RESEARCH ARTICLE

Metabolic response against traffic congestion-induced stress

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Abstract

Background: Traffic congestion is a common problem in large cities, which can induce stress in participants in vehicles as well as pedestrians. In such conditions, several stress hormones, such as catecholamines and cortisol are secreted by the adrenal glands resulting in metabolic responses. Prolonged metabolic changes can lead to metabolic diseases, such as metabolic syndrome and cardiovascular disease. This study aimed to examine the metabolic responses induced by traffic congestion in young adults.

Methods: We enrolled 71 undergraduate students aged 20-30 years from the Faculty of Medicine Universitas Islam Bandung, who regularly go to campus on foot, by public transportation, motorcycles, and cars. Medical history was screened by questionnaires before the testing day. Blood samples were drawn soon after participants arrived on campus. We used bioimpedance analysis to measure fasting blood glucose, lipid profiles, and nutrient body status.

Results: Most participants rode motorcycles (46.48%), and the most common travel time was short (<15 min, 54.9%). Total cholesterol and low-density lipoprotein (LDL) levels were significantly lower in participants using vehicles - either private or public transportation - compared with pedestrians ($p < 0.05$ and $p < 0.02$, respectively). Participants with a short travel duration had significantly higher total cholesterol and LDL levels than those with longer ones ($p < 0.02$ and $p < 0.05$, respectively).

Conclusions: Based on the above results, metabolic responses to traffic congestion such as changes to total cholesterol and LDL levels depended the type of transportation and travel duration.

Keywords

Metabolic responses, Stress, Traffic congestion, Travel duration, Transportation type

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Introduction

A problematic issue in big cities is traffic congestion, which can induce stress in traffic users.¹ Bandung, Indonesia, is ranked 14th on the list of 278 Asian cities with traffic congestion. Until 2013, the number of private four-wheeled vehicles in Bandung was 318,598, and that of two-wheeled ones 1,030,279 (Badan Pusat Statistik Kota Bandung, 2015).²

Stressors can induce both positive and negative responses, determined by a balance between tolerance and sensitivity. Stressors can be helpful; for example, they can motivate the body to produce the energy to get through challenging situations such as exams or job tasks. However, stressors in extreme amounts and over long periods can cause chronic stress affecting the immune system, cardiovascular, neuroendocrine, and central nervous systems.^{3,4}

Stress induces several metabolic responses in the body; for example, the hypothalamus transmits signals directly through the sympathetic nervous system to the adrenal glands, resulting in the production of catecholamines in the adrenal medulla, including epinephrine, which increases the heart rate and respiration.⁵ In addition, norepinephrine, stimulates liver cells to release glucose for cellular respiration.⁵ In contrast, hormones secreted by the adrenal cortex signal a chronic response to stress. In such a case, under stress stimulation, the hypothalamus causes the anterior pituitary to secrete the adrenocorticotrophic hormone (ACTH), which induces cells in the adrenal cortex to produce and secrete corticosteroids, such as glucocorticoids, to break down fat and stimulate glucose synthesis. Cortisol is an example of glucocorticoid involved in this stress response, and regulates or supports various cardiovascular, metabolic, immunological, and homeostatic processes.³

Tsigos (2019) suggested that chronic stress is associated with hypercortisolemia and sympathetic system activation, resulting in metabolic changes. For example, visceral fat accumulation contributes to visceral obesity, type 2 diabetes mellitus (DM-2), and associated cardiometabolic complications.^{6,7} Currently, the prevalence of DM-2 in adults is approximately 6.4% of the world population and will continue to increase to 7.7% of the population by 2030.⁸ The prevalence of DM in urban areas in Indonesia is approximately 5.7%, and the incidence of this disease at young age tends to increase.⁹ This increase deserves particular attention, considering their productive role in society.⁹ However, studies on the effect of chronic stress due to traffic congestion on the metabolism at young age are still limited.

