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Abstract
Cortisol induced memory loss was due to disruption of hippocampal function. It negatively affects the long-term potentiation which is associated with learning and memory. Based on these observations, concern has been raised that prolonged elevation of endogenous glucocorticoids (GCs) caused by chronic cortisol or pharmacological doses of GCs commonly administered to humans with inflammatory or bronchospastic diseases might produce neuronal loss. On the other hand, studies had shown presence of potential neuroprotective plant with flavonoid and coumarin compounds plays important role against oxidative stress because it regulate the cortisol level. This infers the efficacy of flavanoid compound of Fructus Psoreleae (FP) in controlling cortisol level. So, the administration of FP not only regulate the cortisol and also reverse the elevated cortisol levels which was very much useful in the treatment of neuropsychiatric disorders such as depression, schizophrenia and alzheimers disease.

Keywords: Hippocampus, Endogenous glucocorticoids, Schizophrenia and Alzheimer’s disease

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**INTRODUCTION**

Cortisol is a steroid hormone, in the glucocorticoid class of hormones, and is produced in humans by the zona fasciculata of the adrenal cortex within the adrenal gland. It is released in response to cortisol and low blood-glucose concentration. Despite the other organ like heart, liver and kidney, brain is the most affected organ by cortisol. The release of cortisol is controlled by the hypothalamus, a part of the brain. The secretion of corticotropin-releasing hormone (CRH) by the hypothalamus triggers cell in the neighboring anterior pituitary to secrete another hormone, the adrenocorticotropic hormone (ACTH), into the vascular system, through which blood carries it to the adrenal cortex. ACTH stimulates the synthesis of cortisol, glucocorticoids, and dehydroepiandrosterone (DHEA) 1, 2. Rodent studies suggest that prolonged exposure to elevated glucocorticoid (GC) concentrations lowers the threshold for cerebral neuronal degeneration and loss 3. The major effects of prolonged exposure of cortisol in brain are memory loss, confusion and augmentation. It was due to increased free radical production and a resultant state of oxidative cortisol. A few studies suggest GC neurotoxic effects in primates 4, 5. Older animals may be particularly vulnerable to this phenomenon 6. Cortisol induced memory loss was due to disruption of hippocampal function. It negatively impacts the LTP which is associated with learning and memory 7, 8. Based on these observations, concern has been raised that prolonged elevation of endogenous GCs caused by chronic cortisol or pharmacological doses of GCs commonly administered to humans with inflammatory or bronchospastic diseases might produce neuronal loss 9. On the other hand, studies had shown presence of potential neuroprotective plant with flavonoid and coumarin compounds plays important role against oxidative cortisol 10, 11. Flavanoid compound has been shown to rapidly activate adenylate cyclase, increase intracellular [Ca^{2+}], activate phospholipase C to generate inositol 1,4,5-trisphosphate and diacylglycerol, stimulate nitric-oxide synthase to generate nitric oxide, and activate the extracellular regulated kinases 1/2 (ERK1/2) mitogen-activated protein kinase (MAPK) pathway 12.

**AIM OF THIS STUDY**

1. To investigate the effect of FP induced cortisol level in the hippocampal area of the rat brains by hormonal study.
2. Comparing the effect of cortisol and FP in the neurons of the hippocampal area of the rat brains by histological study.

**MATERIALS AND METHODS**

The study was conducted on thirty female healthy adult wistar albino rats in laboratory conditions (i.e. room temperature of 25±2°C; relative humidity 45% to 55% and a 12:12 light/dark cycle). The approval of the Institutional Animal Ethical Committee (IAEC) of Saveetha University (IAEC No.Anat.002/2009) was taken prior to the experiments. All the protocols and the experiments were conducted in strict compliance according to ethical principles and
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guidelines provided by Committee for the Purchase of Control and Supervision of Experiments on Animals (CPCSEA).

Experimental design
The experiment was carried out for a period of 2–4 months with 30 healthy adult wistar albino rats. Before starting the experiment, the rats were made to acclimatize to the laboratory environment for one week. Then rats were randomly assigned into 4 groups of 6 animals with age of 2 months.

