

Predictive Processing of Interoception, Decision-Making, and Allostasis: A Computational Framework and Implications for Emotional Intelligence

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Abstract

Emotional intelligence is composed of a set of emotional abilities, including recognition of emotional states in the self and others, the use of emotions to guide thoughts and behaviours, and emotion regulation. Previous studies have demonstrated that emotional intelligence is associated with mental health, social problem solving, interpersonal relationship quality, and academic and job performance. Although emotional intelligence has received much interest both in basic research fields and applied and clinical fields, the mechanisms underlying the functions of emotional intelligence remain unclear. The aim of the present article was to consider the mechanisms of emotional intelligence using a computational approach. Recent theories of emotion in psychology and neuroscience have emphasized the importance of predictive processing. It has been proposed that the brain creates internal models that can provide predictions for sensation and motor movement, and perception and behaviors emerge from Bayesian computations rooted in these predictions. This theoretical framework has been expanded to include interoceptive perception of the internal body to explain affect and decision-making as phenomena based on interoception. This perspective has implications for understanding issues of emotional intelligence.

Keywords: interoception, decision-making, allostasis, prediction

Emotional Intelligence

Emotional intelligence is a set of emotional abilities that aids adaptation to the external environment, and it is generally considered to be composed of several factors, including recognition of emotional states in the self and others, the use of emotions to guide thoughts and behaviors, and emotion regulation (Hogeveen, Salvi, & Grafman, 2016; Mayer & Salovey, 1997). Previous studies have reported that higher emotional intelligence is associated with better mental health (Kee et al., 2009), greater social problem solving abilities (Barbey et al., 2014), higher quality interpersonal relationships (Brackett, Rivers, Shiffman, Lerner, & Salovey, 2006), and better academic and job performance (Brackett, Rivers, & Salovey, 2011;

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Libbrecht, Lievens, Carette, & Côté, 2014). Although it has received much interest both in basic research fields and applied and clinical fields, there has been substantial debate about whether emotional intelligence is a valid scientific construct, in both methodological and theoretical domains (Conte, 2005; Murphy, 2006).

One of the limitations of emotional intelligence as a valid construct is that the mechanisms underlying the functions of emotional intelligence are currently unclear. Hogeveen et al. (2016) proposed candidate brain regions related to various components of emotional intelligence based on findings from patients with brain lesions. These findings are useful for understanding how the components of emotional intelligence are constructed by the functions of various brain regions. However, the fundamental principles by which emotional intelligence is produced remain to be elucidated. The current article considers the principles supporting emotional intelligence from the perspectives of the psychological constructivist theory of emotion (Barrett, 2017; Barrett, Quigley, & Hamilton, 2016; Barrett & Simmons, 2015) and predictive coding (Friston, 2010; Friston, Kilner, & Harrison, 2006), which are influential theoretical frameworks in modern psychology and neuroscience.

The psychological constructivist theory of emotion is based on the concept of predictive coding, which hypothesizes that the brain constructs internal models with various functional layers, and that every function of the brain emerges from computations of the models and input signals. In psychological constructivist theory, it is hypothesized that internal interoceptive signals from the body also emerge via the same predictive principles. Furthermore, it has been proposed that all mental functions, including perception, motor function, cognition, and affect can be produced based on predictive interoception. Because this theoretical framework is a kind of meta-theory, empirical verification of the whole theory is difficult. However, this theoretical framework is useful because it enables speculation about the functional associations between the brain and body, and supports the development of hypotheses that can be examined empirically (for details, see Ohira, 2018a). The current article first introduces the psychological constructivist theory of emotion, then proposes a computational model explaining the emergence of affect and decision-making based on the prediction of homeostasis (i.e., allostasis), and finally outlines the implications of this model for understanding emotional intelligence.

Predictive Coding of Interoception, Decision-Making, and Allostasis

The brain is not a passive organ that solely responds to input signals from sensory organs. Rather, the brain actively constructs perception based on internal models predicting future input signals, and on computations of differences between predictions and input signals (prediction error). This theoretical principle of brain function is called "predictive coding" (Friston, 2010; Rao & Ballard, 1999). In the

theory of predictive coding, processes of perception in the brain are explained as an analogy of the principles of Bayesian statistics (Ainley, Apps, Fotopoulou, & Tsakiris, 2016). The prediction of perception by an internal model is expressed as a probabilistic distribution, corresponding to the prior distribution in Bayesian statistics. Sensory input can also be expressed as a probabilistic distribution, and prediction error is computed as the difference between the distributions of the prediction and sensory input. This sensory input corresponds to observation or likelihood in Bayesian statistics, and the posterior distribution is computed based on updating in Bayes' theorem. Our subjective experiences of perception can be considered as awareness of the computational processes of the posterior distribution. It is thought that such Bayesian computations are involved in processing in every modality of perception and motor function. Organisms, including humans, construct and maintain integrated and consistent images of self and the world by minimizing the sum of the prediction error. To minimize prediction error, the organism will either update the internal model or actively modulate the sensory input by changing behaviour.

