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Effect of sugar diet on hepatic proteins in albino rats treated with petroleum contaminated diet

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Abstract

Six groups of five albino rats per group were fed *ad libitum* with rat diet contaminated with petroleum at concentrations of 3.88, 7.75, 15.51, 31.01, 62.02 g/kg and the last group fed only rat diet as control. Another six groups of five albino rats were fed *ad libitum* with rat diet contaminated with petroleum at the same concentrations mixed with 20% sugar (Granulated) with the last group fed only rat diet as control to determine glucose effect of sugar on petroleum induced toxicity. The hepatic enzymes aspartate amino transferase (AST), alanine amino transferase (ALT), gamma glutamyl transpeptidase (GGT) and alkaline phosphatase (ALK PHOS) activities with albumin, Total protein and liver /body weight were monitored in the animals. There was dose dependent decrease in enzymes activities (ALT, AST, GGT and ALKPHOS) with dose dependent increase in albumin and Protein in Sugar fed albino rats compared with Petroleum fed albino rats ($P<0.05$). There was dose dependent increase in enzymes activities and dose dependent decrease in proteins in petroleum fed rats compared with their controls ($P<0.05$). The study showed that 20%sugar reduced enzyme induction caused by petroleum contaminated diet through reducing cAMP concentrations. Therefore sugar cane represses enzymes induction through the process of glucose effect.

Keywords: Hepatic Proteins, aspartate amino transferase, alanine amino transferase, Total protein.

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1. Introduction

Crude oil has been described as a complex mixture of over 6000 potentially different hydrocarbons and metal [1] which may be broadly characterised as paraffinic naphtanic or aromatic [2]. It also contains smaller proportions of non hydrocarbon compounds such as oxygen, thiols, heterocyclic nitrogen, sulphur compounds as well as metalloporphyrins [2-4] while vanadium, iron, copper and nickel are found in low concentrations in crude petroleum [5]. The organic substances are primarily and principally compounds comprising of carbon and hydrogen otherwise referred to as hydrocarbons. Crude and refined petroleum and oil field chemicals and emissions are highly complex chemical mixtures. Crude petroleum contains hundreds of compounds and the chemical composition varies between geologic formations [6]. Most of the substances contained in crude petroleum occur naturally due to their presence in rock formation or in saltwater deposits from which the crude oil was drawn [7]. They have also been grouped into types as light, medium (Intermediate) and heavy depending on their density, physical and chemical properties. Crude petroleum can be separated by fractional distillation into gasoline, kerosene, diesel, fuel etc.

Exposure of humans and animal to crude oil, which is increasing in terms of the environmental levels, and application to body, may be toxic. Crude oil is used in folkloric medicine in the Niger-delta area of Nigeria for the

treatment of various ailments including stomach up-set, wound, and burns [8]. In several organs, mainly heart and liver, cell damage is followed by increased levels of a number of cytoplasmic enzymes in the blood, a phenomenon that provides the basis for clinical diagnosis of heart and liver diseases e.g. liver enzymes are usually raised in acute hepatotoxicity but tend to decrease with prolonged intoxication due to damage to the liver cells. The Nigerian Bonny crude oils are classified as light crude oils, with aromatic hydrocarbons accounting for up to 45% of the total hydrocarbons. As aromatic hydrocarbons are relatively soluble in water [9], it is expected that the potential of this light crude oil to have adverse toxic effects is higher than for heavier, less water–soluble crude oils. It is known that lipophilic xenobiotics may have the characteristics of both electron uncouplers and energy inhibitors [10]. Since a large proportion of the crude oil components is lipophilic in nature biological membrane may be the target sites where adverse effects occur.

The liver is considered as the major organ involved in the biotransformation of myriads of structurally and chemically diverse compounds of both endogenous and exogenous nature [11]. A multigene family of haemo proteins, the cytochrome P_{450} (CYP) is the principal enzyme system catalyzing oxidative biotransformation reactions. Liver enzymes such as lactate dehydrogenase (LDH), Aspartate aminotransferase (AST), Alanine aminotransferase (ALT), alkaline phosphatase (Alkphos) and gamma glutamyl transpeptidase (GGT) are considered to be biochemical markers for assessing liver function [12].

