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EFFECT OF ALCOHOL-KOLANUT INTERACTION ON BRAIN MONOAMINE OXIDASE ACTIVITY IN WISTAR ALBINO RATS

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ABSTRACT

Effect of alcohol-kolanut interaction on brain monoamine oxidase activity in wistar albino rats was studied. Sixty wistar albino rats were divided into six groups of ten (10) rats per group. Group 1 was the control, while groups 2 through 6 were treated with alcohol (10%v/v), kolanut extracts, caffeine, alcohol and kolanut, and alcohol and caffeine respectively. The experiment lasted for twenty one (21) days. Brain levels of monoamine oxidase, noradrenaline and adrenaline were assayed. The results showed that alcohol-kolanut interaction increased the monoamine oxidase activity but however, decreased noradrenaline and adrenaline levels. This might suggest that alcohol-kolanut interaction did not damage the genes encoding monoamine oxidase but it induced its expression, slowed down the release of neurotransmitters in the brain, thus, reducing the risk of schizophrenia, stroke and hypertension.

KEYWORDS: alcohol, kolanut, caffeine and monoamine oxidase.

INTRODUCTION

Alcohol and Kolanuts are common items of entertainment in community functions. Kolanut contains constituents, kolanin, quinine, caffeine, theobromine and theophylline (Eteng et al, 1997; Abulude, 2004; Obochi, 2006). These constituents are also constituents of coffee, cocoa, bean seeds and tea leaves (Eteng et al, 1997; Abulude, 2004; Obochi, 2006); and are widely consumed through their beverages such as snacks (coke, schwebbs, bitter lemon, etc), pharmaceutical products, over the counter drugs, and extracts of coffee, cocoa, tea and kolanuts (Obochi, 2006).

Alcohol is widely consumed through alcoholic beverages such as table wines, beers, desert or cocktail wines, cordials, liquors, whisky and brandy (Obochi, 2006). These beverages are valued as foods, medicine and ceremonial drinks. Although, it is negligible as nourishment, alcohol is an energy producing food like sugar.

These drugs (alcohol and kolanuts) have opposing effects on the brain (Obochi, 2006). Brain function involves subtle chemical and electrical processes which can easily be altered and modified with the use of psychoactive drugs. There is possibility of interaction to bring about changes in neurotransmitter uptake or release and or other metabolic alterations which had overall depressing or activating effects. Their metabolic interactions may also be of medical interest for diagnosis and or treatment of neuronal disorders.

Monoamine oxidase is found in the mitochondrial membrane. It is an ubiquitous homodimeric flavin adenine dinucleotide (FAD) – containing redox active disulfide oxidoreductase at the catalytic centre, which catalyzes the oxidation of biogenic amines. Biogenic amines are synthesized in the brain from the precursor, amino acid, tryptophan, to L-Dopa, 3-4 dihydroxyphenethylamine (dopamine), a precursor for the biosynthesis of noradrenaline and adrenaline, are involved in the transduction of nerve impulses resulting in depolarization of nerve cells. These neurotransmitters are linked with an interplay of release, reuptake, metabolism and excretion. Neurotransmitters are synthesized in the cell bodies or axon terminals and must be transported to the synapse terminals before they are functional. Some drugs interfere with neurotransmitter production or transport while other drugs can enhance the release of neurotransmitters into the synapses and this is one of the mechanisms of action of most stimulants and kolanut. Once released, the neurotransmitter must be deactivated inorder to terminate the cell activity, resulting in an enzyme breakdown by changing its structure so that it is no longer capable of occupying receptor sites. Thus, a drug can affect neural transmission by affecting enzyme activity.

Reuptake is another mechanism in which neurotransmitters are inactivated by being taken back up into the axon terminals that release them. Thus, reuptake is an economical mechanism of deactivating neurotransmitters, leading to preservation of the neurotransmitter molecules for future usage. Some drugs such as cocaine exert their action by blocking the reuptake process, thus, leading to accumulation of excess neurotransmitters in the synapses thereby causing excitotoxicity as seen in schizophrenia, strokes and hypertension.

Some drugs directly affect the receptor by mimicking the activity of neurotransmitters – similar to duplicate key that fits into and opens the lock, causing depolarization of nerve cells, and this is one of the mechanisms of action of alcohol. Thus, the metabolic interactions of alcohol and kolanut may be of medical interest for diagnosis and or treatment of neuronal disorders.

The level of monoamine oxidase (MAO) activity therefore reflects the brain function which reflects the activity of the neuron.

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MATERIALS AND METHODS

Brain monoamine oxidase (MAO) activity was determined with modification of the methods of Tabor et al (1954) and that of McEwen and Sober (1967). The procedure was reported by McEwen (1979). This method is based on the production of benzaldehyde from the deaminated benzylamine during the action of monoamine oxidase on it when used as its substrate. Homogenates of the brain samples were made with 5mmol/L TRIS buffer, pH 7.5, and centrifuged. 0.2ml of the supernatant, 0.1ml of buffered 0.1m benzylamine and a sufficient volume of 0.2m phosphate buffer, pH 7.2, to provide a final volume of 3.0ml, were added to a silical cell, 1cm light path. The optical density was measured at 250nm at 35^0 with HACH DR/3000 spectrophotometer, against a reaction mixture blank that contains no benzylamine. Specific activity was expressed as units of enzymes per milligram of protein (mmoles mg^{-1} proteinmn^{-1}).

