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Effect of Caffeine Intake on Blood Glucose Levels and Autonomic Nervous System Response

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Abstract: This study was conducted to determine the acute effects of drinking caffeine on blood sugar levels and responses of the autonomic nervous system in healthy people. The findings proved that the consumption of caffeine at 5 mg/kg body weight produces evident and temporary physiological changes in metabolic and autonomic equilibrium. The blood glucose and insulin levels were found to be significantly high after taking caffeine, with the highest level at 60 minutes, and then there was a partial reduction at 120 minutes and the leveled at 60 was as a result of the acute metabolic response to the caffeine intake and compensation by the body. Also, there was a temporary increase in the Homeostasis Model Assessment of Insulin Resistance (HOMA-IR), which implies a short-term decrease in the insulin sensitivity within the range of the peak response. At the cardiovascular level, the consumers of caffeine achieved a significant rise in HR and systolic and diastolic BP. Moreover, an increase was found in the LF/HF ratio calculated by analyzing the heart rate variability, indicating the stimulation of the sympathetic nervous system and its temporary domination over the parasympathetic activity , In contrast, no significant changes in physiology could be observed in the placebo group during the period of the study, thus confirming that the results were directly due to caffeine intake.Collectively these findings suggest that caffeine is a metabolic and autonomic stimulant that produces acute but reversible physiological effects in healthy individuals, with pronounced ability of the body to restore remission of a homeostatic imbalance after the discontinuation of this effect. These results provide an improvement in the knowledge of the short-term effects of caffeine consumption, especially in people who are at greater risk of metabolic or cardiovascular problems, and also note that the possible long-term effects of regular caffeine intake need to be studied in the future.

Keywords: Caffeine , Blood , levels , Insulin.

INTRODUCTION

Caffeine is the most widely used psychoactive substance worldwide in the form of beverages such as coffee, tea and energy drinks, dietary supplements and even in some medications (Regts, J. M., & Ditzler, T. F. 1987). Partly because of the well-established stimulatory effects on alertness, cognitive efficacy and mood, its application has been widespread. In addition to these central nervous system effects, caffeine has major effects on many physiological systems, including cardiovascular, metabolic and autonomic nervous system functions (Shi, Z. 2025). These effects are mediated mainly by caffeine's antagonistic activity on adenosine receptors with resultant enhanced activity of the sympathetic nervous system, enhanced catecholamine secretion and modulation of cardiovascular and metabolic homeostasis (Marcinek, K. *et al.*, 2024).

It has also been reported that, acute caffeine dosing is linked to short-term increases in blood glucose levels and insulin levels which is probably because of slated rises in hepatic glucose synthesis and temporary decreases in insulin sensitivity in peripheral tissues (Emami, M. R. *et al.*, 2019). These metabolic effects specifically apply since they represent interaction between the activity of the autonomic nervous system and energy metabolism, which are key to glucose homeostasis. Heart rate variability (HRV) is a non-invasive tool to assess autonomic balance where an index of the sympathetic and parasympathetic pre-eminence is often defined as the low-frequency to high-frequency (LF/HF) ratio (Khurana, D. 2025). It is shown that acute caffeine intake changes this balance in favor of sympathetic dominance with ramifications on cardiovascular stress and general autonomic control (Hamad, A. K. S. 2024).

Although a lot of studies have been done, it is not consistently reported about the acute effects of caffeine on metabolic and autonomic markers in healthy adults, as it depends on dose, habitual caffeine intake, time of measurements, and characteristics of the participants, such as age, sex, and body mass index (Abbood, H. A., & Salman, H. A. 2024; Abbood, H. A., & Salman, H. A. 2024). It is important to understand these acute effects considering that caffeine is widely consumed and it may have implications on the populations with different metabolic and cardiovascular risk factors (Mohajan, D., & Mohajan, H. K. 2023).

The aim of the present study is to investigate the acute effect of caffeine intake on blood glucose levels, insulin response, insulin resistance (HOMA-IR) and the autonomic nervous system

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(HRV) in healthy adults. Using a randomized, double blind, placebo-controlled study design this research therefore allows for a comprehensive evaluation of both metabolic and neurocardiac responses to caffeine which will provide information on the immediate physiological adaptations to a commonly consumed stimulant, as well as informing safe consumption practice.

