

Kinetic Potentials of the Effect of Ethanol on Iron Content of Ashed Cow Liver

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Abstract: The kinetics of the effect of ethanol on iron content of ashed cow liver was investigated. The work revealed that the cow liver contained a very high quantity of iron. The effect of alcohol distilled from palm wine was tested kinetically on the measured concentration of the iron in the ashed cow liver. The depletion in the concentration of the iron was monitored spectrophotometrically at specific time intervals. The analysis showed a gradual decrease in the concentration of iron in the ashed cow liver extract. The initial iron content in the ashed cow liver was 22.6130 mg/l. After 10 minutes of reaction of the local ethanol with the cow liver extract, the quantity of the iron reduced to 7.5443 mg/l. at the end of 90 minutes, the concentration of iron further reduced to 5.3624 mg/l. the half life, rate of reaction, rate constant and order of the reaction were determined. The pH of the reaction mixture was almost constant throughout the time interval of measurement. The average pH was measured to be 6.52, being weakly acidic. The results showed that ethanol reduced or destroyed the concentration of iron present in a biological system.

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1.0. INTRODUCTION

The mammalian liver contains high quality iron, and this portion of meat is used mainly for consumption of food. Since it is rich in iron, iron supplements can be synthesised and processed using this portion of meat which can be administered for patients who suffer from iron deficiency. Iron in the body is very essential and is required by the human cell. Iron transports and stores oxygen as haemoglobin in red blood cells (Donovan *et al.*, 2006). Deficiency of iron causes anaemia, which diminishes red blood cells production due to low iron in the stored in the body. Anaemia is associated with dizziness, fatigue, weakness and drowsiness, leading to death in severe cases (Batra and Archana, 2005), while very high iron levels can also cause health problems.

Iron is an essential nutrient required by every human cell, its atomic structure gives rise to a number of bio-chemically useful properties, including the capacity to both donate and accept electrons, and to reversibly bind to ligands such as oxygen and nitrogen. As such, iron plays a vital role in the transport and storage of oxygen, in oxidative metabolism and cellular growth and proliferation. The body iron content is approximately 3-4g, which corresponds to a concentration of 40 – 50 mg of iron per kilogram of body weight. Approximately 60% of this is present in the form of haemoglobin in circulating red blood cells. However, the body exploits the unique properties of iron by incorporating it into hundreds

of different enzymatic and non-enzymatic proteins that are crucial to a wide range of physiological functions. Therefore, the majority of iron in the body is contained within erythrocytes where it is incorporated into haem protein complexes that transport oxygen as haemoglobin and myoglobin (Donovan and Andrews, 2006). The body is able to regulate uptake of iron, so overdose is rare and usually only occur when people take supplements. Foods rich in iron include liver, oyster, pumpkin seeds, beans, whole grains, as well as dark leaf greens.

1.1. Iron and Alcohol

Alcohol consumption is deeply entrenched in the social fabric of the society. It is legal, easily available and affordable. Alcohol (ethanol) consumption has both short term and long term effects on the human body. It affects the organs in the body depending on the quantity consumed. Consistent consumption of alcohol significantly increases the risk of developing alcoholic liver disease (ALD), consequently leading to death. It is a well established fact that both iron and alcohol individually cause oxidative stress and lipid peroxidation, enhancing the production of free radicals that damages DNA (Wu *et al.*, 2003; McCord, 1998; Tsukamoto *et al.*, 1995). Alcohol in the human system leads to increased liver oxidative stress and adducts. Ethanol is oxidised to water and carbon dioxide and this is mediated by hepatic enzymes leading to chemical pathways that produce acetaldehyde as their toxic by-product