Noradrenaline and adrenaline levels were determined with modifications of the method of Fischbach (1988). Catecholamines were isolated by chromatographic separation on alumina at pH 8.5, eluted with dilute acetic acid and oxidized to lutin derivatives by treatment with ferricyanide and zn^{2+}. Ascorbic acid was then added to inactivate excess oxidizing agent. The extract was further centrifuged for 5 minutes and the clear supernatant used for the determination of noradrenaline and adrenaline levels. Six 13 x 100 mm borosilicate tubes containing 0.5ml of the supernatant extract were set and 2 drops of bromethymol blue added, titrated to approximately pH 6 by dropwise addition of 2 N K_2 Co_3, stirred vigorously and 0.5ml of 0.2M phosphate buffer added to each test tube. 10ml of a mixture of 5 N NaoH: 2% ascorbate (9:1) was prepared just prior to use. 0.1ml of 0.25% $ZnSo_4$ and 0.1ml of 0.25% K ferricyanide added to test tubes 2 through 6, and mixed thoroughly. After 2 min, 1ml of the ascorbate reagent was added to the six test tubes and mixed again. Then, the same amounts of the oxidizing agents ($ZnSo_4$ and K ferricyanide) to tube 1, the blank. After 20 min the absorbance of the tubes were read at 420nm for noradrenaline and 485nm for adrenaline levels

RESULTS AND DISCUSSION

Tables 1 and 2 present the results of the effect of the treatment on monoamine oxidase activity, noradrenaline and adrenaline levels respectively. The results for the monoamine oxidase activity showed that there was a significant increase ($P<0.05$) in the kolanut and caffeine treated groups when compared to that of the control while there was a significant decrease ($P<0.05$) in values of the alcohol, alcohol-kolanut and alcohol-caffeine treated groups when compared to that of the control. However, the results for the noradrenaline and adrenaline levels showed a reverse effect.

Table 1: Effects of the treatment on brain monoamine oxidase (MAO) activity in wistar albino rats.

	Group (N)	Monoamine oxidase activity (mmoles mg^{-1} Protein mn^{-1})
1.	Control	10.36±1.29
2.	Alcohol	16.53±1.64*
3.	Kolanut	7.18±1.14*
4.	Caffeine	8.96±1.21*
5.	Alcohol-Kolanut	12.24±1.34*
6.	Alcohol-Caffeine	13.41±1.57*

* Significantly different from control, $P<0.05$ using ANOVA and student 't' test.
Values are expressed as mean ± SD.
N = Number of rats per group = 10.

Table 2: Effects of the treatment on brain Noradrenaline and Adrenaline levels in wistar albino rats.

	Group (N)	Noradrenaline (mg/ml)	Adrenaline (mg/ml)
1.	Control	7.63 ± 0.46	6.48 ± 0.49
2.	Alcohol	5.28 ± 1.67*	4.41 ± 1.23*
3.	Kolanut	10.14 ± 0.34*	8.31 ± 0.32*
4.	Caffeine	12.53 ± 0.47*	9.17 ± 0.28*
5.	Alcohol-Kolanut	5.28 ± 0.48*	4.36 ± 0.37*
6.	Alcohol-Caffeine	6.23 ± 0.51*	5.28 ± 0.41*

* Significantly different from control, $P<0.05$ using ANOVA and student 't' test.
Values are expressed as mean ± SD.
N = Number of rats per group = 10.

DISCUSSION

In this study, alcohol-kolanut interaction increased brain monoamine oxidase activity in wistar albino rats. Kolanut independently decreased the activity of monoamine oxidase, but however acted synergistically with alcohol to increase monoamine oxidase activity. In this study, an increase in monoamine oxidase activity resulted in reduced levels of noradrenaline and adrenaline which showed noradrenaline interplay of release, reuptake, metabolism and excretion of neurotransmitter molecules. High levels of noradrenaline and adrenaline resulted in the alteration in function and cell morphology, inducing neuronal loss and plastic changes in nerve cells and disruption of signaling pathways by loss of neurotrophic factors necessary for growth, development, differentiation, memory and learning.

The significant increase in the activity of monoamine oxidase in rats showed that alcohol-kolanut interaction

however, did not damage the genes encoding monoamine oxidase but might induce its expression. However, the kolanut interaction with alcohol modulated the noradrenaline and adrenaline levels, slowed down the release of these neurotransmitters into the synapse terminals, thus reducing the risk of schizophrenia and hypertension.

Conclusively, alcohol-kolanut interaction increased brain monoamine oxidase activity, induced gene expression and inhibited the release of neurotransmitters into the axon terminals of neuron, suggesting that moderate consumption of alcohol and kolanut concurrently may be useful in the management of schizophrenia, strokes and hypertension, and possible improvement in memory and learning.

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