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Caffeine and Cognition: A Cognitive Architecture-Based Review

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Potential Conflict of Interest Statement

Frank Ritter co-developed CaffeineZone, an app for predicting pharmacokinetic and pharmacodynamics effects of caffeine and has received compensation through the sales of CaffeineZone.

Abstract

Caffeine is a chemical that is commonly ingested by people daily to modify their behavior. Its physiological and psychological effects have been studied extensively for theoretical and applied reasons. We briefly review the current information on caffeine's physiological effects. We then review caffeine's effect on cognition and summarize these effects as changes in cognitive architectures (a fixed set of mechanisms to explain cognition), which provide a unified way to represent the changes. Modeling the effects of caffeine on an individual's physiology, as well as their cognitive function, is a logical addition to cognitive architectures because caffeine moderates cognitive performance. Cognitive architectures have recently been connected with physiological simulators, allowing physiological variables to interact with cognition. This combination provides a natural way to represent caffeine in current cognitive architectures and model how cognition and physiology interact, and use such models in system design. Our review notes how caffeine influences several aspects of users' capabilities that will influence system performance. It also notes gaps in the caffeine literature needed to improve models of users, including studies on the distribution of half-life, the need for the use of dosages vs. doses, and task-based effect studies.

Keywords: Caffeine, Cognition, User Modeling, Cognitive Architecture

Relevance to Ergonomics: Understanding caffeine, one of the most common behavior moderators, will help model users. Better understanding of users will help design systems. This approach to understanding caffeine's effects can also serve as a template for understanding and modeling other moderators of behavior.

Introduction

Caffeine is the most commonly used psychoactive drug. There are numerous reviews of its effects from both theoretical and applied perspectives (Ahluwalia & Herrick, 2015; Amendola et al., 1998; Institute of Medicine, 2001; Lorist & Tops, 2003; McLellan et al., 2016; Nehlig, 2010; Poole et al., 2017; Rees et al., 1999; Smith, 2002; J. L. Temple et al., 2017; Wingelaar-Jagt et al., 2021; Young & Molesworth, 2011). The existing reviews focus primarily on caffeine's physiological effects and present them as non-parameterized effects. These reviews note that caffeine ingestion leads to changes in cognition and in motor physiology but do not focus on these changes nor provide quantitative details that could be used to create a model that simulates the effect of caffeine on cognition for use in designing interfaces (e.g., McMillan et al., 1989; Pew & Mavor, 2007) or for creating agents in simulations that have circadian rhythms (e.g., Pew & Mavor, 1998). Our review focuses on caffeine's effects on cognition and uses a theoretical framework to hold and summarize the results.

A review of how caffeine affects cognition and to cast that review in a representation that allows it to be applied would be useful. Caffeine is consumed by individuals participating in many industrial and governmental environments to moderate their behavior (e.g., Temple, Warm, Dember, Jones, LaGrange, & Mathews, 2000; Wingelaar-Jagt et al., 2021). Its effects have been shown to vary depending on an individual's previous consumption (Crawford et al., 2017; Tharion et al., 2003; Yeomans et al., 2002). A drug with such complexity should be better understood because it is consumed by individuals in high-risk environments to moderate their behavior (e.g., Institute of Medicine, 2001; Wingelaar-Jagt et al., 2021). A cognitive architecture is a useful tool to model these changes because it can use a unified system to model a combination of cognitive effects.

In our review, we will describe how caffeine is commonly consumed. We will evaluate its physiological effects (how its levels vary over time), and then review caffeine's effects on cognition. The results of the review will be represented as a set of changes to cognition, allowing us to illustrate how many of these effects can be implemented in a computational representation—a cognitive architecture. This approach also supports the application of this knowledge in models of users. We will also note future work that is suggested by our review.

Thus, our review should be interesting to caffeine researchers as a summary of the effects of caffeine on cognition and how to make their work more useful to a wider audience. Our review should also be useful to those modeling users as a way to understand and implement the effects of caffeine in a cognitive architecture, and as an example review and implementation of such moderators of cognition. It should also be useful to system designers who need to understand how user's capabilities change with caffeine consumption and decay over the course of a work day.

Our review can also be useful as a template for other moderators that influence behavior in systems. We review the literature about caffeine's sources, time course, and impact. And we also provide a way to include its effects on behavior.

Caffeine

Caffeine, it has been said, is the most commonly used psychoactive substance in the world (Julien, 2001). It is found in several common foods and drinks, either naturally or through additive processes. Although caffeine can be found in many prescribed and over-the-counter drugs, we will focus on caffeine intake through food and beverages in our review.

Coffee, tea, and cola all contain caffeine and are consumed at high levels around the world. American adults, for example, consume an estimated 4 mg/kg of caffeine daily (Barone & Roberts, 1996). Coffee is one of the most commonly consumed beverages, so there has been an interest in understanding the safety of coffee consumption (Poole et al., 2017). Additionally, energy drinks that contain caffeine have become more popular over the past decade, which suggests that caffeine consumption in young adults may increase through this factor (J. L. Temple et al., 2017). Caffeine can also be consumed through foods such as chocolate, but higher doses are typically consumed through beverages such as coffee and energy drinks.

Caffeine is consumed by individuals that work in routine conditions as well as situations where sleep schedules are modified, such as night shifts and in situations where sleep restriction occurs (e.g., Naval Aerospace Medical Research Laboratory, 2000; Wingelaar-Jagt et al., 2021), because caffeine is believed by users and organizations to reduce fatigue and improve cognitive performance (i.e., Cappelletti et al., 2014; Dawkins et al., 2011; Institute of Medicine, 2001). Researchers have considered how varying doses of caffeine influence cognitive performance when there is sleep restriction (Kamimori et al., 1995; Lieberman et al., 2002). The effects of caffeine vary based on other environmental and physiological factors and many such factors will be considered within our review.

The effects of caffeine on an individual vary based on the amount consumed. A common range of doses in studies is between 50 and 400 milligrams (mg), so intake in this range will be the focus of our review. A single cup (8 fl oz/237 ml) of coffee typically contains 85 mg of caffeine, so this range has effects representing most caffeine consumption. Some studies suggest that this range is also a safe range for daily consumption (e.g., Julien, 2001).

Caffeine is not dose but dosage dependent (Smith, 2002). While dose refers to the mass (in mg) of a drug consumed within a serving, dosage refers to the mass per kilogram (mg/kg) of body weight for the individual consuming it, which depicts the proportional amount of the drug metabolized by the consumer. For example, a person weighing 85 kg (187 lbs) who consumes 591 ml (20 fluid ounces) of coffee prepared from ground roasted coffee will have a 340 mg dose of caffeine, with a dosage of 4 mg/kg. Most studies that we have found use dose, a measurement that is underspecified when individual body weight is not reported, and this lack of specification should be kept in mind when considering differences in data reporting the effects of caffeine consumption. Dosage is a better measure to report than dose because dosage takes into account body weight, a factor that affects the action of most drugs, including caffeine. Before proceeding to the review, we explain the theory that we will use to summarize the results.