(Gramenzi *et al.*, 2006). Alcohol consumption has been associated with changes in iron homeostasis ranging from anaemia to iron overload. Iron and alcohol react to cause liver injury. Consumption of up to two alcoholic drinks per day has been shown to exert a protective effect by reducing the risk for iron deficiency anaemia (Ioannou *et al.*, 2004), whereas heavy alcohol consumption (more than two alcoholic drinks per day) elevates the risk of iron overload. Alcohol affects all parts of the body including blood and immune system, bones and muscles, brain, nervous system, eye, heart, kidney and fluid balance, liver, lungs, mental health, mouth and throat, pancreas and digestion of sugar, sexual and reproductive system in men and woman. As well as potentially affecting the physical and mental health of individuals in many ways, chronic and heavy alcohol use can increase the risk of death either directly through acute alcohol poisoning or indirectly through violent death or suicide with alcohol as a factor. Unintentional injury from alcohol use often results in falls, burns, motor accidents, assault and drowning. Chronic alcohol consumption also increases the rate of haemorrhagic stroke, increased blood pressure, reduced fertility, and birth defects in unborn babies (Gill, 2000). A survey of literature reveals that much work has been carried out on the effect of ethanol on iron. Cook *et al.*, 1995 reported that excessive wine intake was the primary cause of the iron disorder – haemochromatosis. They also reported that iron in wine is not well absorbed and that red wine in particular impairs the absorption of non-heme iron in the human body. Wayne (2003) reported that Chronic and heavy drinking, particularly during adolescence and young adult years can dramatically affect bone quality and increase the risk of osteoporosis later in life. It is known to cause severe alcoholic hepatitis (Gyongy and Bala, 2010), alcoholic liver disease (ALD) (Hiromasa *et al.*, 2010; Crist *et al.*, 2007; Osna and Terrence, 2007), and a rise in oestrogen levels in women as well as a decrease in progesterone level in pre-menopausal women (Gill, 2000). The aim of this study is to determine the amount of iron in ashed cow liver and to study the kinetic potential of the effect of ethanol on the iron content of ashed cow liver.

2.0. Materials and Methods

2.1. Ashing of Cow Liver Sample

The cow liver was collected from a slaughter house in Itam; Itu local government area of Akwa Ibom State, Nigeria. It was oven dried and crushed to powder. The powdered cow liver was put in a crucible and ashed at 500°C for 6 hours. The ashed liver powder was agitated occasionally to bring fresh particles to the surface to ensure complete ashing. The ashed cow liver was grinded in a

mortar and then weighed in a weighing balance to be 56 g.

2.2. Extraction of Iron from the Ashed Liver

The ashed cow liver (56 g) was leached with 200 cm³ of distilled water and the volume was made up to one litre solution. The leached sample was kept for 24 hours to ensure optimal leaching and then filtered. The filtrate was labelled and kept for AAS measurement.

2.3. Determination of Iron Present in the Ashed Cow Liver Extract

The concentration of iron was determined spectrophotometrically using Buck 200 Atomic Absorption Spectrophotometer and their absorption compared with absorption of iron in the calibration curve.

2.4. Kinetic Measurement of the Effect of Ethanol on the Concentration of Iron in the Ashed Cow Liver Sample

The percentage purity of the local ethanol obtained was 30%. Based on the percentage purity and relative density of ethanol (789 kg/m³), the required volume of ethanol which contained one mole was determined.

Ethanol (194 cm³) was measured and diluted in 200 cm³ of distilled water and the volume was made up to 1000 cm³ of solution. This solution gave a 1 M solution of local ethanol. Equal volumes (200 cm³) of the prepared ethanol solution and the filtered ashed cow liver solution was reacted together in a 500 cm³ beaker and a stop watch started simultaneously. At every 10 minutes interval, the reaction mixture was bled out with a syringe and the concentration was determined spectroscopically. The pH of the reaction mixture was also taken at every 10 minutes interval.

3.0. RESULTS AND DISCUSSION

Table 3.1 records the effect of ethanol on the concentration of iron extract. It reveals a gradual decrease in the concentration of the iron with increasing time, indicating that ethanol actually reduces or destroys the quantity of iron present in the human system and thereby weakening the strength of the biological system.

3.1. Rate of Reaction of the Kinetics of Ethanol with the Iron Content in the biological system

It was observed that the original iron content in the ashed cow liver extract which was 22.6131 mg/l reduced to 14.2983 mg/l after 10 minutes. At 30 minutes, the iron content was found to be 10.6901 mg/l and this decrease continued gradually as the ethanol persisted in the solution. At the end of 90 minutes of the measurement interval, the iron content left in the reaction mixture was measured to be 5.3624 mg/l.

Table 3.1: Concentration-time data of the effect of ethanol on iron

Time (mins)	0	10	20	30	40	50	60	70	80	90
[Fe ²⁺] mg/l	22.6131	14.2983	12.8053	10.6901	8.8246	7.7283	7.5443	6.2431	5.6705	5.3624
pH	6.49	6.50	6.50	6.51	6.52	6.52	6.52	6.53	6.53	6.54
$\text{Log} \frac{x}{a_0(a_0 - x)}$	0	0.026	0.034	0.049	0.069	0.085	0.088	0.116	0.132	0.142

Table 3.2: Data for variation of the rate of reaction of local ethanol and cow liver extract

