

## **The Error Monitoring and Processing System in Alcohol Use**

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## **The Error Monitoring and Processing System in Alcohol Use**

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### **Abstract**

**Background:** Current data suggest that alcohol might play significant role in error commission. Error commission is related to the functions of the Error Monitoring and Processing System (EMPS) located in the substantia nigra of the midbrain, basal ganglia and cortex of the forebrain. The main components of the EMPS are the dopaminergic system and anterior cingulate cortex. Although, recent data show that alcohol disrupts the EMPS, the ways in which alcohol affects this system are poorly understood.

**Aims & Objectives:** We reviewed recent data that suggest the indirect effect of alcohol use on error commission.

**Methods / Study Design:** Databases were searched for relevant literatures using the following keywords combination – Alcohol AND Error *Commission* (OR *Processing, Monitoring, Correction, Detection*). Literatures were searched in scientific databases (Medline, DOAJ, Embase from 1940 to August 2010), journal website (Psychophysiology, Neuroscience and Trends in Neuroscience). Manual book search, including library information were included in the data collection process. Other additional information was searched through Google.

**Results / Findings:** Blood and brain glucose levels play a vital role in error commission, and are related to error commission, monitoring and processing through the modulation of the activity of the dopaminergic system. To summarize the results

of our findings, here we suggest a hypothesis of Alcohol-Related Glucose-Dependent System of Error Monitoring and Processing (ARGD-EMPS hypothesis), which holds that the disruption of the EMPS is related to the competency of glucose homeostasis regulation, which in turn may determine the dopamine level as a major component of the EMPS. The ARGD-EMPS hypothesis explains the general processes and mechanism of alcohol related disruption of the EMPS.

**Conclusion:** Alcohol may indirectly disrupt the EMPS by affecting dopamine level through disorders in blood glucose homeostasis regulation. The effect of alcohol use on EMPS may be realized through its action on the blood/brain glucose level.

**Keywords:** Alcohol related disruption, error commission, error monitoring and processing system

### Introduction

A phenomenon of increased error commission associated with decrease in blood glucose level was recently reported. In the same study, a significantly higher rate of total error commission among alcohol users, compared to the values for the non-alcohol users was noted. The degree of error commission in various psychophysiological tests/tasks involving the analysis of intellectual capacity on visual, auditory and operative memory and attention significantly increased with decrease in the blood glucose level ( $\rho = -0.9$ ;  $p < 0.0001$ ) (Table 1). This correlation value was noted only after the 4<sup>th</sup> and 6<sup>th</sup> hrs of mental activities, during when significant differences in the glycemic levels of non-alcohol and alcohol users were noted<sup>1</sup> (Table 1 & Fig 1). This strong negative correlation following the decrease in blood glucose level permits us to think about a possible role of glucose homeostasis regulation in error commission<sup>1</sup>. Changes in blood glucose level may affect error commission<sup>1-4</sup>, monitoring and processing<sup>2-4</sup>.

Table 1: Correlation values of the glycemic levels with the total number of errors committed in course of intensive mental activities<sup>1</sup>

$\rho$ values of blood glucose level with the total number of errors committed in every phase of the experiment under intensive mental work			
Initial	After 2hrs	After 4hrs	After 6hrs
-0.10	-0.38	-0.84*	-0.91**

