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A hybrid cognitive architecture with primal affect and physiology

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Abstract—Though computational cognitive architectures have been used to study several processes associated with human behavior, the study of integration of affect and emotion in these processes has been relatively sparse. Theory from affective science and affective neuroscience can be used to systematically integrate affect into cognitive architectures, particularly in areas where cognitive system behavior is known to be associated with physiological structure and behavior. I introduce a unified theory and model of human behavior that integrates physiology and primal affect with cognitive processes in a cognitive architecture. This new architecture gives a more tractable, mechanistic way to simulate affect-cognition interactions to provide specific, quantitative predictions. It considers affect as a lower-level, functional process that interacts with cognitive processes (e.g., declarative memory) to result in emotional behavior. This formulation makes it more straightforward to connect these affective representations with other related moderating processes that may not specifically be considered as emotional (e.g., thirst or stress). An improved understanding of the architecture that constrains our behavior gives us a better opportunity to comprehend why we behave the way we do and how we can use this knowledge to recognize and construct a more ideal internal and external environment.

Index Terms— Cognitive Models, Modeling human emotion, Mood or core affect, Emotion theory

INTRODUCTION

The human mind is a complex biological system that operates as a computational system to behave within its environment. Given this (immense) complexity, it can be useful to breakdown the mind into hierarchies to develop models and simulate behavior of the human mind and human behavior (e.g., [1]). Indeed, this idea has been used to begin to develop integrative models of human physiology relevant for understanding and predicting behavior of several aspects of physiology [2]. Hierarchy is a useful concept when discussing a very difficult aspect of modeling human behavior, computational models of emotion.

Several computational models of emotion or affect have been proposed and developed into systems that can be run through simulations (e.g., see [3, 4] for a useful overview). Progress in developing unified computational models and systems that integrate theory in affect and emotion remains slow relative to the overall activity in unified computational models of human behavior. Nonetheless, there have been useful developments of computational models of emotion that provide unified computational accounts of human behavior [5-9].

Marsella and Gratch [5] and Marinier III, et al. [8] implement computational models of appraisal processes [10] and their effects of cognition by implementing this appraisal process within versions of the Soar cognitive architecture [11]. MicroPsi [9] provides a useful hierarchy of urges (physiological, cognitive, and social) that modulate behavior of the system, albeit in way that is more concerned with intelligent *agent* behavior, than particularly intelligent *human* behavior. Clarion [7] uses lower-level (e.g., related to eating) and higher-level (e.g., relating to *belonging* to a group) drives to approximate emotions. More recently, Juvina, et al. [6] use *core affect* [12] to break down emotion into *valence* and *arousal* and have these modulate cognitive processes.

All these computational systems use hierarchies to model and simulate interactions between emotion and cognitive systems, albeit with different representations and components. It remains less clear how these systems may handle the interaction between emotional processes and other cognitive moderators [13] that are not emotional, but nonetheless may influence affective and cognitive processes. It may be difficult to develop models that can realistically and tractably combine moderators (including emotion) and simulate their effects on behavior (e.g., the combination of being tired, caffeinated, and in a fearful state).

Panksepp [14] offers a useful hierarchy that can begin to approach the previously mentioned problem and can be used to separate emotional experience into increasingly complex levels. They postulate a continuum of processes (Fig. 1) to represent the processing that mediates human thinking and behavior and use the organization of neural systems as a basis for this formulation.

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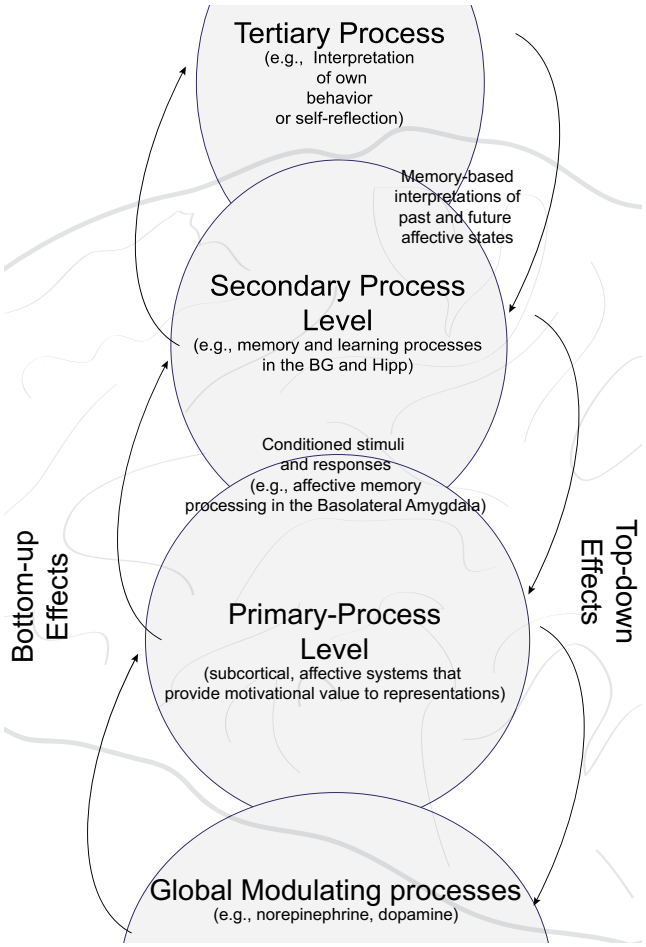

Fig. 1. Panksepp [16] describes levels of processing that mediate behavior. This spans from the more general global modulating processing to the metacognitive processes used to internally reflect upon experience.

Thus, it is useful to think about emotion as the intersection between these levels. Put another way, emotion is the result of feeling some combination of affect and having that affect interact with cognitive processes. This allows us to separate subjective reports of emotional experience, from affective experience that can be inferred from physiological or behavioral change but are nonetheless inaccessible to awareness or unconscious (e.g., [15-17]).