Time (mins)	0	10	20	30	40	50	60	70	80	90
[Fe ²⁺] mg/l	22.6131	14.2983	12.8053	10.6901	8.8246	7.7283	7.5443	6.2431	5.6705	5.3624
Rate	-	0.8314	0.4904	0.3974	0.3447	0.2977	0.2511	0.2339	0.2118	0.1917
Log R	-	-1.080	-1.310	-1.401	-1.463	-1.526	-1.600	-1.631	-1.674	-1.717
Log C	1.3544	1.1553	1.1074	1.0289	0.9457	0.8881	0.8776	0.7954	0.7536	0.7294

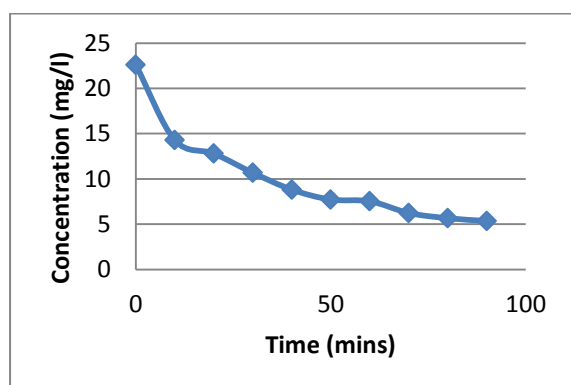


Fig. 3.1: Variation of the concentration of Iron in the ashed cow liver extract with time

Table 3.2 records the rate of reaction obtained for each time interval for the corresponding concentration of iron in the reaction mixture. It was observed that the rate of reaction was very fast in the first 10 minutes of the reaction. This rate seems to be appreciably fast, suggesting that alcohol reduces the iron content in the system fast. A further reduction of the concentration of iron from 22.6131 mg/l, originally present to 10.6901 mg/l after 30 minutes suggests the danger of excessive intake of alcohol as suffered by most alcoholics.

3.2. pH of the Reaction Mixture

The average pH of the reaction from table 3.1 was obtained to be 6.52 compared to 7.00 for pure water. In the reaction mixture, ethanol's hydroxyl group causes the mixture to be slightly basic. Ethanol is a neutral molecule and the pH of a solution of ethanol in water is nearly 7.00, hence the measured value of the pH of the reaction mixture was not far from expectation.

3.3. Order of the Reaction

The concentration-time data obtained from the atomic absorption spectrophotometric measurement was subjected to various integrated rate laws fitted to Zero, First, Second, and Third order reaction kinetics. From the integrated rate equations, it was observed that the concentration-time data fitted into the second order kinetic model which resulted in an

approximately equal and constant value of the various rate constant,

$$k = \frac{x}{ta_0(a_0 - x)} \quad (1)$$

This kinetic test confirms that the reaction of the local ethanol with the iron extract obtained from ashed cow liver follows a second order.

3.4. Rate constant of the Reaction of Ethanol with the Iron Extract

The various rate constants obtained from the experimental fittings of the kinetic data into the integrated rate law were obtained and their average calculated. The average rate constant obtained for the hydrolysis of the iron extract with the ethanol was $1.8 \times 10^{-3} \text{ mg}^{-1} \text{ dm}^3 \text{ min}^{-1}$. This value confirms that the rate of the reaction of the alcohol molecule is expeditious and portends a quick danger at high rate of consumption of alcohol.

Further, a plot of $\text{Log} \frac{x}{a_0(a_0 - x)}$ versus time

(minutes) gives a straight line from the origin with slope $0.0016 \text{ mg}^{-1} \text{ dm}^3 \text{ min}^{-1}$.

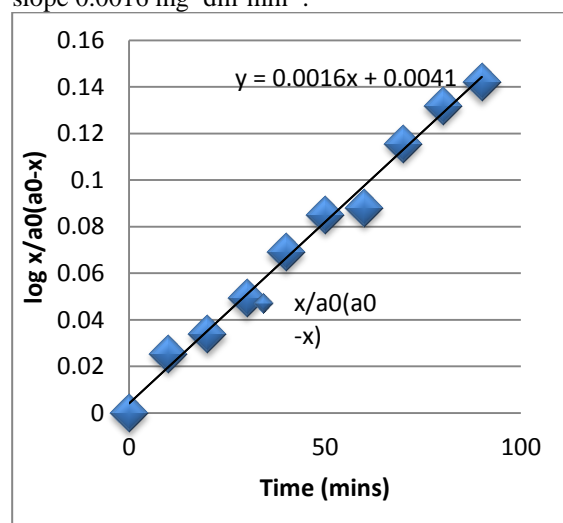


Fig. 3.2: A plot of $\text{Log} \frac{x}{a_0(a_0 - x)}$ against time for the reaction of ethanol with cow liver extract.

