Cognitive Apparatus Simulation Model (CASM) – Formal Specification

Overview: CASM treats perception and cognition as arising from a single embodied generative system that integrates the brain with bodily networks (enteric nervous system, endocrine, immune, and microbiota). In this view, the brain uses hierarchical models to predict both external sensations and internal bodily signals. Recent work emphasizes that the "gut complex" – comprising enteric nerves, gut hormones, immune cells and microbes – is integral to cognition [1] [2]. For example, Boem et al. (2024) argue that embodied cognition must include the enteric nervous, endocrine and immune systems along with gut microbiota [1] [2]. Likewise, Mayer (2011) describes complex bidirectional gut–brain communication: gut signals (via neurons, hormones and immune cells) influence affect, motivation and higher cognition [3] [4]. CASM therefore posits hidden state variables for neural and bodily components, with sensory inputs from both exteroceptive (e.g. vision, hearing) and interoceptive (e.g. gut stretch, hormone levels, cytokines) channels.

System Components: CASM consists of coupled modules for each subsystem, each providing state and sensory variables:

- **Neural Generative Model:** A hierarchy of cortical states $\mathbf{x}(t)$ implements Bayesian inference (predictive coding). It generates predictions for sensory inputs and receives error signals to update beliefs [5].
- **Enteric (Gut) System:** The gut has its own neural plexus (ENS) and afferent pathways. Gut sensors (stretch, nutrients) and enteroendocrine cells send signals (via vagus and hormones) to the brainstem and insula [3] [4]. CASM includes gut state variables $\mathbf{g}(t)$ (e.g. distension, chemical milieu) that produce interoceptive observations.
- **Endocrine System:** Hormones (e.g. cortisol, insulin, gut peptides) are modeled as slow internal state variables $\mathbf{h}(t)$. The hypothalamic–pituitary–adrenal (HPA) axis and other pathways provide top-down control of $\mathbf{h}(t)$, while brain receptors sample actual hormone levels via feedback. These hormonal signals modulate neural gain/precision, thus influencing attention and arousal in the model [6].
- **Immune System:** Cytokines and immune mediators $\mathbf{c}(t)$ are included in $\mathbf{b}(t)$ (bodily state). Immune cells in the gut and periphery sense pathogens and release cytokines, which affect brain activity (microglia, sickness behavior). In CASM, unexpected immune signals act like prediction errors that update internal priors on bodily integrity, coordinating with neural circuits to preserve homeostasis [7].
- **Microbiota:** The gut microbiome is treated as an environmental factor M that influences gut state. Microbes produce metabolites (e.g. short-chain fatty acids, neurotransmitters) that enter circulation or stimulate vagal afferents. These effects are encoded in the generative model as modulatory inputs to $\mathbf{g}(t)$ and indirectly to $\mathbf{x}(t)$. For instance, subliminal microbial signals have been shown to affect memory and emotion via insular networks [8].

This embodied architecture ensures that **each modality of information** (neural, hormonal, microbial, immunological) becomes part of the joint state $\mathbf{w}(t)=(\mathbf{x}(t),\mathbf{b}(t))$, where $\mathbf{b}(t)$ summarizes bodily states (gut, hormones, immune, etc.).

Generative Model and Perceptual Inference

CASM is built on a probabilistic generative model $p(\mathbf{s}_t,\mathbf{i}_t,\mathbf{w}_t)$, where $\mathbf{s}_t$ are exteroceptive observations and $\mathbf{i}_t$ interoceptive observations at time t. Consistent with predictive-coding theory, we assume:

- **Generative Observations:**

$$\mathbf{s}_t = g_e(\mathbf{x}_t;\boldsymbol{\theta}_e) + \boldsymbol{\nu}_t, \quad \mathbf{i}_t = g_i(\mathbf{x}_t,\mathbf{b}_t;\boldsymbol{\theta}_i) + \boldsymbol{\omega}_t,$$

  where g_e,g_i are (generative) mapping functions and $\boldsymbol{\nu}_t,\boldsymbol{\omega}_t$ are Gaussian noise. For example, g_e may encode how visual and auditory causes in $\mathbf{x}_t$ produce retinal inputs $\mathbf{s}_t$, while g_i encodes how combined neural and bodily states produce visceral sensations or hormone readings in $\mathbf{i}_t$. Here $\mathbf{x}_t$ are high-dimensional brain states and $\mathbf{b}_t=(\mathbf{g}_t,\mathbf{h}_t,\mathbf{c}_t,\dots)$ are bodily states.