In this study, we investigated whether stress whilst driving would affect metabolic responses by examining several metabolic parameters such as fasting blood glucose, lipid profiles, and nutrient body status in young adult traffic users, which were active students at UNISBA Faculty of Medicine.

Methods

Ethical considerations

The ethics committee of the university approved this study with the number: 003/KEPK-FK/XI/2017. Interviews were in the UNISBA Faculty of Medicine area, after written consent was obtained from the subjects following an explanation about the study, without any coercion.

Study design and setting

This was a cross-sectional study to determine the correlation between driving stress caused by different types of transportation and travel duration, and lipid profile and glucose. The study was conducted from September 2017 to February 2018 on data from second-year and third-year students at UNISBA Faculty of Medicine, Bandung-Indonesia. Participants were approached by visiting classroom and offering participation in this study. Then, interviews for recruitment were conducted in the UNISBA Faculty of Medicine area after participants provided informed consent. The study materials including participant information sheet and questionnaire can be found as *Extended data*.^{19,20} We collected blood serum samples in the 7th-floor laboratory of the UNISBA Faculty of Medicine.

We followed the STROBE cross-sectional reporting guidelines.¹⁰ Restriction methods (by applying inclusion and exclusion criteria) were applied to variables that biologically possible to compromise the outcome variable, such as metabolic diseases, cholesterol-affecting medications, pancreatic disorders, and smoking-alcohol drinking habits. Valid variable measurements explained later on this section.

Participants

The minimum sample size in this study was 55 people, and calculated using the following correlation test formula:

$$n = \left[\frac{Z\alpha + Z\beta}{0.5 \ln \left[\frac{(1+r)}{(1-r)} \right]} \right]^2 + 3$$

With $Z\alpha$: 1.96 (with α : 0.05), $Z\beta$: 0.842 (with β : 0.2), r : 0.37 (obtained from previous research), N : 55.

To anticipate sample dropouts, the number of samples in this study was 10% of the minimum sample size (60.5) rounded up to 62 people.

We used consecutive sampling to obtain the 71 participants among students in the UNISBA Faculty of Medicine. After conducted interview for recruitment, the selected subjects fulfilled the following inclusion criteria: being active students (active in academic activity during the study period) and having performed academic activities for >1 year (this was to prevent sampling bias due to student adaptation to the campus environment). The exclusion criteria were: taking drugs which can interfere with the results, such as steroids, diazoxide, diuretics, and phenytoin, being active smokers or consuming alcohol, a history of DM-2 or cardiovascular disease, dyslipidemia, obesity, or pancreas abnormalities, family history of DM-2, cardiovascular disease, and dyslipidemia.

Measurement of travel duration

In this study, the travel duration was the time participants needed to go from their residence to campus as measured by themselves using a stopwatch application in their own mobile phone. Subjects were requested to use their usual transportation method to campus. Travel duration data were collected twice on different days within a week before blood sampling collection. As evidence, we asked the participants to share the location twice, namely, when they left the residence and reached the campus lobby. They shared their location and the measurement of their travel duration immediately with the researchers via text messages.

Blood sampling

The participants fasted for 8-10 hours (fasting started 10 PM, night before the blood collection), then blood collection was conducted at 6-8 AM). First, we obtained venous blood 3-5 cc (venipuncture) from the median cubital vein, venous cephalic, or basilica vein. Then, the blood serum was centrifuged (ZENTRIFUGE EBA 200 HETTICH, Tuttlingen, Germany) at $1500 \times g$ for 15 min at 4°C to separate the serum, which was stored at 80°C until further use.

Measurement of body composition

We measured body composition by bioimpedance analysis (BIA) using Omron HBF-375 (Omron Healthcare, Kyoto, Japan). The procedure and normal reference values followed the manufacturer's protocol. Body fat percentage measurement = (body fat mass [kg] / body weight [kg]) $\times$ 100, skeletal muscle ratio = (skeletal muscle mass [kg] / body weight [kg]) $\times$ 100. As hydration status is essential for accurate body composition assessment using BIA.¹¹ The participants were asked to avoid alcohol the night before the test and not exercise on the day before the test.