Barrett and colleagues proposed that, in addition to exteroception (e.g. vision and hearing) and proprioception (perception of the location of the body and bodily movement), interoception (perception of the internal body, such as internal organs and vessels) is established by the principle of predictive coding (Ainley et al., 2016; Barrett & Simmons, 2015, 2016; Seth & Friston, 2016). To maintain homeostasis, organisms must regulate bodily states appropriately. To achieve this, the brain represents the body's current state and its desirable state (set point) and constructs an internal model of the body to satisfy the set points of the bodily states. The model determines desirable ranges of states, including blood pressure, blood glucose level, as well as concentrations of hormones and cytokines, depending on specific situations. When the brain receives bodily signals, the signals are compared with predictions by the internal model, and the differences between them are computed as prediction errors. Organisms regulate their bodily states to minimize prediction errors. To reduce prediction errors, an organism both updates its internal model and alters its bodily states.

In this theoretical framework, interoception can function as a signal to inform whether the current bodily state is within the range of homeostasis, or if it deviates from this range. Deviation of bodily states from the range of homeostasis constitutes a threat for organisms. Thus, in such a case, there is a drive to change bodily states to be within the range of homeostasis. This modification of bodily states in a desirable direction is experienced as pleasant affect, and a lack of change or a change in an undesirable direction is felt as unpleasant affect. Furthermore, it has been argued that impairment of this regulatory process of the body based on interoception results in mental disorders, such as depression, anxiety, developmental disorders, and fatigue (Barrett & Simmons, 2015; Stephan et al., 2016).

Keramati and Gutkin (2014) expanded this idea, describing an association between interoception and decision-making (Figure 1). Although bodily states are high-dimensional, we consider two factors as examples: blood pressure and blood glucose level. The set points of these two dimensions of bodily states are expressed as point H^* on a level surface H in Figure 1. A neural representation of these two factors at time point t , H_t corresponds to interoception. As H_t is distant from H^* , there is a drive, $d(H_t)$, to move H_t into H^* . This drive is expressed as the curved surface whose peak is just above the H^* in Figure 1. If the individual chooses an action that causes the bodily states to move to H_{t+1} at the next time point $t+1$, the drive will reduce to $d(H_{t+1})$. Thus, the difference K_t will be evaluated as reward $r(H_t, K_t)$. The signal of this reward will be conveyed to the brain and utilized to update values of the current states and the action, on the basis of the principle of reinforcement learning. Specifically, a reward is determined as the degree of movement of bodily states in the direction of the desirable set point. In this way, reward is considered to be fundamentally rooted in bodily states. In humans, more abstract representations such as money and favourable reputation from others can function as rewards via the ability to construct representations and to link such representations with bodily states.

The set point of bodily states H^* is not always fixed and can be actively changed by the demands of environments and social contexts. This function involves modulation of the prediction of bodily states produced from the internal model by higher level internal models and corresponds to the physiological concept of "allostasis." Here, I hypothesize that the top-down signal regulating allostasis can be modulated by "reward prediction error" signals in the reinforcement learning algorithm in the brain. Reinforcement learning is one of the basic mechanisms of learning and decision-making. In this algorithm, the values of options are continuously updated using reward prediction error signals. If an individual chooses an option and the obtained outcome is better than the prediction (the current value), the sign of the reward prediction error is positive, and the value of the option is updated upward. If the outcome is worse than the prediction, the sign of the reward prediction error is negative, and the value of the option is updated downward. At the next opportunity, the individual will make a decision by a comparison of the values of the options. When reward prediction error increases, the sign of the reward prediction error is either positive or negative, and some coping behaviours are needed to acquire reward or avoid harm. In this case, the set point of bodily states will be regulated upward to elevate physical energy levels. On the other hand, a decrease in reward prediction error indicates the completion of learning; thus, the set point of bodily states will be regulated downward to save energy.

This theoretical framework can seamlessly integrate concepts of various psychological phenomena, such as bodily states, interoception, reward, value, decision-making, and affect. The well-tuned and functional aspects of these

processes might provide the basis of emotional intelligence.