- **State Dynamics:**

$$\dot{\mathbf{x}}_t = f_x(\mathbf{x}_t,\mathbf{b}_t) + \boldsymbol{\xi}_x(t), \quad \dot{\mathbf{b}}_t = f_b(\mathbf{b}_t,\mathbf{x}_t) + \boldsymbol{\xi}_b(t),$$

  where f_x,f_b describe the (possibly nonlinear) evolution of neural and bodily states, and $\boldsymbol{\xi}$ are process noise. For example, f_x might encode cortical predictive dynamics and recurrent connectivity, while f_b includes physiological laws (e.g. hormone release, cytokine decay, microbiome growth) coupled to brain signals. Equations of this form generalize classical predictive models [5] [9]. In particular, Friston and Kiebel (2009) describe hierarchical dynamical models $y=g(x)+z$, $\dot x=f(x)+w$ which we extend to include interoceptive variables [5] [9].

In Bayesian terms, CASM thus defines a joint distribution $p(\mathbf{s}*t,\mathbf{i}_t|\mathbf{x}_t,\mathbf{b}_t)\,p(\mathbf{x}_t,\mathbf{b}_t)$ whose inversion yields posterior beliefs $p(\mathbf{x}_t,\mathbf{b}_t|\mathbf{s})$. Perception/inference minimizes prediction error: the system computes},\mathbf{i}_{1:t

$$\boldsymbol{\epsilon}^s_t = \mathbf{s}_t - g_e(\hat{\mathbf{x}}_t), \quad \boldsymbol{\epsilon}^i_t = \mathbf{i}_t - g_i(\hat{\mathbf{x}}_t,\hat{\mathbf{b}}_t),$$

and updates its estimates $\hat{\mathbf{x}}_t,\hat{\mathbf{b}}_t$ to reduce these errors. In practice, this is achieved by minimizing a variational free energy F, which Friston shows yields dynamics equivalent to predictive coding [5] [10]. For example, one can define free energy as

$$F = -\ln p(\mathbf{s}_t|\hat{\mathbf{x}}_t) - \ln p(\mathbf{i}_t|\hat{\mathbf{b}}_t,\hat{\mathbf{x}}_t) - \ln p(\hat{\mathbf{x}}_t,\hat{\mathbf{b}}_t)\,,$$

so that $\partial F/\partial \hat{\mathbf{x}},\partial F/\partial \hat{\mathbf{b}}$ drive updates. As Friston (2010) explains, minimizing such free energy implements Bayes-optimal perception and action in a single scheme [10].

In CASM, these updates are continuous in time (often formulated in generalized coordinates) with recurrent feedback loops. Neural populations encode prediction errors which are propagated up the

hierarchy. Crucially, physiological signals serve as additional “top-down” predictions: e.g. the brain’s model predicts hormone levels or gut pressure, and actual endocrine/visceral readings produce interoceptive errors. This links allostatic regulation to cognition. Barrett & Simmons (2015) argue that interoceptive experience mainly reflects such cortical predictions about bodily state, issued by visceromotor cortices [11]. CASM formalizes this by having g_i generate expected bodily sensations from brain states, with agranular cortex areas outputting predictions that are reconciled with true signals.