Panksepp and Biven [18] posit several systems that are on the primary-process level and are implemented by neural circuits that cause affect. Most important for this paper are the SEEKING and FEAR systems that mediate appetitive motivations (e.g., those that are activated by hunger) and some response to aversive stimuli (e.g., a powerful shock), respectively. Though aspects of the FEAR system and circuit have been the most dominantly studied, the SEEKING system may be considered as, if not more, fundamental to human behavior.

I use the primary-process affect theory to connect a unified theory of cognition to an integrated model of physiology. I have implemented this multi-level model in a hybrid cognitive architecture that can be used to simulate and predict interactions between physiological, affective, and cognitive processes and how they mediate behavior. This formulation gives us the opportunity to understand a wide range of behavioral moderators and representations that can be used to tractably understand how multiple moderators (affective or otherwise) may interact to affect behavior.

In the next sections, I provide an overview of some connections between physiological and affective systems, and how these interactions can affect cognitive processes. I then describe a hybrid cognitive architecture with physiology and affect and an affective-cognitive agent that runs within the architecture. Lastly, I discuss limitations and potential future work related to the architecture.

PHYSIOLOGICAL-AFFECTIVE SYSTEMS

Nonlinear physiological processes interact with affective and cognitive processes across time. To develop a realistic, tractable computational theory of emotion that can be applied to real-world situations we often encounter, it is important to directly consider the underlying physiological processes that affect behavior both in the face of and in the absence of affective stimuli.

In the proceeding sections, I discuss two areas of physiological-affective systems that are important to studying human behavior in realistic contexts and thus important for any computational model of human behavior that seeks to develop a comprehensive account for behavior across time in varying contexts. First, I discuss the physiological bases for hunger and thirst, and their interactions with existing affective systems. I also discuss sleep, stress, and arousal from an integrative physiological systems perspective; when taken from this perspective, it becomes clearer how these concepts can all be related, especially as it pertains to developing systematic, integrative computational models of emotion. Lastly, I discuss some of the many interactions these physiological systems have with memory.

Hunger and thirst

Hunger and thirst describe basic physiological-affective processes that govern our behavior, potentially in subtle ways (e.g., [19]). Changes in homeostatic physiological processes modulate peripheral and central systems to create certain SEEKING behavior (or *wanting* behavior, [20]), where behavioral tendencies begin to reflect the homeostatic need of particular physiological systems. (Because of the similarities between the theories, I will use *SEEKING/wanting* when discussing this system.) To understand and quantify how physiological-affective systems may interact to modulate human behavior, it is important to gain an understanding of processes that mediate these systems. Hunger and thirst both represent particularly useful homeostatic processes as they've been extensively studied and heavily involve hypothalamic nuclei. This known connection with neural substrates gives one a more straightforward way to connect existing models in this area with work done with affective (and other behavioral) neural systems.

Hunger behavior involves several peripheral regulators, including leptin [21], ghrelin [22], blood glucose [23], and insulin [24] which cause changes in bodily need states and thus changes to hunger-related appetitive and consummatory behavior [25]. Though these changes sometimes manifest themselves as overt behavior, they can also be more covert throughout the day, potentially biasing affective and cognitive processes to behaviors that encourage (or discourage) behaviors related to food cues (e.g., [26]). This bias of processes is facilitated through the hypothalamus, more specifically through the lateral and ventromedial hypothalamic nuclei (LH and VMH, respectively). The LH acts as an excitatory neural regulator, while the VMH is important for inhibition of hunger [25]. Thus, these densely connected nuclei act as a *middle-layer* between peripheral changes indicating bodily need in the form of hunger, and affective processes that modulate behavior.

Thirst and hunger are linked in many ways including salt appetite, that is, salt appetite and thirst typically have an inverse relationship. This is because homeostasis related to thirst (fluid homeostasis) involves mechanisms that maintain a balance between sodium and water balance. As with hunger, behavioral biases that are generated from homeostatic modulation can often manifest as behavior that is overtly thirst-appetite (e.g., taking a drink of water) or more covert (e.g., causing changes in perception to related stimuli; [27]).

These physiological-affective interactions have consequences for behavior in larger contexts. For example, Danziger, et al. [19] show that hunger may have unintended consequences on something as serious as judicial decisions. The authors showed that the proportion favorable judicial decisions after a food break was approximately 65% while just before the break, the proportion had dropped all the way down to 0%; this is with a beginning proportion of around 65% that steadily declined until the food break was taken. As a way to show general overlap of neural processes that modulate thirst and SEEKING/wanting behavior, Winkielman, et al. [16] studied effects of unconscious affective stimuli (happy or angry faces). They found that those who were unconsciously exposed to happy faces poured and drank more of a sugar beverage. In addition, the authors also found that study participants exposed to happy faces were willing to pay more money for a can of that beverage. For thirsty participants, unconscious stimuli affected water pouring and consuming behavior, as well as the amount participants were willing to pay for the water. In an interesting study (due to its control and objective and subjective thirst), Wright, et al. [28] used a primary motivation and reward (i.e., thirst and water) version of the ultimatum game (which typically uses money as the main reward; [29]). In the study, the participants had a choice that resulted in a tradeoff of rejecting an unfair offer from what they believed to be another participant (a 12.5% distribution of a drink of water for the participant and 87.5% for the *other* participant) versus accepting the offer (and thus getting a drink of water). The authors found that physiological thirst (i.e., osmolarity) failed to make a statistically significant difference on participants' choice. However, *subjective thirst* did affect their choice, with participants who accepted the offer showing a higher subjective thirst that was statistically significant. This shows (in a laboratory setting) that affective processes can modulate higher-level cognitive processes that may balance absolute self-interest and fairness. A person may be more likely to accept unfair offers when those offers are directly related to a physiological need.

Thus, even in cases where a decision or action may not be directly related to a human's hunger or thirst state, such a state may affect the outcome of said decision or choice of said action. When the state and decision are related, there may be an especially pervasive change in behavior. Physiological motivational states can be pervasive and have global consequences on affective and cognitive processes that influence human behavior. It is important to develop models that consider these consequences and how they may affect memory, learning, and decision-making (e.g., [30]). I address how one may represent this affective-cognitive interaction in a computational model and architecture in *Integrating the theory into a computational architecture*.

