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Research Article

Estimation of serum glutamic oxaloacetic transaminase, serum glutamic-pyruvic transaminase, gamma-glutamyl transferase and cholesterol levels in prolonged (30 years) daily consumption coffee in people

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ABSTRACT

Background: Although prolonged (30years) coffee consumption has been associated with reduced or increased frequency of liver (SGOT, SGPT, GGT) enzymes and cholesterol levels, it is unclear whether the effect is from coffee or caffeine.
Methods: A self-administered questionnaire ascertained lifestyle characteristics, including alcohol consumption, cigarette smoking, Diabetes mellitus and Dietary habits. As for drinking habit, examinees were first asked about their current drinking frequency (none, 1-2 times/ week, or almost daily) past or current smokers about the number of cigarettes smoked per day and the duration of smoking in years. As regards coffee, examinees were asked their usual daily intake in cups.
Results: A total No of 200 cases were studied by dividing them into two group's controls and cases. The results so obtain were compared with 50 healthy controls (not to consumed caffeine contained things that include nor coffee, tea, any type of cola's). Statistical evaluation was carried out to confirm any deviation from the normal values. In men the mean serum SGOT, SGPT, GGT and cholesterol of cases is having higher level as compared to the mean value of controls. This increase is statistically highly significant (SGOT<0.0001, SGPT=0.045, GGT=0.0043, cholesterol<0.0001). In women's the mean serum SGOT, SGPT, GGT and cholesterol of Cases is having higher level as compared to the mean value of Controls. This increase is statistically significant (SGOT< 0.05, SGPT=0.0319, GGT <0.0001, cholesterol: <0.0001).
Conclusion: It has been shown from this study that prolonged daily consumption of coffee in many people it results increasing of levels of serum concentrations of the SGOT, SGPT, GGT and Cholesterol. It is observed that liver enzymes and cholesterol could be a target for caffeine or other components of coffee.

Keywords: SGOT, SGPT, GGT, Cholesterol, Caffeine and coffee

INTRODUCTION

Coffee has been for decades the most commercialized food product and most widely consumed beverage in the world. Since the opening of the first coffee house in Mecca at the end of the fifteenth century, coffee consumption has greatly increased all around the world. In 2010, coffee production reached 8.1 million tons worldwide.[1]

The reasons for this continuous increase in coffee consumption include improved cup quality through

selection of varieties and breeding, better agricultural practices; the creation of specialty shops, and a change in coffee's image through the dissemination of information on the health benefits of long-term coffee consumption. Today, coffee is considered a functional food, primarily due to its high content of compounds that exert antioxidant and other beneficial biological properties. The characteristic flavour and richness of coffee aroma make it a unique beverage, with almost a thousand volatile compounds identified in roasted coffee.[2]

The coffee tree belongs to the Rubiaceae family, genus *Coffea*. Although more than 80 coffee species have been identified worldwide only two are economically important.[3] *Coffeaarabica*, also known as Arabica coffee, is responsible for approximately 70% of the global coffee market, and *Coffeacanephora* or Robusta coffee (commercial name of one of the main C. *canephora cultivars*) accounts for the rest.[4,5]

Some health effects of coffee are due to its caffeine content, as the benefits are only observed in those who drink caffeinated coffee. While others appear to be due to other components as exemplified by the presence of the antioxidants that prevent free radicals from causing cell damage.[6]

Due to the importance of caffeine in human health caffeine metabolism has been studied for some time in coffee.[7-9] The world's primary source of caffeine is the coffee bean, from which coffee is brewed. Caffeine, an active ingredient of coffee, is a central nervous system and metabolic stimulant.[10]

Chemistry of the plant: Coffee plants contain two different kinds of alkaloid delivered from nucleotides. One type is purine alkaloids, such as Caffeine (1, 3, 7-*N*-trimethylxanthine) and Theobromine (3, 7-*N*-dimethylxanthine); the other is the pyridine alkaloid, trigonelline (1-*N*-methylnicotinic acid). The distribution of caffeine and Trigonelline in the plant kingdom is different; caffeine is present in both coffee and tea, but trigonelline is found only in coffee.[6]

Caffeine is a naturally occurring stimulant found in coffee, Caffeine acts through multiple mechanisms, the most important of which is the antagonism of adenosine receptors (ADORA1 and ADORA2A).[11]