Cognitive and Physiological Architectures

Cognitive architectures are based on the theory that the mind is an extension of fixed structures that produce intelligent behavior in complex environments. These systems have been used to model human cognition under a number of circumstances for a variety of tasks (Newell, 1990). Architectures use fixed mechanisms to produce intelligent behavior, and can be categorized as agent, symbolic, sub-symbolic, hybrid, or non-generative architectures based on their underlying structure.

Figure 1 shows an overview of the Common Model of Cognition (CMC), which is a theory that describes a common set of mechanisms found in most cognitive architectures. It is based on Soar and ACT-R, and is consistent with others including EPIC (Kieras et al., 1997) and CoJACK (Ritter et al., 2012). While CMC is basic, it will provide enough framework for summarizing the

effects of caffeine, and will provide a framework for applying the effects of caffeine in many architectures.

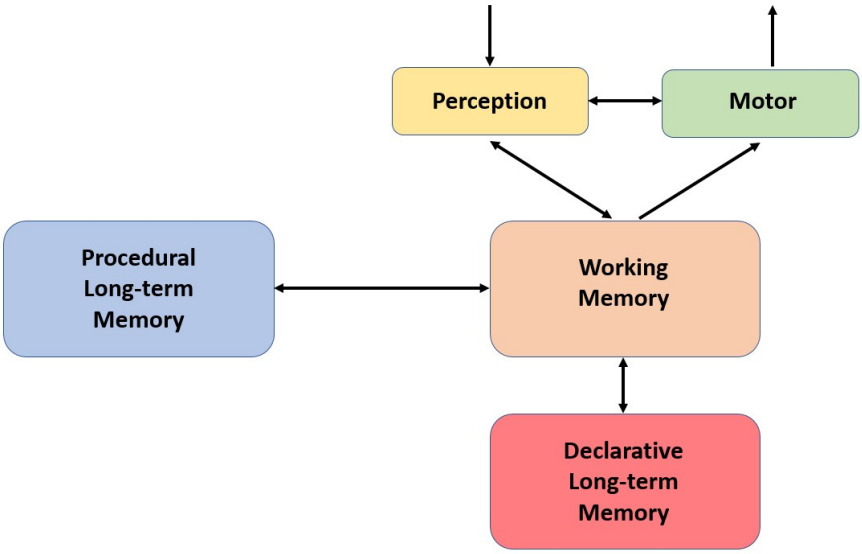


Figure 1. Structural representation of the Common Model of Cognition (formerly, the Standard Model of Cognition, based on Laird et al., 2017).

Models of individual moderators (Ritter et al., 2007; Silverman, 2004) have been created as overlays in cognitive architectures (e.g., Ritter et al., 2012). This approach implements changes to cognition as a set of changes, for example, anger might change a decision process, or sleep deprivation might decrease processing speed. This approach can be useful for individual moderators but does not provide a principled way to combine the effects of multiple moderators (e.g., caffeine and nicotine; caffeine and stress), because the order of applying the overlays can influence their effects.

Recently, physiological simulations have been combined with a cognitive architecture to model how cognition is modified by various internal and external stressors, (e.g., ACT-R/Φ: Dancy, Ritter, Berry, & Klein, 2015). The construction of ACT-R/Φ, which is one of these simulators,

has occurred as biological studies uncover interactions between cognition and physiological measures. Implementing the effects of common factors such as caffeine or stress in combined cognitive/physiological simulations is a way to understanding these complex interactions. We believe that to effectively model the changes of cognition that occur due to caffeine consumption, we must understand the relevant underlying physiological changes that take place.

Other architectures or frameworks for modeling cognition could also be used to summarize the results. These approaches include neural networks (e.g., O'Reilly 2000), which represent the mechanisms on a lower level, and sequential sampling models (e.g., see Forstmann et al., 2016), which represent the effects of moderators on behavior but not the information processing; it uses different mechanisms (e.g., processing speed, caution). We believe using these other approaches as the basis for this review would likely lead to similar results, and that the general results of this review would be useful for creating models of caffeine in these architectures.

Summary

This review is intended for a diverse audience, both people in pharmacokinetics and pharmacodynamics who wish to understand the effects of caffeine on cognition, and people modeling cognition and performance. Our review is intended to serve as a resource for both groups.

The remainder of the paper will address the interactions between caffeine consumption and physiological and cognitive abilities. Effects of caffeine on cognition that can be implemented in existing cognitive architectures will be focused on, as that is the goal of our review. Cognitive architectures will then be reviewed by describing existing features and applications. Finally, a

summary of the changes that can be made to these architectures to simulate caffeine's effects will be presented.

Physiological Effects of Caffeine

A drug's effects on the human body can be described through pharmacokinetics and pharmacodynamics. Pharmacokinetics describes the passage of a drug through the body; pharmacodynamics refers to the results of a drug's effects on the body as seen in behavioral and cognitive changes. Our intention is to summarize the pharmacokinetics as well as the pharmacodynamics of caffeine in cognitive architecture terms so that the effects can be modelled and influence models.

To identify the effects of caffeine, we looked for papers on caffeine and cognition through Google Scholar, and we found 46,000 papers. Using advanced search settings, we narrowed it down to those presenting new data (no reviews), and we found 6,800. Of these, 3,700 mention dose or dosage. When the search was narrowed to cognition and caffeine dosage ("cognition" with the exact phrase "caffeine dosage"), only 330 papers remained. Papers that only mention dose were discarded because they do not support creating parameterized models, but are used to indicate areas for further work. Unfortunately, not all of these papers contained relevant data, as many studies solely look at physical performance. The remaining subset of these papers (~55) and papers found in other ways including recommendations by commentators (~10) are used in our review. Additionally, PubMed and the Penn State University's library system including a librarian were used to find (~10) related papers. We limited the use of papers to those that specifically measured an aspect of cognition with caffeine consumption or those that complemented the work of such papers.

Although many studies have looked at the cognitive effects of caffeine, some of these studies have neglected the physiological impact of caffeine (James, 2014). Caffeine withdrawal and withdrawal reversal are important in the pharmacodynamics that result from caffeine consumption (Childs & De Wit, 2006; Rogers et al., 2013). Past studies that have found significant effects of caffeine on cognition have not always controlled for previous caffeine use, a misstep that would result in the comparison of withdrawal and restoration of caffeine levels (James, 1994; James & Rogers, 2005; Yeomans et al., 2002). By first describing the physiological effects of caffeine, we hope to clearly show what changes occur as a direct result of caffeine.