Sleep, stress, and arousal

Humans are bombarded with stressors that cause physiological (and behavioral) adaptation. Though this adaptation is generally referred to as *stress*, the term stress, itself, is not as useful without specificity of the stressor and the processes that change because of this stressor. Often, stress is characterized by an activation of several systems including the hypothalamic-pituitary adrenal (HPA) axis and the Locus-Coeruleus noradrenergic system [31]. The latter system is a main driver in changes in *arousal* as the system that releases the excitatory neurotransmitter noradrenaline.

Several psychological, physiological, and environmental changes may cause adaptation that lead to stress on the body. One of the common stressors that can lead to pervasive allostatic changes in physiological and cognitive systems is sleep deprivation [32]. Variables associated with the HPA-Axis (e.g., Corticotropin Releasing Hormone [CRH], Adrenocorticotropic Hormone [ACTH], and cortisol) are characterized by a diurnal cycle (i.e., circadian rhythms). This rhythmic release of the hormones is disrupted by sleep deprivation [33], causing physiological and behavioral adaptation to offset these effects. Sleep deprivation and disruption also affects the LC-Noradrenergic system as this system receives input from neural systems important for the sleep-wake cycle, that is, the LC-Noradrenergic system receives inhibitory input from sleep promoting systems [33].

Sleep deprivation is known to cause several behavioral deficits, all of which may be caused by modulation of cognitive processes [34-36]; though see [37] for a counterexample task in which behavioral deficits were not found. Given work linking stress-related physiological variables and memory modulation [38, 39],

and the effects that sleep changes have on these variables, one can begin to construct a computational process model of the effects of sleep changes on cognition. In linking these physiological changes with changes in cognitive processes (through known neural and behavioral modulation) one can develop a more unified process model of sleep-deprivation, stress, and arousal and their effects on behavior. This would be useful for more tractable and nuanced understanding of how these physio-cognitive interactions mediate behavior.

In the next sections I discuss connections between the physiological-affective systems I have discussed thus far, and memory systems. I then describe a computational model that links these physio-affective-cognitive processes and can be integrated into a unified architecture (the implementation of which is discussed later in *Integrating the theory into a computational architecture*).

Physiological-affective systems and memory

Developing a computational understanding of the physiological processes that interact with (and in some cases partially govern) affective and cognitive processes allows one to move beyond a static *noise* representation in a cognitive system. With a computational model, we can trace changes and can begin to understand how physiology may change a cognitive system over-time. Related to the previous sections, I focus on changes to physiology that cause changes in primary affective systems, namely the effect of hunger and thirst on the SEEKING/wanting system, as well as stress-related physiology and effects on memory.

Hunger, Thirst, and Affect

As mentioned previously, primary homeostatic systems (i.e., hunger and thirst here), interact with affective processes to enact behavioral changes that help restore balance of those related systems. It appears these behaviors are related to various brain neural processes that connect the sensing of peripheral homeostatic imbalance with the downstream dopaminergic system that is the primary driver behind the SEEKING/wanting system. Perhaps most important at a high-level is the connections between the lateral hypothalamus (LH) and the ventral tegmental area (VTA), the latter of which is responsible for the widespread release of dopamine, the main neurotransmitter implicated in the SEEKING/wanting system and related affect [18].

These connections are affected by several hormones, with Orexin being one of the more notable mediators in homeostatic-imbalance, affect connections. Orexin is related to several peripheral changes that relay a hunger-based need (e.g., glucose, insulin, and leptin changes; [21]), relay a thirst-based need [40], and it is one of the major excitatory systems involved in sleep onset [33].

Subcortical basal ganglia (BG) structures are important for linking the previously mentioned physiological sensors, the SEEKING/wanting system, and behavioral output. Berridge [20] formulates the incentive salience theory that postulates a separate *wanting* and *liking* system, the former of which is mediated by the discussed dopaminergic system and its effects on the ventral striatum (which is a part of the BG). Berridge [20] notes the importance of the affective (or motivational) effect of this system as it relates to behavior and learning. Thus, though the two systems come from separate theories, the wanting and SEEKING system are functionally similar.

These structures are also important in linking the SEEKING/wanting system with learning, particularly aligning well with reinforcement learning related the dopaminergic system. Incentive salience theory provides an account for *wanting* affect and its effect on reinforcement learning processes, including how wanting and *liking* differ in this learning-behavior process. In addition to effects of SEEKING/wanting system affect on memory related to processing in the BG, this system also modulates declarative memory systems primarily mediated by medial temporal lobe (MTL) system that includes the hippocampus [41, 42]. Dopamine modulates the declarative memory learning process by affecting long-term potentiation (LTP) in the hippocampus, causing more stable memories that are more difficult to forget [41]; this effect on LTP is also relevant to the computational architecture that is discussed in *Integrating the theory into a computational architecture* as the architecture's declarative memory system has a functional account for LTP (see [43] for more discussion on this declarative memory-LTP connection). The modulatory power of novelty on memory elements [44] seems to confirm the importance of affect/motivation in this learning process (e.g., as discussed by [20]) as one may have an intuition that as stimuli become less novel, there would be a decrease in the accompanying positive motivation. Relating to previous discussions, this effect of appetitive affect on declarative memory is seen in tasks as simple as small changes in thirst state resulting in an increased likelihood of retrieving thirst-related declarative memory [27]; this also is likely related to the decision-processing seen in Wright, et al. [28].

Though presented as distinct models, with distinct effects on behavior, these physio-affective-cognitive processes occur with an integrated system. As we continue to accumulate this knowledge, it is useful to pull all of these results together into a unified model (e.g., [45]), ideally into a computational architecture that can be simulated so that we may understand the assumptions of theories and models. Simon [1] noted, "…even when we have correct premises, it may be very difficult to discover what they might imply". In *Integrating the theory into a computational architecture*, I present a computational model that brings together the theory and models previously discussed, and that is implemented into a computational system that can be simulated. This computational model gives an account for how affective processes may interact with cognitive processes to mediate behavior.