Caffeine

The best known N-compound is caffeine (1,3,7 trimethylxanthine) because of its physiological effects (stimulation of the central nervous system, increased blood circulation and respiration). It is mildly bitter in taste (threshold value in water is 0.8-1.2mmole/l), crystallizes with one molecule of water into silky, white needles, which melt at 236.5^0C and sublime without decomposition at 178^0C. The caffeine content of raw Arabica coffee is 0.9-1.4%, while in the Robusta variety; it is 1.5-2.6%. Other purine alkaloids are theobromine (Arabica: 36-40mg/kg, Robusta: 26-82mg/kg) and theophylline (Arabica: 7-23µg/kg, Robusta: 86-344µg/kg). Caffeine forms, inpart, ahydrophobic π-complex with chlorogenic acid in a molar ratio of 1:1. In a coffee drink, 10% of the caffeine and about 6% of the chlorogenic acid present occur in this form.[12]

Trigonelline synthesis: Trigonelline is synthesized through the methylation of nicotinic acid (NAD) by S-adenosyl-L-methionine (SAM)-dependent nicotinate *N*-methyltransferase. Its function is still a matter of debate but it might be a reserve compound for NAD that could be used during germination.[13] During roasting, trigonelline alkaloid gives rise to many aroma (flavor) compounds, like alkyl-pyridines and pyrroles which explains the interest of breeding programs to increase of trigonelline content in Robusta green beans.[14,15]

Trigonelline(N-methylnicotinic acid) is present in green coffee up to 0.6% and is 50% decomposed during roasting. The degradation products include nicotinic acid, pyridine, 3-methyl pyridine, nicotinic acid methyl ester, and a number of other compounds.[12]

Various acids in the plant includes formic and acetic acids predominate among the volatile acids, while nonvolatile acids are lactic, tartaric, pyruvic and citric. Higher fatty acids and malonic, succinic, glutaric and malic acids are only minor constituents. Itaconic (I), citraconic (II) and mesaconic acids (III) are degradation products of citric acid, while fumaric and maleic acids are degradation products of malic acid: Chlorogenic acids are the most abundant acids of coffee.[12]

Chlorogenic acids

Chemical structure and composition of chlorogenic acids

Chlorogenic acids (CGA) are the main phenolic compound found in green coffee beans and have been studied for more than a century**.** [16] which are responsible for substantial part of coffee antioxidants.[17,18] They belong to hydroxycinnamicacids derivative have a promising role in protection from radiation and photooxidation hydroxycinnamic acid classes and chiefly consists of caffeic acid (3, 4-dihydroxycinnamic acid), ferulic acid (3-methoxy-4-hydroxycinnamic acid), pcoumaric (4-hydroxycinnamic acid), and sinapic acid (3,5dimethoxy-4-hydroxycinnamic acid).[19,20]

Antioxidant properties of chlorogenic acids

In addition to the aforementioned, commercially antioxidants are also manufactured from coffee that consists of 55% chlorogenic acids which regulates the blood sugar and perhaps help for the management of obesity.[21] Several studies related to the effects of this compound on blood sugar regulation are also noted. Clinical evolution indicated the chlorogenic groups had

lowered the increase in blood glucose level by 15 to 20% Furthermore, the risks of type-2 diabetes also decreased with higher consumption.[22] It is expected that the chlorogenic acids are the main active component to ant diabetic effect according to pharmacology studies.[21-23]

Table1: Composition of green Arabica and Robusta coffee.[12]

Constituent	Arabica	Robusta	Components
I.Soluble carbohydrates	9–12.5	6–11.5	
Monosaccharides		0.2–0.5	Fructose, glucose, galactose, arabinose (traces)
Oligosaccharides	6–9	3–7	Sucrose (>90%), raffinose (0–0.9%), stachyose (0–0.13%)
Polysaccharides	3–4		Polymers of galactose (55–65%), mannose (10–20%), arabinose (20–35%), glucose (0–2%)
II.Insoluble polysaccharides	46–53	34–44	
Hemicelluloses	5–10	3–4	Polymers of galactose (65–75%), arabinose (25–30%), mannose (0–10%)
III.Cellulose,β(1–4)mannan	41–43	32–40	
IV.Acidsand phenols 1.Volatileacids		0.1	
Nonvolatile aliphatic acids	2–2.9	1 .3–2.2	Citric acid, malic acid, quinic acid
Chlorogenic acid	6.7–9.2	7 .1–12.1	Mono-, dicaffeoyl- and feruloylquinic acid
Lignin		1–3	
Lipids	15–18	8–12	
Wax	0.2–0.3		
Oil	7.7–17.7		Main fatty acids: 16:0 and 18:2
N Compounds	11–15		
Free amino acids	0.2–0.8		Main amino acids: Glu, Asp, Asp-NH2
Proteins	8.5–12		
Caffeine	0.8–1.4	1 .7–4.0	Traces of theobromine and theophylline
Trigonelline	0.6–1.20	0 .3–0.9	
Minerals		3–5.4	

