Towards a physio-cognitive model of slow-breathing

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Abstract

How may controlled breathing be beneficial, or detrimental to behavior? Computational process models are useful to specify the potential mechanisms that lead to behavioral adaptation during different breathing exercises. We present a physio-cognitive model of slow breathing implemented within a hybrid cognitive architecture, ACT-R/Φ. Comparisons to data from an experiment indicate that the physiological mechanisms are operating in a manner that is consistent with actual human function. The presented computational model provides predictions of ways that controlled breathing interacts with mechanisms of arousal to mediate cognitive behavior. The increasing use of breathing techniques to counteract effects of stressors makes it more important to have a detailed mechanistic account of how these techniques may affect behavior, both in ways that are beneficial *and* detrimental. This multi-level understanding is useful for adapting to changes in our physical and social environment, not only for performance, but for physical and mental health.

Keywords: ACT-R/Φ; Physio-cognitive model; Breathing, Adaptation, Cognitive Architecture, HumMod, Stress, Arousal

Understanding the potential effects of slow breathing on cognition is important for effectively using breathing techniques to reduce behavioral decrements during stressful situations. Conscious, controlled breathing may be used to positively moderate behavior, both in terms of performance (Neumann & Thomas, 2011) and for anxiety-related behavior (Bouchard et al., 2012; Brandão et al., 2008).

Previous work on mathematical and computational models of breathing have been completed to explore physiological processes related to breathing (Ben-Tal, Shamailov, & Paton, 2014; Molkov et al., 2017). Ben-Tal et al. (2014), for example, model respiratory modulation of heart-rate as a closed loop system of processes involving lung mechanics and gas exchange. This and related mathematical and computational models (see Molkov et al., 2017 for a comprehensive review on several computational models that control respiration) give a picture of the involved *local* physiological processes.

These physiological models often fall short in having straightforward ways to combine with other mechanistic models. One such computational physiological model that does provide a system-level account for multiple physiological processes is the HumMod system (Hester et al., 2011). This model is useful as it provides a straightforward way to simulate several physiological changes over time, that is, they allow us to explore changes in physiological systems due to perturbations *and* their interactions.

Though studies and theories that exist elucidate several of the process interactions at different levels of analysis (e.g., Brandão et al., 2008; Panksepp & Biven, 2012; Philippot, Chapelle, & Blairy, 2002), relatively few provide an account of interactions between physiological and behavioral (i.e., cognitive or otherwise) processes. Even fewer work has focused on pulling these theories into a computational process model that can be tested and simulated (though some more recent work does exist, for example, Fisher et al., 2017 uses ACT-R with a model of drug pharmacokinetics). This can be a useful step to understand the implications of premises made that detail interactions between physiological, affective, and cognitive processes.

We have used the HumMod system in combination with the ACT-R (Adaptive Control of Thought—Rational) cognitive architecture (Anderson, 2007; Anderson et al., 2008) to begin to study ways physiology, affect, and cognition interact to modulate behavior; we call this combined hybrid architecture ACT-R/Φ (see Dancy, 2013; Dancy, Ritter, Berry, et al., 2015; Dancy, Ritter, & Gunzelmann, 2015; Dancy & Schwartz, 2017 for some previous work using ACT-R/Φ). Using ACT-R and HumMod allows us to explore moderators in a manner that is tractable and that can be combined.

Below, we present a high-level model of physiological and cognitive interactions in slow breathing modulation of behavior. We then present the implementation of that model within ACT-R/Φ architecture. Data from simulating a physio-cognitive model that runs in ACT-R/Φ indicate that the physiological system provides a realistic representation and points to ways controlled slow, deep breathing may modulate memory processes and behavior.

The Physio-Cognitive Model

Physiology behind the model

Respiratory changes are known to cause several changes in the autonomic nervous system (ANS), both in the sympathetic and parasympathetic nervous systems. The short-term modulation of ANS activity appears to be predominantly modulated by pulmonary stretch receptors in the lungs and baroreflex activity (e.g., see Jerath et al., 2006; Russo, Santarelli, & O'Rourke, 2017; St. Croix et al., 1999).

Thus, we can tie short-term respiratory modulation of ANS activity to changes in baroflex, chemoreceptors, and pulmonary stretch receptor activity (in the latter case, that which is more directly related to cardiovascular activity). Figure 1 gives a high-level view of some of the interactions between these two receptor reflexes and ANS activity.

