

1 **Caffeine and Cognition: A Cognitive Architecture-Based Review**

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

24 **Abstract**

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

40 *Keywords:* Caffeine, Cognition, User Modeling, Cognitive Architecture

41

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

46

48 **Introduction**

49 Caffeine is the most commonly used psychoactive drug. There are numerous reviews of its
50 effects from both theoretical and applied perspectives (Ahluwalia & Herrick, 2015; Amendola et
51 al., 1998; Institute of Medicine, 2001; Lorist & Tops, 2003; McLellan et al., 2016; Nehlig, 2010;
52 Poole et al., 2017; Rees et al., 1999; Smith, 2002; J. L. Temple et al., 2017; Wingelaar-Jagt et al.,
53 2021; Young & Molesworth, 2011). The existing reviews focus primarily on caffeine's
54 physiological effects and present them as non-parameterized effects. These reviews note that
55 caffeine ingestion leads to changes in cognition and in motor physiology but do not focus on
56 these changes nor provide quantitative details that could be used to create a model that simulates
57 the effect of caffeine on cognition for use in designing interfaces (e.g., McMillan et al., 1989;
58 Pew & Mavor, 2007) or for creating agents in simulations that have circadian rhythms (e.g., Pew
59 & Mavor, 1998). Our review focuses on caffeine's effects on cognition and uses a theoretical
60 framework to hold and summarize the results.

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

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

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

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

87 *Caffeine*

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

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

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

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

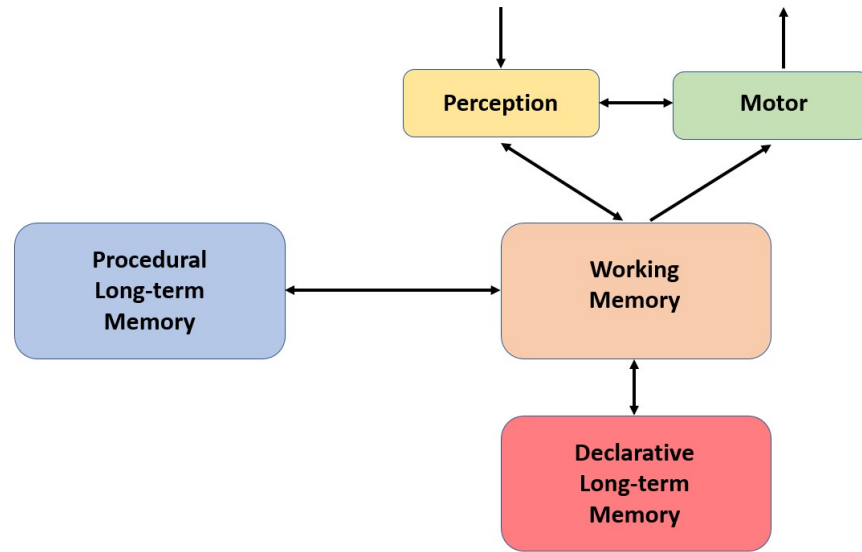
114 Caffeine is not dose but dosage dependent (Smith, 2002). While dose refers to the mass (in mg)
115 of a drug consumed within a serving, dosage refers to the mass per kilogram (mg/kg) of body
116 weight for the individual consuming it, which depicts the proportional amount of the drug
117 metabolized by the consumer. For example, a person weighing 85 kg (187 lbs) who consumes
118 591 ml (20 fluid ounces) of coffee prepared from ground roasted coffee will have a 340 mg dose
119 of caffeine, with a dosage of 4 mg/kg. Most studies that we have found use dose, a measurement
120 that is underspecified when individual body weight is not reported, and this lack of specification
121 should be kept in mind when considering differences in data reporting the effects of caffeine
122 consumption. Dosage is a better measure to report than dose because dosage takes into account
123 body weight, a factor that affects the action of most drugs, including caffeine. Before proceeding
124 to the review, we explain the theory that we will use to summarize the results.