Measurement of blood parameters

Blood glucose levels were measured using a glutes sensor (Sanwa Kagaku, Aichi, Japan). In addition, we measured serum triglyceride levels (Triglyceride E-test, Wako Chemical, Osaka), total cholesterol, low-density lipoprotein (LDL), and high-density lipoprotein (HDL) (NEFA C-test Wako Chemical, Osaka) following manufacturer's protocols.

Statistical analysis

We conducted univariate, bivariate, and multivariate data analysis by STATA/MP 16. First, we used univariate analysis to describe the frequency, distribution, and percentage of each variable in the study and specific measures of average, median, standard deviation, and the maximum and minimum values. Next, we performed bivariate analysis using t-test and ANOVA test followed by Dunnett's post hoc multiple comparison tests to determine the relationship between variables, and Pearson's correlation test to determine the power of the correlation if the data obtained had a normal distribution,^{12,13} with $p < 0.05$ considered as statistically significant. Finally, a logistic regression test was used for the multivariate analysis of the relationship between the variables.

Results

Participant characteristics

Table 1 presents the characteristics of the participants. The total number of participants in the study was 71.¹⁸ Most participants were female, with a mean age of 21 years, and the average body mass index (BMI) was normal. In addition, the participants' average fasting glucose and lipid profiles were normal, but 63.4% of participants had pre-diabetic fasting glucose levels and 12.7% at a diabetic level (Table 2).

Before univariate analysis, the Shapiro–Wilk normality test was conducted. All data had a normal distribution, except for travel duration, HDL, and triglyceride levels. In the variable duration of travel, data transformation to obtain normal data distribution could not be performed, so we converted this variable into a categorical/ordinal variable with the following

Table 1. Participant characteristics.

Category	Frequency (%)	Mean $\pm$ SD	Median (range)
Gender			
Man	29 (40.9%)		
Woman	42 (59.1%)		
Age (years)			21 (20-23)
Level			
Level 3	25 (35.2%)		
Level 4	46 (64.8%)		
Bodyweight (kg)		56.1 $\pm$ 9.1	
Height (cm)		160.6 $\pm$ 8.9	
BMI (kg/m ²)		21.66 $\pm$ 2.41	
Parameter serum			
Fasting glucose (mg/dL)		107.5 $\pm$ 14.8	
Total cholesterol (mg/dL)		168.2 $\pm$ 32.6	
HDL (mg/dL)			55.7 (27.6 – 91.9)
LDL (mg/dL)		91.0 $\pm$ 36.1	
Triglyceride (mg/dL)			107.6 (38.8 – 230.9)

Note. n total participants = 71, SD: Standard deviation.

Abbreviations: BMI, Body mass index; HDL, High density lipoprotein; LDL, Low density lipoprotein.

categories: 1. Short duration (<15 min); 2. Medium duration (15–30 min); 3. Long duration (> 30 min). We performed data transformation using the Log function to conduct a parametric analysis of HDL and triglyceride levels.

Table 2 shows that most respondents had a short travel duration (54.9%), and that motorcycle was the most used type of transportation in this study (46.48%). At the beginning of the research design, we divided public transportation into 2-wheeled and 4-wheeled public vehicles. However, in the study, the participants who used 2-wheeled public vehicles were very few (1 participant), so the categories were combined into general public transportation users. Next, despite the broad range of travel duration in participants using the same means of transportation, the median travel duration was 11 minutes.

Table 3 shows that most respondents had a normal body fat percentage, visceral fat, and skeletal muscle ratio. The mean body fat percentage was $24.4 \pm 7.39\%$, while the mean values of visceral fat and skeletal muscle ratio were 3.5 (0.5-13) and 26.8. There were no significant differences between the types of transportation and duration of travel with the three parameters. Next, we measured the proportion of high and very high body fat percentages. The highest body fat percentage was observed in private car users (46.67%), although the difference was not significant (**Table 4**).