Attention, Precision and Feedback Control

CASM incorporates precision-weighted predictive coding to model attention and salience. Prediction errors are weighted by precision (inverse variance), implemented neurophysiologically as synaptic gain. Friston (2010) notes that minimizing free energy entails adjusting gain on prediction errors, effectively coding the confidence (precision) of predictions [6]. In our equations, let Π^s_t,Π^i_t be precision matrices for exteroceptive and interoceptive errors. Then state updates include terms like

$$\dot{\hat{\mathbf{x}}} \;\propto\; \frac{\partial g_e}{\partial \mathbf{x}}^T \Pi^s_t\, \boldsymbol{\epsilon}^s_t + \frac{\partial g_i}{\partial \mathbf{x}}^T \Pi^i_t\, \boldsymbol{\epsilon}^i_t + \nabla_{\mathbf{x}} \ln p(\hat{\mathbf{x}}_t, \hat{\mathbf{b}}_t),$$

and similarly for $\dot{\hat{\mathbf{b}}}$ using $\partial g_i/\partial \mathbf{b}$. Biologically, neuromodulators like norepinephrine or dopamine adjust these precisions: for instance, arousal could increase Π^s for threat-related exteroceptive cues, while stress hormones (cortisol) might increase Π^i for bodily threat. Thus CASM links physiology to attention: hormonal state modulates which prediction errors dominate the inference process [6].

Feedback loops abound: ascending (bottom-up) signals convey actual interoceptive and exteroceptive errors to higher areas (e.g. insula, prefrontal cortex), while descending predictions from limbic and cortical areas drive autonomic, endocrine and immune adjustments. For example, a high-level prediction of “need glucose” might descend via the hypothalamus to increase insulin, while the resulting blood glucose level is sensed and fed back as an interoceptive signal. The model therefore includes efferent pathways (e.g. autonomic nervous outputs controlling heart/gut, HPA axis modulating hormones) as part of f_x,f_b coupling. In sum, CASM is a closed-loop dynamical system: internal states generate sensory projections, errors update beliefs, and belief changes produce bodily responses (actions or hormone releases) that affect future inputs.

Interoceptive and Physiological Inputs

CASM explicitly incorporates non-neural information sources. Mayer’s review highlights three mechanisms encoding gut state: (1) afferent neurons of the ENS and vagus, (2) immune cell signals, and (3) enteroendocrine hormones [3]. Accordingly, CASM’s interoceptive vector $\mathbf{i}_t$ includes, for instance, vagal nerve activity from the stomach/intestinal wall, cytokine levels from gut-immune interactions, and gut hormone concentrations. These are predicted by the model (via g_i) and compared to actual measures, producing visceral prediction errors that update cortical representations. For example, enteroendocrine cells secrete gut peptides (ghrelin, CCK, GLP-1, etc.) that signal hunger or nutrient status; Mayer notes these regulate CNS circuits via vagal and endocrine routes [4]. In CASM, such hormones are treated as part of $\mathbf{i}_t$ with known generative dependencies on $\mathbf{b}_t$.

The gut microbiota's role is modeled through its effect on $\mathbf{b}_t$. Microbial metabolites (e.g. short-chain fatty acids, tryptophan metabolites) can be considered exogenous inputs that shift the generative mapping g_i. For instance, certain bacteria modulate inflammation (cytokines) or produce serotonin precursors. The model can capture this by letting an internal variable M (microbiome state) influence the parameters of f_b or g_i. Indeed, Mayer et al. report that "intestinal microbes" provide subliminal interoceptive inputs affecting memory and emotional arousal [8]; CASM would treat these as additional hidden causes inferred from the data.

Overall, the generative mappings g_e,g_i and dynamics f_x,f_b explicitly unify neuroanatomy and physiology. For example, allostatic set-points (homeostatic goals) can be encoded as priors $p(\mathbf{x}_t, \mathbf{b}_t)$ that the inference tries to maintain. Barret and Simmons' EPIC model shows that visceromotor cortices issue predictions to regulate heart rate, gut, etc., while sensory cortices receive the sensory consequences [11]. CASM captures this by having cortico-hypothalamic loops in f_b: cortical state $\mathbf{x}$ drives hormone release $\mathbf{h}$ (via f_b), which is then sensed and compared to the predicted $\mathbf{i}_t$.