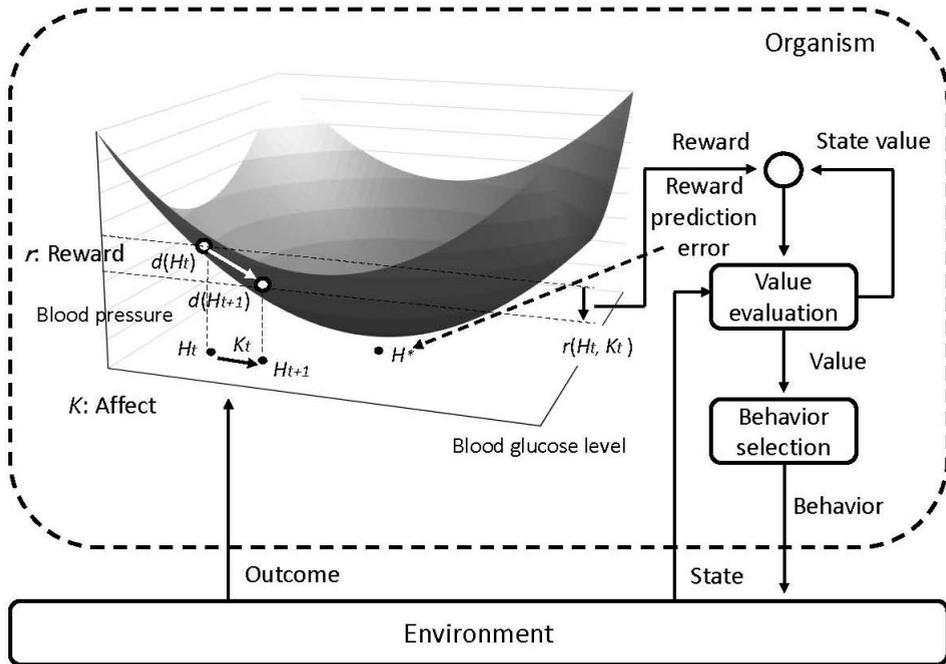


Figure 1. Reward, decision-making, and affect determined by bodily states. Set points of blood pressure and blood glucose level are represented as H^* . As the neural representation of bodily states at a time point t , H_t is distant from the set point, the drive $d(H_t)$ to move H_t to H^* happens. If this organism chooses an action (decision-making) and the bodily state moves to H_{t+1} , the drive reduces to $d(H_{t+1})$ and the difference between $d(H_t)$ and $d(H_{t+1})$ is evaluated as a reward. The difference between the obtained reward and the current value of the option is computed as a reward prediction error. The signal of the reward prediction error is used to update values of the current states and the option, and to shift the set point of bodily states (dashed arrow in Figure 1). As coping is needed in a situation where reward prediction error increases, the set point of bodily states is shifted upward. As the reduction of reward prediction error indicates the completion of learning and adjustment to the environment, no more effort is needed. In such a situation, the set point of bodily states is shifted downward to save physical energy.

Computational Model of Interoception, Decision-Making, and Allostasis

A previous study by the author (Ohira, 2019) proposed a computational model to explain the dynamic functions of the predictive coding of interoception and decision-making expressed in Figure 1, inspired by the work of Stephan et al. (2016) (Figure 2).

