cortical-subcortical interactions – associated with affect generation – allow appraisals of cognitive and perceptual representations (e.g., percepts, thoughts, memories, etc.) to trigger changes in the state of both the body and other cognitive/behavioral control systems. Afferent feedback from the body, in combination with the influence of expectations derived from previous experience, then leads to changes in both subcortical and cortical representations (across many levels of description) of the current state of the organism. At the cortical level, resulting representations of body states, desired actions, emotion concepts, and other aspects of an affective response then compete with other represented information for selection to become consciously accessible. This process model has the resources to account for many aspects of emotion in multiple ways, including individual differences in emotional awareness. Alexithymia may therefore constitute a phenotype that results from any combination of deficits across the processes underlying affect generation, affective response representation, and conscious access. A considerable amount of research is still required, however, to provide a detailed characterization of the role played by each of the processes described above in accounting for such individual differences in actual cases. Such research offers the promise to aid in the development of interventions that could improve awareness of emotions and potentially lead to better physical and emotional health outcomes.

References

Barrett, L., Mesquita, B., & Gendron, M. (2011). Context in Emotion Perception. *Current Directions in Psychological Science*, *20*(5), 286–290. http://doi.org/10.1177/0963721411422522

Barrett, L., & Satpute, A. (2013). Large-scale brain networks in affective and social

neuroscience: towards an integrative functional architecture of the brain. *Current Opinion in Neurobiology*, *23*(3), 361–72. http://doi.org/10.1016/j.conb.2012.12.012

Barrett, L., & Simmons, W. (2015). Interoceptive predictions in the brain. *Nature Reviews. Neuroscience*, *16*(7), 419–29. http://doi.org/10.1038/nrn3950

Barsalou, L. (2009). Simulation, situated conceptualization, and prediction. *Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences*, *364*(1521), 1281–9. http://doi.org/10.1098/rstb.2008.0319

Berns, G., & Sejnowski, T. (1996). How the Basal Ganglia Makes Decisions. In A. Damasio, H. Damasio, & Y. Christen (Eds.), *Neurobiology of Decision-Making* (pp. 101–113). Berlin, Heidelberg: Springer Berlin Heidelberg. http://doi.org/10.1007/978-3-642-79928-0

Brosch, T., & Sander, D. (2013). The Appraising Brain: Towards a Neuro-Cognitive Model of Appraisal Processes in Emotion. *Emotion Review*, *5*(2), 163–168. http://doi.org/10.1177/1754073912468298

Cisek, P. (2007). Cortical mechanisms of action selection: the affordance competition hypothesis. *Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences*, *362*(1485), 1585–99. http://doi.org/10.1098/rstb.2007.2054

Cools, R., Nakamura, K., & Daw, N. (2011). Serotonin and dopamine: unifying affective, activational, and decision functions. *Neuropsychopharmacology*, *36*(1), 98–113. http://doi.org/10.1038/npp.2010.121

Craig, A. D. (2002). How do you feel? Interoception: the sense of the physiological condition of the body. *Nature Reviews. Neuroscience*, *3*(8), 655–666.

Danker, J., & Anderson, J. (2010). The ghosts of brain states past: remembering reactivates the brain regions engaged during encoding. *Psychological Bulletin*, *136*(1), 87–102. http://doi.org/10.1037/a0017937

de M'uzan, M. (1977). Zur Psychologie der psychosomatisch Kranken. *Psyche*, *31*(4), 318–332.

Dehaene, S. (2014). *Consciousness and the Brain*. Viking Press.

Dehaene, S., Changeux, J., Naccache, L., Sackur, J., & Sergent, C. (2006). Conscious, preconscious, and subliminal processing: a testable taxonomy. *Trends in Cognitive Sciences*, *10*(5), 204–11. http://doi.org/10.1016/j.tics.2006.03.007

Dehaene, S., Charles, L., King, J.-R., & Marti, S. (2014). Toward a computational theory of conscious processing. *Current Opinion in Neurobiology*, *25*, 76–84. http://doi.org/10.1016/j.conb.2013.12.005

Fain, M. (1966). Regression and psychosomatic medicine. *Revue Francaise de Psychanalyse*, *30*(4), 451–456.