- Group I (Control group): This group rats were housed and maintained for two months from the start of the experiment. After two months, the rats were sacrificed.
- Group II: Orally treated with FP in a daily dose of 25 mg/kg orally by gavage for 2 months.
- Group III: Induced with synthetic glucocorticoids by intraperitoneal injection of hydrocortisone sodium succinate at 15 mg/kg, respectively, for 20 days (cortisol) daily.
- Group IV: Induced with synthetic glucocorticoids (cortisol) 15 mg/kg BW and orally treated with FP 25 mg/kg by gavage method for 2 months.

Cortisol Assay
The blood from each animal of all the groups were collected and analyzed for cortisol assay using ADVIA Centaur System. The ADVIA Centaur cortisol assay is a competitive immunoassay using direct chemiluminescent technology.

RESULTS
Cortisol level result
The blood from all the rats were collected and analyzed for cortisol assay using ADVIA Centaur System.

Table 1: Mean ± SEM of Cortisol Assay (pg/ml) during the days of training after FP extract and/or cortisol treatment in rats.

<table>
<thead>
<tr>
<th>Group</th>
<th>Cortisol level (pg/ml)</th>
</tr>
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<tbody>
<tr>
<td>Group I</td>
<td>8.7±0.29</td>
</tr>
<tr>
<td>Group II</td>
<td>6.2±0.26</td>
</tr>
<tr>
<td>Group III</td>
<td>14.4±0.41</td>
</tr>
<tr>
<td>Group IV</td>
<td>10.2±0.56</td>
</tr>
</tbody>
</table>

This above results showed that the mean cortisol level was increased in group III as when compared with other groups. But in group II, it was decreased as compared to all the other groups. So, this showed that FP regulating the cortisol level. The ANOVA test done for cortisol level showed that the difference in cortisol level between the groups were statistically significant (F= 62.37; P<0.01). Post hoc test revealed that comparing the mean cortisol level of all the five groups, group II had significant value. This showed that cortisol induced the cortisol level in group IV but in it was regulated by FP. So FP treated group was better than other groups. Cortisol level of FP treated group was significantly different from other group (n=6; F= 62.37; P<0.01).
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Figure 1: Bar diagram: Mean ± SEM of Cortisol (Pg/ml) of all the groups

Figure 2: Topography of toludine blue stained hippocampus (400x magnification)

G I - Blue arrow shows the Pyramidal Shaped Neurons
G II, G IV - Red arrow shows the Increased Pyramidal Shaped Neurons
G III - Black arrow shows the Inactive Circular Shaped Neurons
DISCUSSION

This study showed that cortisol, a hormone produced by the adrenal gland within the HPA-axis in response to synthetic glucocorticoids, was high level in cortisol treated group. Mainly it produces cortisol related loss of neurons and thereby decreases long-term potentiation (LTP) in the hippocampus. The present study reveals that increased cortisol impairs the hippocampal dependent memory, which was proved by the above data (Table 1). So elevation of cortisol must necessarily be associated with learning deficits in rats. These findings were consistent with previous research that cumulative exposure to cortisol had functional and structural effects on the hippocampus and finally end up with decrease hippocampal integrity and hippocampal dependent cognitive task. On comparing group II (9.7±0.28) with group IV (14.4±0.41), cortisol had raised in group IV. So the synthetic glucocorticoids induced elevation of cortisol was due to the activation of HPA which produce more complex effects on cognitive behaviour and also attenuates the normally occurring neurogenesis in hippocampal dentate gyrus, a process that was thought to be necessary to sustain a constant level of neuron density in this region. Prolonged exposure to glucocorticoids was also associated with several adverse effects on brain morphology, particularly in the hippocampus. The exposure may ‘endanger’ neuronal integrity and cause atrophy of apical dendrites in the Ammon’s horn. It has been proposed that also these neurodegenerative changes may induce some of the deleterious behavioral effects. The hippocampus was enriched with two classes of corticosteroid receptors, type I, mineralocorticoid receptors (MRs); and type II, glucocorticoid receptors (GRs). Chronic exposure to glucocorticoids induces changes in adrenal steroid receptor density and or affinity that may account for some of the cognitive effects. This effects of glucocorticoids (cortisol) on the hippocampus depends on the concentration GRs. The adverse effects of cortisol on the hippocampus seems to be mediated largely by the lower-affinity GRs, which become heavily occupied with corticosteroids in response to cortisol.