Carbohydrates are an important source of energy for human being. Feeding varieties of carbohydrates (Glucose, sucrose and fructose) to mammals and bacteria result in blocking the induction of many enzyme systems. Melvin and Goldberg [13] showed that glucose feeding causes in both man and microorganism profound changes in metabolism include inhibition of induction of several enzymes, stimulation of others and blockage of most effects of glucocorticods while Bostford, and Harman [14] studied the reduction of gene expression by glucose (catabolite repression) in various microorganisms. Glucose, usually an excellent carbon source for growth, interferes with the synthesis of many secondary metabolites. Because of parallels with the well known suppression by glucose of catabolic enzymes that use less preferred substrates [15] this has been referred to as catabolite repression.

Glucose represses the induction of inducible operons by inhibiting the synthesis of cyclic Adenosine monophosphate (cAMP) a nucleotide that is required for the initiation of transcription of a large number of inducible enzyme systems including the Lac operon. Glucose represses the synthesis of certain inducible enzymes even though the inducer of the pathway is present in the environment. The aim of this study is to determine the effect of sugar diet on aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase (Alkphos) and Gamma glutamyl transpeptidase (GGT), serum total protein and albumin in the liver of albino rats treated with petroleum hydrocarbon.

2. Materials and methods

2.1 Test animals

Ninety Wistar albino rats of 0.195kg average body weight on normal rat diet were obtained from the animal house of the department of Pharmacology and Toxicology, University of Port Harcourt. These rats were fed *ad libitum* with normal rat pellet and water and acclimatized to laboratory conditions for a period of 14days prior to commencement of study. The granulated sugar (produced by Dangote Sugar Nigeria PLC) used in this study was purchased from Mile 3 Market, Port Harcourt.

2.2 Animal studies

Preliminary animal study was done to ascertain the oral LD_{100} [16] and LD_{50} of crude petroleum [17]. Preliminary study was also done by authors to ascertain the sugar concentration that will cause glucose effect in rats fed different concentration of sugar.

2.3 Biochemical studies

Determination of ALT and AST was done by monitoring the concentrations of pyruvate hydrazone formed with 2, 4 dinitrophenylhydrazine. 0.5ml of buffer solution was dispensed into test tubes labeled blank, sample, control blank and control respectively for AST and ALT respectively. 0.1ml of sample and control was dispensed into their respective test tubes. All the tubes were incubated at 37ºC for 30minutes. 0.5ml of 2, 4 dinitrophenylhydrazine was dispensed into all test tubes. 0.1ml of sample and control was dispensed into their respective blank test tube. The contents of each test tube was mixed and allowed to stand for 20minutes at 25ºC. 5ml of 0.4N sodium hydroxide was added to each tube, mixed and read at 550nm against the respective blank prepared. The activity of the unknown was extrapolated from the calibration curve already prepared [18].

Alkaline Phosphatase activity was done by Phenolphthalein Monophosphate method .The test tubes were respectively labeled sample, standard and control. 1.0ml of distilled water was pipetted into each tube followed by a drop of the substrate into each test tube. All the test tubes were incubated at 37ºC for 5minutes. 0.1ml of sample, standard and control were dispensed into their respective test tubes. The test tubes were incubated at 37ºC for 20minutes. 5ml of colour developer was added to each test tube, mixed, and read at 550nm using water as blank.

The activity of sample was calculated using the absorbance of sample against absorbance of standard multiplied by concentration of standard [19].

Gamma Glutamyl Transpeptidase was done by Modified Szasz method [20]. 2.0ml of working reagent (Substrate dissolved in Buffer according to manufacturer's specification) was pipetted into test tube and incubated at 37 ºC for 3minutes. 0.2ml of serum sample was added into the test tube mixed and transferred into measuring cuvette. The absorbances were read at O, 1, 2 and 3 minutes using water as blank at wavelength of 405nm. The activity of Gamma glutamyl transpeptidase was calculated by multiplying mean change in absorbance per minute with a factor (1158).

Total Protein concentration was carried out using Biuret method. 5.0ml of Biuret reagent was pipetted into tubes labeled blank, standard, test, and control. 0.1ml of distilled water, standard, sample and control were pipetted into their respective tubes, mixed and incubated for 30minutes at 25ºC. The absorbances were measured against the reagent blank at wavelength of 546nm. The concentration of total protein was calculated by dividing the absorbance of sample against absorbance of standard multiplied by concentration of standard [21].