*N/B: Correlation values are calculated from the Spearman rho ( $\rho$ )*

*\* $p < 0.001$ ; \*\* $p < 0.0001$*

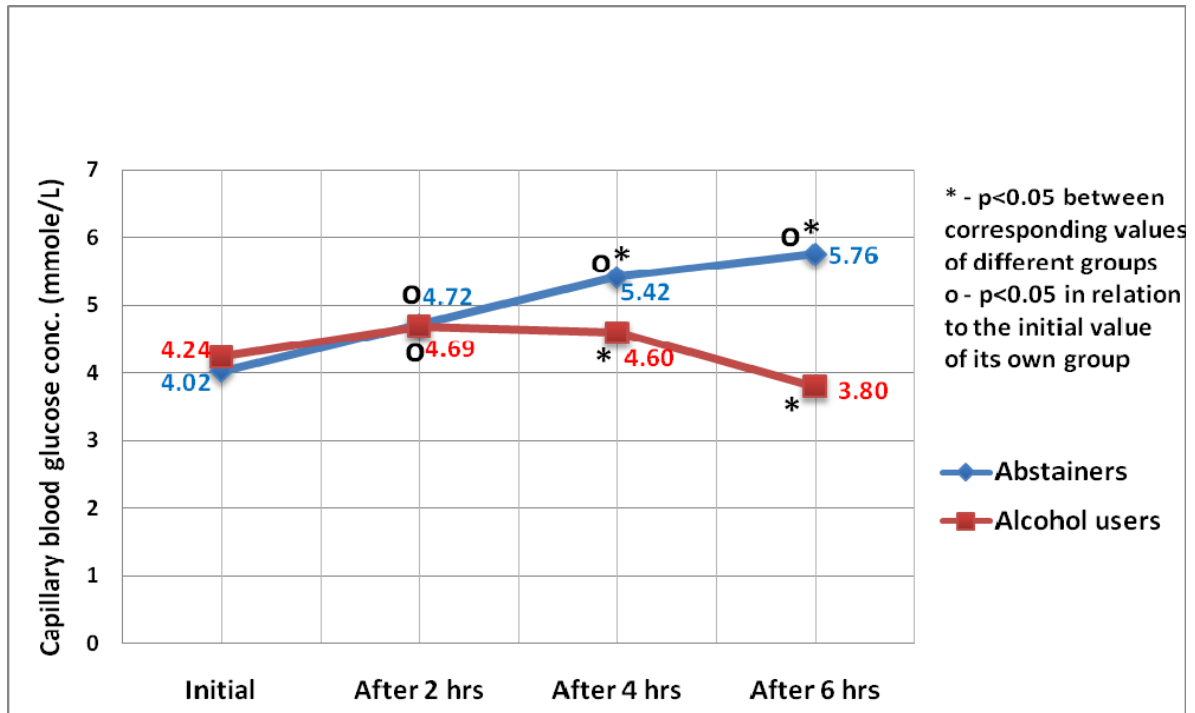


Fig. 1: Initial values of capillary blood glucose level of abstainers (controls) and alcohol users (cases) in course of intensive mental activities<sup>1</sup>

Ridderinkhof and colleagues had earlier reported that alcohol consumption disrupts error monitoring<sup>5</sup>. According to electro-physiological studies, the effect of alcohol on the Error Monitoring and Processing System, EMPS is reflected in the reduced amplitude of the Error Related Negativity, ERN (or Error Negativity, Ne)<sup>6-9</sup>, a negative deflection in the electroencephalogram with a maximum in the midline of the frontocentral region of the scalp having a latent period around 50-150ms<sup>5,10-12</sup>. It has been suggested that alcohol may directly disrupt the system or indirectly by disrupting the stimulus processing system upon which the EMPS depends<sup>10</sup>. The EMPS is a monitoring response system located in the mediofrontal brain<sup>5,13,14</sup>, basal ganglia<sup>11,15</sup> and is responsible for error commission, detection and correction<sup>5,10,16-21</sup>. Although, recently, precise brain regions like the substantia nigra, nucleus accumbens, amygdala, and hypothalamus including the insula have been implicated in modulating the functions of the EMPS<sup>11,22,23</sup>, its major components are the Anterior Cingulate Cortex (ACC) and dopaminergic system<sup>2,5,10,24-27</sup>. The functions of the EMPS are dependent on the degree of phasic dopamine activity on the ACC<sup>10</sup>. Considering the pivotal role of dopamine in regulating the activities of EMPS<sup>2,5,10,11</sup>, it may be assumed that any change in the brain dopamine levels might necessarily affect error monitoring and processing<sup>10</sup>. Importantly, the levels of dopamine have been reported to change when subjects commit error, with subsequent effect on the ACC

activeness<sup>12,20,28,29</sup>.