MATERIALS AND METHODS

Participant Selection and Preliminary Screening

This research study was carried out in the College of Education for Pure Sciences, Physiology Laboratories of University of Kirkuk, during the year 2025. An announcement was carried within the college and thirty healthy volunteers of both genders and aged between 18 and 40 years were enlisted. All subjects were initially subjected to comprehensive initial screening process which included detailed medical history taking to establish absence of chronic diseases especially diabetes mellitus, cardiovascular diseases and endocrine disorders. Information pertaining to drug use, health related habits and habits of caffeine use was also gathered.

Anthropometric measurements were done including height and body weight using calibrated instruments, and body mass index (BMI) was calculated based on standard formulas as to include only normal weight individuals in the study. Systolic and diastolic blood pressure, resting heartbeat were recorded after participants have been seated quietly for 10 minutes in a tranquil environment in order to verify the cardiovascular stability within normal physiological boundaries (Mohajan, D., & Mohajan, H. K. 2023; Faur, M. J. 2025).

Participant Preparation before the Test Day

All the participants were instructed to follow some specific test guidelines, such as complete abstaining from caffeine containing products for 24-48 hours prior to the experiment, complete abstaining from food for 10-12 hours prior to measurements, complete abstaining from strenuous physical activity over the preceding 24 hours, adequate sleeping of at least seven hours. This preparation to standardize basal blood glucose levels, reduce the undesired activation of the sympathetic nervous system due to physical exertion or lack of sleep and eliminate the remains of caffeine's action, which could be to ensure that physiological conditions are standardized in order to evaluate the acute caffeine responses.

Day of Experiment and "Baseline" Measures.

All experimental procedures were carried out in the morning (8:00 to 11:00 a.m) in a cool room (22–24C^{O}) with very low noise and external stimuli in order to keep out the effects of circadian rhythm and the environment on glucose levels, cortisol release, and activity of autonomic nervous system . Upon arrival, the participants were directed to sit quietly and remain inactive and silent in 10 minutes in order to stabilize their autonomic state. Measurement of baseline was then taken, such as heart rate, systolic and diastolic blood pressure, electrocardiographic recordings of the heart rate variability (HRV), the venous blood sampling of the blood glucose, insulin and cortisol concentrations, which was taken as a reference baseline measurement to be compared against later (Vypasniak, I. *et al.*, 2023).

Caffeine Administration and Absorption Period

Participants were given either caffeine in the dose of 5 mg/kg body weight, dissolved in 200 mL of water, or a placebo drink of 200 mL of water containing no caffeine in a double blind design to minimize the potential of psychological bias (Williamson, C. M. *et al.*, 2018). After taking it, the participants sat quietly for an hour without eating or drinking. Caffeine absorption starts within 15-30 min and peak plasma concentrations occur within 45-60 min making this time frame important for monitoring physiological responses associated with glucose metabolism and autonomic nervous system activity (Sharma, V. K. *et al.*, 2023).

Time-Dependent Measurements After Caffeine Intake

All of the measurements were repeated at 30, 60 and 120 minutes after consumption of the beverage. These included measurements of heart rate, blood pressure, HRV measurements, blood sugar levels and blood sampling from a vein to measure some hormones. This timing allowed for the evaluation of the exact onset of the caffeine effects, peak physiological responses and the recovery phase, therefore providing a more complete evaluation of some of the metabolic and autonomic responses to caffeine intake (Zhang, Y. *et al.*, 2024).

Assessment of Derived Indices

Area Under the Curve (AUC) Blood Glucose

Blood glucose levels were measured at baseline, 30, 60 and 120 minutes after consumption of the caffeine or placebo. These values were used to calculate the area under the curve (AUC) by the

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trapezoidal integration method that represent the overall glucose response during the studied time period which allows comparing the physiological effects of caffeine relative to baseline and placebo conditions (Wang, Y. *et al.*, 2022).