a .Values in % of solids, b. Water content of raw coffee: 7–13%, c. Main components: 5-caffeoylquinic acid (chlorogenic acid: Arabica 3.0–5.6%; Robusta 4.4–6.6%).

Table 2: Amino Acid composition of coffee (%).

Amino acid	Arabica		Robusta	
	Green	Roasted	Green	Roasted
Alanine	4.75	4.76	4.87	6.84
Arginine	3.61	0.0	2.28	0.0
Asparagine	10.63	9.53	9.44	8.94
Cysteine	2.89	0.76	3.87	0.14
Glutamic acid	19.88	21.11	17.88	24.01
Glycine	6.40	6.71	6.26	7.68
Histidine	2.79	2.27	1.79	2.23
Isoleucine	4.64	4.76	4.11	5.03
Leucine	8.77	10.18	9.04	9.65
Lysine	6.81	3.46	5.36	2.23
Methionine	1.44	1.08	1.29	1.68
Phenylalanine	5.78	5.95	4.67	7.26
Proline	6.60	6.82	6.46	9.35
Serine	5.88	2.60	4.97	0.14
Theorine	3.82	2.71	3.48	2.37
Tyrosine	3.61	4.11	7.45	9.49
Valine	8.05	6.93	6.95	10.47

Table 3: Composition of Roasted Coffee (medium degree of roasting).[12]

Component	Content (%)	
	Arabica	Robusta
Caffeine	1.32	2.4
Lipids	17.0	11.0
Proteins	10.0	10.0
Carbohydrates	38.0	41.5
Trigonelline niacin	1.0	0.7
Aliphatic acids	2.4	2.5
Chlorogenic acids	2.7	3.1
Volatile compounds	0.1	0.1
Minerals	4.5	4.7
Melanoidins	23.0	23.0

Besides the antioxidant properties and physiological function, chlorogenic acids play a great role in the formation of pigments, taste and flavor of coffee beans, which determine the quality and acceptance of the

beverages. They contribute to the final acidity of the beverages, as a result of maillard and strecker's reaction bitterness and the formation of lactones and other phenol derivatives responsible for flavor and aroma.[24-26]

Table 4: Lipid composition of roasted coffee beans (coffee oil).[12]

Constituent	Content (%)
Triacylglycerols	78.8
Diterpene esters	15.0
Diterpenes	0.12
Triterpene esters	1.8
Triterpenes (sterols)	0.34
Unidentified Compounds	4.0

Table 5: Chlorogenic acid content as a function of the degree of roasting.[12]

Raw/degree of roasting	Arabica	Robusta
Raw	6.9%	8.8%
Light	2.7%	3.5%
Medium	2.2%	2.1%
Dark	0.2%	0.2%

The antioxidant capacity of chlorogenic acid is more potent than of ascorbic acid (vitamin C) or mannitol, which is a selective hydroxy-radical scavenger.[27]

Metabolism

The liver plays a major role in metabolism, synthesis and storage of essential substances in the body, as well as the detoxification of xenobiotics.[28] Caffeine from coffee is absorbed by the small intestine within 45 minutes of ingestion and distributed throughout all bodily tissues.[29] After absorption, caffeine is metabolized in the liver by the cytochrome P450 oxidase enzyme system, in particular, by the CYP1A2 isozyme into three primary metabolites: paraxanthine: 84%; theobromine: 12%, and theophylline: 4% each of which has its own effects on the body (30)Caffeine has a half-life of 4 to 5 hours, which may be prolonged in patients with hepatic diseases, infants and neonates (up to 100 hours), or during pregnancy.[30-32]

METHODS

The study was lasted from 1st January 2015 to 30th November 2015 (11 months) the subjects included both men and women Subjects consisted of 130 Men and 70 women with age between 40-70.