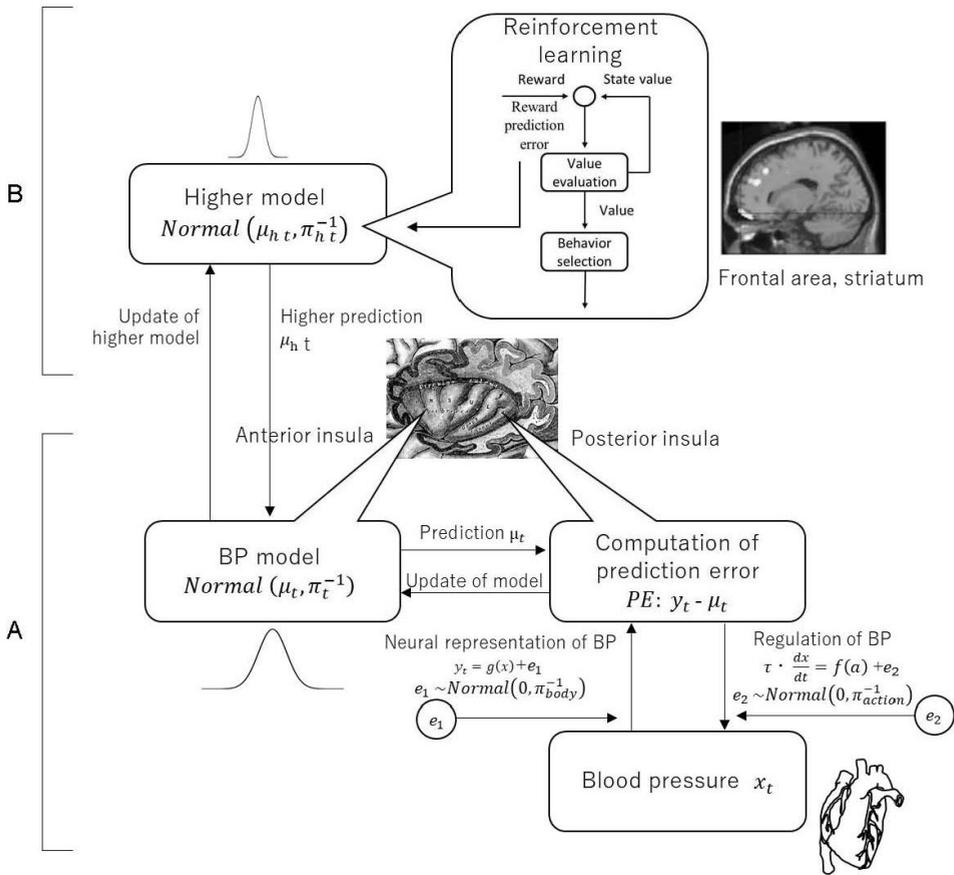


Figure 2. Hierarchical predictive coding of interoception and reinforcement learning. *Normal*: normal distribution, *PE*: prediction error, *e*: noise expressed by normal distribution. A: Regulatory processes of bodily states (blood pressure) by predictive coding of interoception (Ohira, 2018b). B: Influences of reward prediction error in reinforcement learning on higher model of bodily states (blood pressure).

Here, as an example of the regulation of bodily states by interoception, an internal model of blood pressure (x) is considered. This internal model is expressed by a probabilistic distribution (normal distribution), with mean μ_t and variance π_t^{-1} (π is precision, reciprocal number of variance) at a time point t . The mean μ_t is the prediction of blood pressure at this time point and can function as the set point of blood pressure. The real value of blood pressure at this time point is expressed as x_t . This value is conveyed to the brain by a hyperbolic function, with noise, e_1 ($\text{Normal} \sim (0, \pi_{body}^{-1})$). As such, a neural representation of blood pressure at this time point, y_t is formed. By comparison between the prediction μ_t and the neural representation of blood pressure y_t , prediction error $y_t - \mu_t$ is computed.

The principle of predictive coding aims to reduce this prediction error. Therefore, the mean and variance (precision) at the next time point $t + 1$, are updated as follows, using Bayes' theorem.

Mean in model of blood pressure:

$$*(y_t - \mu_t), \quad (1)$$

Precision in model of blood pressure:

$$\pi_{t+1} = \frac{\pi_t + \pi_{body}}{\pi_t * \pi_{body}}, \quad (2)$$

At the same time, an action a works on the body to reduce prediction error. For example, the activities of the sympathetic and parasympathetic nervous systems are modulated to alter blood pressure. This effect is expressed as the following hyperbolic function $f(a)$.

$$f(a) = \tanh\left(\frac{\pi_t}{\pi_t + \pi_{body}} * -(y_t - \mu_t)\right), \quad (3)$$

By this effect, blood pressure continuously changes with a certain temporal delay. This process is expressed by the following differential equation: τ is time constant controlling the temporal delay (here, $\tau = 5$) and e_2 is a noise term expressed by a normal distribution with mean 0 and variance π_{action}^{-1} .

$$\tau * \frac{dx}{dt} = f(a) + e_2, \quad (4)$$

It has been hypothesized that the generation of such prediction of interoception is conducted in the anterior insula, and computation of prediction error is conducted in the posterior insula (Figure 2A; Barrett & Simmons, 2015).

Imagine a situation where an individual performs a typical decision-making task in which reward is stochastically delivered by a choice. In this article, a simple two-armed bandit task is considered, in which one option is linked with a monetary reward at a probability of 70% and the other option is linked with a monetary reward at a probability of 30%. To represent top-down modulation of the internal model of blood pressure accompanying the execution of this decision-making task, a higher-layer model is added (Figure 2B). This higher model is also expressed by a normal probabilistic distribution, with mean $\mu_{h t}$ and variance $\pi_{h t}^{-1}$ at a time point t . At the initiation of the decision-making task, this higher model is applied and its mean $\mu_{h t}$ is output as the higher prediction into the internal model of blood pressure. The higher prediction error is then computed as the difference between the current value of prediction in the internal model of blood pressure μ_t and the higher prediction $\mu_{h t}$. Using this prediction error, the mean and variance (precision) in the higher model are updated as follows.