In the rat hippocampus, corticosterone binding to GRs had been shown to adversely affect neuronal metabolism, cell survival, physiological functions and neuronal morphology. Moreover, changed receptor densities induced by prolonged glucocorticoid exposure may alter autonomic, neuroendocrine and
behavioral responsiveness during learning, which may affect memory consolidation. MR/GR imbalances can occur and the blockade of MR by a specific antagonist increases circulating levels of cortisol under basal and cortisol condition. So the secretion of high level of cortisol was known to affect learning and memory. But in group II and IV, the cortisol level was decreased (6.2±0.26; 10.2±0.56) compared with group III (14.4±0.41). This was mainly due to the effect of FP. It had been suggested that action of FP on dysregulation and normalization of the HPA axis system plays an important role in the pathophysiology of cognition.

Calcium flux through NMDARs was thought to be critical in synaptic plasticity, a cellular mechanism for learning and memory. Corticosterone modulates NMDA receptor–mediated Calcium influx in cultured hippocampal neurons and NMDA-dependent long-term potentiation and these effects were mediated by glucocorticoid receptors. In addition, NMDA receptor plays a critical role in dendritic plasticity and dendritic reorganization occurring in frontal cortex after cholinergic differentiation. NMDA receptors mediate corticosterone’s effects on dendritic morphology in hippocampal CA3 neurons. Thus, glutamatergic transmission at NMDA receptors may play a role in cortisol-induced dendritic reorganization in hippocampus. Consistent with this hypothesis, chronic administration of corticosterone down regulates expression of the NR2B subunit of the NMDA receptor in hippocampus. Given that NMDA receptor activation was crucial for producing remodeling in the hippocampus due to cortisol and for reorganization in frontal cortex due to cholinergic differentiation. NMDA receptors may also play a role in alcoholic cortisol-induced dendritic remodeling in medial prefrontal cortex. So by blocking NMDA receptors during chronic cortisol prevents cortisol-induced dendritic retraction of hippocampus. So FP may act as NMDA receptor blocker, which blocks sodium and T-type calcium channels.

Normal level of cortisol facilitate hippocampal plasticity and promote the survival of dentate gyrus granule cells through activation of MR-mediated effects which stabilize neuronal transmission and appear critical for neuronal integrity of a sub-region of the hippocampal dentate gyrus. This finding suggests that one of the MR-mediated effects of cortisol was the suppression of the activity of the HPA axis by means of increase hippocampal plasticity. In contrast, the elevated cortisol levels of group III (14.4±0.41),
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not only to negate the effects of MR activation but also to promote the debilitating GR effects on hippocampal function, including a dampening of LTP. So this proposed that decreased hippocampal volume was associated with high cortisol level. Intake of FP which regulate the cortisol (group II = 6.2±0.26) resulting in improved short-term memory, working memory processes such as selective attention, memory consolidation, and LTP. This result clearly explains that FP regulate the cortisol level in group II and IV (10.2±0.06). This infers the efficacy of FP in controlling cortisol level caused due to cortisol. So, the administration of FP not only regulate the cortisol and also reverse the elevated cortisol levels which was seen in group V rats. Thus, FP was very much useful in the treatment of neuropsychiatric disorders such as depression, schizophrenia and Alzheimer’s disease.

Activation of the HPA axis was considered to be a characteristic physiological response to alcoholic cortisol. Oestrogen is thought to play a causal role in the gender or menstrual cycle-dependent differential HPA-axis responses, because ovariectomy (OVX) reduces basal CORT levels, whereas estrogenic replacement restores basal plasma CORT concentrations. In addition, these data provide valuable insight into the mechanisms mediating decreased HPA responsiveness, which in-turn regulate the cortisol level. There was a strong relationship between HPA function and monoamine oxidase (MAO) activity that induces oxidative cortisol. Therefore, it had been suggested that MAO inhibitors used for treating cortisol induced alcoholics. Finally, the effect by FP was consistent with previous result that clorgyline, MAO inhibitor, inhibited HPA axis function. Therefore, inhibition of MAO and HPA axis may be responsible for the therapeutic effects of FP to treat depression and Parkinson’s disease. It seems likely that FP exhibit anticortisol activity by inhibiting MAO-A and MAO-B activities. In fact, it had been shown that psoralen and isopsoralen suppressed MAO-A and MAO-B activities in rat brain mitochondria in vitro. It was hypothesized that the FP anti cortisol acts, at least in part, by inhibition of MAO activity.