3.5. Half life of the concentration of iron [Fe²⁺] with respect to the reaction of local ethanol

The kinetics for the reaction of local ethanol with the cow liver extract was found to follow the second order rate law. The half life for the second order reaction is given as

$$t_{1/2} = \frac{1}{k[A]_0} \quad (2)$$

By appropriate substitution, the half life value obtained for the reaction of local ethanol with the ashed cow liver extract is 24.570 minutes. This value showed that if the original iron concentration in the body was 22.613 mg/l, by consuming 200 cm³ of local ethanol, it will take approximately 25 minutes for the original iron concentration to reduce to half the initial quantity.

The full life of the iron concentration showed that at 50 minutes interval after the consumption of the alcohol, the original concentration of the iron in the blood would have been completely depleted by the alcohol leading to complete exhaustion as experienced by most alcohol addicts.

4. CONCLUSION

The kinetics of the effect of ethanol on iron content of ashed cow liver has been studied and the ashed cow liver was found to be rich in iron content up to 22.613 mg/l. The original iron content of the ashed cow liver was observed to decrease with increase in time on addition of the local ethanol. The average pH of the reaction mixture was 6.52, showing that the reaction pH was almost neutral. The rate constant of the reaction was calculated to be 1.8 x 10⁻³ mg⁻¹dm³min⁻¹, revealing a half life of 24.570 minutes. By use of the integrated rate law, the kinetics of the reaction of local ethanol with the ashed cow liver extract was determined to follow a second order kinetics.

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REFERENCES

Batra, J. and Archana, S. (2005). Iron Deficiency Anaemia: Effect on Cognitive Development in Children: A Review. *Indian Journal of Clinical Biochemistry*, 20(2): 119 – 125

Cook, J. D.; Keddy, M. B. and Hurrell, R. F. (1995). The Effect of Red and White Wines on non

heme-iron Absorption in Humans. *Journal of American Society for Clinical Nutrition*, 61(4): 800 – 804

Crist, C.; Klein, E.; Gollan, J.; Findik-Harrison, D. D. and Frye, J. (2007). The Interaction of Alcohol and Iron-overload in the in-vivo regulation of Iron Responsive Genes, *Centaurus*, 15(1): 2 – 6

Donovan, A.; Roy, C. N. and Andrews, N. C. (2006). The Ins and Outs of Iron Homeostasis. *Physiology (Bethesda)*, 21, 115 – 123

Gill, J. (2000). The Effects of Moderate Alcohol consumption on Female Hormone Levels and Reproductive Function. *Alcohol and Alcoholism*, 35(5): 417 – 423.

Gramenzi, A.; Caputo, F.; Biselli, M.; Kuria, F.; Loggi, E.; Andreone, P. and Bernardi, M. (2006). Alcoholic Liver Disease – Pathophysiological Aspects and Risk Factors. *Journal of Alimentary Pharmacology and Therapeutics*, 24(8): 1151 – 1161.

Gyongyi, S. and Bala, S. (2010). Alcoholic Liver Disease and the Gut-Liver Axis. *World Journal of Gastroenterology*, 16(11): 1321 – 1329

Hiromasa, I.; Yoshinori, H.; Yosiyuki, Y.; Hirotohi, E. (2010). Alcoholic Liver Disease and its Relationship with Metabolic Syndrome. *Journal of Japan Medical Association*, 53(4): 236 – 242.

Ioannou, G. N.; Dominitz, J. A.; Weiss, N. S.; Heagerty, P. J.; Kowdley, K. V. (2004). The Effect of Alcohol Consumption on the Prevalence of Iron Overload, Iron Deficiency, and Iron Deficiency Anaemia. *World Journal of Gastroenterology*, 126, 1293 – 1301.

McCord, J. M. (1998). Iron, Free Radicals, and Oxidative Injury. *Seminars in Hematology*, 35, 5 – 12.

Oсна, N. A. and Terrence, M. D. (2007). Implication of Altered Proteasome Function in Alcoholic Liver Injury. *World Journal of Gastroenterology*, 13(37): 4931 – 4937.

Tsukamoto, H.; Horne, W.; Kamimura, S.; Niemela, O.; Parkkila, S.; Yla-Herttuala, S.; Brittenham, G. M. (1995). Experimental Liver Cirrhosis Induced by Alcohol and Iron. *The Journal of Clinical Investigation*, 96, 620 – 630.

Wayne, H. S. (2003). Alcohol and Other Factors Affecting Osteoporosis Risk in Women. *Alcohol Abuse and Alcoholism*, 26(4): 292 – 298

Wu, D. and Cederbaum, A. I. (2003). Alcohol, Oxidative Stress, and Free Radical Damage. *Alcohol Research and Health*, 27: 277 – 284.