Next, we conducted a comparative bivariate analysis and Bonferroni's post hoc test. The results showed a significant relationship between the type of transportation and travel duration with total cholesterol ($p = 0.0492$ and 0.0214) and LDL levels ($p = 0.0197$ and 0.0494 , respectively). However, there was no significant relationship between the two variables and other risk parameters, such as fasting glucose, HDL, and triglycerides (**Table 5**). Respondents with a short travel duration had higher total cholesterol and LDL levels than those with a long travel duration (177.1 vs. 152.4 mg/dL $p = 0.016$, 99.6 vs. 75.5 mg/dL $p = 0.049$, respectively). In addition, we found a significant difference between respondents who walked and those who used vehicles; pedestrians had higher levels of total cholesterol and LDL than other transportation users ($p = 0.049$ and $p = 0.021$, respectively).

Table 6 shows the correlation between variables using the Spearman rank test. There was a statistically significant correlation between duration and type of transportation with total cholesterol and LDL levels. The correlation coefficients for both travel duration and transportation type were negative, which means that the longer the trip, the lower the total cholesterol and LDL levels. However, the strength of the correlation between these variables was low (0.20-0.39).

Table 2. Obtained parameters.

Parameters	N	%	Median (Range)
Travel duration			
Short (<15 minutes)	39	54.9	
Medium (15 – 30 minutes)	12	16.9	
Long (>30 minutes)	20	28.2	
Type of transportation			
Pedestrian	15	21.13	
Private motorcycle	33	46.48	
Private car	15	21.13	
Public transportation	8	11.27	
Travel duration (Range in minutes)			
Pedestrian	15		5 (4 – 9)
Private motorcycle	33		14 (3-58)
Private car	15		36 (8 – 100)
Public transportation	8		6 (4 – 66)
Total of travel duration (Range in minutes)	71		11 (3 – 100)
Fasting glucose			
Normal	17	23.9	
Pre-diabetic	45	63.4	
Diabetes	9	12.7	
Total cholesterol			
Normal	62	87.3	
Borderline	8	11.3	
High	1	0.01	
HDL			
Normal	41	57.8	
Low	5	7	
High	25	35.2	
LDL			
Optimal	44	62.9	
Almost optimal	18	25.7	
Borderline	6	8.6	
High	1	1.4	
Very High	1	1.4	
Triglyceride			
Normal	63	88.7	
Borderline	7	9.9	
High	1	1.4	
Total	71	100	

Abbreviations: BMI, Body mass index; HDL, High density lipoprotein; LDL, Low density lipoprotein.

Table 3. Body fat, visceral fat, and skeletal muscle ratio.

Parameter	Body fat percentage					Visceral fat				Skeletal muscle ratio			
	Low	Normal	High	Really high	Total	Low	Normal	High	Total	Low	Normal	High	Total
Type of transportation													
Pedestrian (n)	1	8	5	1	15	1	13	1	15	1	13	1	15
(%)	6.67	53.33	33.33	6.67	100	6.67	86.67	6.67	100	6.67	86.67	6.67	100
Private motorcycle (n)	1	22	8	2	33	8	25	0	33	8	25	0	33
(%)	3.03	66.67	24.24	6.06	100	24.24	75.76	0	100	24.24	75.76	0	100
Private car (n)	1	7	6	1	15	1	14	0	15	1	14	0	15
(%)	6.67	46.67	40	6.67	100	6.67	93.33	0	100	6.67	93.33	0	100
Public transportation (n)	0	6	1	1	8	2	6	0	8	2	6	0	8
(%)	0	75	12.5	12.5	100	25	75	0	100	25	75	0	100
Total (n)	3	43	20	5	71	12	58	1	71	12	58	1	71
(%)	4.23	60.56	28.17	7.04	100	16.9	81.69	1.41	100	16.9	81.69	1.41	100
Travel duration													
Short (n)	2	25	11	1	39	4	34	1	39	4	34	1	39
(%)	5.13	64.1	28.21	2.56	100	10.26	87.18	2.56	100	10.26	87.18	2.56	100
Medium (n)	1	6	3	2	12	4	8	0	12	4	8	0	12
(%)	8.33	50	25	16.67	100	33.33	66.67	0	100	33.33	66.67	0	100
Long (n)	0	12	6	2	20	4	16	0	20	4	16	0	20
(%)	0	60	30	10	100	20	80	0	100	20	80	0	100
Total (n)	3	43	20	5	71	12	58	1	71	12	58	1	71
(%)	4.23	60.56	28.17	7.04	100	16.9	81.69	1.41	100	16.9	81.69	1.41	100