CONCLUSIONS:
So administration of FP not only regulate the cortisol and also reverse the elevated cortisol levels which was very much useful in the treatment of neuropsychiatric disorders such as depression, schizophrenia and alzhimers disease.

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NANOPARTICLE BASED AMPEROMETRIC BIOSENSOR FOR THE QUANTITATIVE DETERMINATION OF CHOLESTEROL IN HUMAN BLOOD

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ABSTRACT

Total cholesterol monitoring in human blood serum is one of the most important routine analysis performed in clinical laboratory. There is a strong correlation between coronary heart disease and blood cholesterol level. Numbers of cholesterol biosensors have been developed over the past 30 years. Cholesterol is determined enzymatically by Fibre-optic fluorescence, Fibre-optic luminescence, Potentiometric, Spectrophotometric and Fluorometric biosensors. Some of these methods suffer from interference from other substances found in the blood such as ascorbic acid and uric acid. There is need for a method that is sufficiently flexible to yield good results in clinical laboratory. The attributes of the enzyme linked platinum electrode was studied with cholesterol powder solution using cyclic voltammeter 797 VA. The peak value of the current from the electrode was detected with the input potential changes from +0.5 V to -1 V. The Mann Whitney U Test was conducted for the sampled data, the results show that the data is significant with α = 0.05. Fabricated amperometric biosensor was found to be sensitive in determining the cholesterol level in the human blood.

KEY WORDS: Spectrophotometric, Fluorometric, luminescence, Amperometric

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INTRODUCTION

For many decades, scientists have recognized the power of incorporating biological principles and molecules into the design of artificial devices. Biosensors, an amalgamation of signal transducers and biocomponents play a prominent role in medicine. Many kinds of amperometric glucose sensors were fabricated based on Cadmium Sulfide (CdS) nanoparticles modified electrode\(^1\), Cl-plasma treated Ag/AgCl reference electrode\(^2\), immobilization of glucose oxidase in chitosan on a glassy carbon electrode with gold platinum alloy multiwall carbon nanotubes\(^3\), bioelectrocatalytical glucose oxidation with phenoxazine modified glucose oxidase\(^4\), nonenzymatic glucose sensor in alkaline media with carbon nano tube on glassy carbon electrode\(^5\). The glucose concentration ranging from 1 to 26.5 mM was detected using platinum nanoparticles\(^6\). The response sensitivity of the glucose slightly changes at more positive detection potentials. An immuno sensor to detect human immunoglobulin G of cholesterol oxidase (CHOX) crystallite size using sol-gel technique for immobilization sensor containing preferred (002) plane and 10 nm ranging from 1 to 26.5 mM was detected using platinum nanoparticles\(^7\). The dose response was studied at working potential -0.3V. Needle enzyme electrode was used to measure lactate in vivo\(^8\), \(H_2O_2\) biosensor\(^9\), Insulin sensor\(^10\) and NADH sensor\(^11\) were designed to estimate \(H_2O_2\), Insulin and NADH respectively. Researchers have been made attempts to create sensitive, selective, reliable and low cost cholesterol sensors because of the clinical significance in the measurement of blood cholesterol level. Highly selective methods have been developed by utilizing the electrode modified with cholesterol oxidase\(^12\). Nanostructured zinc oxide (nano-ZnO) film onto indium-tin-oxide (ITO) cholesterol sensor containing preferred (002) plane and 10 nm crystallite size using sol-gel technique for immobilization of cholesterol oxidase (CHOX)\(^13\). A novel potentiometric sensor based on the fabrication of ISFET (Ion Selective Field Effect Transistor) coated with molecular imprint of cholesterol on the \(SiO_2\) + \(Si_3N_4\) dielectric gate of the said electrode, poly (pyrrole –co- N- methyl pyrrole)-sensor\(^14\), surface plasmon resonance based biosensor\(^15\), membrane permeability based sensor\(^16\). A high cholesterol level in human blood is related to arteriosclerosis, hypertension, myocardial infarction and many heart disorders\(^17\). There is considerable interest towards the application of silicon to biosensors. This has been attributed to their interesting properties such as biocompatibility, redox characteristics and the possibility direct electron transfer between electrode and active sites of biomolecules\(^18\). The electrochemical reaction was studied by covalently coupling cholesterol oxidase via glutaraldehyde onto electrochemically prepared polyaniline film in presence of Triton-X-100 onto indium-tin-oxide (ITO) glass substrate\(^19\). The photolithography technique is used to fabricate the nanogap based on the CMOS technology\(^20\). Cholesterol oxidase catalysis the aerobic oxidation of cholesterol to \(\Delta_2\) -cholestenone with stoichiometric production of hydrogen peroxide has opened the way for the development of electrochemical sensors for the determination of cholesterol that are based on the amperometric determination of \(H_2O_2\) at the electrode surface\(^21\).