How does alcohol disrupt the EMPS? Literature data suggest that there could be a link between error commission and glucose metabolism<sup>1,15,30-37</sup>. Coupled with the well known fact that alcohol consumption results in hypoglycemia<sup>38,39</sup>, it is therefore, necessary to assume that disorders in glucose homeostasis regulation might change the brain dopamine level<sup>31-37</sup>. This change in dopamine levels may affect the activity of EMPS<sup>5,10,11,31-37</sup>. The possible effects of the changes in blood glucose level on the brain dopamine level (major factor that regulates EMPS)<sup>31-37</sup>, as well as the implication of the dopaminergic system in alcohol use<sup>10</sup> allows to assume that disorders in the competency of glucose homeostasis regulation which might result after alcohol consumption<sup>18,38</sup> could be one major cause for the disruption of EMPS<sup>5,10,31-37</sup>. In fact, current knowledge on brain metabolism, suggests that the degree of error monitoring and processing might depend on the concentration of glucose in extracellular fluid around neurons<sup>30,32</sup>. Besides, current scientific data reveal that decrease in the competency of glucose metabolic regulation negatively affects neuronal functions through decreased dopaminergic activity<sup>30-33,36</sup> which might in turn lead to increase in error commission<sup>10,30,32</sup>.

Based on increasing evidence from a wide range of modern literature data<sup>1-39</sup>, here we proposed that the disruption of EMPS by alcohol might be indirect and realized through its effect on the competency of glucose homeostasis regulation. The indirect disruption of the EMPS is summed up in the hypothesis of alcohol-related glucose-dependent system of error monitoring and processing (Fig 2). Included in this hypothesis are major concepts of Ridderinkhof *et al's*<sup>5</sup>, and Holyod & Yeung's<sup>10</sup> models, and the selfish brain theory<sup>40</sup>. The major concept of this hypothesis holds that the error processing capacity of the ACC depends on the blood-brain glucose proportionality level<sup>30,32,40</sup> which affects the dopaminergic system<sup>29,30,32,34,35,37</sup> as a major component of the EMPS<sup>5,10,11</sup>.

## **Methodology**

### **Search Strategy**

We used five parallel search strategies to obtain relevant information from the following literature sources:

### **Sources of Literatures**

1. Library information were used to collect data of the processes of alcohol related effect on error commission.
2. Manual search of the table of contents of books for relevant chapters on how alcohol affects error commission.
3. Scientific databases: the following databases were included in the search process - Medline, DOAJ, Embase from 1940 to August 2010.

4. The indexes of recognized journals (Psychophysiology, Neuroscience and Trends in Neuroscience) were hand-searched to ensure coverage of those not abstracted in the online databases.
5. Internet searches (other additional information were searched through Google).

### **Search Design/Techniques**

Searches in peer reviewed databases, journal websites, and Google were conducted in the following phases. Once a preliminary list of articles was determined, the databases, Google, and Journals were researched for additional articles that did not appear in the initial searches but may be appropriate for the literature review (see Table 2). Using an analytical and ancestry approach the articles chosen were scanned for further relevant articles. The following techniques were applied to further retrieve relevant articles for the review process.

**Backward References Search:** The references of high quality articles were searched to retrieve important information about alcohol's effect on error commission, monitoring and processing.

**Forward References Search:** Reviewing additional articles that have cited the article, to locate follow-up studies or newer developments related to the phenomenon under study.

The backward and forward searches were terminated when to new idea was found to support the phenomenon under study. The number of literatures retrieved in all searches is given in table 2.

Table 2: Number of literatures in various searches

<b>Search design/technique</b>	<b>Scientific databases</b>	<b>Journal website</b>	<b>Books with RC*</b>	<b>Library information</b>	<b>Google</b>
Initial search	1298	2416	36	46	2860
Second search	1392	2519	–	–	2989
Filter (focused) search	213	1565	–	–	–
Relevant literatures	157	867	36	38	265
Backward search	889	978	87	69	–
Forward search	87	80	–	–	–

*\*Books with Relevant Chapters*

### **Keywords Search Parameters**

This study was limited to keyword searches that resulted in the most relevant results. A keyword combination search was used considering the vast definition of “error” in modern concepts. A single keyword search was not performed as words like “error” or mistake lack clear definition and maybe impossible to complete an exhaustive online search. The combination of keywords could better describe the aim of the study. The following keyword combinations were employed in all cases of the literature search:

1. Alcohol and error detection
2. Alcohol and correction
3. Alcohol and error commission
4. Alcohol and error processing (OR monitoring)

### **Literature Selection Process**

#### **Inclusion Criteria**

We included studies (original communications, review articles, reports, and book chapters) that report on the aim of this study. The articles were selected based on their relevance to the topic areas of study. The results of searches were filtered according to their relevance to the aim of this search. The titles that were logged in the various databases searched were analyzed against the key terms. Reports not wholly focused on the topic of this study were removed. All the retrieved publications were reviewed with emphasis on the effect of alcohol on the blood and brain glucose levels, and cognitive functions and their possible role in error commission, monitoring and processing, including associated theories and hypothesis were examined.

This study included literatures that meant the following criteria:

1. Literatures that meant the parameters of the keyword search.
2. We considered literatures written in English.
3. We focus on studies that explicitly discuss effects of alcohol on the error commission, monitoring, processing, alcohol’s effect on the blood glucose level, and associated theories and hypotheses.
4. We focused on studies that clearly distinguish the role of alcohol on error commission, monitoring and processing from other definition of error in other contexts.
5. We assess the quality of studies based on the clarity with which methodologies and results are described.
6. The study includes clear description of research background and context in which it was conducted.

Literatures that fulfilled the above criteria were stored in separate files: Electronic literatures were saved in their original formats as computer files, while information collected from library and book chapters were stored in a document file. Only a few literatures out of the lots that meet the inclusion criteria are included in the reference list (see Table 2).

### **Data Analysis and Synthesis**

Data were extracted and recorded in Excel 2007. The statistical value for significance was set at  $p < 0.05$ . Literatures that report theories and hypotheses on how alcohol might affect error commission, monitoring and processing were noted. Studies were analyzed and synthesized into theories to explain how alcohol affect error commission and disrupts error monitoring and processing.

### **Results and Discussion**

As reported by majority of literatures, there are at least four hypotheses/theories that could explain how alcohol consumption affects the error monitoring and processing system by reducing amplitude of the Error Related Negativity, ERN. They are hypothesis of error detection of the ERN<sup>10,41,42</sup>; reinforcement-learning theory of the ERN<sup>10,43</sup>; conflict-monitoring theory of the ERN<sup>10,11,25,43</sup>; and the integrated conflict-monitoring and reinforcement-learning theory of the ERN<sup>10,11,25,27,43</sup>. The first three hypotheses/theories listed above were reported by at least 120 studies for each theory or hypothesis. Fewer studies (53) reported the last theory.

#### **Alcohol and the Error Detection Theory of the ERN**

Alcohol disrupts response monitoring and the effectiveness of cognitive capacity<sup>5,10,44-50</sup>. It is known that alcohol reduces the amplitude of ERN and activeness of the dorsal ACC<sup>5,10</sup>. The amplitude of ERN reduces on incorrect responses<sup>5,11,12,20</sup>. Alcohol acts on dopamine receptors by interfering with the activity of dopaminergic system which subsequently leads to the decrease in ERN amplitude<sup>5,10</sup>. Thereby, alcohol may lower the activity of the error detection system, by decreasing the error detection capacity which is associated with the quality of information upon which the error monitoring and processing system depends<sup>10,11,51</sup>. The resultant effect is lowering of response correctness and effectiveness of cognitive capacity<sup>5,10,11,25</sup>. Alcohol intoxication disrupts normal execution of stimulus related activation of the ACC, cerebellum and the prefrontal cortex which in turn leads to cognitive deficit<sup>10,25,52</sup>.

