

Effect of Coffee Daily Consumption on Uric Acid Level and Body Weight to Prevent Metabolic Syndrome

Rosa Lelyana*

Diponegoro University, Semarang City, Central Java, Indonesia

Abstract

Obesity was one of diagnosis criteria of metabolic syndrome. There was increasing mortality case of obesity in recent decade. High uric acid level was the early marker of metabolic syndrome. Coffee was one of favorite drink of obese and non obese people in the world. Thus we investigated the effects of coffee drinking for preventing metabolic syndrome. This study was conducted by true experimental with randomized controlled group pretest-posttest design. Twenty four male wistar rats (10 weeks) were fed a standar density diet with and without 0.72 ml coffee solution daily during 14 days. Treatment and control group were greatest difference in the last day (day-14) or after study ($t=2.24$; $p<0.05$). The difference in body weight decrease was bigger in treatment group. The difference in body weight increase became bigger with longer time in control group. Mean of body weight difference during 14 days, differed significantly ($t=-4.59$; $p=0.00$) between treatment ($n=12$) and control group ($n=12$). Mean of obese uric acid level at the start of study was higher than obese uric acid level at the end of study although the difference was not significant ($p>0.05$). Drinking 0.72 ml (equal with 2 cup of coffee in human) coffee daily during 14 days decreased uric acid level in obese rats model although not significant. Coffee has activity as antiobesity and has preventive action against metabolic syndrome disease.

Keywords: Coffee; Obese model rats; Non obese; Uric acid; Body weight; Metabolic syndrome

Introduction

Obesity is still one of the health problems in the world. Prevalence of metabolic syndrome increased in adult males aged > 45 years and in adult females aged >55 years [1]. Obesity is related with the body's immune response. Accumulation of adipose tissue influence inflammation response so increase body's immune response. Body's immune response is a response from the body's biological systemic structure which protect the body to combat diseases. This system must be able to detect various disease agents such as viruses to parasites (worms) and know how to differ this from organism that possesses healthy tissue [2,3]. Decreasing of body weight is characterized by reduced white adipose tissue as the main source of xanthin oxidase and decreasing of xanthin oksidase activity is the basic decrease of serum uric acid level [4]. High uric acid level is a marker of inflammatory response in metabolic syndrome [5,6].

Results study showed that minimal processing of diet that rich in whole foods such as vegetables, fruits, legumes (including coffee), grains, cereals would be able to slow glucose absorption [7]. The advantage of robusta coffee was could grow in tropical area and the price was not too expensive [8].

The total content of fenolic acid in hot water mixture of pure robusta coffee powder according to the previous study was 45.63 - 47.95 mg/g coffee powder or 456 mg/10 g - 479 mg/10 g robusta coffee powder which was higher than arabica coffee powder (342.3-396.3 mg/10 g) [9]. Previous study showed a decrease of 15% blood uric acid level on hyperuricemia wistar rats after consuming 0.72 ml of coffee solution during 7 days [10].

Results of surveillance on bibliography had not found studies that examined uric acid level in obese and non-obese. This study has chosen on obesity wistar rat model to find out the difference in effects of coffee given in condition of accumulated adipose tissue (pro-inflammatory rats) and without accumulation of adipose tissue. Is there any difference in uric acid level between obese Wistar rats group and non-obese that were given coffee drinking? The novelty of this study was based

on bibliography surveillance that is study on measurement of various endogen antioxidant status i.e., uric acid had never been developed so far. The difference between this study and the previous study was that the study subject used obese rats model and non-obese rats.

The independent variable was giving robusta coffee powder that is equivalent with 2 cups per day which contained polyphenol and caffeine [10]. The dependent variables was uric acid level. The purpose of this study: to prove the difference of uric acid level of obese and non-obese rats between coffee drinking group (treatment group) and aqua drinking group (control group).

Methods and Materials

This study design is true experimental with randomized controlled group pretest-posttest design.

Population and samples

Study population: It was male *Rattus Norvegicus* of Wistar strain, ± 10 weeks age. The blood samples were performed through retroorbitalis vein plexus.

