Towards Adding a Physiological Substrate to ACT-R

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ABSTRACT: Connecting a physiological model to a cognitive architecture presents an attractive option to better simulate a wide range of human behavior. This connection should facilitate both the effects of physiology on cognition (e.g. hunger and decision-making), and the effects of cognition on physiology (e.g. autonomic responses to memory featuring particularly aversive stimuli). To add physiology to a cognitive architecture, it should be represented as a separate module or substrate. We present ACT-R Φ (ACT-R Phi), a connection of the physiology simulation system HumMod (Hester et al., 2011) and the cognitive architecture ACT-R (Anderson, 2007) using an newly created ACT-R module. A model of the startle response and its consequent effects on cognition and physiology is presented to demonstrate an example use of the new substrate. This extended version of ACT-R allows a user to computationally realize theories involving cognition, physiology, and their interaction. This architecture has potential applications to training simulations.

1 Introduction

The connection of a physiological model to a cognitive architecture provides an opportunity to simulate a wide range of human behavior, cognitive, and physiological. Previously, architectures have been developed to implement different forms of moderators of cognition but they often lack a unified approach.

Based on reviewing several previous systems we note some possible connections and design suggestions for connecting a model of human physiology and a cognitive architecture (represented as ACT-R). We then present ACT-R Φ , a modification of ACT-R 6 (Anderson, 2007) that connects it a model of human physiology, the Hum-Mod simulation system (Hester et al., 2011), to ACT-R. HumMod is a simulation system that provides a top-down representation of human physiology. ACT-R Φ allows the user to enact changes to the underlying physiological substrate via buffers (within a new "physio" module) accessible by productions. Change in physiological variables can consequently drive change of cognitive parameters if a connection has been established. We also provide an example use of the system with an example model derived from a previous ACT-R model.

2 Past Implementations of Cognitive Moderators

Here we provide an abbreviated overview of several previous implementations of cognitive moderators in architectures. They provide lessons for creating an architecture with moderators, or modifying existing architectures to accommodate moderation.

2.1 CoJACK

CoJACK is an adaptation of the Beliefs, Desires and Intentions (BDI) architecture Java Agent Construction Kit (JACK). JACK consists of events, plans, belief-sets, and intentions. CoJACK extends JACK by adding moderators and errors(Evertsz et al., 2009); it adds the ability to include noise (modeled after the noise representation in ACT-R) in the belief-set choice process. This introduces the possibility of retrieval of beliefs which are incorrect or only partially match to the conditions presented. The moderators represented in the modified architecture are fear and caffeine. These moderators can be dynamically set, thus they can be modified during a simulation. They are represented as overlays to the existing architecture, this is similar to previous work on defining theories of stress using the idea of overlaying the existing architecture by Ritter et al. (2007).

Work with CoJACK shows that computational physiology is possible and would lead to interesting changes in behavior. It explores some mechanisms involved in physiology moderating cognition (and vice versa) but the work required implementing a full model of physiology to continue. Additionally, combining multiple overlays to represent multiple effects ultimately will become intractable and lead to creating a representation of physiology to resolve the conflicts in how overlay effects combine and affect one another. Models of physiology that provide systematic representations of gross anatomy potentially provide a modeler with an opportunity to combine and represent multiple overlays within one tractable system.

2.2 MicroPSI

MicroPSI (Bach, 2009) is a hybrid architecture with both symbolic and subsymbolic (neural network) representations based on the Principle of Synthetic Intelligence (PSI) theory. The architecture has urges, motives (an urge with a corresponding goal), and demands underlying the motivation schema. MicroPSI has three physiological based motives, two cognitive based motives, and one social motive. The physiological motives (fuel, water, and "intactness") provide higher-level representation of homeostatic-based (fuel and water) and somatosensorybased (intactness) motivations.

Bach (2009) admits the mechanisms in MicroPSI fail to represent many of the complexities of human cognition; however it does provides mechanistic representations important for realistic agent autonomy. MicroPSI provides an example of a middle ground between an agent representing human-like cognitive abilities and underlying modulating mechanisms that interact. This architecture displays some of the important aspects of having an underlying motivational system to modulate behavior and cognition. Providing a systematic representation of human physiology to connect to architectures that represent higher-level concepts of the human mind allows one to traverse levels of representation to provide the facilities for users to model the moderating effects of physiology at an appropriate level.