#### **Alcohol and the Reinforcement Learning Theory of the ERN**

This theory is based on recent findings which suggest that the basal ganglia monitor and steadily predict the result of ongoing events (ability to determine whether the end-result of events will be favorable or not)<sup>11,12,15,24,25</sup>. The theory explains how ACC controls and increases the effectiveness of action and modulates commands with the



help of dopamine signals. ACC receives command information from several neuronal origins, called controllers (basal ganglia, dorsolateral prefrontal and orbitofrontal cortices, amygdala etc)<sup>10,11,25</sup>. In conditions, when incoming commands are conflicted, ACC acts as a signal selector and transfers information which may be more adequate for a successful completion of a set target to the motor systems and controllers. This is why the ACC is regarded as a control filter<sup>10,11,24-26,51,53,54</sup>. Effect of dopamine signal on the apical dendrites of motor neurons of ACC modulates the amplitude of ERN, so that the phasic decrease in dopamine activity (meant that the result of the present action is worse than expected i.e. error) is associated with a high ERN and vice versa<sup>10,25</sup>.

Nucleus accumbens, NA may play significant role in the realization of the action of dopamine on the ACC<sup>10,23</sup>. According to Munte *et al*<sup>23</sup> NA is greatly implicated in error monitoring and processing. NA is a limbic motor interface, which receives information from the prefrontal cortex, hypothalamus, amygdala under which its actions are modulated by dopamine. Besides, the NA can receive information preceding ERN in the ACC<sup>10,12,18</sup>. Furthermore, increase in NA dopamine level is associated with acute alcohol use<sup>12,23</sup>. The reinforcing properties of alcohol are realized through dopamine D1 & 2 receptors<sup>10</sup>. According to the reinforcement learning theory of the ERN, alcohol related disruption of the mesencephalic dopamine system may decrease the amplitude of ERN by increasing the “tonic” activity of mesencephalic dopamine system and subsequently leading to inhibition of ACC activity, the result of which is the reduction of ERN amplitude<sup>10,23</sup>.

### **Alcohol and the Conflict-Monitoring Theory of the ERN**

The theory suggests that ACC trace for response conflict (by simultaneous activation of descending response channels) and sends this information to cognitive control brain regions like the lateral prefrontal cortex<sup>25,43</sup>. Conflict occurs as a result of simultaneous activation of different regions, controlling the activation of different levels of competing motor control units in the motor cortex<sup>10,11,24,26,43</sup>. Processing of stimulus is characterized by constant flow of activity in the pathways that send stimulus related information to the cortex of the hindbrain, and subsequently results in the corresponding response in the motor cortex<sup>10,25,43,51,55-57</sup>. Distractive stimulus may activate incorrect response in this system<sup>10,25,58-60</sup>. As opposed to the reinforcement learning theory, the conflict monitoring theory supports the fact that ACC produces an additional excitability phasic response, N2 (N2 is produced by the neurons of ACC as a conflict monitor, while ERN is produced also by ACC as the control monitor), when it detects a pre-response conflict<sup>10,25</sup>. According to the conflict monitoring theory, ERN is formed when a constant processing stimulus after error commission results in the activation of correct responses, subsequently producing a post-error conflict<sup>10,11,25,43</sup>. Alcohol selectively acts on the ERN, while the N2 amplitude is not affected<sup>10</sup>. Alcohol related disruption of stimulus processing decreases the activation

of correct responses, immediately after when subjects commit an error which in turn decreases the post-error conflict and so decreases the ERN amplitude<sup>10,11,43,61-72</sup>.

### **Integrated Conflict Monitoring and Reinforcement Learning Theory of the ERN**

This theory considers the integration of electrophysiological signals during monitoring of action and reinforcement learning at the biological and cognitive levels<sup>23,73-79</sup>. The model considers ERN as part of the constant process of ongoing monitoring<sup>11,20,23,25,43</sup>. According to this integrated view of the ERN, ACC filters sensory impulses and sends error signals to other brain regions<sup>11,23</sup>. Although not fully understood, it is suggested that these error signals are generated by the basal ganglia (the adaptive critics), which undertake processing of input signals, and are also predictor of event related results<sup>11,18,23,26</sup>. Discrepancy between these processes produces a phasic shift in dopamine signal, leading to “temporal difference error”. These errors are sent through the mesencephalic dopamine system to three brain regions – 1) motor control systems (i.e. amygdala, dorsolateral prefrontal and orbitofrontal cortices); 2) control filter – ACC; 3) and again to the adaptive critics – basal ganglia. Phasic shift in dopamine signal (may be caused by alcohol<sup>80-95</sup>) in these regions disinhibits ACC and modulates the magnitude of ERN-signal<sup>11,20,23,25,43,79,96-102</sup>.