A self-administered questionnaire ascertained lifestyle characteristics, including alcohol consumption, cigarette smoking, diabetes mellitus and dietary habits. As for drinking habit, examinees were first asked about their current drinking frequency (none, 1-2 times/ week, or almost daily) past or current smokers about the number of cigarettes smoked per day and the duration of smoking in years. In this study, past and never drinking, smokers were combined. As regards coffee, examinees were asked their usual daily intake in cups.

The daily consumption of alcohol, cigarettes, and coffee venous blood was taken for the determination of liver enzymes (AST, ALT, GGT) and cholesterol.

Sample collection

Random blood samples (for 11 months from 1st January 2015 to 30th November 2015) were collected from 150 variety population include Diabetes Mellitus, Smokers, Alcohol intake (prolong with limited), and 50 healthy controls (not to consumed caffeine contained things that include nor coffee, tea, any type of cola's).

5 ml of venous blood was collected from each subject and dispensed into lithium heparin bottles. Plasma was obtained by centrifugation for 5 min at 3,000 rpm and separated into plain bottles for analysis

concentrations were assayed at the institution, based on the IFCC (International Federation of Clinical Chemistry) methods with an Semi Auto analyser (Lab life CHEM MASTER, RFCL) using commercial reagents (ERBA for AST,ALT, Cholesterol and Tulip for GGT); the normal ranges are AST 5-34 IU/L, for ALT: 0-40, GGT (Males:10-50 U/L, Females:7-35U/L), Cholesterol: 140-250mg/dl).

1. ***Estimation of serum AST (aspartate aminotransferase) (IFCC Method, Kinetic*):**[33]

Principle

L-Aspertate +2-Oxoglutarate $\xrightarrow{\text{AST}}$ Oxaloacetate + L-Glutamate

MDH

Oxaloacetate + NADH $\xrightarrow{\text{MDH}}$ Malate + NAD

LDH (lactate dehydrogenase)

Sample pyruvate + NADH $\xrightarrow{\text{LDH}}$ L-Lactate + NAD

Reagent composition (Single step enzymatic reagent) includes: 2-Oxoglutarate + L-Aspertate + MDH + LDH + NADH(Yeast) + Tris buffer + EDTA.

Allow the reagent bottle to attain room temperature add the 20 ml of Aqua-4 of each vial. Swirl to dissolve. Do not shake vigorously.

Assay Procedure: Add 1000µl working reagent and 100µl test solution than mix well and aspirate note the value.

Estimation of serum ALT (IFCC Method, Kinetic):[34]

Principle

L-Alanine +2-Oxoglutarate $\xrightarrow{ALT}$ Pyruvate + L-Glutamate

Pyruvate + NADH $\xrightarrow{LDH}$ L-Lactate + NAD

Reagent composition (Single step enzymatic reagent) consists of L-Alanine + NADH (Yeast) + LDH + 2-Oxoglutarate + Tris buffer.

Allow the reagent bottle to attain room temperature add the 20 ml of Aqua-4 of each vial. Swirl to dissolve. Do not shake vigorously.

Assay Procedure: Add 1000µl working reagent and 100µl test solution than mix well and aspirate note the value.

Estimation of serum GGT (Carboxy Substrate Method)[35]

Principle

γ Glutamyl Transferase (GGT) catalyzes the transfer of amino group between L-γ -Glutamyl-3-carboxy-nitroanilide and glycylglycine to form L-γ –Glutamylgly cylglycine and 5-amino -2-nitrobenzoate . The rate of formation of 5-amino -2-nitrobenzoate is measured as an increase in absorbance which is proportional to the GGT activity in the sample.

L-γ -Glutamyl-3 -carboxy-4nitroanilide + Glycylglycine $\xrightarrow{GGT}$ L-γ-Glutamylglycylglycine + 5-amino -2-nitrobenzoate

Reagent composition includes buffer reagent and substrate tablets. And for the working reagent dissolve 1 substrate tablet in 2.2 ml of buffer reagent.

Assay procedure: Add 1ml of working reagent to 0.1 ml of test solution after that mix well and aspirate note the value.