2.3 Gunzelmann model

Gunzelmann and colleagues developed a model that simulates the effects of fatigue (arising from sleep-deprivation) and circadian rhythms by altering ACT-R module parameters (Gross et al., 2006; Gunzelmann et al., 2009). The approach they use is an alteration of the utility of production rules based on alertness or arousal. The authors simulate fatigue and alertness using mathematical models of alertness, the output from these models is used to drive ACT-R module parameters, predominantly those in the procedural system, i.e. those related to utility. Their model of fatigue illustrates the opportunities afforded by tying a mathematical model representation of a physiological component to a cognitive architecture. With this model they have been able to model a deterioration in driving performance based on fatigue (Gunzelmann et al., 2011). While they were able to simulate some of the effects of fatigue, others like a spike in alertness due to a startle/defensive response proved elusive. A more full representation of physiology at macroscopic levels would potentially allow one to simulate these responses as well as fatigue based on other aspects of physiology (e.g. energy need or thirst).

3 HumMod

HumMod (Hester et al., 2011) is a simulation system that simulates human physiology via a model specified in XML schema. The HumMod system is an extension of physiology research of Dr. Guyton who applied engineering systems analysis to the cardiovascular system under normal and pathologically significant physiologic states. His work continues to serve as the basis of contemporary medical knowledge regarding cardiovascular pathophysiology (Guyton et al., 1972; Montani & Van Vliet, 2009). The current HumMod model is a derivative of the original Guyton model (Guyton et al., 1972) that represents an instance of human cardiovascular physiology using a topdown schema that is defined through hundreds of linear and non-linear state equations over 5000 state variables. HumMod has provisions for simulating normal and abnormal physiology in multiple time scales. Additionally, the model provides several points of access to the nervous system through both the endocrine and nervous systems.

There are two ways to change the values attached to variables in HumMod, changing the underlying XML-based model or changing the set values after the model has been loaded into the simulator system. An alteration of the base-model allows the changing of initial variables, derivations, and connections between variables. Changing the set-values has perhaps less systematic power than a change to the actual model, but allows one to work within the given model and quickly view the data arising from these changes.

Tracking changes in data can become cumbersome in the HumMod system. If one chooses to use the full-system with the built-in user-interface, a user will need time to explore the interface to determine what variables should hold their attention. One can also communicate directly with the model solver (the portion that actually digests the XML model) via message passing through commands written to files. If one chooses to simply use the model solver portion of the system, processing the output data becomes difficult as portions of the 5000+ variables are predominantly used to implicitly affect other variables.

How to best represent these variables and corresponding values remains an open problem.

4 Initial Design for Adding Physiology to a Cognitive Architecture

We note a preliminary design here to tie ACT-R to HumMod, and then demonstrate part of this design. There are several ways one can represent the connections between physiology and cognition depending on the contextual factors in which one is interested. As an example, we discuss appetitive mechanisms and the hypothalamicpituitary-adrenocortical (HPA) axis. Homeostatic-Appetitive motivation can cause a modification of behavior and cognition to reduce the particular motivation. The HPA axis plays a major role in reactions (cognitive and physiological) to stressful stimuli (Tsigos & Chrousos, 2002).

4.1 Appetitive motivation

Basic bodily motivations deserve important attention when discussing the physiological moderation of cognition. Homeostatic-Appetitive motivations can begin to take priority over attention resources and affect human behavior and cognition depending on the physiological imbalance being sensed by a person. Innate necessities like hunger and thirst homeostasis ultimately can change memory recall and attention (e.g., (Aarts et al., 2001; Mogg et al., 1998)). The need to void and its effect on cognition has also been previously studied including how it affects both working memory (Lewis et al., 2011) and decision-making (Tuk et al., 2011)