125 *Cognitive and Physiological Architectures*

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

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

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



138
139 **Figure 1. Structural representation of the Common Model of Cognition**
140 **(formerly, the Standard Model of Cognition, based on Laird et al., 2017).**

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

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

151 has occurred as biological studies uncover interactions between cognition and physiological
152 measures. Implementing the effects of common factors such as caffeine or stress in combined
153 cognitive/physiological simulations is a way to understanding these complex interactions. We
154 believe that to effectively model the changes of cognition that occur due to caffeine
155 consumption, we must understand the relevant underlying physiological changes that take place.
156 Other architectures or frameworks for modeling cognition could also be used to summarize the
157 results. These approaches include neural networks (e.g., O'Reilly 2000), which represent the
158 mechanisms on a lower level, and sequential sampling models (e.g., see Forstmann et al., 2016),
159 which represent the effects of moderators on behavior but not the information processing; it uses
160 different mechanisms (e.g., processing speed, caution). We believe using these other approaches
161 as the basis for this review would likely lead to similar results, and that the general results of this
162 review would be useful for creating models of caffeine in these architectures.

163 *Summary*

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

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

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

174 **Physiological Effects of Caffeine**

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

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

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

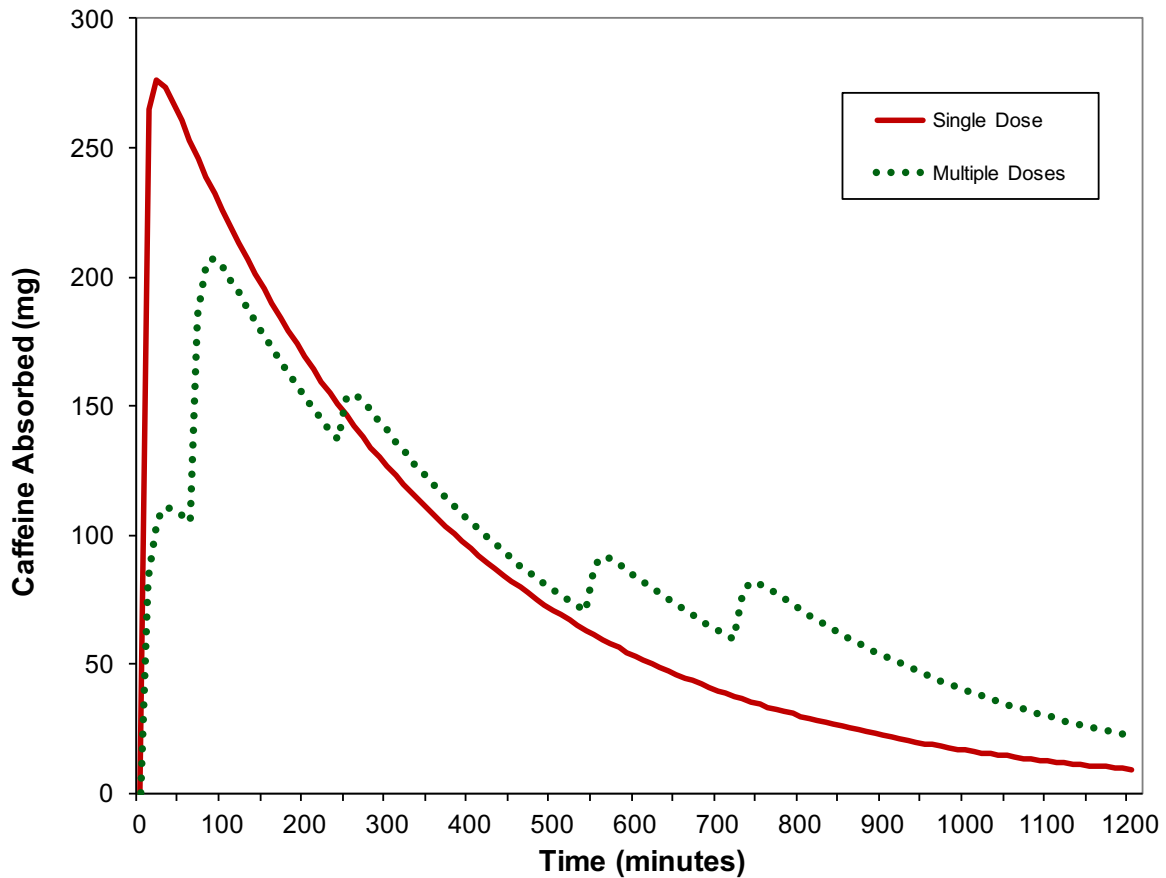
203 *Pharmacokinetics*

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

216 Caffeine binds to adenosine receptors, specifically the A₁ and A_{2a} receptors, and blocks these
217 receptors that prevents them from binding adenosine (McLellan et al., 2016). The antagonistic
218 effects of caffeine on adenosine and its agonistic effects on cortisol and epinephrine are
219 important in understanding the physiological effects of caffeine and its effects on cognition.
220 Antagonists work by inhibiting a drug or its receptor, while agonists activate a drug or its
221 receptor. Adenosine receptors interact with both dopaminergic and glutamatergic transmission,
222 as is seen in the neuromodulation of various neuropsychiatric functions (Shen & Chen, 2009).
223 Adenosine's depressive effects in the central nervous system are antagonized by caffeine,
224 resulting in some of the stimulating effects of caffeine. Caffeine's influence on adenosine
225 function is seen in animal models as increased spontaneous electrical activity, an enhancement of
226 neurotransmitter release, increased locomotor activity (increased energy in humans), and
227 increased operant response rates (Garrett & Griffiths, 1997). Because caffeine crosses the blood-
228 brain barrier, the upregulation of adenosine receptors in the brain is considered the cause of
229 cognitive changes resulting from caffeine consumption.

230 Caffeine absorption is rapid and complete, within 45 minutes of administration. We have
231 previously modeled caffeine absorption as an exponential process with a 7-minute absorption
232 half-life (Ritter & Yeh, 2011). Therefore, it is effectively fully absorbed within approximately
233 one hour, and peak concentration occurs in the saliva within 45 minutes of consumption and in
234 blood after approximately two hours (Arnaud, 1987; J. L. Temple et al., 2017). Caffeine levels
235 are considered to be evenly distributed across tissue and blood. Caffeine has a metabolic half-life