Study samples: Size of minimal sample in experimental study referred to the WHO standard (1993). Requirement for experimental animal i.e., each treatment were at least 5 animal. It should be based on Federer rule $\rightarrow (k-1)(n-1) \geq 15$. The size of sample in each group were at least 6 rats. Drop-out size was estimated 10% ($d.o=0.1$). Minimal size

*Corresponding author: Lelyana R, Medicine Faculty of Diponegoro University, Semarang City, Central Java, Indonesia, Tel: 0815764-5736; Fax: 024-8453708; E-mail: r3lyana@gmail.com

Received September 24, 2016; Accepted October 05, 2016; Published October 13, 2016

Citation: Lelyana R (2016) Effect of Coffee Daily Consumption on Uric Acid Level and Body Weight to Prevent Metabolic Syndrome. J Nanomed Nanotechnol 7: 400. doi: 10.4172/2157-7439.1000400

Copyright: © 2016 Lelyana R. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

of sample was: $n_{do} = 5 / (1 - do)^2 = 5 / (1 - 0.1)^2 = 5 / 0.81 = 6.2 \approx 6$. Allocation of rats was performed in simple random sampling by giving number to the tail. Then the numbers were taken through lottery. This study was performed on 4 groups (2 treatment groups and 2 control groups). Each group consisted of 6 rats, so the number of total sample were 4 groups \times 6 rats=24 rats.

Inclusion criteria: Normal body weight were 150-200 g and obesity were 251-300 g. Healthy, clean, white feathers, no abnormality in the eye, the movement was agile, no anatomic physiologic abnormalities.

Exclusion criteria: Suffering diarrhea or disease before treatment.

Study variable and operational definition

Independent variable was coffee drink 0.72 ml/day. Dependent variable was uric acid level. Confounding variables were age, sex, food/beverage consumed and rat species.

Dosage calculation

The amount of coffee consumption per day used table of comparison of experimental animal body surface area. Body weight's rat of 200 g were comparable with 0.018 human dose. Previous study results, 2 cups of coffee in human was equivalent with 0.72 ml/day in experimental animal test was able to decrease uric acid level as much as 15% (0.53 mg/dl) [10]. Coffee powder was given by mixing with boiling hot water. After the water temperature was not too high, the mixture was given to rats using sonde. Coffee drinking during 14 days according to the previous study that had ever been performed in human [11].

Data processing and analysis

The difference between before and after giving coffee drink of 0.72 ml daily during 14 days between treatment group and control group was tested using independent t test or Mann Whitney. A difference was considered as significant if the value of $p < 0.05$ with 95% confidence interval. Statistical analysis used SPSS program for Windows v.16.01.

Ethical clearance

Before performing the study, we needed to get ethical clearance and this would be presented to Health Study Ethical Commission in Medicine Faculty of Diponegoro University, Semarang city, Central Java, Indonesia.

Results and Discussion

Study location

The study was performed in MIPA Biological Laboratory at Semarang State University and also for examination of uric acid level on experimental animal.

Characteristic of experimental rats

The obese rats used as subject in this study were obese rats as a result of food high in carbohydrate given to the mice [11-13] that was combined with standard food of more than 20 g/day (40 g/day). Experimental animals were maintained in separated cages, each experimental animal lived in 1 cage. We gave 0.72 ml coffee drink daily for 14 days to 6 obese treatment group and 6 non obese treatment group. In addition, other study result showed that consumption of 2 cups per day was positively associated with higher plasma SHBG level (Serum Hormone Binding Globulin) and also associated with risk reduction of diabetes mellitus type 2 than consuming coffee less than 2 cups per day [14]. During the study, the entire rats in obese and non-obese group consumed the entire food available in each cage.

Coffee was the second most drink consumed in the world after aqua water [15]. Therefore in this study rats were also given aqua water consumption. Consumption of hygienic/aqua water that was made available in each cage was not consumed entirely by each experimental animal. Rats did not change into aggressive behavior during the study after consuming coffee. Rats could sleep well during study, both treatment and control rats. Consumption of coffee drink was given through sonde as much as 0.72 ml coffee solution and 2.28 ml warm clean water solution (3 ml in total). These coffee solution and 20 g standard food were given daily to obese treatment group and non-obese every morning for 14 days. Obese and non-obese control rats just consumed aqua water ad libitum and 20 g of standard food daily for 14 days. No rats dead as a result of the treatment.