In ACT-R one can begin to implement appetitive motivation mechanisms by tying rule and memory parameters to the current states of receptors represented by HumMod. Hunger can be represented with the food portion of the "GILumen" variable. HumMod also provides a representation of the effect of osmoreceptors, which measure changes in extracellular body fluid and are known to be responsible for thirst (McMorris, 2009). These variables can be tied to procedural utility (e.g., :iu and :nu) and declarative memory (e.g., :ans, :bll, and :mp) to have results of physiology control the probability of rule selection and memory is retrieved during activation of these motivations. This change should enact attentional biases. With these motivations and change in utility one also has a mechanism to represent a type of "pleasure" based on whether or not (and to what extent) an action satisfies a motivation. Procedural partial matching also presents an opportunity to represent effects of hunger or thirst. Mogg et al. (1998) determined that there is a selection attention bias related to a hungry state, i.e. participants were found to have a bias in selection of words related to food while in a hungry state.

4.2 The HPA axis

The relation between stress, endocrine responses, and cognition is a good connection to explore for the modeling of effects of physiology on cognition (or vice versa). Stress affects the hypothalamic-pituitary-adrenocortical (HPA) axis and this axis dictates the release of hormones that can subsequently affect cognitive processing. Stimulation of this axis can cause a systematic release of cortisol from the adrenal cortex as well as a release of epinephrine and norepinephrine from the adrenal medulla. The systematic release of cortisol is known to have latent effects on cognition, however it can also have a more rapid effect on stress-related information consolidation and appraisal of new situations (Groeneweg et al., 2011). Cortisol also affects the HPA axis itself by reducing future activation leading to further release of glucocorticoids.

One can use previous studies of stress representation in ACT-R to guide parameter modulation via a more complex physiological system. Ritter et al. (2009) chose to change memory retrieval and vocal module parameters (activation noise, base level constant, and seconds-persyllable) to better match human experimental data in a serial subtraction task being completed in conjunction with the Trier Social Stress Test (TSST) (Kirschbaum et al., 1993). Additional potential parameters exist in the rule utility facilities represented in ACT-R, e.g. initial utility of a user-defined function (:iu) or initial utility for a learned function (:nu). Upon completion of a stressful task, model production utility can be assessed not only based on reward for task-completion, but also for a return to a homeostatic state. Thus, for this reward we could monitor the delta between physiological variables in HumMod related to a stress response, e.g. adrenal hormones, heart rate, and blood pressure. An increased attention on the contextual stressor could potentially dictate a change in the rule retrieval values for rules relating to shifting attention away from the context. The initial declarative memory activation values during the stressor may also change depending on the input module that contains information on the stressful stimuli, i.e. if the model's stress is triggered by predominantly visual stimuli then assign a higher initial activation value to that declarative memory when the memory is harvested.

4.3 Summary of discussed modulating effects of physiology

Homeostatic-based appetitive motivation can potentially affect procedural rule choice and memory encoding/retrieval. As motivations are failed to be satisfied, effect on cognition intensifies further causing more extreme changes to cognition and behavior. Stress causes activation in the hypothalamus, eventually triggering sympathetic system activation through mechanisms like cortisol release, which acts as a moderator on activation of neuronal activity important to cognitive function. With innervations of the adrenal medulla by efferent fibers, hormonal release of epinephrine and norepinephrine is initiated as a mechanism for short-term adaptation to the stress; this also has an effect on other aspects of human physiology like heart-rate and blood-pressure. These effects can cause a change in cognitive mechanisms like memory retrieval and rule choice.

4.4 Module vs. Substrate

The question of whether one should use a module or an underlying substrate arises when realizing physiology in a cognitive architecture. Due to a possible difference in answers partially depending on the cognitive architecture, we ground our answer to this question in applying physiology to a modular architecture. We use ACT-R for specific examples.

Modules in a cognitive architecture are utilized to represent functional centers for different aspects of human behavior and cognition. A motor module, for example, would represent certain aspects of human motor behavior like the cognition behind the motor behavior or the outcome of this cognition. Modules in cognitive architectures are useful because they allow the separation of functionality and thus minimize interdependency among aspects of the cognition, essentially allowing the modeler to choose which features of cognition they wish to manipulate.