Mean in higher model:

$$\mu_{h\ t+1} = \mu_{h\ t} + \frac{\pi_t}{\pi_{h\ t} + \pi_t} * (\mu_t - \mu_{h\ t}), \quad (5)$$

Precision in higher model:

$$\pi_{h\ t+1} = \frac{\pi_{h\ t} + \pi_t}{\pi_{h\ t} * \pi_t}, \quad (6)$$

The prediction error in the higher model also affects updating of the internal model of blood pressure. Thus, equations (1) and (2) are replaced as follows.

Mean in model of blood pressure:

$$\mu_{t+1} = \mu_t + \frac{\pi_{body}}{\pi_t + \pi_{body}} * (y_t - \mu_t) + \tanh\left(\frac{\pi_{h\ t}}{\pi_{h\ t} + \pi_t} * -(\mu_t - \mu_{h\ t})\right), \quad (7)$$

Precision in model of blood pressure:

$$\pi_{t+1} = \frac{\pi_t + \pi_{body}}{\pi_t * \pi_{body}} + \frac{\pi_{h\ t} + \pi_t}{\pi_{h\ t} * \pi_t}, \quad (8)$$

Figure 4A shows a typical result of simulation for changes of blood pressure accompanying the execution of the decision-making task. At the initiation of the task (① in Figure 4A), the higher model ($\mu_{h\ t} = 0.2, \pi_{h\ t} = 10000$) was applied, resulting in elevation of blood pressure by an upward shift of the prediction in the internal model of blood pressure ($\mu_t = 0, \pi_t = 100$). At the termination of the task (② in Figure 4A), the influence of the higher model was removed and blood pressure returned to the baseline. However, this simulation of blood pressure is fixed, with no influence of the context on decision-making. Thus, a typical algorithm of reinforcement learning, Q learning, is then introduced into the model (Lee, Seo, & Jung, 2012).

$$Q_{(a(t))}(t+1) = Q_{a(t)}(t) + \alpha (R(t) - Q_{a(t)}(t)), \quad (9)$$

$$\text{RPE}_t = R(t) - Q_{i(t)}(t), \quad (10)$$

$$P(a(t)) = \frac{1}{1 + \exp[-\beta(Q_{a(t)} - Q_{b(t)})]}, \quad (11)$$

$Q_{(i(t))}(t)$ in equation (9) indicates the value of each option, $i = a, b$, at a time point t . If reward $R(t)$ is acquired by choosing option a, the difference between the reward prediction $Q_{(a(t))}(t)$ and $R(t)$ is computed as the reward prediction error: RPE_t (equation 10).

Here, reward is manipulated as 1 or 0 for simplicity. Positive RPE indicates that the outcome was better than the prediction; thus, the value of the option is updated upward. Negative RPE indicates that the outcome was worse than the prediction; thus, the value of the option is updated downward. Based on current values of the options, the probability of choosing option "A" from the two options is expressed by equation (11). The parameter α in the equation (9) is called the "learning rate", and controls the degree to which the value is updated by RPE in a single trial. An excessively low learning rate results in slow learning; however, an excessively high learning rate is linked with unstable learning that is substantially influenced by each single outcome. The parameter β in equation (11) is called "inverse temperature", and controls the weighting of the difference of values in choice of options. Larger inverse temperature values indicate a strategy of choice that is sensitive to differences in values; this strategy is called exploitation. Smaller inverse temperature indicates greater randomness in choice; this strategy is called exploration. Combination of these parameters determines the characteristics or individual differences of decision-making. For example, depressive individuals typically show lower learning rates and inverse temperature, compared with healthy individuals (Kunisato, Katahira, Okimura, & Yamashita, 2019). This pattern of the two parameters in depressive people might indicate slower and inefficient learning, as well as unstable choice.

As described above, RPE is hypothesized to affect the higher model. Specifically, when the absolute value of RPE increases, the mean of the higher model is shifted upward. When the absolute value of RPE decreased, the mean of the higher model is shifted downward. RPE within n trials ago (here, $n = 4$) would be expected to affect the higher model with a discounting rate, δ (here, $\delta = 0.1$). Thus, an accumulated RPE at time point t is determined as follows (equation (12)). The denominator in equation (12) is included for standardization.

$$RPE_{int t} = \frac{|RPE(t)| + \delta|RPE(t-1)| + \delta^2|RPE(t-2)| + \dots + \delta^n|RPE(t-n)|}{1 + \delta + \delta^2 + \dots + \delta^n}, \quad (12)$$

A change in accumulated RPE at the time point t is expressed as follows:
 $\Delta RPE_{int t} = RPE_{int t} - RPE_{int t-1}, \quad (13)$

This change of accumulated RPE is thought to affect the higher model. Equation (5) is then replaced as follows.