Wistar rats were adapted for 1 week. Then, underwent fattening for more than 1 month. Twenty four rats that full filled inclusion criteria as obese and non-obese subject were randomly separated into treatment and control group. Treatment group was the group that was given coffee drink of 0.72 ml/day for 14 days + standard food. Control group was the group that was given aqua water ad libitum for 14 days + standard food. Each experimental animal was placed in individual cage, where room temperature and illumination and comfort in cage always maintained for mice.

Body weight characteristic during treatment

In obese group, table showed statistically significant difference in mean difference of body weight between treatment group and control group in day-0 and day-14 after study ($t = -3.19$; $p < 0.05$). Whereas non-obese group showed significant differences in mean body weight difference for every week between treatment and control group. Clinically, treatment group showed a decrease difference that was increasingly bigger. On the contrary the control group showed an increase difference that increasingly bigger with study time.

Table 1 said that the treatment group showed a decrease in mean body weight. The control group experienced an increase in body weight every week. The treatment group and control group were in greatest difference in the last day (day-14) after study ($t = -2.24$; $p < 0.05$). The difference in body weight decrease was bigger in treatment group. The difference in body weight increase became bigger with study time in control group. Mean of body weight difference during 14 days, differed significantly ($t = -4.59$; $p = 0.00$) between treatment group ($n = 12$) and control group ($n = 12$).

Body weight decrease occurred because coffee was a beverage with low glycemic index. This was supported by the fact that the amount of daily carbohydrate consumed did not influence BMI but the type of carbohydrate consumed [16,17] influenced BMI. Study result in several brands of packaged coffee powder showed that coffee was a type of grain that contained high carbohydrate [18]. The type of carbohydrate found in coffee was carbohydrate with low glycemic index [16,17].

Group	n	BW 0-1	BW 1-2	BW 0-2
		Mean \pm SD	Mean \pm SD	Mean \pm SD
Treatment	12	-3.5 \pm 9.43	-5.33 \pm 10.58	-8.83 \pm 16.6
Control	12	9.0 \pm 15.56	13.79 \pm 16.46	22.79 \pm 17.24
(t/z, p)		- 2.38 (0.02*) ^a	-3.38 (0.003*) ^a	-4.59 (0.00*) ^a

Explanation:

BW 0-1=BW difference day 0 and day 7th

BW 1-2=BW difference day 7th and day 14th

BW 0-2=BW difference day 0 and day 14th

* Signifikan $p < 0.05$; ^a Independent t test; [†] Mann Whitney test

Table 1: Change in body weight per week (n=12).

Normal daily caffeine consumption was able to cause 3% increase in Total Energy Expenditure (TEE). Caffeine in coffee was like drugs such as amphetamine, ephedrine, and several antidepressants, it was able to stimulate sympathetic nerve system so increases body metabolism [19]. The potential of coffee had been studied in body weight reduction as thermogenic agent. Caffeine mechanism affects thermogenesis by inhibiting phosphodiesterase that stimulates degradation of intracellular cyclic AMP (cAMP) [20].

Uric acid level

Hyperuricemia is very strongly associated with obesity and metabolic syndrome and predicts visceral obesity and insulin resistance. Hyperuricemia has a role in adipokine production. Uric acid that is soluble in water will directly stimulate redox of pro inflammation signal in adipose tissue [21]. Uric acid is one of endogen antioxidant. Nonenzymatic antioxidant that is a scavenger of free radical formed from purine degradation is excreted to extra cellular fluid including the lung, blood and saliva [22]. Hyperuricemia induces endothelium dysfunction and cardiovascular disease [23].

Mean of uric acid level in all obese groups at the start of study was 2.40 ± 0.81 . Mean of uric acid level in all non-obese groups at the start of study was 1.22 ± 0.63 . Mann Whitney test between obese and non-obese group at the start of study showed significant difference ($z = -3.32$; $p < 0.05$).