4.5 Using both concepts together in a cognitive architecture

If one chooses to follow a theory like that instantiated in ACT-R, modules and an underlying substrate should be used to add physiology to a cognitive architecture. The underlying physiological substrate should be used to represent functional aspects human physiology. This substrate should provide a fairly robust simulation of the processes occurring in the body during cognitive actions; at the very least, homeostatic processes should be available to represent primitive motivations like energy need and body temperature regulation.

Anderson (2007) proposes the encapsulation of the (normal) cognitive/behavioral function of neurological structures within architecture modules with ACT-R. Adding a module to represent the receiving and sending of signals to the body, in addition to the underlying substrate, follows an idea of module representation presented in ACT-R. It is less clear if more than one module should be used for this function; encapsulating this functionality within one module is feasible, given that one module avoids the heavy fixed interdependency issues one may hit by using two modules. However, it is also likely that encapsulating functionality in two modules may improve longevity and feasibility of expansions to architecture. We instantiate the ideas presented in the connection of ACT-R and HumMod by representing the HumMod system as a substrate running in conjunction with the ACT-R architecture. To realize this substrate within the ACT-R architecture, we created a module that represents a physiological substrate and applies its effects to other modules. The physiology portion of the module is provided by the HumMod simulation system.

4.6 ACT-RΦ: ACT-R with physiology

ACT-R Φ is an extension of the cognitive architecture ACT-R that provides an additional representation of human physiology and allows for corresponding bidirectional connection between cognition and physiology. This allows one to model both the environmental and internal effects on central and peripheral physiology and the corresponding effects on cognitive parameters. We accomplish this physiological representation through the use of a system that presents a dynamic model of human physiology, HumMod. HumMod gives a user the option to use the included model of integrative model of physiology or alter that model via XML. The schema for the modified architecture is represented below in Figure 4.1.



Figure 4.1: An overall schema of ACT-R Φ . The light links represent potential connections between within the extended architecture.

4.7 The physio module

We have developed a module for the representation of physiology and connection of this representation to other ACT-R modules. This module represents an underlying physiological substrate that dynamically obtains data for physiological variables from the HumMod simulation. In this case, a module is used as software construct not as another type of working memory buffer, although it has some memory-like aspects of remembering how recently the body has eaten, for example.

The "physio-substrate" buffer is used to request the module to begin retrieving physiological variable data from the HumMod simulation. One can accomplish this task by putting a chunk of type *phys-var* into the physio-substrate buffer. One can also explicitly request the value of any physiological variable by sending a request to the "physsubstrate" buffer. Physiological variables in HumMod can be explicitly set to a certain value by adding a chunk to the "efferent" buffer. A request can be sent in the form similar to buffers of existing modules. Currently, users must enter the exact variable name representation from HumMod to enact changes via the efferent buffer. This can prove cumbersome and requires a fairly low-level understanding of the model in HumMod to actually use the buffer. Alternative ways to provide a powerful, but more usable change to values is currently being explored.

These functionalities allow the user to model and simulate both cognitive effects on physiology and conversely effects of physiological variables on aspects (e.g., module parameters in ACT-R) of cognition and behavior. Currently, only implicit representations of connections between the physiological variables and ACT-R exist, accessible by function calls; examples of these connections are represented in table 4.1. A modeler may also write their own functions and simply use the module to get and set variable values.

Table 4.1: Example connections built into ACT-R Φ

Concept	ACT-R	HumMod
Startle Response	:ans	SympNS, Epi
Hunger	:mp, goal-focus,	GI-Lumen
Thirst	:mp, goal-focus, :ppm	Osmo-Rec, BodyH20.vol

5 A Extension of an Existing ACT-R 6 Model

We have developed a modified version of the ACT-R 6.0 subtraction model developed by Ritter et al. (2009). This model in ACT-R Φ includes provides a representation of the CNS-PNS loop afforded by the addition of the physio module, i.e. an action affects the central nervous system that consequently affects the peripheral nervous system, and this result feeds back to affect the CNS. With the extended model, we assume a fluctuation of sympathetic nervous system activity due to a scheduled sound that causes a form of a startle reflex. This startle reflex causes

sympathetic nervous system activation in HumMod and affected variables feedback to affect the noise in retrieving declarative knowledge.