Pharmacokinetics

The binding of caffeine to receptors, its absorption through the body, and factors that affect these processes are important in understanding individual variation in the effects of caffeine consumption. An understanding of the time course of how caffeine is processed is necessary for modeling its effects in a computer simulation because the amount of caffeine present in the body increases with absorption, decays with a half-life, and because its effects (pharmacodynamics) are dependent on the current caffeine level. It is also important to note that interactions between heart rate variability, prefrontal neural function, and cognitive performance have been found (Thayer et al., 2009). These are physiological systems that are influenced by caffeine consumption, and suggest that the effects of caffeine on cognitive function may have interactions with other physiological systems as well. This is important to know if one later wishes to combine other moderators with caffeine. We provide a very brief review to help readers headed into this literature for the first time.

Caffeine binds to adenosine receptors, specifically the A_1 and A_{2a} receptors, and blocks these receptors that prevents them from binding adenosine (McLellan et al., 2016). The antagonistic effects of caffeine on adenosine and its agonistic effects on cortisol and epinephrine are important in understanding the physiological effects of caffeine and its effects on cognition. Antagonists work by inhibiting a drug or its receptor, while agonists activate a drug or its receptor. Adenosine receptors interact with both dopaminergic and glutamatergic transmission, as is seen in the neuromodulation of various neuropsychiatric functions (Shen & Chen, 2009). Adenosine's depressive effects in the central nervous system are antagonized by caffeine, resulting in some of the stimulating effects of caffeine. Caffeine's influence on adenosine function is seen in animal models as increased spontaneous electrical activity, an enhancement of neurotransmitter release, increased locomotor activity (increased energy in humans), and increased operant response rates (Garrett & Griffiths, 1997). Because caffeine crosses the blood-brain barrier, the upregulation of adenosine receptors in the brain is considered the cause of cognitive changes resulting from caffeine consumption.

Caffeine absorption is rapid and complete, within 45 minutes of administration. We have previously modeled caffeine absorption as an exponential process with a 7-minute absorption half-life (Ritter & Yeh, 2011). Therefore, it is effectively fully absorbed within approximately one hour, and peak concentration occurs in the saliva within 45 minutes of consumption and in blood after approximately two hours (Arnaud, 1987; J. L. Temple et al., 2017). Caffeine levels are considered to be evenly distributed across tissue and blood. Caffeine has a metabolic half-life of three to seven hours in adults and significantly longer in younger individuals (Bonati et al., 1982; J. L. Temple et al., 2017).

Figure 2 thus shows two major pharmacokinetic effects. In the single dose, the caffeine level rises as the caffeine that is ingested is absorbed. The absorption rate is an exponential curve, and its parameter is the half-life for absorption. Even as the caffeine is being absorbed, it is being metabolized and is being excreted in an exponential process, where the parameter is a half-life. The second line in Figure 2 shows the difference in absorbance of one versus multiple smaller doses of caffeine over time. In the multi-dose curve, as subsequent doses are consumed, the resulting peak grows smaller, but the active dose remains higher than the single dose. This pharmacokinetic model has been used in CaffeineZone™, an iPhone application that was developed to help individuals track their caffeine consumption in an effort to bring about awareness of how caffeine influences daily life, as well as to develop an understanding of caffeine (Ritter & Yeh, 2011).

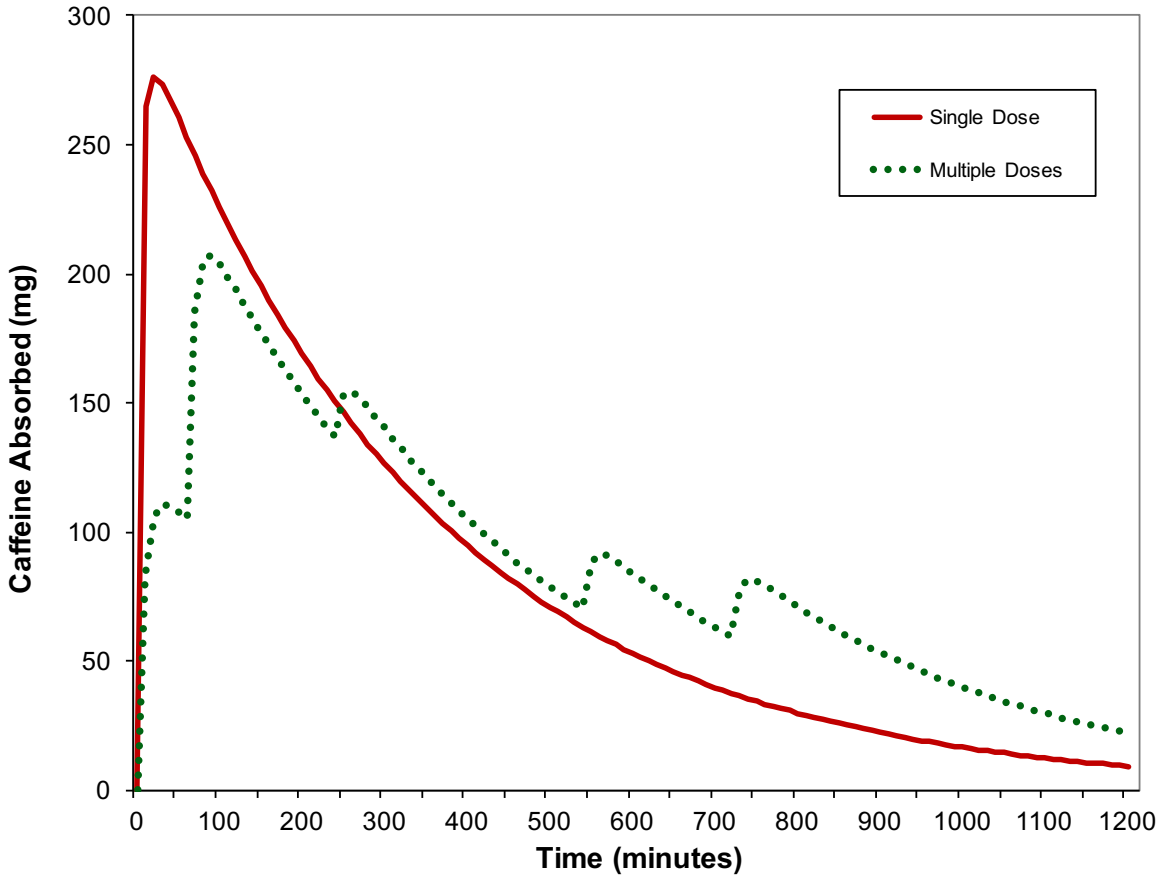


Figure 2. The pharmacokinetic time course of caffeine for single and multiple doses of the same total amount.