Mean of uric acid level at the start of study in obese treatment group was 2.45 ± 0.91 and in obese control group was 2.33 ± 0.78 . Mean of uric acid level in both obese group have normal distribution and there are no significant difference in the result of parametric non-paired differential test Independent t test ($t = 0.26$, $p > 0.05$). Mean of uric acid level at the start of study in non-obese treatment group was 1.10 ± 0.18 and in non-obese control group was 1.35 ± 0.9 ; the result of non-paired parametric differential test Independent t test did not show significant difference ($t = -0.67$, $p > 0.05$).

Mean of uric acid level after 14 days of treatment in obese treatment group decreased -0.18 ± 1.11 to 2.09 ± 1.85 and also in control group there was a decrease of -0.80 ± 1.14 to 1.20 ± 0.57 . Mean of uric acid level in non-obese treatment group experienced a decrease of -0.75 ± 1.38 to 1.10 ± 0.44 and control group also experienced a decrease of -0.23 ± 0.84 to 0.88 ± 0.40 . Mean of uric acid in all obese and non-obese group experienced a decrease but it was not significant statistically ($p > 0.05$) (Table 1).

In the Table 2 it appears that mean uric acid level in all treatment group (obese and non obese) at the start of study was 1.78 ± 0.95 . Mean of uric acid level in all control group (obese and non obese) at the start of study was 1.84 ± 0.95 and there was abnormal data distribution, so the requirement for parametric non-paired differential test Independent t test was not fulfilled so we used alternative test with non-parametric Mann Whitney and the result was not significant difference ($z = -0.23$; $p > 0.05$).

Obesity increased uric acid level to become significantly different as compared with non-obese condition. Uric acid level in obese rats decreased more but was not significantly different after coffee was given as compared with non-obese group. Uric acid level in non-obese group decreased not significantly. This finding meant that coffee was able to decrease uric acid level if the value was above normal, although not significantly. If the uric acid level had been stable, then coffee consumption could not decrease uric acid level. Mean of uric acid level in all treatment groups experienced a decrease of -0.18 ± 1.11 from

Group	n	BW 0	BW 1	BW 2
		Mean \pm SD	Mean \pm SD	Mean \pm SD
Treatment	12	236.58 \pm 51.02	233.1 \pm 48.04	227.75 \pm 45.6
Control	12	239.54 \pm 38.49	248.5 \pm 34,1	262.33 \pm 27.75
(t/z, p)		0.16 (0.87) ^a	0.91 (0.4) ^a	-2,24 (0.03) ^a

Explanation:

BW 0=body weight day 0

BW 1=body weight day 7th

BW 2=body weight day 14th

* Signifikan $p < 0.05$; ^a Independent t test; [†] Mann Whitney test

Table 1 and 2 showed that the treatment group showed a decrease in mean body weight. The control group experienced an increase in body weight every week. The treatment group and control group were in greatest difference in the last day (day-14) after study ($t = -2.24$; $p < 0.05$). The difference in body weight decrease was bigger in treatment group. The difference in body weight increase became bigger with study time in control group. Mean of body weight difference during 14 days, differed significantly ($t = -4.59$; $p = 0.00$) between treatment group ($n = 12$) and control group ($n = 12$).

Table 2: Difference in body weight per week ($n = 12$).

1.78 ± 0.95 to 1.60 ± 1.38 . The entire control group also experienced a decrease of -0.80 ± 1.14 from 1.84 ± 0.95 to 1.04 ± 0.50 . Mean of uric acid level in all obese group experienced a decrease of -0.75 ± 1.38 , from 2.40 ± 0.81 to 1.65 ± 1.39 and in all non-obese group experienced a decrease of -0.23 ± 0.84 from 1.22 ± 0.63 to 0.99 ± 0.42 . Uric acid level decreased after coffee drink consumption and standard diet were provided but the decrease was not significant ($p > 0.05$).