Mean in higher model:

$$\mu_{h t+1} = \mu_{h t} + \frac{\pi_t}{\pi_{h t} + \pi_t} * (\mu_t - \mu_{h t}) + \tanh(\theta * \Delta RPE_{int t}), \quad (14)$$

θ is a parameter that determines the degree of influence of RPE on the higher model (here, $\theta = 0.5$). As such, the dynamics of allostasis which regulate bodily states by alteration of the set point of bodily states based on RPE in decision-making (Figure 1) has been modelled.

Parameters in The Computational Model and Allostatic Regulation

Through simulation using the computational model described above, characteristics of decision-making and allostatic regulation in healthy individuals and those in emotionally non-functional individuals, such as patients with depression, were compared. Depression is associated with less functional styles of emotional intelligence. Typical symptoms of depression, such as negativity bias in attention and thoughts, impaired emotion regulation, and behavioural deficits caused by lack of motivation are closely associated with failures in aspects of emotional intelligence. Imagine a situation in which a depressive individual and a healthy individual perform the two-armed bandit decision-making task described above. Based on previous studies (Kunisato et al., 2019; Toyama, Katahira, & Ohira, 2019), learning rate (α) and inverse temperature (β) in reinforcement learning are set to lower values in the depressive individual ($\alpha = 0.2, \beta = 3.0$) than in the healthy individual ($\alpha = 0.05, \beta = 0.8$). Figure 3 shows typical results of simulation of 120 trials of the decision-making task using these parameters. The healthy individual rapidly learned the contingency between options and outcomes. Thus, the advantageous option became chosen more often, resulting in a reduction of RPE. In contrast, in the depressive individual, the speed of learning was slow due to the smaller learning rate and the disadvantageous option was also chosen more often, due to the lower inverse temperature, resulting in maintenance of larger RPE.

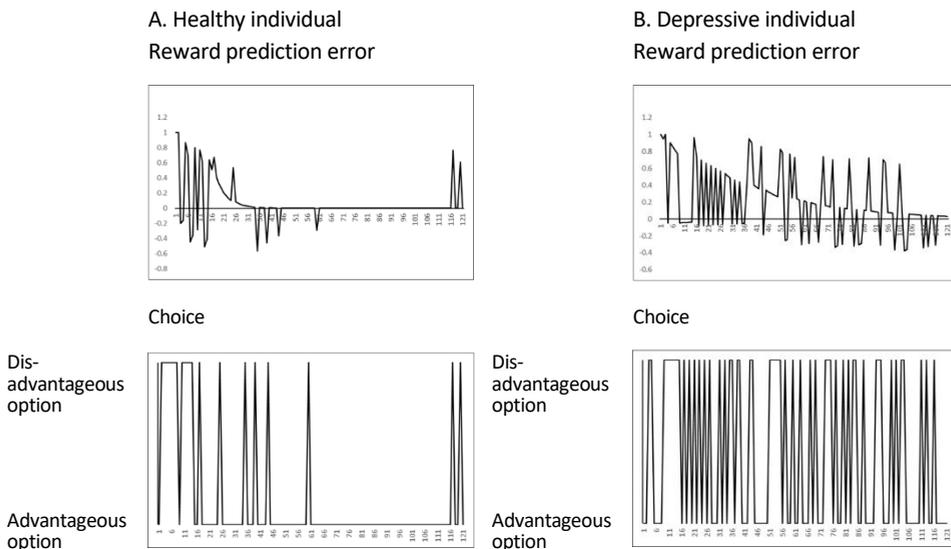


Figure 3. Simulation of the reinforcement learning underlying decision-making. A healthy individual (A) and a depressive individual (B) conduct 120 trials of a decision-making task. The learning rate and inverse temperature were lower in the depressive individual than in the healthy individual ($\alpha: 0.2, 0.05; \beta: 3.0, 0.8$, respectively). Figures show a typical example of changes in reward prediction error and choices of advantageous and disadvantageous options.

Figure 4 indicates the responses of blood pressure during the decision-making task in both individuals. In the healthy individual (Figure 4B), a large RPE that occurred at the initiation of the decision-making task affected the internal model of blood pressure, resulting in an upward shift of the set point of blood pressure, and substantial elevation of blood pressure. Then, as RPE gradually reduced as learning progressed, the set point of blood pressure was modulated downward, leading to a rapid decline in blood pressure. Psychologically, this phenomenon can be interpreted as habituation of physiological responses to a situation. Finally, blood pressure returned to baseline at the termination of the decision-making task. A previous study in the author's laboratory revealed a similar pattern of real temporal changes of blood pressure during the same decision-making task in humans (Kimura, Ohira, Isowa, Matsunaga, & Murashima, 2007). The consistency between the results of the simulation and real experimental data support the validity of the computational model proposed in the current article.