Age, genetics, exercise, pregnancy, disease, smoking, and medications are all known factors that influence one's caffeine metabolism, as shown in Table 1. The genetic variation of metabolism in individuals results in variations of half-life and clearance rates of caffeine, and variance in adenosine receptors leads to different central nervous system effects in individuals (J. L. Temple et al., 2017; Yang et al., 2010). Although there are understood to be differences in caffeine metabolism in children and young adults, age and gender have been found to have no influence on the effects of caffeine in adults (Amendola et al., 1998). Our review does not focus on these differences, but rather looks at the standard psychological measures of cognition (e.g., reaction

time, learning, memory) and how caffeine affects human performance assessed by these measures.

Table 1. Factors that influence caffeine metabolism

Factor	Change	Reference(s)
Age	Infants: 40 hr half-life Adults: 3-7 hr half-life	Temple et al. (2017) Rees, Allen, & Lader, (1999) Arnaud (1987)
Gender	No effect	Amendola et al. (1998)
CYP1A2 genetic variability	Individual differences in caffeine response	Temple et al. (2017) Yang et al. (2010)
Smoking	Nicotine doubles clearance rate	Temple et al. (2017) Arnaud (1987)

Pharmacodynamics on low-level and motor behavior

Caffeine's effects on the nervous system are mediated through changes in synaptic transmission and plasticity in the hippocampus (Lopes et al., 2019). Cortisol secretion is known to increase after caffeine administration in individuals at rest and those enduring mental stress, but this response has been shown to be reduced when an individual consumes caffeine on a daily basis (Lovallo et al., 2005).

Caffeine has been shown to have effects on motor performance that vary by type of exercise. Overall, caffeine appears to improve performance of endurance exercise through the reduced perception of effort and lowered sensations of pain, while also aiding in muscle strength exercises. This exertion was measured using the Borg scale (Watt & Grove, 1993). The ergogenic properties of caffeine have been studied for years and have been summarized in

multiple reviews. The majority of studies have found caffeine consumption to improve performance on endurance and on strength exercises (e.g., McLellan et al., 2016).

Arousal has a significant effect on cognition and behavior, and caffeine, as a stimulant, is known to influence arousal. Caffeine interacts with dopamine and adenosine receptors to produce neural (measured through EEG and ERP) as well as behavioral effects on fatigued individuals (Lorist & Tops, 2003). Additionally, caffeine has been shown to restore cognitive functioning in sleep restricted individuals (Lieberman et al., 2002; Wingelaar-Jagt et al., 2021). This application seems promising, as cognitive architectures have previously been used for modeling sleep restriction (Gunzelmann et al., 2015).

We do not have parameterized results for these motor and other cognitive effects, so we add them to our table of not-yet-parameterized effects, Table 2.

Table 2. Non-cognitive and not yet parameterized cognitive effects of caffeine.

Non-cognitive effects	**Cognitive effects**
Decreased physical fatigue (McLellan et al., 2016)	"Increased energy" (e.g., Lorist & Tops, 2003)
	Increased operant response rates (Garrett & Griffiths, 1997)
	Increased perceptual processing (Lorist & Tops, 2003)

Summary

It is the case that caffeine levels decay with time, so models of users that include caffeine will have to adjust the level of caffeine over time. The pharmacokinetics of caffeine in humans has been found to be relatively fixed, with a number of influencing factors. Some studies of these effects have not been well controlled, either through reporting dose instead of dosage or with inconsistent or subjective behavioral measures, and they therefore do not readily support

simulating the time course of effects of caffeine within a computational model. We did not find publications with the distribution of caffeine's half-life or the distribution of uptake rates. The rest of the review focuses on the pharmacodynamics of caffeine with respect to cognition, how caffeine influences cognition.

Cognitive Effects of Caffeine

Among the other pharmacodynamics of caffeine discussed above, the effects of caffeine on cognition have been studied for decades, which is reflected in the references. Caffeine has been shown to affect attention, learning, memory, and appraisal, depending on the dosage consumed. These effects can be measured through reaction time, semantic processing, vigilance, and working memory tasks. Nehlig (2010) comments on these effects, saying "caffeine apparently cannot be considered a 'pure' cognitive enhancer. Its indirect action … contributes in large part to its cognitive enhancing properties". The indirect effects of caffeine, such as changes in appraisal, can be seen through changes in behavior, cognition, mood, or affect. Many studies have found inconclusive results for how caffeine influences cognition (Miller et al., 1995) or provide only directional effects (e.g., Boff & Lincoln, 1988). A review of caffeine's effects on sleep-restricted military members showed only weak effects on a few aspects of cognition (Crawford et al., 2017). Due to the complexity of these effects, various aspects of cognition will be reviewed separately. We have selected these specific aspects because of their current representation in a broad range of cognitive architectures, including the Common Model of Cognition (Laird et al., 2017). These effects will then be summarized in a table to support implementation.

Attention

Caffeine acts as a stimulant, and its consumption leads to increased energy and alertness according to subjective reports (e.g., Lorist & Tops, 2003). Individuals use caffeine to aid in waking up in the morning and for increasing alertness during fatigue (Barone & Roberts, 1996). It is used on an individual and organizational level (e.g., Institute of Medicine, 2001; Naval Aerospace Medical Research Laboratory, 2000). The most common use of caffeine is to increase general task alertness. However, Lorist and Tops' review is based on multiple studies, and showed that a common scale of alertness has not been universally used for study comparisons, particularly non-vigilance attention.

A number of studies have shown that caffeine has specific effects on the attention system. Event-related potentials (ERPs), which are measures of electrical activity in the brain, have been used to measure the neural activity underlying attention during caffeine consumption. Both the latency and amplitude of ERPs are affected by caffeine, and these effects suggest that caffeine accelerates perceptual processing (Lorist & Tops, 2003). Additionally, sleep restriction has been shown to impact sustained attention and top-down processing as measured through ERPs (Kusztor et al., 2019). This neural measure allows cognitive changes to be more clearly described in a quantitative way than behavioral measures that have also been used to describe the effects of caffeine.

Visual reaction time can be observed as a behavioral measure of one's attention. A number of studies have used visual attention tasks to measure reaction time with different doses of caffeine (K. J. Anderson & Revelle, 1983; Frewer & Lader, 1991; Kenemans & Verbaten, 1998; Lieberman et al., 2002; Oei & Hartley, 2005; J. G. Temple et al., 2000; Wilhelmus et al., 2017). In general, reaction time was shown to decrease by about 3.5%, but the extent to which this

occurred varied in each study, and dosages and doses were not always well reported for use in plotting a dose-response curve. Other studies have shown that accuracy increases with caffeine consumption on the Stroop Task while reaction time does not reliably change (Dawkins et al., 2011).