Frewen, P., Lane, R., Neufeld, R., Densmore, M., Stevens, T., & Lanius, R. (2008). Neural correlates of levels of emotional awareness during trauma script-imagery in posttraumatic stress disorder. *Psychosomatic Medicine*, *70*(1), 27–31.

Friedman, B. (2010). Feelings and the body: the Jamesian perspective on autonomic specificity of emotion. *Biological Psychology*, *84*(3), 383–93. http://doi.org/10.1016/j.biopsycho.2009.10.006

Gazzaniga, M., Ivry, R., & Mangun, G. (2014). *Cognitive Neuroscience: The Biology of*

the Mind (Fourth Edi). NY: W. W. Norton & Company, Inc.
Gupta, R., Koscik, T., Bechara, A., & Tranel, D. (2011). The amygdala and decision-making. *Neuropsychologia*, *49*(4), 760–6. http://doi.org/10.1016/j.neuropsychologia.2010.09.029
Kano, M., & Fukudo, S. (2013). The alexithymic brain: the neural pathways linking alexithymia to physical disorders. *Biopsychosocial Medicine*, *71*(1), 1.
Khalsa, S., Rudrauf, D., Feinstein, J., & Tranel, D. (2009). The pathways of interoceptive awareness. *Nature Neuroscience*, *12*(12), 1494–6. http://doi.org/10.1038/nn.2411
Kiefer, M., & Barsalou, L. (2013). Grounding the human conceptual system in perception, action, and internal states. In W. Prinz, M. Beisert, & A. Herwig (Eds.), *Action science: Foundations of an emerging discipline* (pp. 381–407). Cambridge, MA: MIT Press.
Kojima, M. (2012). Alexithymia as a prognostic risk factor for health problems: a brief review of epidemiological studies. *Biopsychosocial Medicine*, *6*(1), 21.
Kreibig, S. (2010). Autonomic nervous system activity in emotion: a review. *Biological Psychology*, *84*(3), 394–421. http://doi.org/10.1016/j.biopsycho.2010.03.010
Lane, R., Reiman, E., Axelrod, B., Yun, L., Holmes, A., & Schwartz, G. (1998). Neural correlates of levels of emotional awareness. Evidence of an interaction between emotion and attention in the anterior cingulate cortex. *Journal of Cognitive Neuroscience*, *10*(4), 525–535.
Lane, R., Weihs, K., Herring, A., Hishaw, A., & Smith, R. (2015). Affective agnosia: Expansion of the alexithymia construct and a new opportunity to integrate and extend Freud's legacy. *Neuroscience and Biobehavioral Reviews*, *55*, 594–611. http://doi.org/10.1016/j.neubiorev.2015.06.007
Lindquist, K., & Barrett, L. (2012). A functional architecture of the human brain: emerging insights from the science of emotion. *Trends in Cognitive Sciences*, *16*(11), 533–540. http://doi.org/10.1016/j.tics.2012.09.005
Maclean, P. (1949). Psychosomatic disease and the visceral brain; recent developments bearing on the Papez theory of emotion. *Psychosomatic Medicine*, *11*(6), 338–353.
MacLean, P. (1970). The Triune Brain, Emotion, and Scientific Bias. In F. Schmitt (Ed.), *The Neurosciences: Second study program* (pp. 336–349). New York: Rockefeller University Press.
Marty, P., & de M'uzan, M. (1963). La pensee operatoire. *Revue Francaise Psychoanalyse*, *27*, suppl. 1345.
Mather, M., Clewett, D., Sakaki, M., & Harley, C. (2015). Norepinephrine ignites local hot spots of neuronal excitation: How arousal amplifies selectivity in perception and memory. *Behavioral and Brain Sciences*, 1–100. http://doi.org/10.1017/S0140525X15000667
McRae, K., Reiman, E., Fort, C., Chen, K., & Lane, R. (2008). Association between trait emotional awareness and dorsal anterior cingulate activity during emotion is arousal-dependent. *NeuroImage*, *41*(2), 648–55. http://doi.org/10.1016/j.neuroimage.2008.02.030
Niv, Y., Daw, N., Joel, D., & Dayan, P. (2007). Tonic dopamine: opportunity costs and