The summary of result in Table 2 above showed that there were no significant difference in body weight, uric acid level in treatment and control group in the beginning of study ($p > 0.05$), this meant that both treatment group and control group were in condition of no difference. But there was significant difference on body weight in treatment and control in the end of study ($t = -2.24$; $p < 0.05$), also significant difference in all control group and treatment group ($t = -4.26$; $p < 0.05$). This meant that at the start of study the body weight in the entire treatment and control group was not different, but coffee consumption was able to decrease body weight in treatment group because in all treatment group that consumed coffee had lower body weight (227.50 ± 45.89) compared with control group that only drank aqua water (262.33 ± 27.75).

Uric acid level at the start of study between control and treatment group ($z = -0.23$; $p > 0.05$) was not different significantly, but there was significant difference in uric acid level in control group between the start and the end of study ($t = 2.41$; $p < 0.05$)[§]. This meant that consumption of aqua water was more able to decrease uric acid level so differed significantly than coffee consumption because there was no significant difference in uric acid level between the start and the end of study in treatment group that consuming coffee ($z = -0.86$; $p > 0.05$) although the decrease of uric acid level also occurred in treatment group. In entirety, there was no significant difference between control and treatment group ($t = 1.33$; $p > 0.5$). This meant that coffee consumption was not different with aqua water consumption in influencing the difference of decrease in uric acid level, but aqua water consumption was better in influencing uric acid level decrease because the difference was significant as compared with consuming coffee that decreased uric acid level but the difference was not significant.

Discussion

Influence of coffee consumption on body weight of rats

Body weight reduction is a way for individual with obesity to improve body composition with the purpose to increase immune response so reducing the risk of early mortality and the occurrence of chronic disease as a result of obesity. A person with obesity has

hormone and enzyme that is different with person with normal body weight in controlling muscular growth and fat accumulation, so would effectively help controlling hormone naturally and positive change in body composition that simultaneously would be able to improve health in long term [24].

Table of differential test for mean body weight difference in obese treatment group at the start of study and at the end of study showed significant difference ($p < 0.05$). This meant that by consuming coffee drink for 14 days then obese mice only experience mean body weight increase of 2.83; whereas control group that did not consuming coffee drink experienced mean body weight increase of 20.83. So, coffee consumption and standard diet would able to stabilize body weight, than consuming standard diet only that unable to stabilize body weight.

Decrease of body weight occurred because coffee was a kind of drink with low glycemic index. This was supported by a statement that the amount of daily carbohydrate consumed did not affect BMI but the type of consumed carbohydrate [16,17] would affect BMI.

Uric acid level

Table 3 on the result of differential test of uric acid showed uric acid level of obese mice before treatment/at the start of study significantly higher than non-obese uric acid level ($p < 0.05$). Excessive adipose tissue accumulation would increase the secretion of pro inflammation product so uric acid level would increase. The increase of uric acid level or hyperuricemia was strongly associated with accumulation of visceral fat tissue [25].

This was different with non-obese condition that without adipose tissue accumulation. The difference in uric acid level of obese and non-obese mice in this study was in line with the previous study that said that BMI and eating behavior pattern such as behavior in consuming food and beverage would significantly affect the increase in uric acid level, but age and sex of adult person did not significantly affect uric acid level [26]. Other study gave different statement i.e., hyperuricemia was strongly associated with sex. The risk of hyperuricemia in male increased 5.19 times than in female [27]. The increase of uric acid would also activate renin angiotensin systemically, then this increased sodium resorption and influencing direct cellular effect (vascular smooth muscle cell/VSMC and endothelial cell) so caused the occurrence of hypertension and renal microvascular disease. So reduction of uric acid

would reduce hypertension. Uric acid reduction was also influenced by low salt diet and low purine diet [22].

Result of study also showed that mean of obese uric acid level at the start of study was higher than obese uric acid level at the end of study although the difference was not significant ($p > 0.05$). Decrease of uric acid level occurred in treatment group that was given coffee as well as control group, this meant that giving coffee of 0.72 ml and standard diet consumption for 14 days did not increase uric acid level as compared with clean water consumption and standard diet. This was in accordance with previous study that coffee consumption of 0.72 ml for 7 days could reduce uric acid level of 15% in rats with hyperuricemia [10]. Coffee consumption was associated with reduction of hyperuricemia prevalence as compared with person that did not consume coffee [28,29].