Estimation of serum Cholesterol (CHOD-PAP (with LCF) Method)[36]

Principle

The estimation of cholesterol involves the following enzyme catalysed reaction.

Cholesterol ester $\xrightarrow{CE}$ Cholesterol + Fatty acid

Cholesterol + O_2 $\xrightarrow{CHOD}$ Cholest-4-en-3one + H_2O_2

$2H_2O_2$ + 4AAP + Phenol $\xrightarrow{POD}$ 4 H_2O + Quinoneimine

Reagent composition (Single step reagent) consists of Cholesterol esterase + Cholesterol Oxidase + Peroxidise + Sodium phenolate + 4-Aminoantipyrine + Phosphate buffer + Lipid clearing Agent.

Cholesterol standard used is 200mg/dl.

Allow the reagent bottle to attain room temperature add the 20 ml of Aqua-4 of each vial. Swirl to dissolve. Do not shake vigorously.

Assay Procedure: For the blank reading pipette 20µl distilled water into 1000µl working reagent, for standard reading pipette 20µl standard solution into 1000µl working reagent and for the test pipette 20µl test solution into 1000µl working reagent after that mix well incubate at 37°C for 10 minutes. Aspirate blank followed by standard and test, and note the values.

RESULTS

During the period from 1st January 2015 to 30th November 2015 (11 months) in the Department of Medicine, Gayatri Vidya Parishad Health Care and Medical College, Marikavalasa, Visakhapatnam. A total No of 200 cases were studied by dividing them into two groups Controls and Cases and observation made were tabulated.

Table 6: Sex wise distribution of groups.

Group	Total number	Male		Female	
		Number	Percentage	Number	Percentage
Cases	150	110	73%	40	27%
controls	50	20	40%	30	60%

The sex distribution among the cases and controls were 73%, 40% respectively in Males 27% and 60% in Females respectively.

Table 7: SGOT (AST) IU/L, levels in men in both cases and controls.

Group	SGOT	Mean±SD	Z - Value	P- Value
Cases	110	24.44 ±11.68	3.9403	< 0.0001
controls	20	14.05 ± 3.12		

The above table shows that mean serum SGOT of Cases (24.44±11.68) is having higher level as compared to the mean value of Controls (14.05±3.12). This increase is statistically highly significant (< 0.0001).

Table 8: SGPT (ALT) IU/L, levels in men in both cases and controls.

Group	SGPT	Mean±SD	Z - Value	P- Value
Cases	110	23.51±11.06		
controls	20	18.90±5.22	2.0198	0.045

The above table shows that mean serum SGPT of Cases (23.51±11.06) is having higher level as compared to the mean value of Controls (18.90±5.22). This increase is statistically significant (0.045).

Table 9: GGT (IU/L), levels in men in both cases and controls.

Group	GGT	Mean±SD	Z - Value	P- Value
Cases	110	13.20 ±2.93		
controls	20	11.20±2.17	2.9077	0.0043

The above table shows that mean serum GGT of Cases (13.20±2.93) is having higher level as compared to the mean value of Controls (11.20±2.17). This increase is statistically significant (0.0043).

Table 10: Cholesterol (mg/dl) levels in men in both cases and controls.

Group	Cholesterol	Mean ±SD	Z - Value	P- Value
Cases	110	197.88± 24.52	8.6486	< 0.0001
controls	20	147.35± 21.08		

The above table shows that mean serum cholesterol of Cases (197.88±24.52) is having higher level as compared to the mean value of Controls (147.35±21.08). This increase is statistically highly significant (<0.0001).

Table 11: SGOT (AST) IU/L, levels in women in both cases and controls.

Group	SGOT	Mean ± SD	Z - Value	P- Value
Cases	40	24.85±19.26		
controls	30	18.76± 5.84	1.885	< 0.05

The above table shows that mean serum SGOT of Cases (24.85±19.26) is having higher level as compared to the mean value of Controls (18.76±5.84). This increase is statistically significant (<0.05).

Table 12: SGPT (ALT) IU/L, levels in women in both cases and controls.

Group	Sgpt	Mean ± SD	Z - Value	P- Value
Cases	40	19.75 ± 5.54		
controls	30	16.87± 5.32	2.1909	0.0319

The above table shows that mean serum SGPT of Cases (19.75±5.54) is having higher level as compared to the mean value of Controls (16.87±5.32). This increase is statistically significant (0.0319).