the control of response vigor. *Psychopharmacology*, *191*(3), 507–20. http://doi.org/10.1007/s00213-006-0502-4
Panksepp, J., Lane, R., Solms, M., & Smith, R. (2017). Reconciling cognitive and affective neuroscience perspectives on the brain basis of emotional experience. *Neuroscience & Biobehavioral Reviews*, *76*, *Part B*, 187–215. http://doi.org/10.1016/j.neubiorev.2016.09.010
Pereira, M., de Oliveira, L., Erthal, F., Joffily, M., Mocaiber, I., Volchan, E., & Pessoa, L. (2010). Emotion affects action: Midcingulate cortex as a pivotal node of interaction between negative emotion and motor signals. *Cognitive, Affective & Behavioral Neuroscience*, *10*(1), 94–106.
Pessoa, L. (2013). *The Cognitive-Emotional Brain: From Interactions to Integration*. MIT Press.
Pobric, G., Jefferies, E., & Lambon Ralph, M. (2010). Category-specific versus category-general semantic impairment induced by transcranial magnetic stimulation. *Current Biology*, *20*(10), 964–8. http://doi.org/10.1016/j.cub.2010.03.070
Reisenzein, R. (1983). The Schachter theory of emotion: Two decades later. *Psychological Bulletin*, *94*(2), 239–264.
Ruesch, J. (1948). The infantile personality. *Psychosomatic Medicine*, *10*, 134–144.
Ruesch, J. (1957). Principles of human communication. *Dialectica*, *11*(1–2), 154–166.
Scherer, K. (2009). The dynamic architecture of emotion: Evidence for the component process model. *Cognition & Emotion*, *23*(7), 1307–1351. http://doi.org/10.1080/02699930902928969
Shiota, M., & Kalat, J. (2012). *Emotion* (2nd ed.). Cengage Learning.
Smith, R., Killgore, W., & Lane, R. (2018). The structure of emotional experience and its relation to trait emotional awareness: a theoretical review. *Emotion*.. 18(5), 670-692.Smith, R., & Lane, R. (2015). The neural basis of one's own conscious and unconscious emotional states. *Neuroscience & Biobehavioral Reviews*, *57*, 1–29. http://doi.org/10.1016/j.neubiorev.2015.08.003
Smith, R., & Lane, R. (2016). Unconscious emotion: A cognitive neuroscientific perspective. *Neuroscience and Biobehavioral Reviews*, *69*, 216–238. http://doi.org/10.1016/j.neubiorev.2016.08.013
Smith, R., Lane, R., Alkozei, A., Bao, J., Smith, C., Sanova, A., … Killgore, W. (2017). Maintaining the feelings of others in working memory is associated with activation of the left anterior insula and left frontal-parietal control network. *Social Cognitive and Affective Neuroscience*, *12*(5), 848–860. http://doi.org/10.1093/scan/nsx011
Steffen, A., Fiess, J., Schmidt, R., & Rockstroh, B. (2015). "That pulled the rug out from under my feet!" - adverse experiences and altered emotion processing in patients with functional neurological symptoms compared to healthy comparison subjects. *BMC Psychiatry*, *15*, 133. http://doi.org/10.1186/s12888-015-0514-x
Taylor, G., Bagby, R., & Parker, J. (1997). *Disorders of affect regulation: alexithymia in medical and psychiatric illness*. Cambridge: Cambridge University Press.
van der Velde, J., Gromann, P., Swart, M., Wiersma, D., de Haan, L., Bruggeman, R., … Aleman, A. (2015). Alexithymia influences brain activation during emotion