MATERIALS AND METHODS

Reagents

Cholesterol Powder (E.C. Number – 200-353-2) - 3β-Hydroxy-5-Cholestene, \(C_{27}H_{46}O\). Molecular weight is 386.65 g/mol and Cholesterol Oxidase (CHOX) is a monomeric flavor protein containing FAD (E.C. Number – 1.1.3.6). Molecular mass is 55 kDa, \(K_m = 3.5x10^{-4}\) M (Cholesterol). One unit will convert 1.0 µMole of cholesterol to 4-cholesten-3-one per minute at pH 7.5 at 25°C purchased from Sigma-Aldrich.

Preparation of Potassium Buffer Solution

1M of Potassium Phosphate buffer solution with pH 7.0 was prepared by dissolving 17.48 gm. of potassium phosphate dibasic in 100 ml distilled water and 13.609 gm. of potassium phosphate monobasic also in 100 ml distilled water. Then volume of 1M of \(K_2HPO_4\) was mixed with 1 M of 38.5 ml of \(KOH\) to get 0.1 M potassium phosphate buffer solution. This buffer solution was converted to 50 mM by adding 50 ml of 0.1M potassium phosphate buffer with 50 ml of distilled water.

Preparation of Cholesterol oxidase solution

Cholesterol oxidase solution was prepared by dissolving the 100 UN of cholesterol oxidase in 50 mM of potassium phosphate buffer solution with pH 7.0

Synthesis of Titanium oxide

\(TiO_2\) nano powders was prepared by dissolving 8 ml of titanium tetraisopropoxide \([Ti(OCH(CH_3)_2)_4]\) in 50 ml ethanol under constant magnetic stirring. The solution obtained after 45 minutes was converted into a gel by adding 100 ml of deionized water. The white precipitate thus obtained was filtered and washed with distilled water to remove impurities. Finally, the powder was dried at 100°C.

Preparation of Cholesterol solution

The cholesterol solution was prepared by dissolving 100 mg of cholesterol powder was dissolved in ethanol.

Preparation of Titanium oxide solution

Titanium oxide nano powder solution was prepared by dissolving 100 mg of \(TiO_2\) Nano powder in diluted sulphuric acid, heated to 185°C and the dissolved titanium oxide solution color turns into brownish yellow. The UV and Raman spectrum is shown in Fig.1 and Fig.2.

\[
H_2O_2 \xrightarrow{\text{ELEcTRODE}} 2H^+ + O_2 + 2e^- \quad (1)
\]

\[
\text{Cholesterol} \xrightarrow{\text{O}_2} \text{cholesten-4-en-3-one} + H_2O_2 \quad (2)
\]
Apparatus
All electrochemical experiments were carried out on a cyclic voltameter 797 VA Computrace (Metrohm, USA). A conventional three electrode system was used in this work. The enzyme coated platinum electrode was used as a working electrode with 2mm diameter. A platinum electrode was used as a counter electrode and an Ag/AgCl electrode was used as a reference electrode. Sodium Phosphate buffer solution (0.1M) was always employed as supporting electrolyte.