In the depressive individual (Figure 4C), as RPE did not converge, its influences on the internal model of blood pressure continued; thus, habituated reduction of blood pressure did not occur. Furthermore, a high level of blood pressure was maintained even after the termination of the task. This pattern can be interpreted as a state called allostatic load (McEwen, 1998). It is thought that chronic allostatic load can cause systemic inflammation, leading to an increased risk of high blood pressure, heart disease, cerebral infarction, and diabetes. In addition, it has been argued that disturbance of interoception and dysregulation of bodily states due to allostatic load is a major cause of depression (Barrett et al., 2016; Barrett & Simmons, 2015; McEwen, 2003; Stephan et al., 2016). Indeed, depression is sometimes accompanied by somatic disorders such as cardiac, immunological, and metabolic disturbances (Joynt, Whallen, & O'Connor, 2003; Renn, Feiciano, & Segal, 2011). In the framework of predictive coding, this can be explained as persistent effects of impaired high-level internal models predicting uncontrollability and uncertainty of environments with greater precision on functions in allostatic control regions, such as the insula and anterior cingulate cortex, or on autonomic effector regions, such as the hypothalamus and periaqueductal gray, leading to an allostatic load resulting in somatic disturbances. These concepts are consistent with the results of the current simulation, suggesting that accumulation of allostatic load in everyday life by biases of parameters in reinforcement learning might be a critical factor in depression.

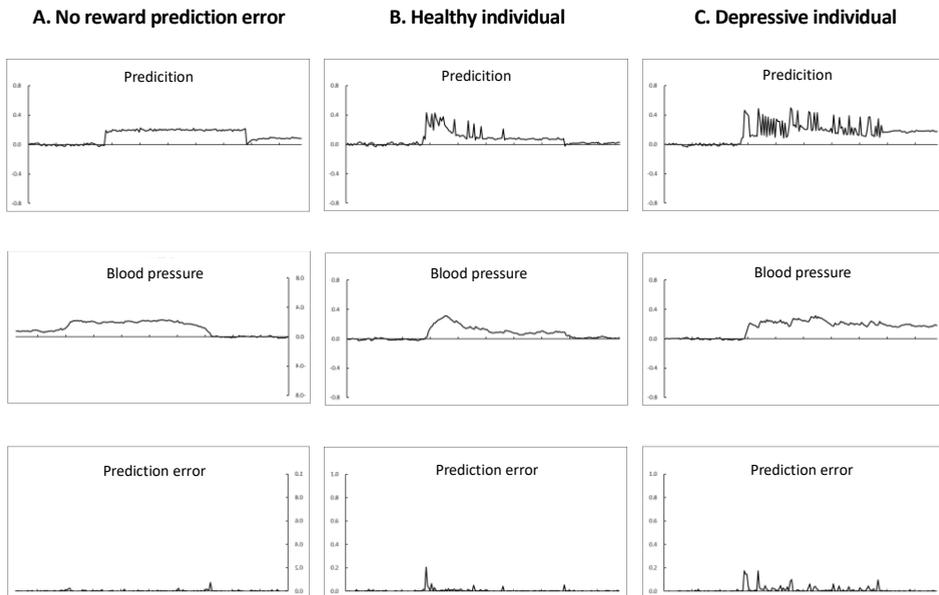


Figure 4. Simulation of the responses in blood pressure influenced by decision-making. A: At the initiation of decision-making task (①), the set point of blood pressure is shifted upward and blood pressure elevates. At the termination of the task (②), the set point is shifted downward and blood pressure returns to baseline. B: In a healthy individual, by introducing signals of reward prediction error in reinforcement learning (Figure 3) into the higher model, rapid elevation of blood pressure and a gradual decline of blood pressure (habituation) are shown, accompanying learning progress. C: In a depressive individual, slow and unstable decision-making results in the maintenance of greater reward prediction error, resulting in a sustained high level of blood pressure, even after the termination of the task.