236 of three to seven hours in adults and significantly longer in younger individuals (Bonati et al.,
237 1982; J. L. Temple et al., 2017).

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



249

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

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

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

262 **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)

263

264 ***Pharmacodynamics on low-level and motor behavior***

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

270 Caffeine has been shown to have effects on motor performance that vary by type of exercise.

271 Overall, caffeine appears to improve performance of endurance exercise through the reduced
272 perception of effort and lowered sensations of pain, while also aiding in muscle strength
273 exercises. This exertion was measured using the Borg scale (Watt & Grove, 1993). The
274 ergogenic properties of caffeine have been studied for years and have been summarized in

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

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

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

286 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)

287 ***Summary***

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

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

297 **Cognitive Effects of Caffeine**

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

314 *Attention*

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

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

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

337 occurred varied in each study, and dosages and doses were not always well reported for use in
338 plotting a dose-response curve. Other studies have shown that accuracy increases with caffeine
339 consumption on the Stroop Task while reaction time does not reliably change (Dawkins et al.,
340 2011).

341 The psychomotor vigilance task (PVT) is a commonly but not universally used measure of
342 vigilance that is mentioned in a number of studies we refer to (Doty et al., 2017; Irwin et al.,
343 2020; Kamimori et al., 2015; Killgore & Kamimori, 2020; Sanchis et al., 2020; Wilhelmus et al.,
344 2017). In this task, the participant presses a button when a light appears. The light appears for a
345 few seconds every 5-10 min. The main measure is not the reaction time but rather how many
346 times the light is not noticed. This task has been shown by many studies to be a reliable measure
347 of vigilance. However, not all studies use the PVT to measure vigilance, but instead rely on other
348 visual vigilance tasks to measure attention. The PVT is also not considered to be practical for use
349 in applied contexts and is only feasible in controlled laboratory settings (Basner et al., 2011).

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

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

359 model as fatigue because the effects of caffeine 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

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

383 The field of learning and memory has many studies of caffeine, but some studies show that just
384 the expectation of caffeine intake can affect attention, which introduces a placebo effect that is
385 not addressed in all studies (Dawkins et al., 2011). Other studies have found that caffeine affects
386 the speed of vocalization to indirectly influence declarative memory performance. Kase et. al
387 (2017) found, with a 400 mg dose of caffeine, a 10% increased utterance speed in the Trier
388 Social Stressor Test (TSST, Kirschbaum, Pirke, & Hellhammer, 1993). Caffeine does not show
389 any effects against the decline of memory from aging (Van Boxtel et al., 2003). This suggests
390 that caffeine acts on the memory system through a different pathway than the normal
391 consolidation pathway, because the normal consolidation pathway declines with age and there
392 are no age effects seen in caffeine's cognitive effects.