Cognitive States and Behavior

The posterior distribution $p(\mathbf{x}*t,\mathbf{b}_t|\mathbf{s}*},\mathbf{i*{1:t})$ constitutes the model's internal state or "beliefs". From these, the system can compute cognitive outputs and decisions. In formal terms, we can augment the model with a value or utility function $U(\mathbf{x},\mathbf{b},a)$ that evaluates states and actions. For example, by defining preferred (homeostatic) levels of $\mathbf{b}$ (e.g. normal glucose, no infection), any deviation yields a "surprisal" cost. The agent then selects action policies a_t to minimize the expected future free energy (combining expected surprisal and information gain). This aligns with active inference: choosing a_t to minimize $E[F*t,a_t]$. }|\mathbf{x}_t,\mathbf{b

Practically, CASM can implement decision-making in a Bayesian or reinforcement-learning style. One may compute predictive distributions of future observations under candidate actions and pick the action that maximizes expected reward or minimizes expected prediction error. Attention and valuation also emerge: Friston notes that perception under FEP maximizes mutual information between sensations and their causes (infomax) [12]. In CASM, this means the model continually refines its beliefs to capture as much structure from the multimodal inputs as possible. Affective valence is then tied to how well predictions match key bodily needs: e.g. a positive affect when homeostasis is achieved, negative when large prediction errors persist (pain, anxiety).

Importantly, *all* forms of information converge on the belief state. Neural spikes, hormone levels, microbial signals and cytokines are each treated as data within $p(\mathbf{s},\mathbf{i}|\mathbf{x},\mathbf{b})$. The same inference rules apply: each signal influences the posterior. For example, an unexpected spike in cortisol would enter $\mathbf{i}_t$ and increase error, leading to updated $\mathbf{x}_t$ (perhaps interpreting the situation as stressful) and adjustments in behavior (e.g. heightened vigilance).

Simulation Architecture

The CASM architecture can be implemented algorithmically. At each time step t, the model maintains estimates $\hat{\mathbf{x}}_t,\hat{\mathbf{b}}_t$. A prototypical update loop is:

- **(1) Observe:** Receive exteroceptive input $\mathbf{s}_t$ and interoceptive input $\mathbf{i}_t$ from sensors and bodily measures.
- **(2) Predict:** Compute predicted observations $\hat{\mathbf{s}}*t=g_e(\hat{\mathbf{x}}*)$, $ \hat{\mathbf{i}*t=g_i(\hat{\mathbf{x}}*)$ using the generative model. },\hat{\mathbf{b}}_{t
- **(3) Error:** Calculate prediction errors $\boldsymbol{\epsilon}^s_t=\mathbf{s}_t-\hat{\mathbf{s}}_t$ and $\boldsymbol{\epsilon}^i_t=\mathbf{i}_t-\hat{\mathbf{i}}_t$.
- **(4) Update States:** Update beliefs via predictive coding (e.g. gradient descent on free energy [5]):

$$\dot{\hat{\mathbf{x}}}_t \;\propto\; \frac{\partial g_e}{\partial \mathbf{x}}\Big|^T_{\hat{x}_t} \Pi^s_t\,\boldsymbol{\epsilon}^s_t + \frac{\partial g_i}{\partial \mathbf{x}}\Big|^T_{\hat{x}_t,\hat{b}_t} \Pi^i_t\,\boldsymbol{\epsilon}^i_t + \nabla_{\mathbf{x}} \ln p(\hat{\mathbf{x}}_t,\hat{\mathbf{b}}_t),$$

$$\dot{\hat{\mathbf{b}}}_t \;\propto\; \frac{\partial g_i}{\partial \mathbf{b}}\Big|^T_{\hat{x}_t,\hat{b}_t} \Pi^i_t\,\boldsymbol{\epsilon}^i_t + \nabla_{\mathbf{b}} \ln p(\hat{\mathbf{x}}_t,\hat{\mathbf{b}}_t).$$

  These differential updates are integrated to adjust $\hat{\mathbf{x}}*{t+1},\hat{\mathbf{b}}*$.
- **(5) Action:** Optionally compute an action a_t (motor command or internal adjustment) that will minimize expected future free energy. For instance, predict outcomes under different a and choose the one that best restores homeostasis.
- **(6) Effector Update:** Apply a_t to the body/environment, affecting future states (e.g. muscle movement, hormone secretion in f_b).
- **(7) Iterate:** Move to $t+1$ with updated states and repeat.