Table 13: GGT (IU/L), levels in women in both cases and controls.

Group	GGT	Mean ± SD	Z - Value	P- Value
Cases	40	12.65±2.12		
controls	30	10.47±1.81	4.5319	< 0.0001

The above table shows that mean serum GGT of Cases (12.65±2.12) is having higher level as compared to the mean value of Controls (10.47±1.81). This increase is statistically significant (<0.0001).

Table 14: Cholesterol (mg/dl) levels in women in both cases and controls.

Group	Cholesterol	Mean ± SD	Z - Value	P- Value
Cases	40	205.58± 19.01	14.806	< 0.0001
controls	30	137.23± 19.25		

The above table shows that mean serum cholesterol of cases (205.58±19.01) is having higher level as compared to the mean value of Controls (137.23±19.25). This increase is statistically highly significant (<0.0001).

Table 15: consolidated chart showing statistical analysis of various parameters between cases and controls.

Serum concentrations	Males		Z – Value	P- Value	Females		Z - Value	P- Value
	Cases	controls			Cases	Controls		
SGOT	24.440	14.050	3.9403	< 0.0001	24.85	18.76	1.885	< 0.05
SGPT	23.51	18.90	2.0198	0.045	19.75	16.87	2.1909	0.0319
GGT	13.20	11.20	2.9077	0.0043	12.65	10.47	4.5319	< 0.0001
CHOLESTEROL	197.88	147.35	8.6486	< 0.0001	205.58	137.23	14.806	< 0.0001

DISCUSSION

Most of the previous studies of caffeine's health effects have been focused largely on coffee consumption rather than total caffeine intake; from the previous studies it was concluded that increased coffee consumption is associated with lower liver enzymes, reduced rates of liver cancer and possibly even reduced hepatic decompensation and liver-related mortality.[37-39]

In careful observation, it was found that daily intake of prolonged (30years) 2 cups of coffee consumption on liver enzymes and cholesterol levels raises (SGOT, SGPT, GGT, and cholesterol) in the serum was significantly raised. This confirms a case study carried out by Urgert R et al, which showed that unfiltered coffee causes an increase in alanine aminotransferase and aspartate aminotransferase.[40]

Urgert R et al, compared the long term effects of cafetiere coffee (unfiltered coffee andfiltered coffee) in a randomized controlled trial.[40] Cafetiere coffee raised alanine Aminotransferase concentration in healthy people. The diterpenescafestol and kahweol in unfiltered coffee are responsible for this effect (Weusten-Van der Wouw MP et al).[41]

A high intake of strong unfiltered coffee might explain some cases of raised alanine aminotransferase concentrations in apparently healthy people (Carola R et al).[42] For the millions of people who depend on coffee to start their day, cholesterol is probably the last thing on their mind as they wait for the morning surprise of caffeine to kick in. In the past few years, though, more and more evidence hints that coffee can increase cholesterol levels.

Coffee increases serum levels of total and LDL cholesterol. Klag and his colleagues reviewed more than a dozen studies that looked at the relationship between coffee consumption and cholesterol levels.[43] They found that drinking an average of six cups of coffee a day was associated with increased total cholesterol.[44] According to Klag, the increase in cholesterol is believed to be caused by oils called terpenes that are found in coffee, but are mostly removed by filters.

Persons who drink unfiltered coffee should get their cholesterol checked to make sure it is not elevated, says klag. The johns hopkins researcher found an association between coffee consumption and an increased risk of heart disease. But most of the increased risk was linked to coffee drinking before 1975. It was during the mid1970s, klag points out, that drip coffee makers became widely used in the united states, making filtered coffee the norm.

First reports of a relationship between coffee and liver biochemistry dates back 20 years.[45] since then, copious studies have reported the association of coffee consumption with reduced levels of liver enzymes in sundry geographical regions extending from north America, through Europe and into Asia.