The psychomotor vigilance task (PVT) is a commonly but not universally used measure of vigilance that is mentioned in a number of studies we refer to (Doty et al., 2017; Irwin et al., 2020; Kamimori et al., 2015; Killgore & Kamimori, 2020; Sanchis et al., 2020; Wilhelmus et al., 2017). In this task, the participant presses a button when a light appears. The light appears for a few seconds every 5-10 min. The main measure is not the reaction time but rather how many times the light is not noticed. This task has been shown by many studies to be a reliable measure of vigilance. However, not all studies use the PVT to measure vigilance, but instead rely on other visual vigilance tasks to measure attention. The PVT is also not considered to be practical for use in applied contexts and is only feasible in controlled laboratory settings (Basner et al., 2011).

The inconsistency of these results suggests that this area needs more research with specific caffeine dosages reported to fully understand caffeine's influence on cognition and to support modeling its effects on cognition in a quantitative way. This inconsistency could be due to the task being used or due to the nature of caffeine's effects at increasing dosages.

The Fatigue Avoidance Scheduling Tool (FAST) has been used to model the effects of caffeine on performance of the PVT (Hursh et al., 2004; Van Dongen, 2004). It has an equation that predicts alertness based on previous work-rest schedules. It is widely used in applied settings. It has been shown to be useful in past studies but has the limitations of the PVT itself (i.e., it predicts alertness in only general terms). Additionally, the effects of caffeine are not as easy to

359 model as fatigue because the effects of caffine varies based on the dose consumed, with time,
360 and with the user's weight).

361 ***Learning and Memory***

362 Both acute and chronic effects of caffeine consumption on memory have been studied through a
363 number of memory tasks, in both humans and animal models (e.g., Rizwan et al., 2017). While
364 the acute effects have generally been inconclusive, habitual caffeine use is suggested to be
365 beneficial to memory retention (McLellan et al., 2003, 2016). Animal models have shown that
366 caffeine improves memory and decreases memory decay, with implications of acting on memory
367 consolidation (Angelucci et al., 2002). These effects have also been seen in humans, particularly
368 in a study that looked at sleep-restricted Navy Seals that were given caffeine and performed
369 memory tasks (Lieberman et al., 2002).

370 Although many studies have reported improvement in learning tasks following caffeine
371 consumption, Nehlig (2010,pg. S86) boldly states that "caffeine facilitates learning in tasks in
372 which information is presented passively; in tasks in which material is learned intentionally,
373 caffeine has no effect". Active and passive learning is not a core concept in learning theory and
374 appears to not be addressed in the field of learning and memory from a cognitive psychology
375 perspective, but it is studied in educational psychology (J. R. Anderson, 2007; Chi et al., 1989).
376 This result does suggest that levels of caffeine should be measured if not controlled in training
377 studies, and caffeine might be suggested to learners of complex systems, particularly where
378 passive learning occurs. One study (Young & Molesworth, 2021) showed that a moderate dose
379 of caffeine (3 mg/kg) lead to faster learning than 0 and 5 mg/kg doses on a perceptual-motor-
380 cognitive task (Space Fortress). This result does suggest that levels of caffeine should be

measured if not controlled in training studies, and caffeine might be suggested to learners of complex systems.

The field of learning and memory has many studies of caffeine, but some studies show that just the expectation of caffeine intake can affect attention, which introduces a placebo effect that is not addressed in all studies (Dawkins et al., 2011). Other studies have found that caffeine affects the speed of vocalization to indirectly influence declarative memory performance. Kase et. al (2017) found, with a 400 mg dose of caffeine, a 10% increased utterance speed in the Trier Social Stressor Test (TSST, Kirschbaum, Pirke, & Hellhammer, 1993). Caffeine does not show any effects against the decline of memory from aging (Van Boxtel et al., 2003). This suggests that caffeine acts on the memory system through a different pathway than the normal consolidation pathway, because the normal consolidation pathway declines with age and there are no age effects seen in caffeine's cognitive effects.

The complexity of caffeine's effects on cognition surprisingly makes it a good fit for representation in a cognitive architecture, because cognition emerges from the fixed mechanisms of the architecture. The complicated influence of caffeine can be implemented through the behavioral changes that have been seen in these studies.

Appraisal

Appraisal is a personal assessment of how relevant a situation is to an individual, and whether gain or loss will arise from it (Maier et al., 2003). A person's appraisal of a situation shapes their emotional response, which is an important behavioral aspect that is observed through individual differences. This is a cognitive concept that should be included as a part of cognitive architectures, but typically is not one that is affected by caffeine use. Lazarus' (2006) theory of

appraisal considers one's goals to be important in both stress arousal and emotion. In the Trier Social Stressor Task (TSST), caffeine has been shown to influence appraisal (Kase et al., 2017). Behavioral measures such as the TSST are important in validating cognitive models that incorporate stress, arousal, and affect.

The effects of caffeine consumption on mood have been measured in a number of studies, and it has been shown that caffeine positively influences mood even without abstinence from caffeine (Warburton, 1995). This suggests that an architecture that incorporates emotional and cognitive processing would be beneficial in modeling caffeine's effects. A computational model has been developed that implements appraisal and emotional processing in decision making (Gratch & Marsella, 2004). Some aspects of this architecture could be implemented to model caffeine's effects on cognition by including caffeine (e.g., coffee) as a resource during appraisal.

Introversion vs. extroversion

An individual's natural level of arousal is of interest when considering the effects of caffeine because caffeine acts through changes in arousal. It is believed that introverts have a higher baseline arousal level than extroverts have, which itself leads to differences in cognitive efficiency (Ackerman, 2012). Introverts and extroverts exhibit differences in vigilance tasks as a result of differences in arousal and this is reflected in individual responses to caffeine (Boff & Lincoln, 1988).

Extroverts show greater behavioral changes in response to caffeine consumption. This finding suggests that considering individual differences, particularly with respect to introversion, is also necessary in considering the effects of caffeine on an individual's cognitive processes, and intro/extroversion of participants is reported in relatively few papers (Ackerman, 2012; Boff & Lincoln, 1988; Keister & McLaughlin, 1972; Revelle et al., 1980).

Sleep restriction

It has been suggested that caffeine can have greater effects on individuals who are under significant stress. The cognitive effects of sleep restriction have been shown to vary among individuals, but are typically considered to be due to the effect of fatigue (Durmer & Dinges, 2005). Sleep restriction is considered to be the most frequent stressor that is present in individuals who consume caffeine.

Sleep restriction lowers an individual's arousal level, which magnifies the effects of caffeine on the body. This has been studied by measuring the cognitive function of individuals who are deprived of sleep and subsequently given caffeine (Lieberman et al., 2002; Tharion et al., 2003). These dynamics have also been modeled with cognitive architectures, specifically using ACT-R to predict the temporal dynamics resulting from fatigue (Walsh et al., 2017). The effects of sleep deprivation on motor tasks have also been modeled (Bolkhovsky et al., 2018). Studies that look at caffeine's effects on sleep restricted individuals have shown that the recovery of cognitive function is caffeine dose-dependent (Beaumont et al., 2001; Kamimori et al., 1995, 2015). This recovery does not persist through five days of sleep restriction, however (Doty et al., 2017). Other studies have shown that caffeine does not allow functioning to recover to its normal capacity, though it does improve functioning overall during sleep restriction (Killgore & Kamimori, 2020). Risky decision making is not recovered in this capacity and is shown to be especially sensitive to sleep restriction (Killgore et al., 2007).