This loop embodies feedback: errors influence belief updates, which change predictions and actions, which in turn alter both external inputs and internal physiology. The simulation thus captures real-time dynamics, prediction-error minimization, and probabilistic inference. (See Friston et al. for similar predictive-coding schemes [5].)

Relation to PRT, DIM, ISM, ESM, GCM

CASM serves as a unified generative submodel consistent with broader cognitive frameworks. In the **Projection Rendering Theorem (PRT)** perspective, one imagines internal representations "rendered" into sensory experience. Formally, CASM's generative mappings g_e,g_i *are* the projection operators: they map internal state $(\mathbf{x},\mathbf{b})$ to predicted sensations. Thus each sensory prediction $ \hat{\mathbf{s}}_t=g_e(\hat{\mathbf{x}}_t)$ is the "informational projection" of the brain's model into exteroceptive space, and similarly $\hat{\mathbf{i}}_t=g_i(\hat{\mathbf{x}}_t,\hat{\mathbf{b}}_t)$ is the interoceptive projection. Prediction-error minimization then corresponds to correcting this rendering. CASM thus instantiates the core PRT idea in mathematical form using Bayesian inference (see Friston on generative models as the basis of perception [10] [6]).

Similarly, any **Dynamic Interaction Model (DIM)** or **Embodied Systems Model (ESM)** can be seen as special cases of CASM's dynamics: DIM arises by focusing on the coupled differential equations for $

\mathbf{x}$ and $\mathbf{b}$, showing how they interact over time, and ESM by including the full bodily coupling. An **Interoceptive State Model (ISM)** would be the submodel concerning only $\mathbf{b}$ and $\mathbf{i}$ (fixing neural priors), while a **Generative Cognitive Model (GCM)** is CASM's neural hierarchy $\mathbf{x}$ and g_e. In practice, CASM's modular specification allows one to "project" onto these subsystems by marginalizing or constraining parts of the generative model.

In all cases, CASM provides the mathematical infrastructure (Bayesian updates, dynamical equations, information measures) that defines how internal states give rise to observable projections and how various information channels are integrated. It ensures that neural, hormonal, microbial, and immune data are transformed into unified cognitive representations by the same probabilistic inference engine.

References: Key concepts are drawn from predictive coding and embodied cognition literature [5] [10]. For example, Friston (2009, 2010) formalizes perception as free-energy minimization with generative models [5] [10]; Barrett & Simmons (2015) develop the EPIC model of interoception [11]; and Mayer (2011) and Boem et al. (2024) review gut–brain–microbe influences on cognition [1] [3]. Kiverstein et al. (2022) explicitly integrate neural, endocrine and immune systems into a single predictive framework (the NEI model) [7]. ICASM's equations and architecture generalize these ideas into a formal, simulable specification.

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[1] [2] Frontiers | Minding the gut: extending embodied cognition and perception to the gut complex
https://www.frontiersin.org/journals/neuroscience/articles/10.3389/fnins.2023.1172783/full

[3] [4] [8] Gut feelings: the emerging biology of gut–brain communication | Nature Reviews Neuroscience
https://www.nature.com/articles/nrn3071

[5] [9] doi:10.1098/rstb.2008.0300
https://www.fil.ion.ucl.ac.uk/~karl/Predictive%20coding%20under%20the%20free-energy%20principle.pdf

[6] [10] [12] The free-energy principle: a unified brain theory? | Nature Reviews Neuroscience
https://www.nature.com/articles/nrn2787

[7] An Embodied Predictive Processing Theory of Pain Experience | Review of Philosophy and Psychology
https://link.springer.com/article/10.1007/s13164-022-00616-2

[11] Interoceptive predictions in the brain
https://www.periodicos.capes.gov.br/index.php/acervo/buscador.html?task=detalhes&id=W1620203002