Homeostasis Model Assessment of Insulin Resistance (HOMA-IR)

Fasting blood samples were taken to determine glucose (mg/dL) and insulin (mcU/mL) levels. HOMA-IR was calculated using the equation standard equation:

HOMA-IR = Glucose (mg/dL) × Insulin (µU/mL) / 405

This index provides an estimation of insulin sensitivity, the higher the value, the greater the insulin resistance (Yamamoto, J. M. *et al.*, 2020).

LF/HF Ratio from Heart Rate Variability Analysis

Electrocardiographic recordings have been carried out in the conditions of rest as well as the analysis of the HRV, from special programs was carried out. Low frequency (LF) and high frequency (HF) components were extracted and LF/HF ratio calculated to represent the autonomic balance between the sympathetic (LF) and parasympathetic (HF) activity. Increased LF/HF values are suggestive of sympathetic predominance and decreased values of increased parasympathetic activity (Khan, A. A. *et al.*, 2019)

Statistical Analysis

The SPSS was used to analyze data. ANOVA of repeated measures was employed to determine time-dependent variations between caffeine and placebo sessions, whereas paired t-tests were used to compare them pairwise. The correlation analysis was used in examining the relationship between blood glucose levels and HRV parameters. Mean ± SD were used to represent results, and the statistical significance level was $P \leq 0.05$.

RESULTS

Blood Glucose Levels

Blood glucose levels were determined at four important intervals, which include baseline, 30, 60 and 120 minutes following caffeine or placebo consumption. In Table 1, there was a great rise of glucose levels with the acute intake of caffeine which peaked at 60 minutes and glucose in the placebo group maintained comparatively steady levels (MacKenzie, T. *et al.*, 2007).

Table 1: Mean ± SD of blood glucose levels across time after caffeine vs. placebo (n = 30)

Time (min)	Caffeine (mg/dL)	Placebo (mg/dL)
Baseline	95 ± 5	96 ± 4
30	110 ± 7	97 ± 5
60	120 ± 8	98 ± 5
120	115 ± 7	97 ± 4

According to figure 1, the blood glucose levels have become significantly higher after caffeine and attains the peak at 60 minutes and levels off partially at 120 minutes. According to this acute rise, this research is in line with the previous study that demonstrated temporary reduced insulin sensitivity and sympathetic stimulation after caffeine intake .

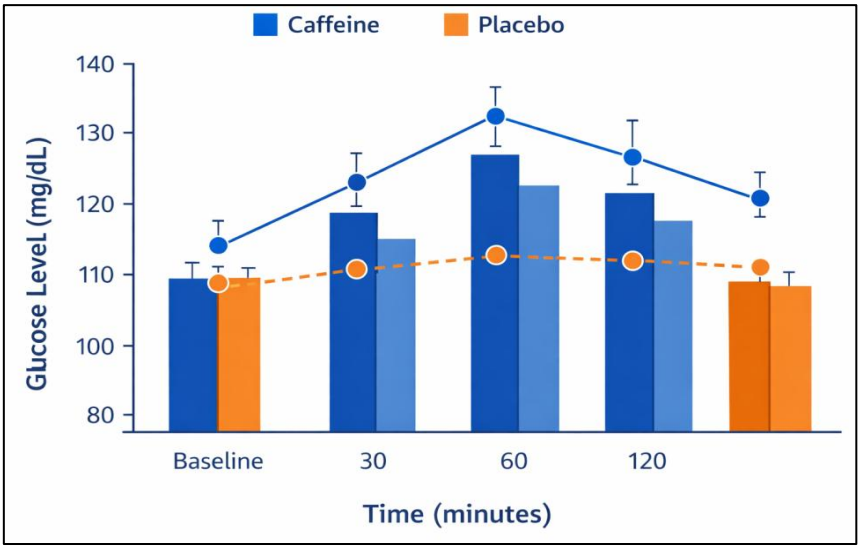

Figure 1: The values of blood glucose at various time intervals following caffeine and placebo (Mean SD)

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Insulin Levels

At the same four time points, insulin levels were determined. Findings showed that there was a great increase after ingesting caffeine over the placebo (MacKenzie, T. 2007) (Table 2, Figure 2).