Stress and memory

Many studies on stressors focus on particularly negative affective stimuli (stressors) to induce a physiological change in stress-related variables. These negative stimuli often affect portions of what Panksepp and Biven [18] calls the FEAR system; some researchers have changed their wording to emphasize the survival nature of these neural circuits more recently (e.g., [46]).

The amygdala is, perhaps, the most well-known structure in the FEAR system, however nuclei in the central portion of the amygdala (CeA) appear to be the central structure in causing behavioral and affective changes related to stressors [18, 47]; indeed, the amygdala is made up of several areas that are functionally separable [48]. The CeA has connections with structures such as the previously discussed lateral hypothalamus, periaqueductal gray (PAG), and the paraventricular nucleus (PVN), all of which are key structures in physiological, affective, and behavioral change (some of these connections are shown in Fig. 2). The PAG has been associated with the neural processing of several behaviors, perhaps most notably to add to this discussion is its relation to breathing and stress (e.g., [49, 50]), as well as anxiety ([47]). Taken together, these neural structures and corresponding systems operate on the *primary-process* level (i.e., Fig. 1) and interact with global modulating and secondary processes to mediate human behavior.

Stressors associated with conditioned learning of fear, which involve the basolateral and lateral amygdala substructures [46, 51, 52], are often used to induce aversive physiological change. Physiological changes normally seen due to stressors can cause various effects memory, many of which are dependent upon the timing and nature of the stressor [38, 39]. Stress appears to cause a switch in the use of types of memory-systems [38], that is, it modulates the use of declarative memory and procedural memory in behavior when both may be used for a given task. This switch is facilitated with an increase in both glucocorticoids (cortisol) and (neural) norepinephrine, but typically not seen when either of those components are absent [38]. In this formulation, declarative memory facilitates more flexible learning behavior, whereas procedural memory facilitates more constrained behavior. Indeed, this relates to the ties between exploration, exploitation, and locus coeruleus activity: moderate activity in the locus coeruleus (and norepinephrine levels) allows more accurate behavior, whereas higher-than normal activity can result in impulsive behaviors that are more habitual (greater use of procedural memory) and may be less relevant to the goals of the task [53].

This effect on uses of memory and learning systems interacts with timing of the stressor, and thus the physiological variables that mediate the stress response. This importance of timing also applies to more affective memory mediated by the basolateral amygdala [39]. Joëls, et al. [39] gives a useful indication of when and how noradrenergic effects on BLA-mediated affective memory will interact with glucocorticoid effects on the same memory. Schwabe et al. [38] provides a related model that provides the timing from the perspective of (declarative-based) memory performance, learning, and retrieval, Table 1 gives this learning-stress timing formulation. Table 1 predicts that, for example, stress followed by a short break (roughly 1 hour) before a learning session results in a decreased declarative memory performance as does stress directly before retrieval, but after learning the item previously.

Table 1. Timing of physiological change due to stressors interacts with the effects those stressors have on behavior (adapted from figure in [38]). Here, $break_S$ denotes a shorter break time (1 hour), while $break_L$ denotes a longer break (a few hours to a day).

Stress-Behavior order	Effect on memory
stress-break$_S$-learn-break$_L$-retr	worsened
stress-learn-break$_L$-retr	improved
learn-stress-break$_L$-retr	improved
learn-break$_L$-stress-retr	worsened

Thus, stress affects both, learning based on the physiological systems recruited during exposure to the stressor, and timing of that recruitment relative to the learning. Effects on specific memory systems used during behavior are also seen: more procedural dependent memory is used when stressors cause increases in glucocorticoids and norepinephrine (e.g., stress from tasks like the Trier Social Stressor Task or TSST; [54]). This has important implications not only for the point-in-time behavior on a task which may be more suited for a specific type of memory, but also for learning stages related to declarative and procedural memory (e.g., those described by [55]). I describe how these ideas are integrated into a computational model and how this model has been implemented to make a hybrid cognitive architecture.

INTEGRATING THE THEORY INTO A COMPUTATIONAL ARCHITECTURE

Physiological need can cause changes in affective systems, which, in turn, can modulate the likelihood of thinking about memory related to that need; for example, thoughts related to water occur more often when one is thirsty. Conversely, thinking about an item that has been associated with affect (through learning) can cause an increase in related affect, that is, when thinking about a stressful situation one may experience the affect and physiological changes associated with that stressor. The theory, models, and results given in previous sections leads to a coherent computational model, which may describe how some of these physiological, affective, and cognitive processes interact.

With this model, we've focused on the FEAR system and the SEEKING/wanting system. Fig. 2 gives a high-level view of neural systems, as well as their relation to physiological, affective, and cognitive systems; some general connections between affective levels (and lower

levels, for example as indicated in Fig. 1) are also shown in the figure. I use an existing unified theory of cognition (ACT-R, [43, 56]) to frame the cognitive systems listed in the Fig. 2.