5.1 Connections made between ACT-R and Hum-Mod

With this modified subtraction model, there exists a connection between production rules for fast-response to sudden sounds and modifications to physiological variables. The concept behind these new productions is related to those discussed by Kennedy and Bugajska (2010), who have modeled inhibition of fast-responses by immediately enacting activity in other buffers. In this model, the response to the sudden sound is not an inhibition response, but a physiological response. The model responds to a sudden sound with a change in attention and underlying physiology, i.e. a partial activation of the sympathetic nervous system. The sound also consequently leads to a change in memory. This creates partial simulated sympathetic nervous system response, e.g. increased heart rate, blood pressure, and epinephrine (adrenaline) levels that affect memory retrieval noise in ACT-R.

5.2 Modifications made to original ACT-R 6 Model

Production rules are added to handle the fast processing of the sudden sound stimulus. After the sensing of the loud noise in the aural-location buffer, the model clamps (sets to 1) the central nervous system autonomic nerve integration variable (via the efferent buffer in the physio module) that positively affects the adrenal nerve activation, thus simulating a feature of sympathetic system activation. The epinephrine HumMod variable is tied to Equation 1. The ansMultiplier variable was determined by solving for the equation when the :ans parameter was equal to the value found in the non-caffeine parameter set found by Ritter et. al. (2009), and the (current level) epinephrine value is equal to that which is the result of HumMod adrenal nerve activity leading to sympathetic activity (e.g., heart-rate) similar to that found in the original TSST study (Kirschbaum et al., 1993).

$$\frac{(Current Value)_{Epi} - Baseline_{Epi}) * ansMultiplier}{Max_{Epi}}$$
(1)

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Physiological change is accomplished by sending a query to the efferent buffer in the physio module that specifies the specific variable to be changed and corresponding values; we change *SympsCNS.ClampSwitch* to value 1 and *SympsCNS.ClampLevel* to value 2.9. The sensing of the sound also results in a relatively short processing of the specific sound. The startle response and corresponding physiological change is relatively short-lived, and rules dealing with the subtraction task continue firing shortly after the encounter with the noise.

Table 5.1: Task Performance and number of attempts (avg.) by the original models (Ritter et al., 2009) and modified model.

Parameter Set	Performance (%)	Attempts
09 Average	81.4	48.1
09 Threatened	77.4	40.3
Modified	78.6	35.98

The modified model run 75 times produced output results (table 5.1b & figure 5.1) similar to the original subtraction model (threat condition), and consequently were lower than overall performance of the original model (signified as *09 Average* by Ritter et al., 2009). Though the average performance is similar between variables we do notice an expected drop in performance after the sound has been reduced and subsequent memory noise increases due to an increase in SNS activity. On average, the modified model also outputs less subtraction attempts than the original models in Ritter et al. (2009) (table 5.1). With this altered model we have shown that one can reproduce similar performance of a model with statically set parameters using a model that has parameters dynamically set via an underlying physiological substrate.



Figure 5.1: The task performance frequency distribution of the *09 average* model (a) and the *modified* model (b) N=75.

6 Discussion and Conclusions

We have introduced ACT-R Φ , a modified version of the ACT-R (6.0) architecture that utilizes a new module to send information to and receive information from Hum-Mod, a system that simulates human physiology using a top-down level of human physiology. Currently, hardwired connections in the system are allowed to be turned off to give the modeler more flexibility using the provided mechanisms or developing their own. This should aid in allowing users to computationally realize conflicting theories involving human cognition and physiology, but ultimately may force the modeler to self-code even more simple physiological based moderation of cognition. Reasonable hard-wired connections are currently being explored for implementation in the near future.