Table 2: The mean SD of insulin levels in the various time intervals following the caffeine intake and the placebo (n = 30) respectively

Time (min)	Caffeine (µU/mL)	Placebo (µU/mL)
Baseline	8 ± 1	8 ± 1
30	12 ± 2	8 ± 1
60	15 ± 2	9 ± 1
120	13 ± 2	8 ± 1

The secondary increases in insulin are in line with the acute compensatory hyperinsulinemia on the basis of decreased peripheral glucose uptake by caffeine (Keijzers, G. B. *et al.*, 2002).

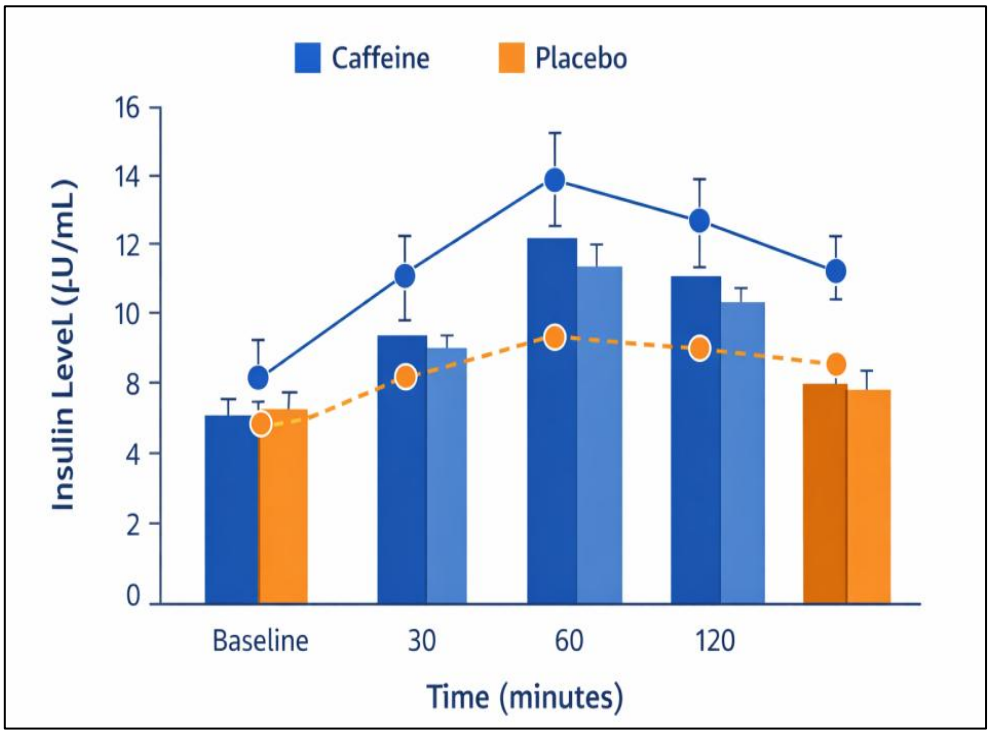

Figure2: The comparison of blood insulin at different point after caffeine and placebo

HOMA-IR

The insulin resistance was measured with HOMA IR, glucose, and insulin levels, Caffeine consumed dramatically raised HOMA IR reaching the maximum at 60 minutes (Lee, S. *et al.*, 2020) (Table 3, Figure 3).

Table 3: The mean and SD of HOMA IR compared to caffeine and placebo (30-subjects).