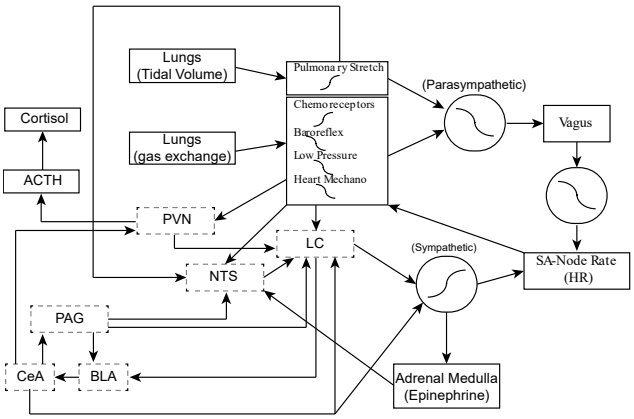

Figure 1. A high-level model of structures involved in effects of controlled breathing.

Though the picture is quite complex (as one may expect), there are structures that prove to be useful in understanding how interactions between cognitive and physiological processes may occur. Pulmonary stretch receptors, baroreceptors, and chemoreceptors (which respond to changes in lung gas exchange) all modulate nucleus tractus solitarus (NTS) activity, which modulates activity in the locus coeruleus (LC) downstream (Russo et al., 2017; Sampaio et al., 2012). NTS activity is also modulated by peripheral epinephrine via vagal efferent nerves (Miyashita & Williams, 2006); epinephrine is a peripheral stress hormone that is released via the sympathetic activation of the adrenal medulla. It also is useful to break the amygdala into two structures for the purposes of modeling respiratory, and indeed arousal, modulation of cognition and behavior. The basolateral amygdala nuclei (BLA) receives much of the input (and communicates with neural structures, though they are not shown above for simplicity purposes). The central amygdala (CeA) nuclei tend to be responsible for the output of signals to other neural structures that enact actions typically ascribed to *fight or flight* (LeDoux, 2007).

The Model within ACT-R/Φ

The ACT-R/Φ architecture extends the ACT-R cognitive architecture with HumMod physiological simulation system. In addition to the general modulatory functions of physiological systems (e.g., many of those implicated above) the cognitive and physiological models are connected using an *affect system* that acts as a functional connecting layer (Dancy, 2013). Though those mechanisms aren't used in this model, they likely are important for aspects related to controlled breathing (e.g., anxiety, see *discussion* for more on this topic).

Physiology

The HumMod physiological system has the many of representations shown in Figure 1. All representations that have a solid outline in Figure 1 are directly represented in HumMod. The effects of pulmonary stretch receptors (due to changes in tidal volume) were added to represent the short-term (i.e., seconds) effects of breathing on ANS activity, especially as it pertains to deep slow breathing (Jerath et al., 2006; Russo et al., 2017; St. Croix et al., 1999). These pulmonary stretch receptors cause a change in cardiovascular activity, which initially is tied to respiration rate and tidal volume (i.e., an increase in heart rate during inspiration and decrease during expiration). Due to the feedback in the system, this change effects primarily the baroflex and the chemoreceptors.

Though pulmonary stretch receptors primarily affect parasympathetic, cardiac variables in the model, the feedback-related changes in other afferent variables (primarily baroreflex and chemoreceptor reflex here) cause systematic changes in the model. This includes modulation of epinephrine release and the HPA-axis.

As mentioned above, changes to the HPA-axis due to deep, slow breathing are mediated through baroreflex and chemoreceptor activity. These afferents modulate corticotropic releasing hormone (CRH through the PVN), which mediates release of adrenocorticotropic hormone (ACTH released in the anterior pituitary). This increase in ACTH causes release in cortisol from the adrenal cortex.

Affect & Cognition

In ACT-R/Φ, concepts like *stress* are represented with homeostatic changes in physiology that modulate subsymbolic properties of memory elements. These physiological changes mediate a central *arousal* parameter (as represented in Eq. 1). The model is meant to take into account the effects of epinephrine, CRH, and cortisol on LC-Noradrenergic activity and on memory-related structures (see Joëls, Fernandez, & Roozendaal, 2011; Sara & Bouret, 2012; Schwabe et al., 2012; Schwabe & Wolf, 2013, for reviews of some of these effects and evidence for effects on both procedural memory and declarative memory).

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

In equation 1, *f(cort), g(CRH),* and *h(epi)* represent transformation of raw values of cortisol, CRH, and epinephrine (respectively). In this case, the functions are simply values normalized according to initial state baseline, such that each function gives an output of 1 when in a normal state. This representation of arousal is used to modulate probabilities to retrieve best matching declarative memories and fire the procedural rules that have the best matching conditions to a given cognitive state (for a theoretical view of these mechanisms, see Anderson, 2007).