Uric acid in normal level was an antioxidant that prevented stress that induced cell transformation and oxidant that stimulated cardiovascular disease and renal toxicity. Uric acid could become prooxidant that would increase free radicals in the circulation, so would drive lipid oxidation, the result was vascular endothelial dysfunction. Inflammation occurred and also NO production disorder, atherosclerosis, and thrombogenesis. Hyperuricemia (uric acid level > 7 mg/dl in male and > 6 mg/dl in female) was an independent risk factor for all cases of cardiovascular mortality, also patient with hypertension. The risk for the occurrence of cardiovascular disease mortality increased 8-13% for every mg/dl of increase in uric acid level [27]. The consensus on uric acid level with the characteristic as protective factor was not known yet, but acute increase in uric acid level was a protective factor, whereas chronic increase in uric acid level was a risk for disease [30].

The limitation and advantage of study

This study evaluated uric acid level, but did not make comparison with medication so we did not know the effectivity of drug therapy to reduce uric acid level if this was performed for 14 days. This was a limitation of this study but it had been based on consideration that the researcher wanted to use natural coffee drink consumption of 0.72 ml (equivalent with 2 cups of coffee per day in human) for 14 days. In addition, with the proved benefit of consuming coffee drink of 0.72 ml per day, this was a proof that coffee consumption of 0.72 ml per day for 14 days was good enough in supporting body health. Result of observation during study also showed that consuming 0.72 ml or

Variables	Group	N	Mean \pm SD		Differential test start and end t/z (p)	Differential test all treatment and control t/z (p)
			Start	End		
Body Weight	Treatment	12	236.58 \pm 5 1.02	227.50 \pm 45.89	1.85 (0.09) [§]	-4.26 (0.00*) [¶]
	Control	12	239.54 \pm 38.49	262.33 \pm 27.75	-4.61 (0.001*) [§]	
	t/z (p)		-0.16 (0.87) [‡]	-2.245 (0.03*) [‡]		
Uric Acid	Treatment	12	1.78 \pm 0.95	1.60 \pm 1.38	-0.86 (0.39) [‡]	1.33 (0.19) [¶]
	Control	12	1.84 \pm 0.95	1.04 \pm 0.50	2.41 (0.03*) [§]	
	t/z (p)		-0.23 (0.82) [¶]	-1.36 (0.17) [¶]		

Explanation: * Significant: $p < 0.05$; § Paired t-test; ‡ Wilcoxon test; ¶ Independent t test; ¶ Mann Whitney test

The summary of result in Table 3 above showed that there were no significant difference in body weight, uric acid level in treatment and control group in the beginning of study ($p > 0.05$). It means that both treatment group and control group were in condition of no difference. There was significant difference on body weight in treatment and control in the end of study ($t = -2.24$; $p < 0.05$)[¶], also significant difference in all control group and treatment group ($t = -4.26$; $p < 0.05$)[¶]. It means that at the start of study the body weight in the entire treatment and control group was not different, but coffee consumption was able to decrease body weight in treatment group because in all treatment group that consumed coffee had lower body weight (227.50 \pm 45.89) compared with control group that only drank clean water (262.33 \pm 27.75).

Uric acid level at the start of study between control and treatment group ($z = -0.23$; $p > 0.05$) was not different significantly, but there was significant difference in uric acid level in control group between the start and the end of study ($t = 2.41$; $p < 0.05$)[§]. It means that consumption of aqua water was more able to decrease uric acid level so differed significantly than coffee consumption because there was no significant difference in uric acid level between the start and the end of study in treatment group that consuming coffee ($z = -0.86$; $p > 0.05$)[‡] although the decrease of uric acid level also occurred in treatment group. In entirety, there was no significant difference between control and treatment group ($t = 1.33$; $p > 0.5$). It means that coffee consumption was not different with aqua water consumption in influencing the difference of decrease in uric acid level. Aqua water consumption was better in influencing uric acid level decrease because the difference was significant as compared with consuming coffee that decreased uric acid level but the difference was not significant.