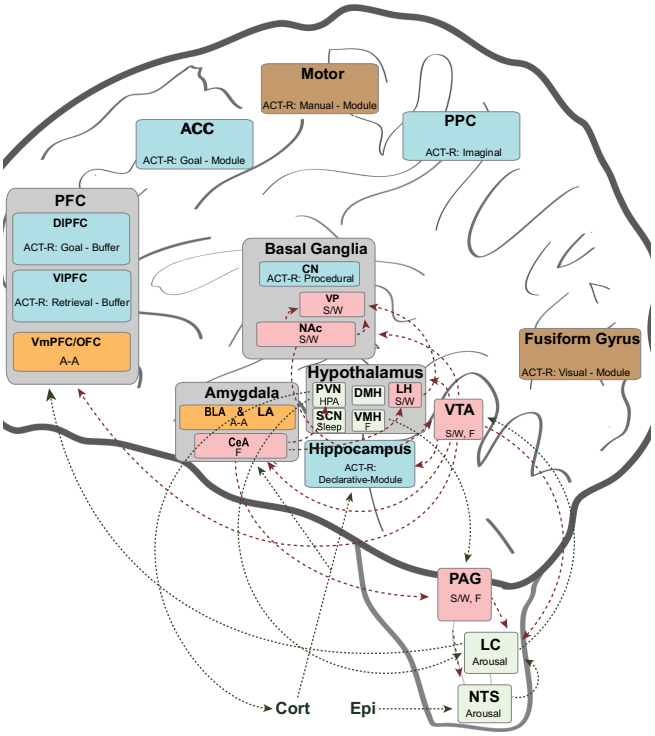

Fig. 2. Functional systems, associated neural structures, and some of the bottom-up connections from the primary-process affect and global/physiological systems. A-A: Affective-Associations; ACC: anterior cingulate cortex; BLA: basolateral amygdala; CeA: central amygdala; CN: caudate nucleus; DlPFC: dorsolateral prefrontal cortex; DMH: dorsomedial hypothalamus; F: FEAR; LA: lateral amygdala; LC: Locus Coeruleus; NAcc: nucleus accumbens; NTS: nucleus tractus solitarus; OFC: orbitofrontal cortex; PAG: periaqueductal gray; PVN: paraventricular nucleus; PPC: posterior parietal cortex; SCN: suprachiasmatic Nucleus; S/W: SEEKING/wanting; VlPFC: ventrolateral prefrontal cortex; VMH - ventromedial hypothalamus; VmPFC - ventromedial prefrontal cortex; VP - ventral pallidum; VTA - ventral tegmental area.

Both the SEEKING/wanting and FEAR system have wide ranging effects on declarative memory and procedural memory systems (e.g., [41]). The many studied effects make these two systems a useful starting point for integrating primary-process affect theory into any cognitive architecture.

Implementing the continuum: a unified computational architecture

To develop this architecture and computational model of emotion, we've connected the HumMod physiological model [57] to the ACT-R cognitive architecture [43], using theory from primary-process affect theory [18] to represent basic affective systems (i.e., at the primary-process level in Fig. 1). These three models all have two advantages for implementation in a computational system: 1) they have a strong theoretical and empirical basis, coming from separate disciplines (which can be useful in providing a fairly diverse perspective); 2) the theories have representations of either neural structures to go along with functional systems (e.g., [56, 58]) or, in the case of HumMod, representations at the levels of organs and hormones. The latter provides an advantage when using data from existing research to verify interactions between functional systems (e.g., we can use existing theory on the effects of stress and arousal on behavior [38, 39, 53]).

ACT-R/Φ

ACT-R/Φ extends the ACT-R architecture with a physiological system and an affective system. The physiological system is composed of the HumMod model of physiology physio module (in the ACT-R system) that communicates between the model of physiology and the other modules in the system (e.g., the physiological components of arousal and their functional effects on the declarative memory module).

The affect system is composed of the SEEKING module, FEAR module, and affective-associations module. The SEEKING and FEAR modules are meant to represent behavioral functionality attributed to the SEEKING and FEAR neural circuits posited by Panksepp and Biven [18]. The affective-associations module is not associated with a specific primary-process affect circuit, and instead represents functionality of systems that operate between the primary and secondary levels specified in Fig. 1. In the next few sections, I discuss the systems, including different equations used. For a summary of the parameters used (and their function) see the appendix.

The physiological system

The physiological system uses the HumMod physiological model to simulate bottom-up physiological modulation of behavior. The physiology module serves as a communication/timing system between the physiological model, affect system, and cognitive system. In addition, it is used to calculate the effects of the HPA-Axis and sympathetic arousal (i.e., epinephrine) on arousal (1).

$$arousal = f(cort) * [\alpha * g(CRH) + \beta * h(epi)] \quad (1)$$

In (1) *f(cort)*, *g(CRH)*, and *h(epi)* represent transformation of raw values of cortisol, corticotrophin releasing hormone (CRH), and epinephrine (respectively). In this case, the functions are simply values normalized according to initial state baseline, that is, each function gives an output of 1 when in a normal state. This representation affects both procedural and declarative memory noise (see Dancy, et al. [59] for related work on simulating the impact of stress on memory during a serial subtraction task). Arousal also modulates the ACT-R production rule firing threshold when below a nominal value.

Though the physiological model has ways to adjust arousal related physiological variables, the canonical model is noticeably missing an account for circadian rhythms and sleep deprivations, as well as cyclic effects

of breathing (i.e., changes in breathing rate and their effects on related physiological receptors, and sympathetic and parasympathetic nervous system arousal). We've added to the physiological model itself by developing extensions for circadian rhythms, sleep, and breathing (see [34, 60] for a more detailed account of these extensions and physio-cognitive models that complete tasks while being modulated by these mechanism). This is useful not only for their effects as more global modulators on cognition (e.g., cortisol effects on arousal and memory), but also to open the architecture to computational representation of interactions between sleep and affect or breathing and affect (e.g., computational modeling sleep or breathing effects on anxiety) and subsequent interactions with cognitive systems.

The SEEKING module

As one may expect, the SEEKING module is based on the SEEKING/wanting system previously discussed. In the architecture, this system and module are key to appetitive motivation and behavior, for example, feeling thirsty and changing one's goals to more readily reflect this affective state of thirst.