Arousal modulates these subsymbolic values by affecting procedural memory utility noise (*:egs*) and declarative memory noise (*:ans*) using Equation 2. This equation gives a recognizable *inverted-u* like behavior, where the non-

linearity can be found within the physiological change. By default, a_{nom} is 1 and a_{max} is 2 in ACT-R/Φ

$$noise = \begin{cases} \frac{a*noise_{nom}+(a_{nom}-a)*a_{max}}{a_{nom}}, & a \leq a_{nom} \\ \frac{(a_{max}-a)*noise_{nom}+(a-a_{nom})*a_{max}}{a_{nom}}, & a > a_{nom} \end{cases} \quad (2)$$

Thus, both low and high arousal can increase noise, which would then make it difficult to retrieve the correct memory chunks (declarative memory) or fire the correct rules (procedural memory). Arousal also has an additional effect on utility values (procedural memory). Arousal modulates production rule firing threshold when below the nominal value. Both noise and threshold are altered; this was done because of previous work (Aston-Jones & Cohen, 2005), which indicates that an increase in distractibility occurs when arousal (reflected by activity in the LC-noradrenergic system) is below normal values.

Simulation results

Below we first present simulation results from running the physiological portion of the model for a period. This allows the understanding of how the mechanisms are functionally changing variables in the system (physiological and otherwise). We also present simulation results from running the physio-cognitive model with controlled breathing during a mental arithmetic task that has been used to induce stress (Kase et al., 2017; Kirschbaum, Pirke, & Hellhammer, 1993).

To test out the physiological model, we ran it under conditions similar to Critchley et al. (2015). In their study Critchley et al. (2015) record physiological data (peripheral measures and fMRI data) and subjective experience measures while study participants were either breathing freely or using a controlled breathing technique. During these different breathing exercises, participants were also either exposed to a normal (consistent with a normal environment breathing) gas mixture, or a *hypoxic* gas mixture (13% O_2).

Table 1. Mean heart rate values for breathing patterns from Critchley et al. (2015) and from the model.

Breathing	Human(*SD*)	Model
$Free_{Hyp}$	72.8 (2.5)	76.6
$Normal_{Hyp}$	74.2 (2.4)	74.2
$Slow_{Hyp}$	72.5 (2.5)	73.6
$Free_{Norm}$	68.3 (2.4)	72.4
$Normal_{Norm}$	70.1 (2.5)	69.6
$Slow_{Norm}$	68.6 (2.4)	71.2

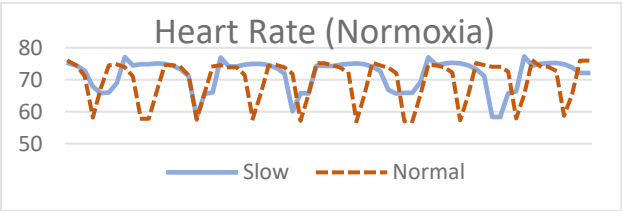

Figure 2. Simulated time course (1 minute) for heart rate in the model.

Those data collected by Critchley et al. (2015) did not show a large difference in heart rate. As, one may expect the physiological variables did not show a large difference when using the respiration rate and tidal volume shown by participants as parameters in the controlled breathing mechanism of the model.

Table 2. Mean systolic blood pressure values for breathing patterns from Critchley et al. (2015) and from the model.

Breathing	Human (*SD*) $_{(n=20)}$	Model
$Free_{Hyp}$	131.7 (1.7)	124.1
$Normal_{Hyp}$	129.6 (1.6)	122
$Slow_{Hyp}$	127.4 (2.8)	119
$Free_{Norm}$	132.1 (1.4)	119.6
$Normal_{Norm}$	129.9 (2.2)	118.4
$Slow_{Norm}$	130.2 (2.9)	118.8

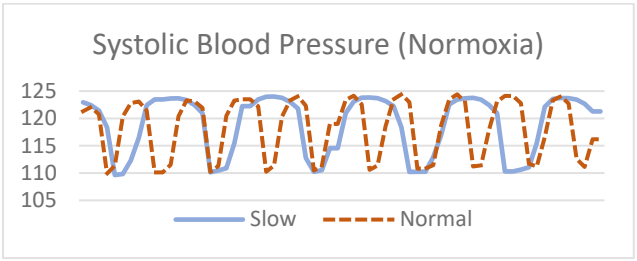

Figure 3. Simulated time course (1 minute) for systolic blood pressure in the model.