Table 3: Summary of body weight and uric acid level from group and treatment.

the equivalent of 2 cups per day in human did not give side-effect in the form of sleep disorder in experimental animal moreover if it was not consumed before sleep time and continued consuming clean water. Therefore in this study, the researcher did not make comparison with standard drugs and only made comparison with clean water consumption.

The advantage of this study in comparison with other study were (1) True experimental study design. (2) Obese rats were compared with non-obese rats. (3) This study analyzed parameter based on treatment group that was given coffee drink and control group that was only given clean water and standard diet. Experimental animal with obesity was chosen because there was accumulation of adipose tissue. Excessive accumulation of adipose tissue would affect inflammation response. Therefore, we needed to know the difference in inflammation response between excessive and not excessive accumulation of adipose tissue. (4) Dose of 0.72 ml/day was equivalent with 2 cups per day in human and this was found from previous study. (5) Duration of study i.e., 14 days was chosen because the result of study from previous researcher showed that consuming 0.72 ml of coffee for 7 days was able to decrease uric acid level 15% in mice with hyperuricemia. This was the advantage and the difference of this study with the previous study.

Influence of coffee drink on uric acid level

The result of this study showed decrease of uric acid level in obese mice although not significant statistically ($p > 0.05$). Previous study observed that coffee consumption was proved as having protective effect against hyperuricemia [29]. Previous study also showed result that coffee consumption of 0.72 ml per day for 7 days was able to decrease uric acid level as much as 15% in mice with hyperuricemia [10].

Conclusion

Drinking 0.72 ml coffee daily (equal with 2 cup of coffee in human) for 14 days could decreased uric acid level in obese rats model although not significant. Coffee has beneficial effects as antiobesity. Coffee has prevention action against metabolic syndrome disease.