In addition to the parameters in the reinforcement learning model, the parameters in the model of interoception may also be related to impaired emotional states (Ohira, 2018b). For example, depression can be caused by a reaction to an initial somatic disease. Systemic inflammation mediated by increased proinflammatory cytokines, such as IL-6 and TNF- α , can affect brain functions and are linked with the onset of depression (Dantzer, O'Connor, Freund, Johnson, & Kelley, 2008). This might cause greater prediction errors in the allostatic control regions, leading to updating of the internal models in a negative direction, accompanied by hedonically negative affective experiences. In addition, it has been argued that noisy bodily signals to the brain and unstable fluctuation of interoception might cause subjective experiences of anxiety (Farb et al., 2015; Stewart, Buffett-Jerrott, & Kokaram, 2001). In a previous study, the author attempted to simulate this phenomenon by increasing the variance of the noise term $e_1(\text{Normal}\sim(0, \pi_{body}^{-1}))$ shown in Figure 2, specifically, introducing a large value of π_{body}^{-1} . Using this

manipulation, the predictions generated by the internal model of blood pressure became unstable and noisy (Ohira, 2018b). If changes of prediction might be felt as subjective and affective experiences, such unstable fluctuation might be essential for experiences of anxiety.

Implications for Emotional Intelligence

The computational model proposed in this article describes basic and abstract mechanisms of association among interoception, decision-making, and regulation of bodily states. This model has implications for various issues related to emotional intelligence. One of the important factors of emotional intelligence is the use of emotions for guidance of adaptive thoughts and behaviors (Hogeveen et al., 2016; Mayer & Salovey, 1997). This factor is closely related to decision-making, as evaluation of the values of available behaviors and choosing one specific behavior that is appropriate for a specific case is an important function of decision-making in everyday life. Emotions have been thought to play critical roles in decision-making. Because we occasionally face situations in which the values of available options and contingencies between options and outcomes are not clear (uncertainty), we sometimes rely not only on deliberate and thoughtful processes, but also on affective or intuitive processes in decision-making (Damasio, 1994). The model proposed in the current article explicitly explains the mechanisms underlying such processes. Changes of bodily states to a predicted set point are evaluated as reward and produce hedonically positive affective states. Reward determined in such a way can then update the values of options, which can be used in decision-making at the next opportunity. When bodily signals can be precisely conveyed to the brain, and parameters of reinforcement learning are within an appropriate range, learning processes rapidly converge and appropriate decision-making and regulation of bodily states are possible. In contrast, disturbances in these mechanisms can be linked with impaired mental and physical well-being. In this sense, the well-tuned functions of the brain and body described in the model might provide the minimal bases of emotional intelligence.

According to the psychological constructivist theory of emotion (Barrett, 2017; Barrett et al., 2016; Barrett & Simmons, 2015), subjective experiences of emotions are generated by categorization of interoceptive affective states using concepts based in cognitive structure as well as information from the external context. This process corresponds to another component of emotional intelligence, recognition of the emotional states of the self. Such processes of awareness of one's emotional states constitute the basis of recognition of emotional states of others. Furthermore, emotion regulation is another important factor of emotional intelligence, and is based on appropriate emotional awareness. Theorists have recently begun to discuss how such concepts of emotions are generated and shared between people, particularly during processes of socializing in the period of infancy (Hoemann & Barrett, 2018;

Hoemann et al., 2020). Although the model proposed in the current article does not explain the processes of generation of experienced emotions, it has implications for the consideration of such processes. Atzil and Gendron (2017) argued that concepts of emotions play an important role in maintaining ongoing physiological balance and allostasis, and that infants learn emotion concepts for allostasis regulation via bio-behavioural synchrony with caregivers. If this reasoning is accurate, the mechanisms of interoception described in the currently proposed model may develop through such a co-action process in infancy, and concepts of emotions should be created to efficiently regulate the mechanisms. In this sense, emotional intelligence is a set of abilities for appropriately operating these mechanisms of interoception.

Conclusion

The current article introduced a basic conceptual framework integrating interoception, decision-making, and allostasis based on the perspective of predictive coding, and it proposed a computational model to explain the mechanisms of those psychological, behavioural, and physiological phenomena. Furthermore, the article discussed how the mechanisms of interoception are related to factors of emotional intelligence. A tentative conclusion of this article is that the ultimate purpose of allostasis regulation is to maintain life, while the mechanisms of interoception have been developed to serve this purpose, and emotional intelligence can be considered to constitute a set of abilities for efficiently operating these mechanisms. Although the model proposed in this article is simply a theoretical model that does not provide explanations or predictions for concrete psychological and physiological indices in real situations, the model is useful for providing an integrated description of the functions of the brain and body to explain accumulated empirical findings regarding emotions. Specifically, such a computational perspective will be useful for elucidating how basic physiological components are related to facets of emotional intelligence, how emotional intelligence is linked with well-being and illness as regulation and dysregulation of allostasis, and how emotional intelligence can be improved by adjustment of parameters in computational processes.

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