Time (min)	Caffeine	Placebo
Baseline	1.88 ± 0.15	1.90 ± 0.14
30	3.26 ± 0.20	1.92 ± 0.16
60	4.44 ± 0.25	2.05 ± 0.18
120	3.70 ± 0.22	1.95 ± 0.15

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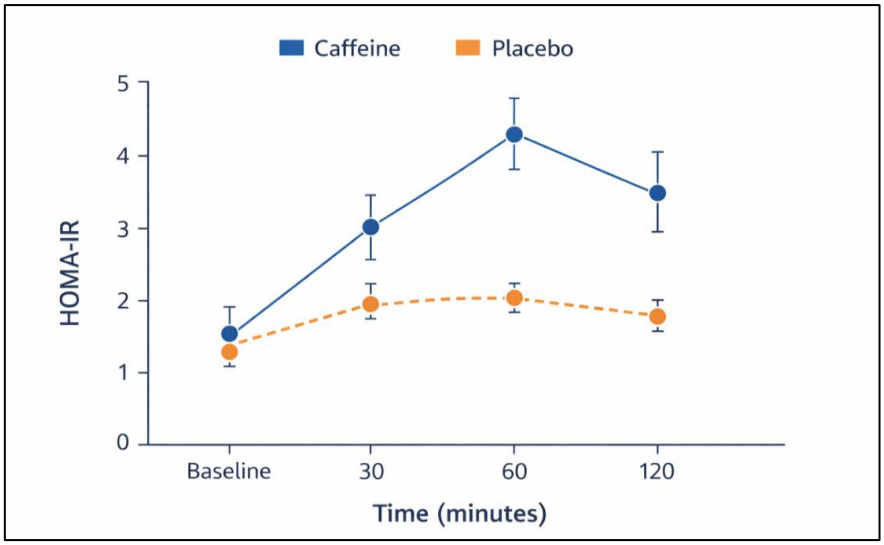

Figure3: Comparison of blood insulin level of caffeine vs placebo in various points
the acute caffeine-induced low insulin sensitivity will be supported by the peak in HOMA IR among healthy adults (Shi, X. *et al.*, 2016).

Heart Rate (HR)

The caffeine intake led to a significant increase in the heart rate (Table 4, Figure 4).

Table 4: Heart rate (mean ± SD) at varying time points (n =30).

Time (min)	Caffeine (bpm)	Placebo (bpm)
Baseline	72 ± 5	71 ± 4
30	78 ± 6	72 ± 4
60	82 ± 6	73 ± 5
120	76 ± 5	71 ± 4

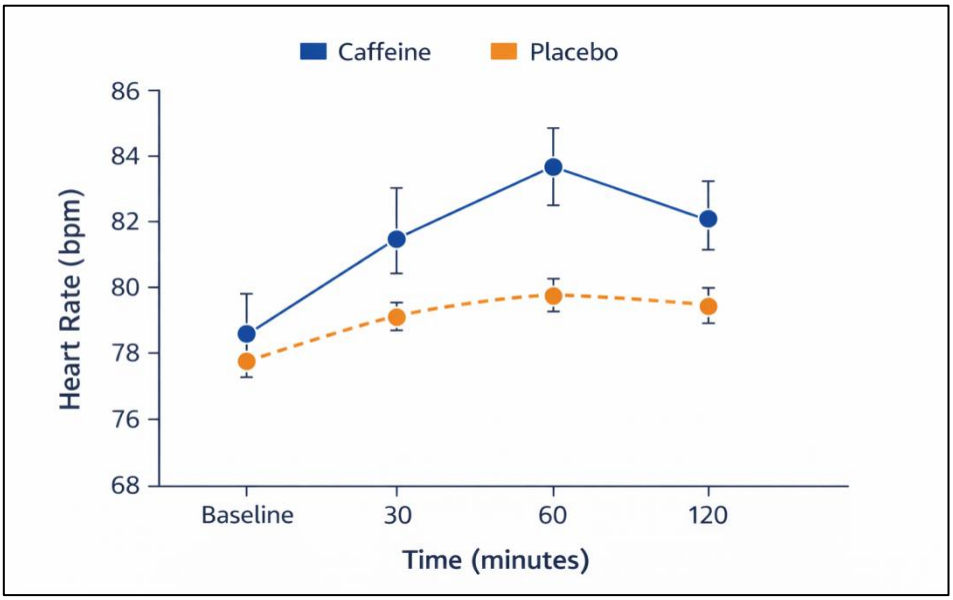

Figure 4: Observation of change in heart rate with time caffeine vs placebo

This increase is an indication of sympathetic activity caused by caffeine and catecholamine discharge (Corti, R. *et al.*, 2002).