Bromocresol green (BCG) method by Doumas *et al.* [22] was used for albumin estimation. Bromocresol green (3ml) reagent was pipetted into tubes labeled blank, standard, sample and control. 0.01ml of distilled water, standard, sample and control was pipetted into their respective tubes, mixed and incubated at 25 ºC for 5minutes. The absorbances were measured at 578nm against the reagent blank. The concentration of Albumin was determined by dividing the absorbance of sample against absorbance of standard multiplied by concentration of standard.

The liver to body weight ratio was determined by taking the weight of the whole liver and comparing it with the final body weight as described by Sunmonu and Oloyede [23].

2.4 Statistical analysis

The biochemical data were subjected to some statistical analysis. Values were reported as Mean±SEM while student's t-test was used to test for differences between treatment groups using Statistical Package for Social Sciences (SPSS) version 16.A value of $P<0.05$ was accepted as significant.

3. Results

The acute toxicity results in albino rats are as presented in Table 1.The LD_{100} obtained for Bonny light crude Petroleum was 255.6± 0.195g/kg while the LD_{50} obtained by Arithmetic method of Karber was 124.04±0.195g/kg.

Table 1: Lethal dose (LD_{50}) of albino rats treated with bonny light crude petroleum

Group	Dose level g/kg	No of death(s) recorded	Average time of Death (Hour)
1	0.00	0	0.00
2	63.90	1	20.00
3	109.00	2	15.00
4	127.80	3	13.00
5	191.70	4	9.00
6	255.60	5	6.00

n = 5, **LD_{50} (g/kg) =** 124.04±0.195g/kg.

The Alkaline phosphatase activity (U/L), aspartate amino transferase and alanine amino transferase showed dose dependent increase. The alkaline phosphatase activity of Petroleum treated albino rats of the control was 43.80±4.37. At 3.88g/kg of petroleum treatment, the alkaline phosphatase activity was 41.80±6.66, while it increased to 51.80±4.94, 64.00±4.57, 63.60 ±8.82 and 66.20±9.30 at concentrations of 7.75, 15.51, 31.01 and 62.02g/kg respectively. The alkaline phosphatase activity of sugar treated albino rats of the control was 41.20±4.42. At 3.88g/kg of sugar treatment, the alkaline phosphatase activity was 27.20±3.93, while it increased to 39.60±2.50, 41.80±3.86, 43.00 ±12.87 and 43.20±6.15 at concentrations of 7.75, 15.51, 31.01 and 62.02g/kg respectively as shown below in table 2.

The Aspartate amino transferase activity of 14.40±3.19 was obtained in the control of petroleum treated albino rats which increased to 19.00± 4.72 at 3.88g/kg. The Aspartate amino transferase activity further increased to 27.00±5.27, 32.40±5.24, 38.40±4.96 and 51.40±5.81 at concentrations of 7.75, 15.51, 31.01 and 62.02g/kg respectively. The sugar treated albino rats also had dose dependent concentration of aspartate amino transferase activity (U/L) lower than the petroleum treated albino rats. The Aspartate amino transferase activity of 14.20±2.73 was obtained in the control of petroleum treated albino rats which increased to 13.40± 3.64 at 3.88g/kg. The Aspartate amino transferase activity further increased to 17.60±3.53, 20.80±6.29, 24.60±8.90 and 25.60±7.31 at concentrations of 7.75, 15.51, 31.01 and 62.02g/kg respectively as shown below in table 2.

The alanine amino transferase activity of 12.00±0.58 was obtained in the control of petroleum treated albino rats which increased to 14.00± 1.14 at 3.88g/kg. The Aspartate amino transferase activity further increased to 21.00±3.77, 24.20±5.50, 26.00±4.01 and 29.20±6.26 at concentrations of 7.75, 15.51, 31.01 and 62.02g/kg respectively. The sugar treated albino rats also had dose dependent concentration of aspartate amino transferase activity (U/L) lower than the petroleum treated albino rats. The Aspartate amino transferase activity of 11.80±1.93 was obtained in the control of petroleum treated albino rats

which decreased to 9.80±1.28 at 3.88g/kg. The Aspartate amino transferase activity further increased to 10.00±2.17, 11.80±1.43, 18.40±4.41 and 20.40±6.32 at concentrations of 7.75, 15.51, 31.01 and 62.02g/kg respectively as shown below in table 2.