We have demoed a way in which one may use the ACT-R Φ system. The open-source nature and maneuverability of ACT-R (and consequently ACT-R Φ) allows for a user with experience to enact relatively fast changes in the ACT-R system without necessarily having to reload the entire system. This control, however, does come at the price of usability and physiological connections will likely be better served to have a higher-level representation.

As one continues to consider physiological ties to cognition and the consequent representation of these ties for modeling of human behavior, one is likely to continuously be confronted with issues and concerns arising from considering behavior and cognition on separate levels. We offer non-exhaustive points of discussion related to insights, implications, and possible concerns arising from opportunities afforded by ACT-R Φ .

6.1 Cognitive-physiological timing

HumMod allows one to continuously simulate physiology over time. Timing is now not only important on a subsecond scale, but on minute and even hourly scale. Effects can occur hours after original stimulus and can induce unforeseen effects in the model, providing variability. Homeostatic-appetitive motivation (e.g., energy needs) should be represented over a longer time-scale to better simulate continuous homeostatic mechanisms. This allowance also has implications for the simulation of effects of different causes of fatigue and effects of biological rhythms.

6.2 Visceral perception conflict

With an abundance of data from the HumMod simulation, one is likely to run into conflicts of differing sensations affecting cognitive parameters. An example of a possible conflict that has been studied in visceral sensory research is the idea of pain vs. hunger. It has been shown that there is a lower activation of pain processing regions of the brain if the source of the pain is directly related to the fulfillment of nutrient deficiency, i.e. hunger (Coen, 2011). Integrating multiple lines of visceral sensory data into perceptual data within the system needs further exploration.

How should a simultaneous sensation and resulting motivations of (e.g., pain and hunger) be resolved within this system? Others have suggested a model that involves a higher priority placed to the more biologically relevant (e.g., Gregory et. al., 2003) but a definitive answer has yet to be reached. The PSI (Bach, 2009) theory perhaps offers some guidance on the matter as is provides separate motives to represent hunger (fuel) and pain (intactness) and reconciles concurrent activation of these representations by a MicroPSI agent in its environment.

6.3 Visualization and ease of use of ACT-RΦ

ACT-R Φ will have additional variables and be a more complex system. Presentation of the system's state will be important. A new visualization system will need to be added to better communicate what is going on in the combined architecture. It will remain difficult to sustain a good balance between showing the maximum amount of information without overloading the modeler with data. This is especially apparent with the integration of models of human physiology as one must not only content with the representation of human physiology, in the case of HumMod 5000+ variables, but also the existing cognitive architecture data and the corresponding connections between them.

As the physiological system continues to evolve a higherlevel representation of knowledge, physiological variables, and their connections should be explored. The Herbal (Cohen et al., 2010) language is a good example of such a high-level language with graphic displays that might be extended to support this work. Herbal allows users to represent agents in its own XML-based language and can compile the model code into Soar, ACT-R, and JESS agents. Alternate representations also should be explored to allow for better validation of the models. At the present juncture, it remains difficult for independent validation of representations due to the sheer size, availability of data, and complexity of the system; high level representations may also aid with validation.

6.4 Developing models for military and game simulation

With a physiological connection, one is afforded more representations to simulate environmental effects on human behavior and cognition. This is important in developing more human-like agents for simulation environments like those in military or gaming. Increased autonomy and variability in models will allow for more realistic environments in military-based simulations like VBS2; Evertsz et al. (2009) also discuss the opportunity afforded by including models with moderators, albeit with a different level of representation.

From a military perspective one can now also begin to simulate more behaviors related to fear and anxiety and cognition (e.g., PTSD). Representing a constant anxiety state could potentially be accomplished by changing baseline parameters in the physiological parameters to represent the constant physiological activation (both central and peripheral) present in those in an anxiety/fear state and ensuring a hard-wired connection to cognitive parameters. These states could potentially be elicited due to internal factors (e.g., PTSD), or environmental factors (e.g. battle conditions).

6.5 Final remarks

Minds need brains to support them and brains need bodies to support them. As we become interested in more accurate models of cognition we will need to add a physiological representation of the substrates that on multiple levels support and implement cognition. ACT-R Φ is a step in this direction, and has provided some insights already.

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