### **Hypothesis of Alcohol-Related Glucose-Dependent System of Error Monitoring & Processing (ARGD-EMPS Hypothesis)**

Recent studies suggest the possibility that alcohol’s effect on the EMPS is related to its action on glucose homeostasis regulation, especially in tasks requiring high cognitive control<sup>1,15,18,31-39,44,49</sup>. The number of errors committed in an experiment is inversely correlated with the glycemic levels, especially among alcohol users (Table 1)<sup>1</sup>. Our analysis<sup>1,15,18,31-39,51,103</sup> suggests the possibility that hypoglycemia might be necessary for the disruption of the activity of EMPS among alcohol users.

The ARGD-EMPS hypothesis (Fig 2) is based on the notion that the effects of alcohol consumption on the system are the result of disorders in glucose homeostasis regulation<sup>1,9,15,18,31-39,44,49,57</sup> which in turn is associated with decrease in cognitive functions (may be manifested as increased in error commission)<sup>5,10,11,15,20,30</sup>. This is also confirmed by the correlation analysis between the academic performance and the number of errors committed by alcohol users in a cognitive task<sup>1,54</sup>. According to the ARGD-EMPS hypothesis, it is envisaged that alcohol consumption, especially during tasks requiring high cognitive control, might result in glucose homeostasis dysregulation<sup>1,9,15,18,31-39,51,103</sup> which might lower the activity of the dopaminergic system<sup>15,31-35,37</sup>, with subsequent effect on the ACC activeness<sup>9,18,57</sup>, leading to low ERN amplitude<sup>1,9,15,18,31-39,51</sup>. The brain glucose level may determine the degree of error commission, monitoring and processing<sup>9,15,31-37</sup>. In fact, decreased glucose metabolism in ACC closely correlates with the results of neurophysiological tests<sup>9</sup>.