Table 2: Effect of sugar on alkaline phosphatase, aspartate amino transferase and alanine amino transferase in albino rats treated with petroleum

	Alkaline Phosphatase (U/L)			Aspartate Amino Transferase (U/L)			Alanine Amino Transferase (U/L)		
Concentration (g/kg)	**Petroleum Treated**	**Sugar Treated**	**P Value**	**Petroleum Treated**	**Sugar Treated**	**P Value**	**Petroleum Treated**	**Sugar Treated**	**P Value**
0.00	43.80±4.37	41.20±4.42	0.756	14.40±3.19	14.20±2.73	0.965	12.80±0.58	11.80±1.93	0.704
3.88	41.80±6.66	27.20±3.93	0.137	19.00±4.72	13.40±3.64	0.133	14.00±1.14	9.80±1.28	0.105
7.75	51.80±4.94	39.60±2.50	0.125	27.00±5.27	17.60±3.53	0.029	21.00±3.77	10.00±2.17	0.082
15.51	64.00±4.57	41.80±3.86	0.002	32.40±5.24	20.80±6.29	0.320	24.20±5.50	11.80±1.43	0.098
31.01	63.60±8.82	43.00±12.87	0.344	38.40±4.96	24.60±8.90	0.132	26.00±4.01	18.40±4.41	0.249
62.02	66.20±9.30	43.20±6.15	0.080	51.40±5.81	25.60±7.31	0.001	29.20±6.26	20.40±6.32	0.314

The gammaglutamyl transpeptidase activity (U/L) of Petroleum treated albino rats of the control was 557.40±52.18. At 3.88g/kg of petroleum treatment, the gammaglutamyl transpeptidase activity was 523.80±97.27, while it increased to 612.00±182.09, 617.60±77.56, 807.60±93.25 and 825.00±131.84 at concentrations of 7.75, 15.51, 31.01 and 62.02g/kg respectively. The gammaglutamyl transpeptidase activity of control in sugar treated albino rats was 577.00±55.87. At 3.88g/kg of sugar treatment, the gammaglutamyl transpeptidase activity was 315.80±93.80, while it increased to 425.40±74.28, 459.80±76.38, 573.20±81.58 and 682.20±43.63 at concentrations of 7.75, 15.51, 31.01 and 62.02g/kg respectively as shown below in table 3.

Table 3: Effect of sugar on gamma glutamyl transpeptidase in albino rats treated with petroleum

Concentration (g/Kg)	Petroleum Treated	Sugar Treated	t	p
0.00	557.40±52.18	577.00±55.87	-1.292	0.266
3.88	523.80±97.27	315.80±93.80	1.279	0.270
7.75	612.00±182,09	425.40±74.28	0.535	0.621
15.51	617.60±7756	459.80±76.38	0.358	0.738
31.01	807.60±93.25	573.20±81.58	0.745	0.497
62.02	825.00±131.84	682.20 ±43.63	1.380	0.240

Total protein concentration (g/l) and albumin (g/l) showed dose dependent decrease in both petroleum and sugar treated albino rats with the petroleum treated lower in concentration than the sugar treated. The control albino rats had protein concentrations (g/l) of 66.00±3.81 and 67.20±4.88 in both petroleum and sugar treated rats respectively. The protein concentration at 3.88g/kg of petroleum treated albino rats was 76.40±4.68. At 7.75g/kg the protein concentration was 66.60±1.89, which decreased to 63.00±3.36, 61.00±2.36 and 61.40±2.31 at 15.51, 31.01 and 62.02g/kg respectively. The total protein concentration (g/l) of sugar treated albino rats was 76.40±4.68, 71.20±5.85, which decreased to 70.40±3.53, 68.60 ±3.73 and 64.80±3.98 at concentrations of 3.88, 7.75, 15.51, 31.01 and 62.02g/kg respectively as shown below in table 4.

The control albino rats had albumin concentrations (g/l) of 38.60±0.68 and 39.40±3.04 in both petroleum and gari treated rats respectively. The albumin concentration at 3.88g/kg of petroleum treated albino rats was 38.40±1.91. At 7.75g/kg the albumin concentration was 36.60±2.31, which decreased to 36.40±0.68, 35.40±1.66 and 35.20±1.02 at 15.51, 31.01 and 62.02g/kg respectively. The albumin concentration (g/l) of sugar treated albino rats was 43.60±1.20, 42.60±1.43, 41.00±2.17, 40.20±1.28, and 39.80±2.09 at concentrations of 3.88, 7.75, 15.51, 31.01 and 62.02g/kg respectively as shown below in table 4.