Sleep loss and its cognitive effects have been studied through simulation to help inform the field about sleep scheduling for improved safety of shift workers (Gunzelmann et al., 2009, 2015; Walsh et al., 2017). Because caffeine is often consumed by individuals who are sleep restricted

or fatigued, the results from the sleep restriction literature are necessary to consider when studying the effects of caffeine.

The effects of both sleep restriction and caffeine consumption on cognitive functioning have been modeled using a tool called "2B-Alert Web" (Reifman et al., 2016). This tool uses inputs of an operator's sleep schedule and caffeine consumption to predict measures including response time over the course of days. This simulation is based on work that has created mathematical models of cognitive performance in response to caffeine consumption (Ramakrishnan et al., 2013, 2014). While this simulation focuses on caffeine's effects in combination with sleep restriction, it can still be used as a starting point for the implementation of caffeine's effects on cognition in a cognitive architecture.

The results from these data and these models of sleep restriction's effect on cognition show that there are also strong circadian (time-of-day) effects on cognition (i.e., less alert after midnight, afternoon dip). This suggests that the effects of circadian rhythms should be included in an architecture when caffeine's effects are included.

Self-reported alertness levels

Previous studies have found that alcoholics report feeling more relaxed by simply being offered a drink, before even consuming it (Leigh & Stacy, 1991). The anticipation of an addictive substance can also be considered in the consumption of caffeine. It has been shown that the expectation of coffee can improve both accuracy and reaction time in cognitive tasks, while caffeine consumption itself only leads to improvements in accuracy (Dawkins et al., 2011). This change as a result of expectation is also suspected to apply to an individual observing a cup of coffee being poured. This finding suggests that the effects of caffeine on cognition may be magnified by changes in appraisal or mood. This further supports the idea that affect and mood

are necessary additions to cognitive architectures and models. It is possible that the visual/audio modules of the ACT-R/Φ architecture could, through expectations that are raised for consuming caffeine, provoke changes in response to the presentation of coffee to an individual separately from the effects that occur as a result of caffeine consumption (Dancy & Schwartz, 2017). This would be another pathway between cognition and physiology.

Summary

Table 3 shows a summary of the cognitive measures in which an effect of caffeine has been observed. The physiological effects of caffeine can be represented as changes in the levels of adenosine and cortisol, with dosage and previous caffeine use as reliable predictors of these changes. The cognitive effects of caffeine consumption are represented by decreases in reaction time and an improvement in memory tasks, although these measures seem to be particularly dependent on the level of consumption. Ideally, these changes arise from changes at the physiology level. The changes seen in learning and memory make the use of a cognitive architecture an appropriate choice for implementation.

Few papers have established equations that describe the effects of caffeine on cognition, which is necessary for application in a cognitive architecture. The effects of caffeine following sleep restriction have been modeled, and these equations may shed light on the equations that need to be used (Ramakrishnan et al., 2013). In general, dosage curves suggest that as dosage increases improvement is always followed by a decrease in performance, resulting in an inverted U-shaped curve. Low dosages have smaller effects improving performance, and too high a dosage leads to a decrease in performance. Overall, our review of the literature has found incomplete descriptions of caffeine's cognitive effects, which has been seen in other reviews as well (Cornelis et al., 2020).

Table 3. Summary of Cognitive Measures

Cognitive measure	Dose/dosage	Effect of caffeine	Referenced studies
Attention	250 mg	10% faster completion	Frewer & Lader (1991)
	60 mg	Increased attention	Wilhelmus et al. (2017)
Reaction time	200 mg & 300 mg	Decrease	Lieberman et al. (2002) Nehlig (2010)
		Tenfold decrease in errors	Rizwan et al. (2017)
Vigilance	200 – 300 mg	Increase, 40-50% hit increase; 60% false alarm decrease	McLellan, Caldwell & Lieberman (2016) Lieberman et al. (2002) Sanchis, Blasco, Luna, & Lupiáñez (2020)
	60 mg	Improved sustained attention	Wilhelmus et al. (2017)
Memory load task	4 mg/kg	Improved low-load performance Hindered high-load performance	K. J. Anderson & Revelle (1983)
Appraisal	200 mg	Increased challenge appraisals	Kase et al. (2017) table 3
	400 mg	Increased threat appraisals	
Memory retention for spatial memories (in rat model)	0.3 – 10 mg/kg	Improved retention	Angelucci et al. (2002)

Implementation of Caffeine in Cognitive Architectures

We next consider how to implement these effects in computational cognitive models for applications and to create a more unified theory of performance. Cognitive architectures as models of human mental processing have been developed in an effort to form a unified theory of cognition (Kotseruba & Tsotsos, (in press), 2020; Newell, 1990). Architectures such as ACT-R (Anderson, 2007) and Soar (Laird, 2012) simulate cognitive behavior with a focus typically on speed of simulation or performance accuracy. These architectures use fixed mechanisms to generate behavior from their initial knowledge, goals, and perceptual inputs They are typically

realized as a computer program. When processes change, however, these fixed mechanisms have to change (or represent the change in processing).

To implement these changes, a simple method is to summarize changes to cognition that arise from a moderator as a fixed set of changes. The use of overlays to represent moderators is a useful but incomplete model (Ritter et al., 2012). The inclusion of effects in an underlying theory of physiology, as is seen in ACT-R/Φ, allows more parameters that can express individual differences and better model moderators of behavior and how these moderators interact.

These architectures are useful for scientific reasons (Newell, 1990). They provide a way to hold together what we know about human behavior. Models of users created in an architecture by adding task knowledge have also been seen as useful components for engineering design tools that help design systems using models of individuals (e.g., Elkind et al., 1989; McMillan et al., 1989; Pew & Mavor, 2007; Ritter, 2019; Tehranchi et al., 2023) and groups (Pew & Mavor, 1998), as a way to model their behavior.

Two aspects of caffeine need to be modeled or available to the model. First, that caffeine level will change over time (pharmacokinetics), and second, how the level of caffeine influences cognition (pharmacodynamics). We will need both aspects to model the effect of caffeine on tasks or a series of tasks that last more than 10 minutes, to measure and represent the effect of caffeine's half-life. The dual aspect of the caffeine dosage and that caffeine level changes over time makes this work difficult to perform, and the data must be prepared and collected with these aspects (recording dosage and time course of the experiment) in mind.