Given the connections between heart-rate and blood pressure in the physiological model, it is of no surprise that we also see the same general pattern when looking at systolic blood pressure. Both variables are the result of an adaptation to perturbations in the physiological system (from a steady state) over time. Looking at Figure 2 and Figure 3, this becomes clearer as blood pressure and heart rate change are similar for each type of breathing.

Given the model performs in line with the participants in this example study (though there were slight deviations, including with blood pressure, these may be explained by a difference in initial state due to variables such as age, Hall, 2011), we present a simulation of the physo-cognitive model completing a mental arithmetic task below.

Breathing, stress, and mental arithmetic

We ran an existing cognitive model of mental arithmetic in the modified ACT-R/Φ architecture. The model completes four blocks of serial subtraction, with each block lasting roughly 4 minutes (for a more detailed account of the model itself and past results, see Dancy, Ritter, Berry, et al., 2015; Ritter et al., 2009). Figure 4 gives a simplified view of the processing steps in the model, as well as the physiological changes that affect arousal. There are two main effects present in the model, an ability to become stressed due to outside factors (e.g., those that would occur before this task; see Kirschbaum et al., 1993) and due to being of incorrectness while vocalizing answers and encouraged to hurry during the task itself, the latter of which occurs on a

much shorter timescale. The model has also been modified to represent the stress that typically occurs as a part of this mental arithmetic task.

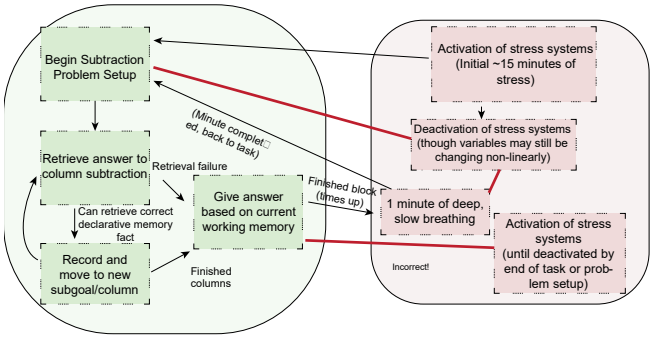

Figure 4. A simplified picture of the cognitive and physiological processing in the model. The black directed lines show processing changes that affect the task directly, while the thicker non-arrowed lines represented parallel physiological change due to changes in state.

The model shows similar performance on the task, albeit with a lower number of attempts. The model, however, does show a deviation in the 3rd block. Overall the model predicts an improved score that one would expect given the effects stress reduction effects of slow breathing.

Table 3. Comparison between human performance (from threat group from Kase et al., 2017) and model performance

	Human	Model
B1 (%)	78.6 (9.1)	83.8
B2 (%)	84.0 (4.6)	85.9
B3 (%)	74.9 (9.6)	88
B4 (%)	79.3 (9.7)	79.7
Total (%)	79.2 (5.0)	83.4

Discussion

General stress effects due to the task make it more difficult for the model to retrieve the *correct* facts due to an increase in declarative memory noise. Controlled deep slow breath has a two-fold effect on reducing stress in the model. The refocusing to control breathing causes a reduction in activation of excitatory mechanisms that increase arousal, which are themselves caused by focus of attention on the arousing/stressful stimuli. Slow deep breathing also causes peripheral physiological change that leads to a decrease in (sympathetic) excitatory through afferent receptor reflexes.

In future iterations and improvements of this model, using potential connections between existing ACT-R modules and neuroimaging (Anderson et al., 2008) may be useful for further prediction and specification. Though we did not use the functionality for this set of simulations, ACT-R provides a built-in model for associating activity in brain areas with module activity. We plan to explore using this to better specify modulations of systems due to physio-cognitive interactions.

The physio-cognitive model is a useful start, however there still are many areas of potential expansion. Though we represent several of the direct physiological changes in the computational implementation, many of the affective components are still lacking, some of which will be important for exploring interactions between deep breathing and other common practices (e.g., slow breathing as it is integrated with yoga-related practices Bhavanani et al., 2016). We highlight some areas and ways they will likely play an important role in affective modulation of behavior in future iterations of this work in Figure 5.