CHOLESTEROL MEASUREMENT SYSTEM BASED ON ARM PROCESSOR
The amperometric cholesterol biosensor system designed for the measurement consists of several hardware modules, which include: Enzyme coated platinum electrode, ARM processor LPC 2148 and LCD display. The current from the working electrode for the input voltage can be read by the processor with the application of the cholesterol powder solution in sodium phosphate buffer solution. Further the same data related to the cholesterol concentration can be transferred it to any android system using wireless technology. Fig.3 shows the block diagram of the system.

FIGURE 3
Block diagram of the biosensor system
METHODOLOGY

The biosensor system consisting of ARM processor continuously reads the data from the electrode and displays the value on the LCD screen. The data was validated with Easy life GCU system. The observed values were stored in a register for further analysis. The processing and display software was written in C using Keil µVision3 software and the Hex code was downloaded to the processor LPC 2148. Electrochemical behavior of the sensor was identified by using cyclic voltammetry techniques. The stability of the nanoparticle mixed cholesterol oxidized biosensor has been analyzed for various temperature, pH, and cholesterol concentration. This fabricated biosensor has been characterized for cholesterol detection in the concentration range between 10 mg/dl and 1 gm/dl cholesterol by cyclic voltammetry measurement. The linear relationship between the analyte concentration and response current of the electrode was observed.

Figure 4 shows an ultra-low bias current monolithic operational amplifier. The non-standard pin out of the operational amplifier was to achieve lowest possible input bias current. The negative power supply was connected to pin 5 to reduce the leakage current from the V- supply (Pin 4) to the op-amp input terminal. With this new pin out, sensitive inputs were separated from both power supply pins. The ARM7TDMI-S (LPC 2148) is a general purpose 32-bit microcontroller, which offers high performance and very low power consumption. The current developed from the working electrode based on the cholesterol present in the blood was read by the ARM processor and displayed in the display unit.

RESULTS

The characteristics of the enzyme linked Platinum electrode (Titanium oxide + cholesterol oxidase) were studied using cyclic voltammetry in various condition. The electrode is placed in the 0.1 M sodium phosphate buffer solution and the current developed in the electrode was observed with the potential from +0.5 to -1V. The cholesterol powder solution was added and the output current from the cyclic voltameter was studied. Initially the output was studied without the nanoparticle linked at the electrode. Then we changed the electrode (nanoparticle linked), the pH value of the buffer solution and the concentration of the cholesterol solution, the output characteristics were studied using cyclic voltammetry as shown below in the Fig.5 and Fig.6. By comparing the determined concentration of cholesterol with the Easy Life GCU system, it was found that there is a linear relation between the cholesterol concentration of blood and the calculated cholesterol concentration measured from the biosensor device. The percentage mean relative error of the sampled data calculated as 0.069. The comparison curve of the sampled data is shown in the figure 7.
DISCUSSION

A novel enzyme electrode was developed for the determination of cholesterol concentration in human blood using titanium oxide nanoparticle embedded in the platinum electrode. This study shows that the titanium oxide nanoparticle acts as a catalyst between the cholesterol solution and the enzyme electrode. The sweep voltage in the cyclic voltammetry 797 VA was changed to study the features of the enzyme electrode. At one voltage, the current in the nanoparticle coated electrode is maximum. This voltage acts as the reference voltage to measure the cholesterol concentration in the human blood.

CONCLUSION

A novel enzyme-electrode based cholesterol biosensor system has been developed that can measure the cholesterol level in the blood. The system is mainly built up with ARM processor. The 797 VA computrace instrument is used to measure electrochemical behavior of the enzyme coated platinum electrode. This study has shown that the titanium oxide nanoparticle acts as effective mediator between the cholesterol oxidase and the platinum electrode. After taking reading from many samples the sensor was calibrated to know the cholesterol concentration in the blood. The results showed that the developed biosensor was sensitive, stable and cost effective method for the determination of cholesterol in blood.

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CONFLICT OF INTEREST

Conflict of interest declared none.
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