Currently, data are often not collected with this theory in mind. Dosages are often not provided; the times of caffeine administration are not always reported; and the time course of behavior is

rarely reported (e.g., summary measures at 10, 20, 30 min. into the experiment because caffeine levels vary over time due to uptake and excretion). To better simulate the effects of caffeine we need further data and analysis. This report provides some of the parameters of interest that should be recorded or controlled in future studies of caffeine.

Existing models of caffeine

A model of fatigue and the sleep/wake cycle was modified to reflect the effects of caffeine consumption with the inclusion of individual differences (Puckeridge et al., 2011). This model is based on sleep-active neurons of the hypothalamus, and it can predict performance on an individual basis.

Models of sleep restriction and fatigue that use both biomathematical models and cognitive architectures currently exist (Gunzelmann et al., 2009, 2015). Biomathematical models of sleep and sleep restriction have been able to implement the effects of caffeine on cognitive performance (Rajdev et al., 2013; Ramakrishnan et al., 2013, 2016). While these models contribute to our understanding, they are not a suitable replacement for being able to simulate how caffeine consumption affects the details of task performance. Caffeine consumption alone must be studied before it can be accurately combined with the effects of fatigue.

Table 4 shows what is needed from research on caffeine consumption to create a quantitative theory of caffeine's effect on cognition. To create a baseline pharmacokinetic equation, we need studies in which dosages are reported. To reduce variance in participants, we need to know participants' previous caffeine use and sleep schedule. To create testable predictions, we need quantitative reports of performance on a task, preferably with at least three data points if the data have or describe a curve, for example, an inverted U-shaped curve. Finally, we will need a

548 replicable way to measure alertness and attention because one of caffeine's major effects is self-
549 reported alertness and subjective and objective measures of attention.

Table 4. What is Needed from Caffeine Studies to Create Quantitative Theories of Caffeine

550 Dosages (measured in mg/kg)
551 Previous caffeine usage (to control for withdrawal) of participants
552 Recent sleep data of participants
553 Quantitative reports of performance including range and standard deviation
554 Distributions of measures
555 Task appraisal, alertness, and related measures, pre- and post-task

556

557 ***Implementing caffeine in the Common Model of Cognition***

558 Because cognitive architectures vary in their implementation of moderators, we will focus on
559 using the CMC, which can be applied more broadly. This approach (perhaps best described as a
560 framework for describing architectures) describes architectures as containing perceptual and
561 motor components, a working memory, declarative long-term memory and procedural long-term
562 memory (Laird et al., 2017). All of these components are represented uniquely in various
563 cognitive architectures, but this theory allows modeling the effects of caffeine on cognitive
564 processes without first implementing it within an architecture.

565 One way to model caffeine is to just note its effects. This has been done for some static effects
566 (Ritter et al., 2012). Caffeine has interactions, for example, with nicotine, as noted above. So, we

will want a way to characterize those interactions across moderators of cognition. We will need a more complex representation of physiology.

Luckily, such models of physiology exist. These effects can be summarized as a set of changes to cognition that can be implemented in a cognitive and physiological architecture that can be used to predict performance for use in system design and will be discussed next. We include one example implementation of the CMC with caffeine included.

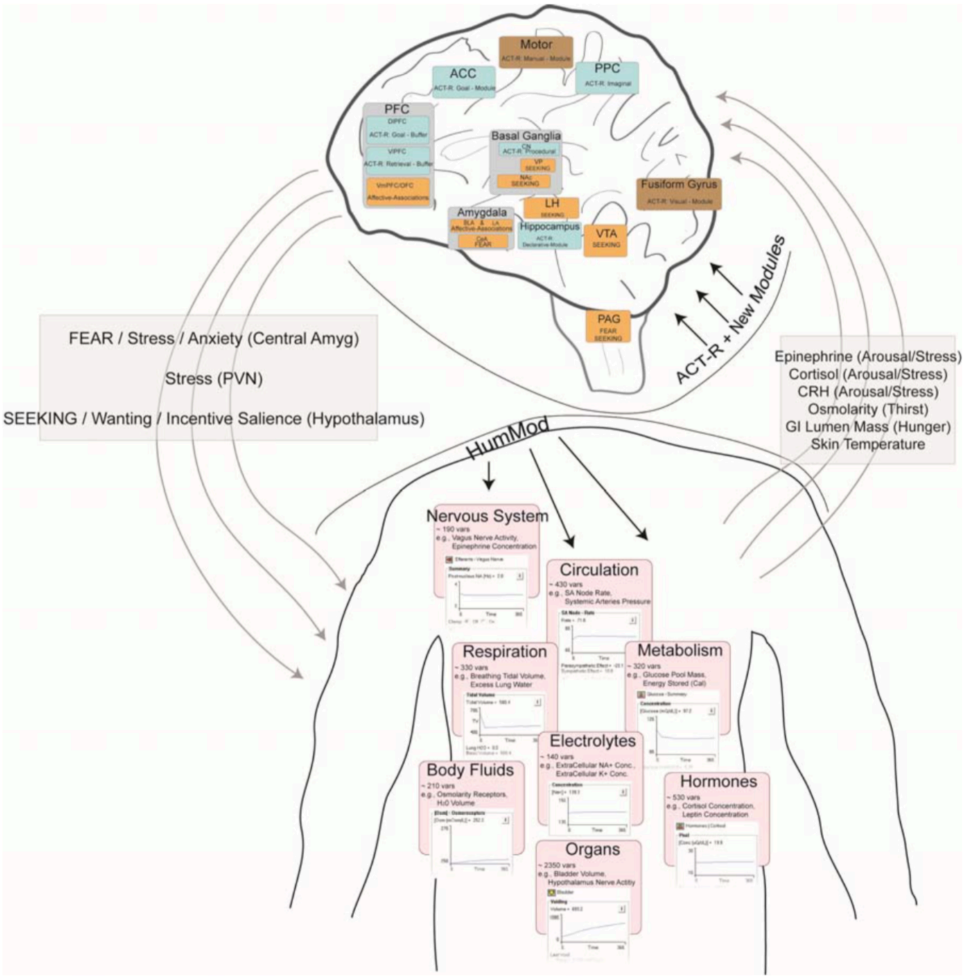
HumMod

HumMod is a physiological simulator that uses initial state, environmental inputs, and the effect of time to model and simulate changes in an individual's physiological state over time (Hester et al., 2011, 2019; Matejak & Kofranek, 2015). This simulation includes around 5,000 variables and equations representing the components of the major biological systems in the human body, including the circulation, metabolism, and respiratory systems with variables that allow these systems to dynamically respond to external stimuli. This model can simulate an individual's physiology over time and can be modified by pathophysiological states. While this model only allows a limited number of pharmacological agents to be administered, any drug can be simulated if the pharmacodynamic and pharmacokinetic relationships are understood as equations. A simulator such as HumMod would be useful in modeling caffeine's effects, as seen in Figure 3. We explain an example system to show what a model of caffeine can look like that combines a cognitive architecture with a physiological model (Hester does not claim it to be an architecture, but it might best be described as a physiological architecture).