Table 4. Differences in high and very high body fat percentages.

Parameters	%	P value
Type of transportation		>0.05
Pedestrian	40	
Private motorcycle	30.3	
Private car	46.67	
Public transportation	25	
Travel duration		
Short	5	
Medium	8	
Long	25	
Total	100	

Fisher's Exact test.

The adequacy of the sample size for each metabolic parameter between travel duration and type of transportation was analyzed based on their correlation coefficient properties. The number of samples in the fasting glucose and HDL groups was inadequate for both travel duration and type of transportation classification (Table 7), as was the triglyceride sample for the type of transportation.

Discussion

According to Jovanović *et al.* (2008), the driver's serum glucose, total cholesterol, LDL cholesterol, and triacylglycerol concentrations increase according to the work stress index.¹⁴ However, research identifying the metabolic responses to driving stress, primarily due to traffic congestion, is limited. The current study showed a correlation between travel duration and type of transportation with total cholesterol and LDL levels. Pedestrians showed the highest total cholesterol and LDL compared with other groups. In travel duration, the shortest travel duration was associated with the highest total cholesterol and LDL cholesterol levels. In the group with the shortest travel duration, >50% were pedestrians. The reason for this finding might be that during physical activity, substrate reserves are released to accommodate energetic needs. The primary sources of energy substrates are glucose and fatty acids. When needed, fatty acids can be obtained from both nutrients and adipose tissue reserves (triglycerides) by lipolysis. Triglycerides, an LDL content, are distributed to body tissues, including the skeletal muscle.¹⁵ Although we found no difference in triglycerides, it may be because the triglycerides in serum were found in the LDL fraction (LDLs consist of cholesterol, cholesterol esters, triglycerides, and phospholipids).

We also found that >50% of participants were pre-diabetic with a fasting glucose level >10% in the classification of diabetes. In addition, many risk factors influence DM-2. One of these is a modifiable behavioral factor. Therefore, it is essential to find pre-diabetes at a young age, because at this age, the average insulin sensitivity is still good; therefore, behavioral factors can be modified as soon as possible, such as diet, physical activity, and stress, to reduce the risk of DM-2.¹⁶

Traffic congestion can cause stress. We suspected that external stress stimuli cause increasing metabolic stress at the cellular level and activate the hypothalamic-pituitary-adrenocortical (HPA) axis, which subsequently causes insulin resistance.⁶ Several studies have suggested that cortisol, as a product of the HPA axis, plays a role in the processes associated with DM-2.^{3,17} Nevertheless, this study showed no significant differences in fasting glucose levels associated with vehicle type and travel duration.

The limitations of this study are that we only focused on the metabolic responses of lipids and glucose. Furthermore, traffic congestion is a physiological stressor with different effects among people; some people may feel stress when they face this condition, while others may not because they face traffic congestion every day for instance. In addition, subjects in this study were not randomly pooled from the general population hence the results of this study only applicable to this group only.

Table 5. Relationship between travel duration and transportation type with fasting glucose, total cholesterol, HDL, LDL, and triglyceride levels.