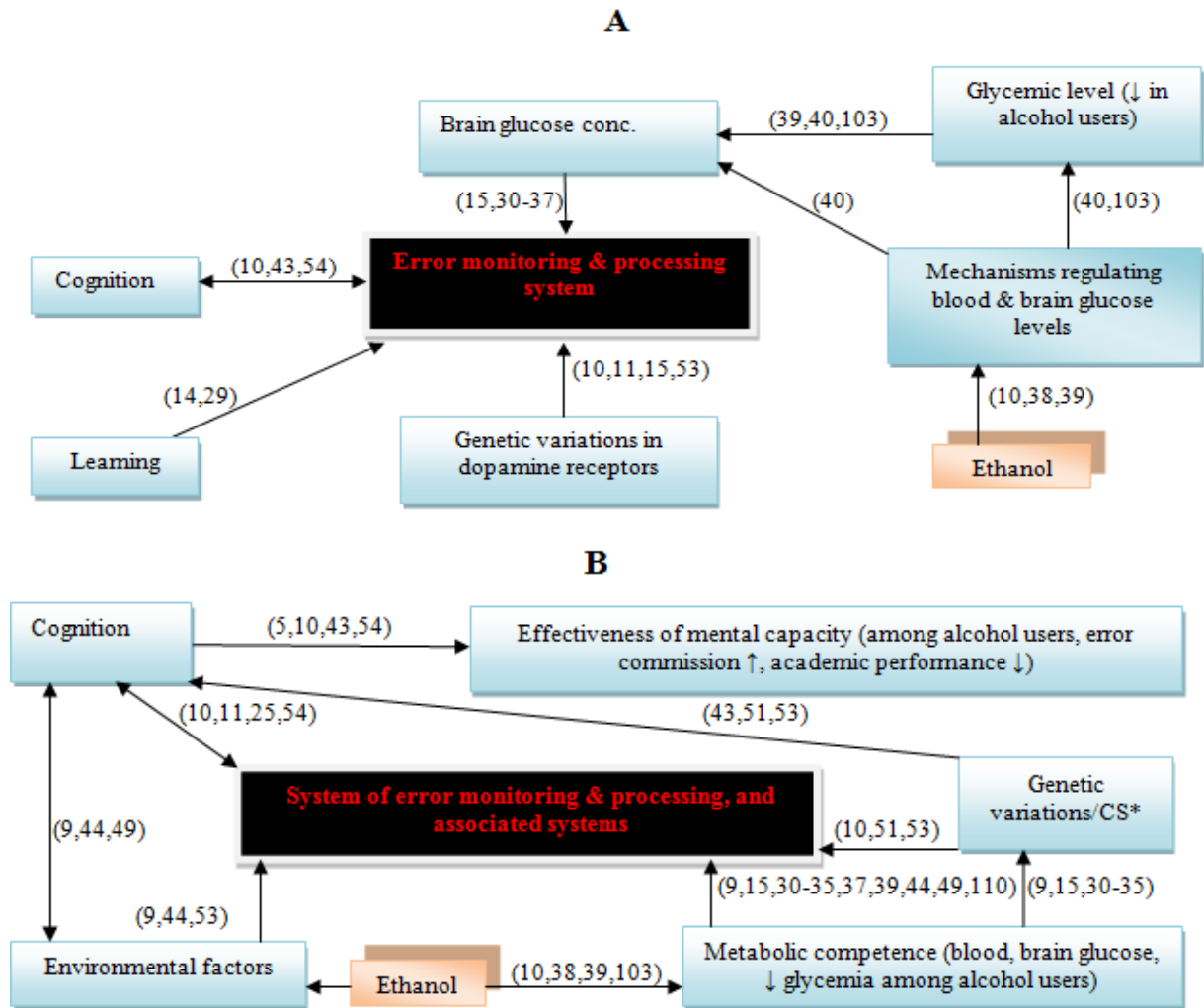


Fig 2: Hypothesis of the indirect disruption of the error monitoring & processing system by ethanol

N/B: ↓ – decrease; ↑ – increase; CS\* - Change in the Sensitivity of dopamine receptors. References to each concept are shown in bracket by the arrow.

Fig 2A: Ethanol affects the functions of the EMPS by altering the brain and blood glucose levels through its action on the mechanisms that regulate the blood-brain glucose concentration. Genetic variations in dopamine receptors can also affect the activities of the EMPS. Learning affects this system by decreasing the degree of error commission (erroneous actions). The resultant effect of all these components on EMPS indirectly affect cognition, at the same time as the level of cognition can affect the activity level of EMPS.

Fig 2B: Ethanol as a component of environmental factors can affect cognition, as the level of cognition may affect the level of alcohol use. Ethanol reduces the glycemic level of alcohol users, especially in tasks requiring high cognitive control, and

*subsequently affecting EMPS by (a) its action on the system (b) and may cause genetic variations or may change the sensitivity of dopamine receptors, thereby, affecting the degree of gene expression. Genetic variations (e.g. in dopamine receptors) could affect the level of cognition. Also, the level of cognition may determine (or reflected in) the effectiveness of cognitive activities.*

It is established that the brain glucose level is proportional to the blood glucose level<sup>40,103</sup>. That is why a decrease in blood glucose concentration leads to a decrease in brain glucose concentration, and subsequently a decrease in cognitive functions, which may be marked by increased in error commission<sup>9,11,103</sup>. The fact that decrease in blood glucose level, caused by alcohol consumption in a cognitive task might affect the activities of the EMPS by increase in the number of error commission<sup>5,9,10,11,15,18,31-39,51,57,103</sup> is evident in the hypothalamic control of blood and brain glucose levels by dopaminergic system<sup>36,37</sup>. The blood glucose level increases with increase in homovanillic acid (metabolite of dopamine) on fasting<sup>36</sup>. Effect of glucose on dopamine is realized through the activities of GLUT- 2 receptor (also similar to the pancreatic  $\beta$  cell) located in hypothalamic neurons<sup>33,36,37</sup>.