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

397 *Appraisal*

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

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

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

414 *Introversion vs. extroversion*

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

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

426 *Sleep restriction*

427 It has been suggested that caffeine can have greater effects on individuals who are under
428 significant stress. The cognitive effects of sleep restriction have been shown to vary among
429 individuals, but are typically considered to be due to the effect of fatigue (Durmer & Dinges,
430 2005). Sleep restriction is considered to be the most frequent stressor that is present in
431 individuals who consume caffeine.

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

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

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

450 The effects of both sleep restriction and caffeine consumption on cognitive functioning have
451 been modeled using a tool called “2B-Alert Web” (Reifman et al., 2016). This tool uses inputs
452 of an operator’s sleep schedule and caffeine consumption to predict measures including response
453 time over the course of days. This simulation is based on work that has created mathematical
454 models of cognitive performance in response to caffeine consumption (Ramakrishnan et al.,
455 2013, 2014). While this simulation focuses on caffeine’s effects in combination with sleep
456 restriction, it can still be used as a starting point for the implementation of caffeine’s effects on
457 cognition in a cognitive architecture.

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

462 *Self-reported alertness levels*

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

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

476 ***Summary***

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

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

494 **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)

495

496 **Implementation of Caffeine in Cognitive Architectures**

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

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

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

511 These architectures are useful for scientific reasons (Newell, 1990). They provide a way to hold
512 together what we know about human behavior. Models of users created in an architecture by
513 adding task knowledge have also been seen as useful components for engineering design tools
514 that help design systems using models of individuals (e.g., Elkind et al., 1989; McMillan et al.,
515 1989; Pew & Mavor, 2007; Ritter, 2019; Tehranchi et al., 2023) and groups (Pew & Mavor,
516 1998), as a way to model their behavior.

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

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

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

530 *Existing models of caffeine*

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

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

542 Table 4 shows what is needed from research on caffeine consumption to create a quantitative
543 theory of caffeine's effect on cognition. To create a baseline pharmacokinetic equation, we need
544 studies in which dosages are reported. To reduce variance in participants, we need to know
545 participants' previous caffeine use and sleep schedule. To create testable predictions, we need
546 quantitative reports of performance on a task, preferably with at least three data points if the data
547 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