The SEEKING module acts as a major interface between procedural memory in ACT-R and homeostatic imbalance. The module equations below draw on work by Zhang et al. [61] to integrate SEEKING/wanting behavior into the existing ACT-R procedural memory system. Equations (3) and (4) show that the utility updated by an affective component that is determined by the current SEEKING system value.

$$U_A = U + \log(k) \tag{3}$$

$$k = (\mathrm{S} + \varepsilon) * e^{reward_{max}} \; s.t. \; S \in [0,1] \tag{4}$$

In (4), k is specified by taking the input value from a specific sensor system (e.g., osmolarity levels/thirst), which I call S; a noise component is also added to S as represented in ε. $reward_{max}$ is the maximum expected reward that is set according to the situation by the agent developer (see [62] for a useful discussion on setting and scaling reward values for cognitive agents that use utility and reinforcement learning). Functionally, these equations allow the agent to change utility values of procedural memory elements (rules) as the affective context changes and environmental (internal or external) needs change. Procedural memory elements also have affective values directly provided by the SEEKING module that are independent of those described in the section on the Affective-Associations module (see (5) for the equation which describes the almost identical FEAR module version of the equation).

Given that it should bias our behavior towards items that relate to certain affective states (especially when, for example, in a physiological state of need like being thirsty or hungry), the SEEKING module should influence goal selection. While I could have developed a special mechanism to adjust goal state directly, without an account for the whole system, the *memory for goals* [63] model provides a much more parsimonious and tractable way to achieve this functionality. When combined with the *offsets* used in the affective-associations module it becomes clearer how the activation bias (which is used to determine one's own goal state) could then begin to affect the goals a cognitive agent would pursue; see the final paragraph of the Affective Associations Module section for an explanation of what I mean here by offsets.

The SEEKING module provides an integrated functional account of the interaction between appetitive motivations and cognitive behavior (in concert with the affective-associations module). Another important system and module is, in some ways, at the opposite end of the spectrum.

The FEAR module

Where the SEEKING module represents approach, appetitive affect related behavior, the FEAR module represents avoid, aversive related behavior. Thus, the FEAR module encapsulates low-level processing of aversive stimuli (e.g., a painful shock) the module can be directly affected by nociceptive or aural stimuli and indirectly affected by visual stimuli. Visual stimuli do not directly change the FEAR module state as the function of the primary neural substrates that are involved in low-level processing of aversive visual stimuli are represented in the *affective-associations* module.

The FEAR module principally operates independent of the learning systems but can be affected by them depending on the affective content in the specific memory elements. As with the SEEKING module, the state of the FEAR module subsymbolically affects the declarative and procedural memory systems. The FEAR module also has direct connections with the physiological system and affects stress system variables (i.e., those implicated in the architectural representation of *arousal*).

Equation (5) is the production updating equation that updates production-affect values.

$$F_i(n) = F_i(n-1) + \alpha(r + \left(\frac{F_{i+1}(n-1)}{1+kt}\right) - F_i(n-1)) \tag{5}$$

$$r = \log(FEAR_{current} * e^{FEAR_{max}}) \tag{6}$$

In (5) the delayed reward parameter k and the learning rate α can be set as a parameter in a model. As with the similar TD-inspired equations discussed in the previous two sections, $1/1 + kt$ is a discount function (e.g.,) that decreases the weight of the new chunk-value pair on the current memory hyperbolically as time between the update of the chunk-value pair and its last update increases. The affective (FEAR) value of the next production from the most recent production trace is represented by $F_{i+1}(n-1)$ and the variable t is the time elapsed since the same rule was last fired. $FEAR_{current}$ (6) is the current state (value) of the FEAR system and $FEAR_{max}$ is a parameter that allows the agent to be calibrated to the expected maximum FEAR values given

an environment; that is, the max expected FEAR affect to be experienced in an environment.

The Affective Associations Module

The affective-associations module contains internal memory systems that link affective states (at a point in time) to visual, auditory, and declarative representations. Fig. 3 gives a high-level view of these systems. I chose to represent separate module-affect systems, focusing on perceptual systems and the declarative memory system (which, itself, can be considered a system to perceive the past, [43]).

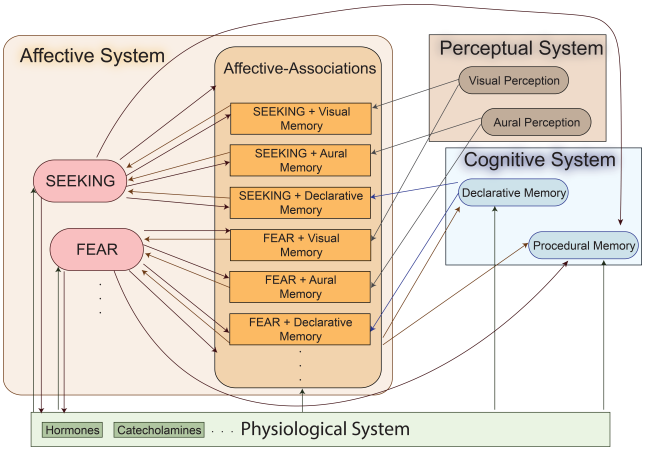

Fig. 3. The affective-associations module provides multiple affect-memory representations and thus gives affective value to memory elements to modulate their processing.

Separate systems were used due to existing evidence for multiple affective memory systems [51]. These internal memory systems can modulate both the underlying affective state due to the context of the cognitive system, and the cognitive state by biasing the cognitive system towards certain behaviors due to the current affective state; these changes operate principally at the subsymbolic level (i.e., values that affect probabilities of using memory representations as opposed to directly changing the symbolic portions of memory representations). Equations (7) and (8) show how affect values are updated for memory representations (I use similar sets of equations for the SEEKING system and related values.).

$$A_i(n) = A_i(n-1) + \alpha\left(r + \left(\frac{F}{1+kt}\right) - A_i(n-1)\right) \quad (7)$$

$$r = \text{argmax}(FEAR_x) \quad (8)$$

$A_i(n-1)$ is the previous affective value (paired with a chunk from a perceptual system or the declarative memory system). The α variable is the learning rate that controls how much each instance of a chunk-value pair affects the overall chunk-value memory of that chunk. r is some primary reinforcer that has the max effect on the FEAR system at the time that the reinforcement process is initiated (e.g., if a loud noise and a painful shock are sensed simultaneously, the higher value produced between the two is used as the reward for r) and F represents the current state of the FEAR system (given by the FEAR module). As previously discussed, $1/1 + kt$ is a discount function used (here) to discount the FEAR value over time.