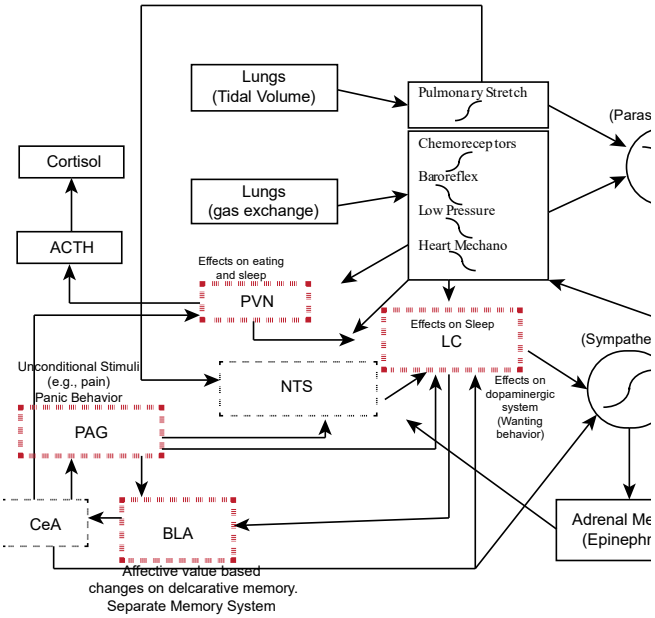

Figure 5. There are several behavioral functions that are likely mediated by structure in the general model presented. These are likely avenues of continued work for this computational system

Though general arousal and stress are useful representations, further specification seems possible and indeed is likely useful for further elucidating interactions between processes such as sleep, circadian rhythms, and eating (all of which are at least affected by the excitatory neuropeptide Orexin, CRH, and activity in the PVN and the Suprachiasmatic Nucleus, SCN; Saper, Scammell, & Lu, 2005.) The BLA (and several of the amygdala nuclei in general) seem to be very important for assigning affective value to memory and this interacts with PAG behavior, which is important for behavior and value associated with unconditioned stimuli, like pain and (central) hypoxia. Many of these structures also have direct and indirect connections to the hippocampus and basal ganglia, mediating declarative and procedural memory.

Breathing and stages of learning

Changes in the human physiological system subsymbolically affect memory and, thus, the process of skill development. The current understanding of the learning stages (e.g., declarative to procedural) needs to be extended. That is, actions like breathing can be considered as non-task-related

memory elements that may have both cognitive and physiological effects while performing a task.

For example, tactical breathing techniques (Grossman & Christensen, 2008) can have a physiological effect that interacts with both attention memory systems. With many tasks, individuals who are a novice use mostly declarative knowledge elements from memory to complete that task. Breathing is an action that may prevent successful declarative memory retrieval or an element that helps the memory retrieval process, depending on a person's arousal state, and breathing speed and volume.

Weak activation strength in declarative memory elements caused by a global change in arousal could lead to distraction. After a sufficient amount of practice, individuals, who are an expert on a task, use more procedural knowledge. In this stage, production rules that specify what the learner should do are to be compiled, and declarative memory activation values have been strengthened. Practicing the correct breathing technique (given certain physiological states) would help the learner move to later stages in learning faster as they would be less affected by noise in the learning process due to stressors. We have begun to explore this topic, and how this improved model may help intelligent tutoring for psychomotor tasks (Kim, Dancy, & Sottilare, Accepted), though much work remains in this area.

Modeling breathing and anxiety

Given the neural and peripheral structures involved in respiratory-related changes in the central and peripheral nervous system (Sampaio et al., 2012), and the association between many of these structures and anxiety (Brandão et al., 2008; Panksepp, Fuchs, & Iacobucci, 2011), it is no surprise that different breathing techniques have been used as a potential method to counteract anxiety (Jerath et al., 2015). Many of these processes may also mediate behavioral effects of mindfulness, which has been suggested to be useful not only for anxiety (see Van Vugt et al., 2012 for a mechanistic model), but also depression (Bellinger, DeCaro, & Ralston, 2015).

Conclusion

Breathing is a continuous mediator of physiological adaptation, affecting the whole nervous system, and often interacting with continuous homeostatic change due to stressors through spontaneous change. Developing computational mechanistic models of these adaptations, especially when under conscious controller breathing, and how they interact with learning and memory are important for more clearly comprehending the consequences of these physiological changes on behavior over time. This physio-computational model brings us closer to developing a straightforward, tractable, and unified model of physiological, affective, and cognitive behavior. This type of multi-level understanding is useful for adapting to changes in our physical and social environment, not only for performance, but for physical and mental health.

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