Parameter	Fasting glucose (SD)		Total cholesterol		HDL		LDL (SD)		Triglyceride	
	Mean $\pm$ SD	P value	Mean $\pm$ SD	P value	Range	P value	Mean $\pm$ SD	P value	Range	P value
All participants	107.5 $\pm$ 14.8		168.2 $\pm$ 32.6		53.7 (27.6-91.9)		91.0 $\pm$ 36.1		103.3 (38.8-230.9)	
Type of transportation										
Pedestrian	104.1 $\pm$ 17.3	0.7347	185.3 $\pm$ 29.6	0.049*	53.8 (27.6-73)	0.4522	109 $\pm$ 31.9	0.021*	100.5 (48.3-182.1)	0.438
Private motorcycle	109.3 $\pm$ 16.4		169.3 $\pm$ 32.7		48.7 (34.7-91.9)		94.2 $\pm$ 36.1		98.2 (38.8-230.9)	
Private car	106.9 $\pm$ 11.2		156.0 $\pm$ 32.4		58.1 (45.4-85.2)		74.9 $\pm$ 35.9		103.3 (65.7-199.9)	
Public transportation	107.3 $\pm$ 7.6		154.5 $\pm$ 27.2		56.9 (40.5-70)		72.7 $\pm$ 27.2		131 (93.7-161.6)	
Travel duration										
Short	109.2 $\pm$ 13.8	0.2917	177.1 $\pm$ 30.8	0.019*	53.3 (27.6-88)	0.9121	99.6 $\pm$ 24.5	0.049*	101.7 (38.8-230.9)	0.915
Medium	101.5 $\pm$ 18.4		165.9 $\pm$ 35.4		50.9 (45.6-91.9)		89.0 $\pm$ 39.2		97.9 (60.5-165.6)	
Long	107.8 $\pm$ 14.1		152.4 $\pm$ 29.6		55.6 (36.2-85.2)		75.5 $\pm$ 33.5		103.8 (52.3-199.9)	

*One way-ANOVA test.

Abbreviations: HDL, High density lipoprotein; LDL, Low density lipoprotein.

Table 6. Correlation between total cholesterol and LDL with travel duration and transportation type.

Parameter	Travel duration		Type of transportation	
	P value	Correlation coefficient(r)	P-value	Correlation coefficient (r)
Fasting glucose	0.399	−0.1016	0.9562	0.0066
Total cholesterol*	0.0118*	−0.2975	0.0076*	−0.3142
HDL	0.8141	0.0284	0.3888	0.1038
LDL*	0.0235*	−0.2687	0.0013*	−0.3744
Triglyceride	0.7995	−0.307	0.1797	0.1611

*Statistically significant difference between groups, *Spearman rank correlation test*.
Abbreviations: HDL, High density lipoprotein; LDL, Low density lipoprotein.

Table 7. Power analysis by correlation coefficient and sample size.

Parameter	Travel duration	Type of transportation
	Power	Power
Fasting glucose	0.13	0.05
Total cholesterol	0.72	0.75
HDL	0.05	0.13
LDL	0.72	0.89
Triglyceride	0.75	0.26

Conclusions

Metabolic responses such as total cholesterol and LDL levels were affected by traffic congestion depending on the type of transportation and travel duration. Our study gives another perspective that traffic condition could affect metabolic dynamic changes. Further study in older respondents and or longer traffic jam condition can be potential to see the possibility of traffic jam affect metabolic changes. Moreover, exploration of chronic stress pathway and other metabolic markers associated with stress, such as cortisol and catecholamine, is necessary to see the correlation between chronic stress and metabolic profile.

Data availability

Underlying data

Figshare: Data Measurement of The Metabolic Response Against Stress Induced Traffic Congestion. <https://doi.org/10.6084/m9.figshare.16529886>.¹⁸

Extended data

Figshare: Questionnaire Correlation of Driving Stress with Fasting Blood Sugar Level and Lipid Profile. <https://doi.org/10.6084/m9.figshare.16959637.v1>.¹⁹

Figshare: Information sheet and consent form of Correlation of Driving Stress with Fasting Blood Sugar Level and Lipid Profile. <https://doi.org/10.6084/m9.figshare.16959649.v1>.²⁰

Data are available under the terms of the [Creative Commons Zero “No rights reserved” data waiver](#) (CC0 1.0 Public domain dedication).

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