perception but not regulation. *Social Cognitive and Affective Neuroscience*, *10*(2), 285–293. http://doi.org/10.1093/scan/nsu056
Van der Velde, J., Servaas, M., Goerlich, K., Bruggeman, R., Horton, P., Costafreda, S., & Aleman, A. (2013). Neural correlates of alexithymia: A meta-analysis of emotion processing studies. *Neuroscience & Biobehavioral Reviews*, *37*(8), 1774–1785.
Wilson-Mendenhall, C., Barrett, L., Simmons, W., & Barsalou, L. (2011). Grounding emotion in situated conceptualization. *Neuropsychologia*, *49*(5), 1105–1127. http://doi.org/10.1016/j.neuropsychologia.2010.12.032
Yeo, B., Krienen, F., Sepulcre, J., Sabuncu, M., Lashkari, D., Hollinshead, M., … Buckner, R. (2011). The organization of the human cerebral cortex estimated by intrinsic functional connectivity. *Journal of Neurophysiology*, *106*(3), 1125–65. http://doi.org/10.1152/jn.00338.2011

NEW REFERENCES TO ADD

Lane, R. D. (2020). Alexithymia 3.0: Reimagining alexithymia from a medical perspective. *BioPsychoSocial Medicine*, *14*(1), 1-8.
Lane, R. D., Anderson, F. S., & Smith, R. (2018). Biased competition favoring physical over emotional pain: A possible explanation for the link between early adversity and chronic pain. *Psychosom Med*, *80*, 880-890.
Lane, R. D., & Smith, R. (2021). Levels of Emotional Awareness: Theory and Measurement of a Socio-Emotional Skill. *J Intell*, *9*(3). https://doi.org/10.3390/jintelligence9030042
Lane, R. D., Solms, M., Weihs, K. L., Hishaw, A., & Smith, R. (2020). Affective agnosia: a core affective processing deficit in the alexithymia spectrum. *BioPsychoSocial Medicine*, *14*(1), 20. https://doi.org/10.1186/s13030-020-00184-w
Lane, R. D., Solms, M., Weihs, K. L., Hishaw, A., & Smith, R. (2021). Is the concept of affective agnosia a useful addition to the alexithymia literature? *Neurosci Biobehav Rev*, *127*, 747-748. https://doi.org/10.1016/j.neubiorev.2021.05.012
Smith, R. (2020). The three-process model of implicit and explicit emotion. In R. Lane & L. Nadel (Eds.), *Neuroscience of Enduring Change: Implications for Psychotherapy*. Oxford University Press.
Smith, R., Alkozei, A., Bao, J., Smith, C., Lane, R. D., & Killgore, W. D. S. (2017). Resting state functional connectivity correlates of emotional awareness. *Neuroimage*, *159*, 99-106. https://doi.org/10.1016/j.neuroimage.2017.07.044
Smith, R., Bajaj, S., Dailey, N. S., Alkozei, A., Smith, C., Sanova, A., . . . Killgore, W. D. S. (2018). Greater cortical thickness within the limbic visceromotor network predicts higher levels of trait emotional awareness. *Conscious Cogn*, *57*, 54-61. https://doi.org/10.1016/j.concog.2017.11.004
Smith, R., Gudleski, G. D., Lane, R. D., & Lackner, J. M. (2020). Higher Emotional Awareness Is Associated With Reduced Pain in Irritable Bowel Syndrome Patients: Preliminary Results. *Psychol Rep*, *123*(6), 2227-2247. https://doi.org/10.1177/0033294119868778
Smith, R., Kaszniak, A. W., Katsanis, J., Lane, R. D., & Nielsen, L. (2019). The importance of identifying underlying process abnormalities in alexithymia:

Implications of the three-process model and a single case study illustration. *Conscious Cogn*, *68*, 33-46. https://doi.org/10.1016/j.concog.2018.12.004
Smith, R., Lane, R. D., Alkozei, A., Bao, J., Smith, C., Sanova, A., . . . Killgore, W. D. S. (2018). The role of medial prefrontal cortex in the working memory maintenance of one's own emotional responses. *Sci Rep*, *8*(1), 3460. https://doi.org/10.1038/s41598-018-21896-8
Smith, R., Lane, R. D., Parr, T., & Friston, K. J. (2019). Neurocomputational mechanisms underlying emotional awareness: Insights afforded by deep active inference and their potential clinical relevance. *Neuroscience & Biobehavioral Reviews*, *107*, 473-491. https://doi.org/10.1016/j.neubiorev.2019.09.002
Smith, R., Lane, R. D., Sanova, A., Alkozei, A., Smith, C., & Killgore, W. D. S. (2018). Common and Unique Neural Systems Underlying the Working Memory Maintenance of Emotional vs. Bodily Reactions to Affective Stimuli: The Moderating Role of Trait Emotional Awareness. *Front Hum Neurosci*, *12*, 370. https://doi.org/10.3389/fnhum.2018.00370
Smith, R., Parr, T., & Friston, K. J. (2019). Simulating Emotions: An Active Inference Model of Emotional State Inference and Emotion Concept Learning. *Frontiers in psychology*, *10*, 2844. https://doi.org/10.3389/fpsyg.2019.02844
Smith, R., Persich, M., Lane, R. D., & Killgore, W. D. S. (2022). Higher emotional awareness is associated with greater domain-general reflective tendencies. *Scientific Reports*, *12*, 3123.
Smith, R., Sanova, A., Alkozei, A., Lane, R. D., & Killgore, W. D. S. (2018). Higher levels of trait emotional awareness are associated with more efficient global information integration throughout the brain: a graph-theoretic analysis of resting state functional connectivity. *Soc Cogn Affect Neurosci*, *13*(7), 665-675. https://doi.org/10.1093/scan/nsy047
Smith, R., Steklis, H. D., Steklis, N., Weihs, K., Allen, J. J. B., & Lane, R. D. (2022). Lower emotional awareness is associated with greater early adversity and faster life history strategy. *Evolutionary Behavioral Sciences*, ebs0000282. https://doi.org/https://doi.org/10.1037/ebs0000282
Smith, R., Steklis, H. D., Steklis, N. G., Weihs, K. L., & Lane, R. D. (2020). The evolution and development of the uniquely human capacity for emotional awareness: A synthesis of comparative anatomical, cognitive, neurocomputational, and evolutionary psychological perspectives. *Biological psychology*, *154*, 107925. https://doi.org/10.1016/j.biopsycho.2020.107925
Smith, R., Thayer, J. F., Khalsa, S. S., & Lane, R. D. (2017). The hierarchical basis of neurovisceral integration. *Neurosci Biobehav Rev*, *75*, 274-296. https://doi.org/10.1016/j.neubiorev.2017.02.003
Smith, R., Weihs, K. L., Alkozei, A., Killgore, W. D. S., & Lane, R. D. (2019). An Embodied Neurocomputational Framework for Organically Integrating Biopsychosocial Processes: An Application to the Role of Social Support in Health and Disease. *Psychosomatic medicine*, *81*(2), 125-145. https://doi.org/10.1097/PSY.0000000000000661

Figure Legends

Figure 1. (A) Illustrates the various aspects of the affect generation process discussed in the text. (B) Illustrates the various represented aspects of an affective response, as well as routes by which they may consciously or unconsciously influence action selection. Dashed lines indicate connections that are only effective if a representation is selected for becoming consciously accessible.

Figure 2. Illustrates different large-scale neural networks (based on Yeo et al., 2011), and how different emotion related processes may reflect interactions between different network functions. (A) Illustrates affect generation processes, whereas (B) illustrates affect representation processes and the competition for conscious access. Dashed black lines indicate connections that are only effective if a representation is selected for becoming consciously accessible.