The liver/body weight ratio of Petroleum treated albino rats of the control was 10.00±1.05 while it was 10.20±0.86 in the sugar treated albino rats. At 3.88g/kg of petroleum treatment, the liver/body weight ratio was 9.90±0.84 which reduced further to 8.36±0.31, 5.98±1.67, 5.36±0.26 and 5.22±0.48 at concentrations of 7.75, 15.51, 31.01 and 62.02g/kg respectively. The liver/body weight ratio in sugar treated albino rats was 10.28±0.55 at 3.88g/kg. At 7.75g/kg sugar treatment, the liver/body weight ratio was 9.36±0.31, 6.84±1.17, 5.58±0.51 and 5.56±0.42 at concentrations of 15.51, 31.01 and 62.02g/kg respectively as shown below in table 4.

Table 4: Effect of Gari on total protein, albumin and liver/body weight ratio in albino rats treated with petroleum

	Total protein(g/l)			Albumin (g/l)			Liver/Body Weight Ratio		
Concentration (g/kg)	Petroleum Treated	Sugar Treated	P Value	Petroleum Treated	Sugar Treated	P Value	Petroleum Treated	Sugar Treated	P Value
0.00	66.00±3.81	67.20±4.88	0.864	38.60±0.68	38.40±2.11	0.923	10.00±1.05	10.20±0.86	0.749
3.88	66.20±2.13	76.40±4.68	0.172	38.40±1.91	43.60±1.20	0.065	9.90± 0.84	10.28±0.55	0.771
7.75	66.60±1.89	71.20±5.85	0.551	36.60±2.31	42.60±1.43	0.171	8.36± 0.31	9.36±0.31	0.087
15.51	63.00±3.36	70.40±3.53	0.132	36.40±0.68	41.00±2.17	0.040	5.98 ±1.67	6.84±1.17	0.655
31.01	61.00±2.36	68.60±3.73	0.132	35.40±1.66	40.20±1.28	0.063	5.36±0.26	5.58 ±0.51	0.641
62.02	61.40±2.31	64.80±3.98	0.423	35.20±1.02	39.80±2.09	0.094	5.22 ±0.48	5.56±0.42	0.544

Overall, there was significant difference in 57.48±4.66 alkaline phosphatase (U/L) activity of Petroleum fed rats compared with 38.96±3.00 of sugar fed rats. Also ALT (U/L) activity of 22.88±2.59 in petroleum treated rats was significantly different from 14.08±2.22 in sugar treated rats. The AST (U/L) activity of 33.64±5.47 in petroleum treated rats was significantly different from 20.40±2.26 in sugar treated albino rats. The GGT activity (U/L) of 677.20 ±59.24 in petroleum treated rats was significantly different from 491.28±62.94 in sugar treated rats. The total protein (g/l) also was significantly different between the 63.56±1.21 of petroleum treated rats and 70.28±1.89 of sugar treated rats. The albumin (g/l) was significantly different between the 36.40±0.57 of petroleum treated rats and 41.00±1.03 of sugar treated rats. There was also significant difference in 6.96±0.92 liver/body weight ratio of Petroleum fed rats compared with 7.52±0.98 of sugar fed rats as shown in table 5 below.

Table 5: Overall effect of sugar on biochemical parameters in albino rats treated with petroleum

Parameter	Crude petroleum	Sugar fed	T	Sig
ALKPHOS(U/L)	57.48±4.66	38.96±3.00	3.916	0.001
ALT (U/L)	22.88±2.59	14.08±2.22	3.695	0.001
AST(U/L)	33.64±5.47	20.40±2.26	4.257	0.000
GGT(U/L)	677.20±59.24	491.28±62.96	3.671	0.001
T. PROT(g/l)	63.56±1.21	70.28±1.89	-2.743	0.010
ALB (g/l)	36.40±0.57	41.00±1.03	-4.417	0.000
Liver/Body weight	6.96 ±0.92	7.52± 0.98	- 3.615	0.022