In case of unsuccessful response (i.e. error), the basal ganglia and hypothalamus is actively engaged with the working system of cognitive control formed by the interaction between dopaminergic system and ACC<sup>11,15,18,104</sup>. The increased error commission associated with alcohol consumption might be related to decrease in dopaminergic functions<sup>10</sup>, which is likely caused by decreased competence of glucose homeostasis regulation<sup>34,36,37</sup>. Several studies have shown a link between the dopaminergic system and glucose homeostasis regulation<sup>15,30-39</sup>. In fact the link between the dopaminergic system and the blood glucose level is manifested in Parkinson disease, schizophrenia, and tardive dyskinesia in which disorders in dopaminergic functions are associated with disorders in peripheral glucose metabolism<sup>30,32,35,105-108</sup>. The link is further justified by the hyperglycemic effect of bromocripton (dopamine agonist) administration<sup>44</sup>. The mechanism of these processes is presently not fully understood, although it is known that antipsychotics act not only on dopamine receptors, but also on other neurotransmitter systems<sup>44</sup>. Putting into consideration the aforementioned role of blood glucose level in the dopaminergic system and maintenance of brain functions<sup>30-35,37,49</sup>, as well as the vital role of dopamine as a major component in the error monitoring and processing system<sup>5,10,11,30,32</sup>, it may be suggested that disorders in glucose homeostasis regulation may lead to disruption of the EMPS<sup>9,14,30,32</sup> (Fig 2). This is the main concept of the hypothesis of alcohol-related glucose-dependent system of error monitoring and processing (Fig 2). This hypothesis explains a general view of the processes and mechanism involved in error commission under alcohol consumption. The central components of this hypothesis which determine the degree of error commission are the blood and brain glucose concentrations, as well as the brain dopamine level.

The difference in the amplitude of ERN (including individual differences) that is reflected in the magnitude of phasic dopamine response in the process of error monitoring and processing<sup>51,53</sup>, may be related to genetic variations in dopamine receptors, especially DRD2 and DRD4, as well as other genes coding for enzymes and transporters of dopamine like cathechol-O-methyltransferase (COMT) and dopamine transporter, DAT<sup>36,51,105,109</sup> (Fig 2). Glucose receptors, including the insulin-like growth factor (IGF-1 & 2) are located in significant numbers in DAT-expressing dopamine neurons of the midbrain<sup>31,34,36,37,110</sup>. Glucose, insulin and the IGF-1 & 2 possess a unique role in modulating the functions of the dopaminergic system<sup>31,34,35,36,37</sup>. In fact, the amygdala dopamine level increases immediately after injection of glucose<sup>11,33,34</sup>.

### **Study Limitation/Weakness**

Since our search was based on English literatures, we cannot claim to have completed a comprehensive and international review. This limitation may be mitigated by the reality that English has been the lingua franca of the majority of the web. Furthermore, nature and content of databases and web of literatures increase daily.

### **Conclusion**

Blood and brain glucose levels play a vital role in error commission, and are related to the activity of the Error Monitoring and Processing System (EMPS) through the modulation of the activity of the dopaminergic system. The suggested hypothesis of alcohol related glucose dependent system of error monitoring and processing (ARGD-EMPS hypothesis) holds that the disruption of the EMPS is related to the competency of glucose homeostasis regulation, which in turn may determine the dopamine level as a major component of the EMPS. The ARGD-EMPS hypothesis explains the general processes and mechanism of alcohol related disruption of the EMPS. Alcohol may indirectly disrupt the EMPS by affecting dopamine level through disorders in blood glucose homeostasis regulation. The effect of alcohol use on EMPS may be realized through its action on the blood/brain glucose levels.

### **Future Research**

Since decreased EMPS activity is related to disorders in glucose homeostasis regulation (blood and brain glucose levels play a vital role in error commission), through the modulation of the activity of the dopaminergic system, future research will examine the electrophysiological components of error commission in relation to varying mental states and functions, with special attention to the activity of the dopaminergic system and ACC, as well as blood and brain glucose levels after alcohol consumption (in various doses and intervals of use) using various cognitive tasks.

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