Blood Pressure (Systolic/Diastolic)

There was a temporarily increased systolic and diastolic BP with caffeine (Table 5, Figure 5)

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Table 5: Mean SD of BP at various time points (n = 30)

Time (min)	Caffeine S/D (mmHg)	Placebo S/D (mmHg)
Baseline	120/75 ± 5/4	119/74 ± 4/3
30	128/80 ± 6/5	120/75 ± 5/4
60	130/82 ± 7/5	121/76 ± 5/4
120	125/78 ± 6/4	119/74 ± 4/3

The effects of caffeine on acute BP increase indicate short-time cardiovascular stimulation and are in line with previous evidence (Gualberto, P. I. *et al.*, 2024).

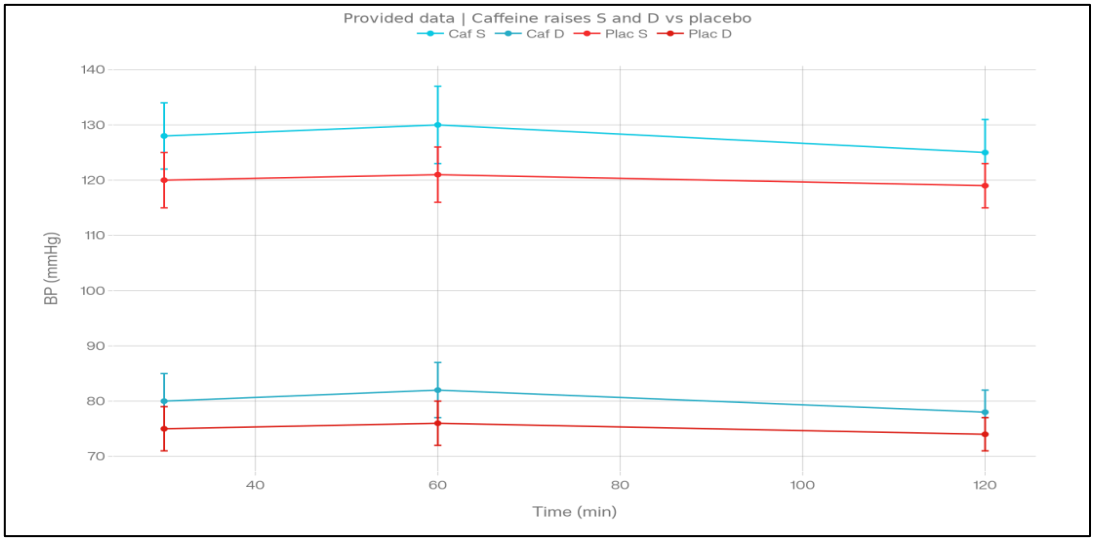

Figure 5: systolic and diastolic changes of blood pressure following caffeine compared to placebo

LF/HF Ratio from HRV

After use of caffeine, LF/HF ratio changed significantly (Table 6, Figure 6), which means that sympathetic dominance shifted (Crooks, E. *et al.*, 2019).

Table 6: LF/HF ratio mean SD at various time points following the intake of caffeine and placebo (n = 30)

Time (min)	Caffeine	Placebo
Baseline	1.8 ± 0.2	1.7 ± 0.2
30	2.6 ± 0.3	1.8 ± 0.2
60	3.0 ± 0.4	1.9 ± 0.2
120	2.4 ± 0.3	1.7 ± 0.2

These results are consistent with sympathetic predominance of HRV caused by acute caffeine-induced autonomic modulation as reported in previous controlled studies (da Silva Rolim, P. *et al.*, 2019).