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

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

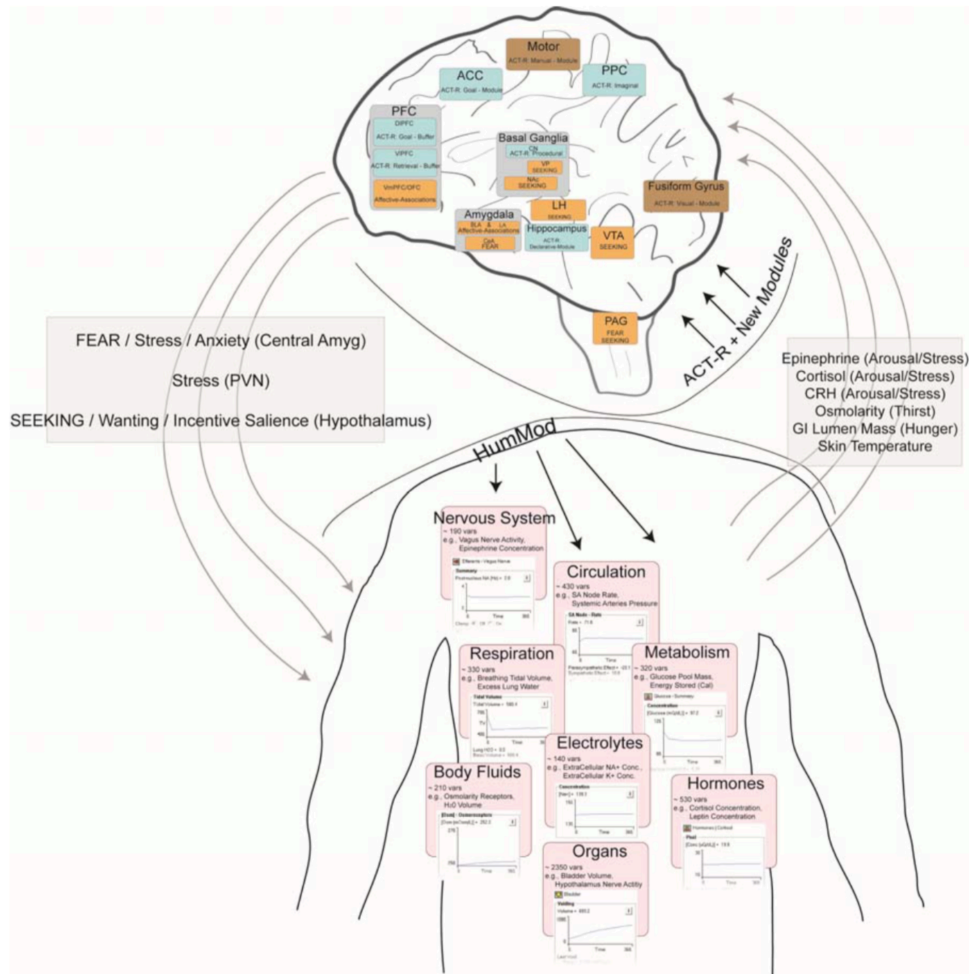
573 *HumMod*

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

587 ***ACT-R/Φ***

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

593 Currently, as ACT-R/Φ in Figure 3 shows, cognition can modify several aspects of the
594 physiology system; and the physiology can influence the processing mechanisms of cognition.
595 Links between hydration and micturition from physiology have been made to the cognition
596 components; and there are links from cognition stress and affect of images to stress variables in
597 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.**

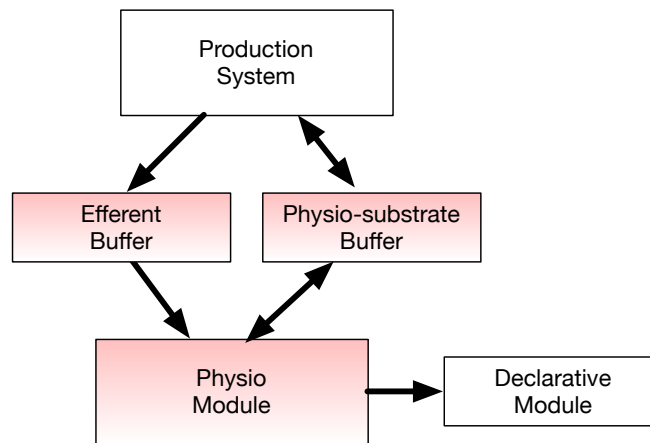
601 The interactions of the physiological module and the ACT-R modules are even more complex
602 than this figure illustrates. The affect system has been shown to have effects on motor and
603 speech, which is not yet represented. This influence will be important to consider when modeling
604 caffeine's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

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

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



616

617 **Figure 4.** How HumMod interacts with ACT-R in ACT-R/ Φ , and how it could work with the

618

CMC. Adapted from Dancy et al. (2015).

619 **Table 5.** Changes to be implemented in ACT-R/ Φ , showing how caffeine influences physiology
 620 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

621

622 **Conclusion**

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

633 ***Limitations***

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

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

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

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

650 We suggest new requirements for caffeine research. Papers should not just report that caffeine
651 had an effect (with ANOVA), but also report percent changes, effect sizes, and standard
652 deviations. These are needed for the computational modeling of these effects. Similarly, papers
653 should report dosages, not just doses, because dosages allow computational models and thus
654 predictions (Table 3). Planners and designers need not only to know that an operator's attention
655 is increased, but also need to know by how much. Reduced variance in predictions also occurs
656 with the reporting of dosages because caffeine's effects are dosage not dose related (Julien,
657 2001). For modeling and applying this data, knowing the data's distribution, such as for

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

660 *Future Work*

661 For our specific implementations, the HumMod architecture must be modified to include cortisol
662 and adenosine parameters to represent the changes that occur from caffeine consumption.

663 Additionally, an ACT-R implementation could add vigilance and appraisal to better represent
664 how caffeine influences behavior. ACT-R's representation of brain regions could also be made
665 more detailed to represent the role of hormones and receptors. Once these variables are
666 established, caffeine use can be modeled more accurately alongside cognitive tasks.

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

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

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