The Affective-Associations module also modulates declarative memory retrieval by affecting levels of activation for those chunks that are in the corresponding internal memory system. It adds *offsets* to the subsymbolic values of a chunks based on their affect value. Functionally, this makes it so that the more similar the current affect state is to the one when the memory was encoded (given the previously mentioned update equation for multiple encodings), the more that chunk memory element is to be retrieved (i.e., being in a fearful state, biases the cognitive agent to retrieve memories of other times it was afraid).

THE INFLUENCE OF AFFECT ON RESPONSES TO UNFAIRNESS

I used these physio-affect systems to better understand the influence of affect on choice. Particularly, I modeled the ultimatum game (UG), to understand how homeostatic affect may interact with a choice that also involves the influence of alternative goals. The ultimatum game is a task where a *proposer* is given an endowment and must propose a division of that endowment to a second player (the *responder*). The responder may accept the proposal or reject the proposal, the latter of which results in neither participant getting any of the money. Thus, the task involves competing goals. of fairness/reciprocity and maximizing the amount of money one may receive in any given round. I focus on modeling the respondent as there exists both respondent choice behavior data during normal ultimatum game under a variety of proposals [64], but also choice behavior while participants have homeostatic imbalance that characterizes thirst [28].

Modeling the UG with and without Affect

A high-level view of the cognitive process model is given in Fig. 4. The model processes the *offer* and *keep* (the amount proposer offers and the amount the proposer will keep if the offer is accepted, respectively). The model then uses either declarative memory (past experience) or the imaginal system (transformation of the offer-keep into a representation of fairness) to determine whether it is fair. It then retrieves a decision and reports either accept or reject. The declarative memory subsymbolic process (i.e., selection based on activation) is affected by both the base-level activation (which is biased by both recency and frequency) and spreading activation (which is biased by related chunks that may be in other buffers.

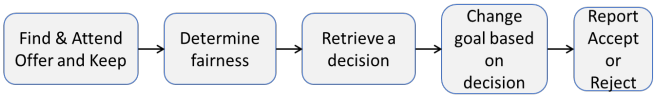

Fig. 4. A high-level diagram of the information processing in the cognitive model

I used MindModeling@Home [65] to run simulations and find the parameter set that produced model behavior closest to human data (see the appendix for more information on the simulations). After finding the best parameter combinations, I ran the model with the highest performing set of parameters with the affective system turned on and while varying only the $reward_{max}$ (i.e., from equation (4)). I used the physiological system in the architecture to simulate hypertonic saline infusion and create a homeostatic imbalance and run the simulation to match the exact experimental parameters used in the human study by Wright, et al. [28]. This homeostatic imbalance (characterized by a change in osmolarity similar to that reported in the aforementioned study) then triggered a change in the SEEKING system that primarily caused downstream effects on the model's decision to report an accept of the offer.

Model Results

The top three subsymbolic parameter combinations produced an $RMSE$ of 0.058864 and R^2 of 0.97 when compared to those human data from [64]. Fig. 5 gives a plot of the model with the parameter-set that produced the highest match to human data, as well as those human data.

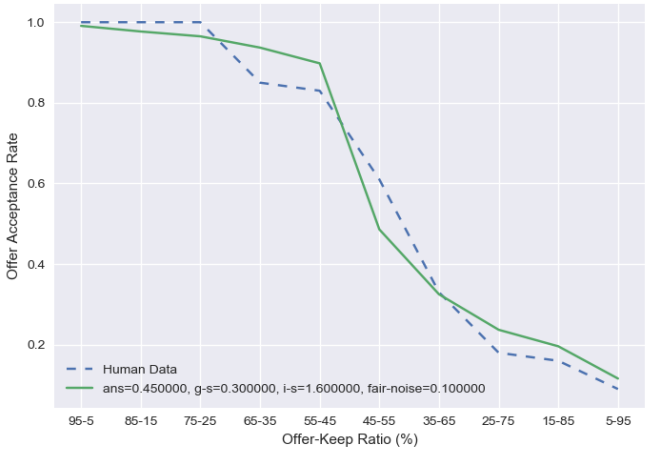

Fig. 5. Acceptance Rate vs Offer-Keep for the model and Human Data. The parameters listed above stand for the following ACT-R parameters: ans = :ans, g-s = :ga, i-s = :imaginal-activation, fair-noise = custom noise for fairness calculation.

When using the model from Fig. 5 (with the same set of parameters) to simulate the primary reward version of the ultimatum game, I found that a $reward_{max}$ of 1.4 showed the closest acceptance rate (48.9%) to the 50% reported by Wright, et al. [28] for the hypertonic condition, while the isotonic model showed a slightly lower 26.2% acceptance rate (vs 27% shown by [28]) when the $reward_{max}$ parameter is set to 1.75. Fig 6 also shows a predicted higher acceptance rate for the hypertonic model in other offer-keep conditions, principally due to the influence of thirst-based homeostatic affect (as shown by the general difference between the hypertonic and isotonic models.)

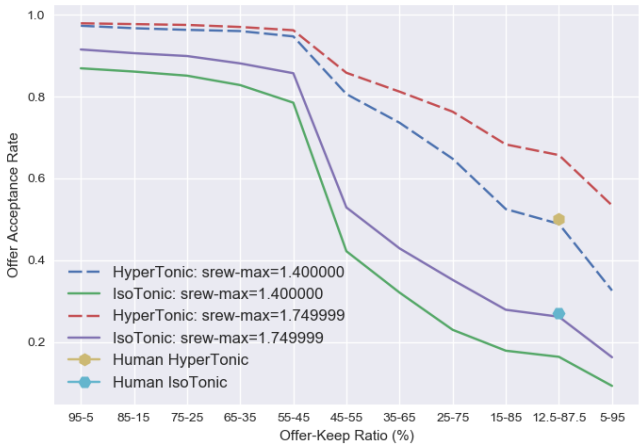

Fig 6. Acceptance Rate vs Offer-Keep for the Hypertonic and IsoTonic models (as well as Human Data). *srew-max* represents $reward_{max}$ from equation (4).