4. Discussion

The oral lethal dose 100 (LD_{100}) and lethal dose 50 (LD_{50}) of 124.04 ±0.195g/kg and 255.60±0.195g/kg respectively obtained in this study is similar to the report of Akaninwor *et al.,* [16] for Bonny light crude petroleum (LD_{100}=254.80g/kg and LD_{50}=108.30g/kg) and Forcados crude petroleum (LD_{100}=254.60g/kg and LD_{50}= 150.70g/kg). Most of the substances contained in crude petroleum occur naturally due to their presence in rock formation or in saltwater deposits from which the crude oil was drawn [7]. However some of these are also introduced from the drilling pipes and drilling fluid additives while others are introduced during pumping, preparing and transporting of crude oil (24). They are also introduced greatly in the relative concentrations of different components and thus show substantial variability in solubility, dispensability, persistence and toxicity [25]. LD_{50} values have been reported to depend on the route of administration. Values are found to increase with the following sequences of route: intravenous, intraperitoneal, subcutaneous and oral [26]. The highest dose of crude petroleum selected for the study was half of LD_{50} which was considered tolerable for the period of study.

There was dose dependent increase in alkaline phosphatase (ALK), aspartate amino transferase (AST), Alanine amino transferase (ALT) and gamma glutamyl transpeptidase (GGT) of rats fed crude petroleum contaminated diet compared with their controls. Similar observations have been reported [12, 27-30]. The significant dose dependent decrease in total protein (TPROT) and albumin (ALB) concentrations in crude petroleum treated albino rats obtained in this study is similar to the work of Sunmonu and Oloyede [23]. Albumin is a major protein manufactured in the liver that circulates in the blood stream [31]. The dose dependent reduction in serum albumin concentration may be a consequence of poor diet or an indication of liver dysfunction amongst others. Thus, it is possible that the contaminated diet consumed by the rats which contains toxic compounds like polycyclic aromatic hydrocarbons (an important constituent of crude oil) may affect the liver thereby preventing it from manufacturing enough albumins for release into the serum. There was significant reduction in the liver to body weight ratio of the crude petroleum treated when compared to the control. Berepubo *et al*., [32], Ovuru *et al.*, [33] and Sunmonu and Oloyede [23] reported similar observations.

There were significant dose dependent lowering of the ALKPHOS, AST, ALT and GGT in the sugar study groups compared with petroleum study group. Also significant dose related increases in the Total Protein and albumin levels were observed in sugar study groups when compared to the petroleum study group while there was no significant difference in the liver to body weight ratio of the two groups. This is similar to other studies [34-38] that showed that feeding carbohydrates suppressed elevated enzyme activities. Glucose feeding in both man and microorganisms causes profound changes in metabolism including inhibition of induction of several enzymes, stimulation of others and blockage of most effects of glucocorticoids [39]. Glucose is known to repress a large number of inducible enzymes in many different bacteria.

There was overall reduction in the activities of ALKPHOS, AST, ALT and GGT in sugar fed albino rats compared with petroleum fed albino rats while there were overall significant increases in albumin and total protein levels of sugar study group compared to the petroleum study group. Glucose represses the induction of inducible operons by inhibiting the synthesis of cyclic Adenosine monophosphate (cAMP) a nucleotide that is required for the initiation of transcription of a large number of inducible enzyme systems including the Lac operon. Cyclic AMP (cAMP) is required to activate an allosteric protein called catabolite activator protein (CAP) which binds to the promoter CAP site and stimulates the binding of Ribonucleic acid (RNA) polymerase to the promoter for the initiation of transcription, but cAMP must be available to bind to CAP which binds to Deoxyribonucleic acid (DNA) to facilitate transcription. In the presence of glucose, adenylase cyclase (AC) activity is blocked. AC is required to synthesize cAMP from Adenosine Triphosphate (ATP) [40,41]. Therefore if cAMP levels are low, CAP is inactive and transcription does not occur. In the absence of glucose, cAMP levels are high, CAP is activated by cAMP and transcription occurs (in the presence of lactose). Thus the effect of glucose in suppressing these inducible enzymes is by lowering cyclic AMP level.

5. Conclusion

This study has shown that feeding on certain concentrations of sugar will repress the induction of certain enzymes due to glucose effect. These repressions depend on the concentration of carbohydrate.

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Conflict of interest

No conflict of interest associated with this work.

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