ACT-R/Φ

ACT-R/Φ is an extension of ACT-R that connects a cognitive architecture to a physiological simulator (HumMod), as is shown in Figure 3. It extends the CMC theory in Figure 1. This approach and system allows cognitive effects on physiology as well as physiological effects on cognition to be simulated (Dancy, 2014; Dancy et al., 2015). ACT-R/Φ provides a way for caffeine's effects to be modelled in a cognitive architecture.

Currently, as ACT-R/Φ in Figure 3 shows, cognition can modify several aspects of the physiology system; and the physiology can influence the processing mechanisms of cognition. Links between hydration and micturition from physiology have been made to the cognition components; and there are links from cognition stress and affect of images to stress variables in the physiology simulator.



598

599 **Figure 3. ACT-R/Φ combines the modules from HumMod and ACT-R.**

600 **Used from Dancy (2014, Figure 3-5) with permission.**

601 The interactions of the physiological module and the ACT-R modules are even more complex
602 than this figure illustrates. The affect system has been shown to have effects on motor and
603 speech, which is not yet represented. This influence will be important to consider when modeling
604 caffeine's influence on cognition.

605 ***A Design for Including Caffeine in Architectures***

606 Although ACT-R/Φ contains the necessary overlay features for implementing caffeine
607 consumption into a cognitive architecture, changes to its current parameters need to be modified

for this to be done more accurately. The production system within ACT-R interacts with the efferent and physio-substrate buffers to influence the physiology within HumMod, shown in Figure 4. The influence of caffeine on cognition would have to be added to the physio-substrate buffer to influence the physiological factors that are affected by caffeine. The effects on motor/sensory systems would be added to the efferent buffer.

A benefit of using the Common Model of Cognition to model the effects of caffeine is that theoretical ideas can be implemented without first being realized in a complete cognitive architecture (Laird et al., 2017). Table 5 shows future directions using this approach.

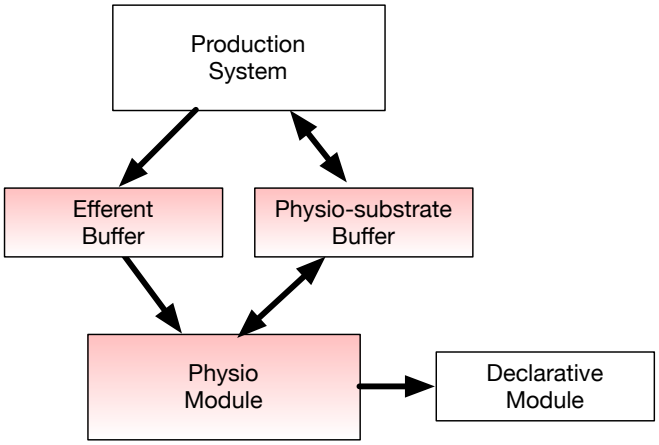


Figure 4. How HumMod interacts with ACT-R in ACT-R/Φ, and how it could work with the CMC. Adapted from Dancy et al. (2015).

Table 5. Changes to be implemented in ACT-R/Φ, showing how caffeine influences physiology and cognitive constructs.

Feature	Current Implementation	New Implementation
Cortisol	Stress	Increased cortisol upon caffeine consumption
Adenosine	None	Adenosine receptors blocked by caffeine
Epinephrine	Included as a catecholamine	Increased epinephrine upon caffeine consumption
Visual model	Shifts attention based on constraints	Faster shift with caffeine
Declarative memory chunks	Chunks have defined speed and retrieval accuracy	Smaller chunks have faster and more accurate retrieval with caffeine

Conclusion

Research on caffeine consumption in humans has accumulated some results on how caffeine affects physiology and behavior. The physiological effects of caffeine can vary based on individual differences in genetics, metabolism, and daily caffeine intake. This can, in turn, result in differences in caffeine's effects on cognition. The average effects of caffeine can be summarized as changes in cortisol and adenosine, as well as changes in attention and memory. These changes can be modeled through the use of a cognitive architecture that simulates physiological as well as cognitive change. This modeling can be useful for scientific and engineering uses. We have described and summarized these changes in a manner that will ease the implementation of caffeine's effects on the cognitive and physiological simulators that are currently in use.

Limitations

Our review discussed the physiological and cognitive effects of caffeine that have been found in past studies. This resulted in the discovery of factors that have not previously been included in

cognitive architectures. For this reason, several parameters need to be introduced into the existing cognitive architectures to model the effects of caffeine. Additionally, the HumMod physiological simulator does not have all of the parameters needed to represent the pharmacodynamics of caffeine use. These variables must be incorporated before caffeine use can be modeled in this simulation.

The experiments that were used did not have consistent measurements of caffeine intake and cognitive variables, so comparison across them is difficult. We noted some ways to improve studies in this area, including recording participants' weights.

Forstman, Ratcliff, and Wagenmakers (2016) note several aspects of behavior that a review like this should address, including response bias and caution. These are aspects of behavior that are not included in the CMC or its related architectures directly that other approaches do study more directly. We also note that these aspects of cognition (e.g., response bias) that change with caffeine are not always studied either, and that will also have to be addressed by studies on caffeine.

We suggest new requirements for caffeine research. Papers should not just report that caffeine had an effect (with ANOVA), but also report percent changes, effect sizes, and standard deviations. These are needed for the computational modeling of these effects. Similarly, papers should report dosages, not just doses, because dosages allow computational models and thus predictions (Table 3). Planners and designers need not only to know that an operator's attention is increased, but also need to know by how much. Reduced variance in predictions also occurs with the reporting of dosages because caffeine's effects are dosage not dose related (Julien, 2001). For modeling and applying this data, knowing the data's distribution, such as for

halflives, is also helpful, for example, for building scheduling and system testing tools (Hursh et al., 2004; Pew & Mavor, 2007; Van Dongen, 2004).

Future Work

For our specific implementations, the HumMod architecture must be modified to include cortisol and adenosine parameters to represent the changes that occur from caffeine consumption. Additionally, an ACT-R implementation could add vigilance and appraisal to better represent how caffeine influences behavior. ACT-R's representation of brain regions could also be made more detailed to represent the role of hormones and receptors. Once these variables are established, caffeine use can be modeled more accurately alongside cognitive tasks.

Our review found that there are several details that future studies of caffeine must consider to accurately measure the effects of caffeine in a way that caffeine's time course and effects can be implemented in a model or even summarized. These include recording participants' weights, controlling and recording the time course of the experiment, and taking baseline measurements before caffeine administration.

This approach to understanding caffeine's effects can also serve as a template for understanding and modeling other moderators of behavior for predicting performance. It could be useful to create similar reviews for nicotine and other stimulants, and also for other behavioral moderators.

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