References

1. Ford E, Giles W, Dietz W (2002) Prevalence of the metabolic syndrome among Adults: findings from the third National Health and Nutrition Examination Survey. *JAMA* 16: 356-359.
2. Cortez M, Carmo LS, Rogero MM, Borelli P, Fock RA, et al. (2013) A high fat diet Increases IL-1, IL-6 and TNF- α production by increasing NFkB and attenuating PPAR gamma expression in bone marrow mesenchymal stem cells (MSCs). *Inflammation* 36: 379-386.
3. Nix S (2009) William's basic nutrition and diet therapy (13th edn). Elsevier Inc India.
4. Oberbach A, Neuhaus J, Schlichting N, Kugler J, Bavmann S, et al. (2013) Sleeve gastrectomy reduces xanthin oxidase and uric acid in a fat model of morbid obesity. *Surg Obes Relat Dis* 10: 684-690.
5. Soltani Z, Rasheed K, Kapusta DR, Reisin E (2013) Potential role of uric acid in metabolic syndrome, hypertension, kidney injury and cardiovascular disease: is it time for reappraisal? *Curr Hypertens Rep* 15: 175-181.
6. Lu W, Xu Y, Shao X, Gao F, Li Y, et al. (2015) Uric acid produces an inflammatory response through activation of NF- κ B in the hypothalamus: implications for the pathogenesis of metabolic disorders. *Sci Rep* 5: 12144.
7. Mahan LK, Stump SE, Raymond JL (2012) Krause's food and the nutrition care process (13th edn). Elsevier Inc USA.
8. Kempf K, Herder C, Erlund I, Kolb H, Martin S, et al. (2010) Effect of coffee consumption on subclinical inflammation and other risk factor type 2 DM: a clinical trial. *Am J Clin Nutr* 91: 950-957.
9. Lelyana R, Cahyono B (2015) Total phenolic acid content in some commercial brands of coffee from Indonesia. *J Med Plant Herb Ther Res* 3: 27-29.
10. Lelyana R, Wijayahadi N, Kusmiyati DK, Puwanto AP, Hardian, et al. (2015) The influence of coffee consumption to decrease uric acid level an experimental study on hyperuricemia wistar strain rats. *International Journal of Current Research* 7: 19147-19153.
11. Van Dam RM, Scaidell JC (2007) Carbohydrate intake and obesity. *Eur J Clin Nutr* 61: S75-79.
12. Bell RR, Spencer MJ, Sherriff JL (1995) Diet induced obesity in mice can be treated without energy restriction using exercise and/or low fat diet. *J Nutr* 125: 2356-2363.
13. Panchal SK, Poudyal H, Iyer A, Nazer R, Alam A, et al. (2011) High carbohydrate diet high fat diet-induced metabolic syndrome and cardiovascular remodelling in rats. *J Cardiovasc Pharmacol* 57: 51-64.
14. Goto A, Song Y, Chen BH, Manson JE, Buring JE, et al. (2011) Coffee and caffeine consumption in relation to sex hormone-binding globulin and risk of type 2 diabetes in postmenopausal women. *Diabetes* 60: 269-275.
15. Yesil A, Yilmaz Y (2013) Review article: Coffee consumption, the metabolic syndrome and non alcoholic fatty liver disease. *Aliment Pharmacol Ther* 38: 1038-1044.
16. Ma Y, Olendzki B, Chiriboga D, Hebert JR, Li Y, et al. (2005) Association between dietary carbohydrates and body weight. *Am J Epidemiol* 161: 359-367.
17. <http://articles.mercola.com/sites/articles/archive/2005/03/05/obesity-carbs.aspx>
18. Lelyana R (2014) The Role of coffee's content for preventing diabetes mellitus risk factor incidence. Proceeding and Abstract Book Presented at 9th Symposium on Nutri Indonesia: Breaking the Boundaries to Optimize the Benefits for Patients. Indonesia University, Jakarta.
19. Ross AC, Caballero B, Cousins RJ, Tucker KL, Ziegler TR (2014) Modern nutrition in health and disease (11th edn). Wolters Kluwer, Lippicott Williams and Wilkins, Philadelphia. pp: 477-484.
20. Diepvens K, Westerterp KR, Westerterp-Plantenga MS (2007) Obesity and thermogenesis related to the consumption of caffeine, ephedrine, capsaicin and green tea. *Am J Physiol Regul Integr Comp Physiol* 292: R77-85.
21. Baldwin W, McRae S, Marek G, Wymier D, Baylis C, et al. (2009) Hyperuricemia as a mediator of the proinflammatory endocrine imbalance in the adipose tissue in a murine model of the metabolism syndrome. *Diabetes* 60: 1258-1269.
22. Hediger MA, Johnson RJ, Miyazaki H, Endou H (2005) Molecular physiology of urate transport. *Physiology (Bethesda)* 20: 125-133.
23. Balakumar P, Sharma R, Kalia AN, Singh M (2009) Hyperuricemia: is it a risk factor for vascular endothelial dysfunction and associated cardiovascular disorders? *Current Hypertension Reviews* 5: 1-6.
24. Busch A (2014) How to control fat accumulation.
25. Tshusima Y, Nishizawa H, Tochino Y, Nakatsuji H, Sekimoto R, et al. (2013) Uric acid secretion from adipose tissue and its increase in obesity. *J Biol Chem* 288: 27138-27149.
26. Yadav BK, Chhetri GB, Poudel B, Sigdel M, Gyawali P, et al. (2009) Serum uric acid level in obese and non-obese individuals. *Journal of Nepal Association for Medical Laboratory Sciences* 10: 27-30.
27. Chen JH, Chuang SY, Chen HJ, Yeh WT, Pan WH (2009) Serum uric acid level as an independent risk factor for all-cause, cardiovascular, and ischemic stroke mortality: a chinese cohort study. *Arthritis Rheum* 61: 225-232.
28. Choi HK, Curhan G (2007) Coffee, tea and caffeine consumption and serum uric acid level: the third national health and nutrition examination survey. *Arthritis Rheum* 57: 816-821.
29. Pham NM, Yoshida D, Morita M, Yin G, Toyomura K, et al. (2010) The relation of coffee consumption to serum uric acid in Japanese men and women aged 49-76 years. *J Nutrition Metab* 2010: 930757.
30. Oliveira EP, Burini RC (2012) High plasma uric acid concentration: causes and consequences. *Diabetol Metab Syndr* 4: 12.