The study by Japanese investigators also addressed the potential relationship between coffee consumption and AST/ALT levels. In this study, 7313 males attending for health examination were recruited; as before, individuals with previous history of liver disease and former consumption of alcohol were excluded.[46] 415 (58%) of subjects had elevated AST/ALT. Following categorization of subjects according to coffee intake, analysis demonstrated a stepwise reduction in odds ratio (or) of having liver inflammation (i.e. Raised AST/ALT) with increasing levels of coffee use. Compared with subjects not drinking coffee

Ruhl CE and Everhart JE published the results of a large population based study examining whether elevated serum alt activity was less common with increasing coffee intake.[47]

The study by tanaka K et al *a*lso investigated the potential relationship between coffee consumption and alanine (ALT) and aspartate (AST) aminotransferase. As with GGT, coffee intake was significantly related to decreased serum concentrations of both enzymes among males, whereas the corresponding relationship amongst females did not achieve statistical significance.[48]

Ruhl CE and Everhart JE published the results of a large population-based study examining whether elevated serum alt activity was less common with increasing coffee intake.[47]

Recently, a group from California published results of a 22-year follow-up of 125580 individuals, again without known liver disease at recruitment.[38] In addition to a cohort study examining a potential role played by coffee consumption on the development of alcoholic cirrhosis. Following stratification of levels of alcohol intake, logistic models demonstrated an inverse relationship between coffee drinking and enzyme levels within these alcohol consumption categories.

Arnesen E et al was the first to report an inverse relationship between coffee drinking and serum gamma-glutamyltransferase (GGT) in the tromso heart study. Tanaka K et al. conducted a cross-sectional study of 12687 healthy Japanese subjects, excluding those with pre-existing liver disease or abnormal liver enzymes.[45,48] This group demonstrated an inverse association between coffee consumption and serum GGT levels.

Multivariate analysis showed a highly significant inverse relationship between coffee and GGT in men ($p<0.0001$) with a much less, however still significant, relationship for females ($p<0.002$). The investigators postulated that the weaker association observed the mean intake levels

for females were lower than males within alcohol consumption categories. Taking into account the observation that the inverse relationship between coffee and GGT was maximal for high alcohol drinkers, the lower alcohol consumption amongst females in the study may account for the less significant results for this gender.

Several investigators have suggested that the inverse relationship which has been described between coffee and liver disease may not be attributable to a coffee effect at all. Given the well-documented impairment of caffeine metabolism in those with cirrhosis.[49] A number of other studies, including those by Tanaka K et al, Corrao R et al and Inoue M et al do not support this hypothesis. Using other caffeine containing beverages such as green tea or cola to substitute for coffee, these groups failed to demonstrate a significant association between these beverages and reduction in liver-related endpoints.[48,42,50]

The study by Urgert R et al and Weusten-van der wouw et al, have shown that coffee constituents actually increase liver function tests (AST/ALT), which is contrary to the results of Tanaka K et al, Honjo S et al And Klatskyet AL al, all of which have been shown a significant inverse relationship between coffee consumption and transaminase levels.[40,41,48,46,38]

Not all types of coffee may be beneficial in liver disease. Numerous studies have shown a hepatoprotective role for filtered coffee, and a potentially deleterious effect for unfiltered coffee.[40,51]

Coffee preparation methods include filtered, unfiltered, and espresso and can also vary in its roast profile (medium and dark). Differences in preparation method (filtered, unfiltered, espresso) as well as type of roast play a role in the composition of coffee. Our data showed a robust inverse relation of coffee drinking to peril of liver enzymes and cholesterol levels in diabetes mellitus, smokers, alcohol intake, and independent of several potential confounders. In population studies, reference is usually made to average national consumption. This is calculated from raw coffee production import and may not represent an accurate portrait of the local coffee.

CONCLUSION

In persuasion and through study of various aspects of coffee consumption people I have taken into consideration of prolonged coffee consumed people more than 30 years, it is clear some of the eminent people stated that the coffee consumption is good for health it reduces the stress on the brain and nerves, most of people state that the consumption of coffee Is not good because of caffeine is a poisonous substance hence I have taken an opportunity to discuss in various issues in coffee consumers.

It has been shown from this study that prolonged daily consumption of coffee in many people it results increasing of levels of serum concentrations of the SGOT, SGPT, GGT and cholesterol. It also effects the veracity of the liver functions studied. It is observed that liver enzymes could be a target for caffeine or other components of coffee; however, the mechanism of the effect presently remains unclear. Further study regarding the mechanism through which coffee affects the liver is exceptionally needed.

Coffee itself does no harm but spoiled coffee might have favourable effects exhibited so far by a moderate and sustained coffee consumption represents an important body of evidence. However possible contaminants which are might be present in coffee as in many other commodities.

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