Discussion

By developing a cognitive model that accounted for the differences in acceptance rates across offer-keep ratios in human data, I constrained the parameter space of the physio-affective version of the model. This is a useful exercise as it allows one to assume those parameters would be reasonable for versions of the task that would incur less behavioral change due to affective processes.

With this model and simulation, one also can now explore more complex questions, like how the same system might respond to different offers given a similar physiological imbalance. I began to explore that question by expanding the validated non-affective model to the primary reward conditions studied by Wright, et al. [28]. The data from Wright, et al. [28] are limited in that they do not indicate how thirsty individuals acceptance rate changes over time.

The thirsty model's acceptance rate declines as the ratio skews towards the proposer, showing a large decline after the offer is no longer fair. Thirsty individuals are predicted by the model to be noticeably more likely to accept offers that give the proposer just a bit more water (81% acceptance rate vs 53% acceptance rate). Thus, though there certainly is an effect of reciprocity predicted after crossing that threshold, that effect is greatly reduced by bottom-up homeostatic processes. This may be counter-intuitive if one expects that bottom-up processes should not have a large effect on decisions that involve fairness and reciprocity. The model predicts that thirsty individuals will show a qualitative difference in how they treat reciprocity, especially in cases where decisions involve quantities deemed to be close to fair.

Future work can now include using these simulations to understand how these physio-cognitive processes may interact with cognitive systems in the ultimatum game over time with repeated proposer-respondent interactions. These simulations would prove as a useful complement to similar human studies that would prove complex and expensive, which would limit the scope of what could be addressed in such studies.

Limitations

While this architecture begins to pull together how we might regard something as emotional given some set of affective states and physiological states, the system likely needs more specification in how top-down appraisal may interact with the systems. This would manifest itself as a tertiary process (e.g., "*how do I feel?*"). Though the affective-associations modules does provide a way to connect *an* affective state to a memory element, the pattern-matching that leads to that affective state (or in the implementation's case, the affective value in a specific system) is underspecified and will need to be expanded to provide a more encompassing computational model of emotion; taking lessons from existing appraisal models (e.g. [5]) may prove useful here.

In addition, functional connections between physiological and cognitive (as well as affective) systems can be difficult to determine. Careful analysis of existing literature and theory related to function/structure being explored can be very useful here, but this remains a difficult task and relies on the assumption that data and theory exist.

From a practical standpoint, as the hybrid architecture continues to add representations and uses the already complex physiological model, computational simulations of behavior take longer to run. This decreases opportunities to explore the parameter space of the system. Though it is doubtful that the system will grow at a pace where the (computing) complexity it such that it takes an unreasonable amount of time to run, it could limit its uses on personal machine. The use of many of these computational cognitive architectures on general personal computers (especially such as ACT-R/Φ that are available to be used by those who request it) is an advantage for those trying to gain an initial understanding of the systems to understand assumptions of these implementations of theory and the behavioral results of those assumptions.

Using the architecture to understand addiction

Both primary-process affect theory and incentive salience theory have been used to explain addiction behavior [58, 66]. Given this has already been explored at a high-level, using the architecture to simulate these processes would be useful to study how the SEEKING/wanting system might interact with cognitive systems. This would be useful for understanding how psychiatric interventions may affect behavior in the short and long-term, under different circumstances.

Given the theoretical model that is implemented in the architecture, one could hypothesize that addiction would manifest at the subsymbolic level. Causing difficulty in avoiding rumination related to the addiction (e.g., [67]), a constant subversion of normal goals for those related to addition due to these memory dynamics (e.g., [63]), and an automatic use of basal-ganglia mediated procedural memory and action, resulting in behavior that occurs without much constant thought. This continues to be a complex topic, but systems and theories like that introduced here are useful for better understanding and teasing apart these processes in a systematic manner.

Evolving the affect in more complex environments

I have noted the need for a more encompassing way to generate affect in a previous section. Simulating an intelligent agent over in an expansive and complex environment to interact with potentially affective stimuli may be a useful way to build these affective value associations. I could use a tertiary process-level system to train on top-down representations, while also using the physiological system to associate physiological changes (e.g., pain) that feed directly into affective systems.

There are several environments for intelligent agents, though there are less that are usable and provide the possibility to expand enough to be useful in multiple contexts for the evolution approach just mentioned. Project Malmo [68], which extends Minecraft to make it more accessible for AI research, may be one such system that is useful in this context. The flexibility would be useful, its representations translate particularly well to ACT-R's perceptual-attention mechanism, and Minecraft is used by multiple age groups. The latter point provides an interesting opportunity to explore developmental-related questions with a computational model of emotion, which is an underexplored topic in the area.

Conclusion

That emotion affects our behavior is an intuitive idea to many people. What is not so intuitive is just how connected are our physiological, affective, and cognitive states. Though we may regard physiological, affective, and cognitive processes separately, they interact to form an intricate and complex stream of behavior over time. I have begun to make understanding and predicting these interactions more straightforward and tractable with a computational model of physiology, emotion, and cognition that is implemented with the ACT-R/Φ architecture.

ACT-R/Φ combines theory from the various areas in cognitive systems, cognitive science, psychology, neuroscience to begin to simulate and predict the hierarchy of processes that result in emotional behavior. An improved understanding of the architecture that constrains our behavior gives us a better opportunity to comprehend why we behave the way we do and how we can use this knowledge to recognize and construct a more ideal internal and external environment.

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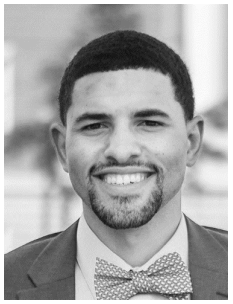
Author Bios



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