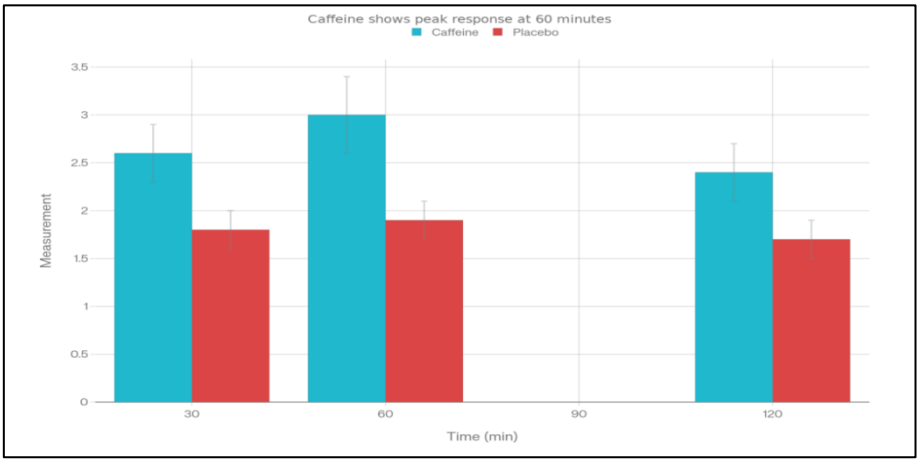

Figure 6: Effect caffeine versus placebo over minutes

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DISCUSSION

The results of the current study suggest that acute ingestion of caffeine at a dose of 5 mg/kg body weight elicit synchronous physiological responses that include both metabolic and autonomic physiological systems (Harpaz, E. *et al.,* 2017). This is attested by transient increases in blood sugar, insulin and insulin resistance index (HOMA-IR), activation of the sympathetic nervous system as indicated by an increase in heart rate, blood pressure and LF/HF ratio, obtained from heart rate variability (HRV) (Yu, Y. *et al.,* 2024). These observations lend support to the notion of caffeine to be a neuro-metabolic stimulant which can modulate autonomic regulation and glucose homeostasis by means of its ability to modify function of both can occur simultaneously in healthy individuals (Moaket, O. S. *et al.,* 2025).

The temporary increase in glucose and insulin following the consumption of caffeine can be explained by its antagonistic effect on the adenosine receptors that increase the release of catecholamines, especially norepinephrine and epinephrine from the sympathetic nerve endings and the adrenal medulla (Sharma, V. K. *et al.,* 2023). This catecholaminergic response elevates hepatic glucose output because of the combination of elevated glycogenolysis and glucagon secretion and an acute loss of peripheral insulin sensitivity (Hartmann, C. *et al.,* 2017). Such mechanisms provide a physiological basis for the peak HOMA-IR found at approx. 60 min after caffeine intake that has been reported in the most recent studies in humans (Lee, S. *et al.,* 2020).

Alterations in heart rate and blood pressure also seen in this study are further an indicator of sympathetic activation by caffeine (Bitar, A. 2015). Increased sympathetic outflow causes an increase in cardiac chronotropy and inotropy resulting in short-term increases in both systolic and diastolic blood pressure (Karim, S. *et al.,* 2023). However, the slow decline back to baseline values of these cardiovascular parameters within 120 minutes indicates effective compensative regulatory mechanisms, such as the baroreflex sensitivity and autonomic modulation, in maintaining cardiovascular homeostasis of healthy subjects (Khurana, D. 2025).

The LF/HF ratio derived from HRV showed a significant change towards sympathetic dominance over parasympathetic activity with increase in the HRV due to caffeine intake resulting in a shift to the sympathetic system response period (Almeida, B. A. 2024). This increase was accompanied by an elevation of glucose and insulin levels and thus point to a couple tight interaction between the autonomic nervous system activity and metabolic regulations (Russo, B. *et al.,* 2021). Previous research has established that better sympathetic tone is well correlated with acute dysfunctions in glucose metabolism and cardiovascular responsiveness, which is possibly consistent with the present results (Carnagarin, R. *et al.,* 2018).

Collectively these results show that caffeine triggers an orchestrated series of acute physiological responses which encompass transient increments in glucose, insulin and insulin resistance, as well as significant sympathetic cardiac and autonomic activation, to return to homeostasis over time (Hamad, A. K. S. 2024) This pattern would suggest that caffeine is working as a short acting neuro-metabolic stimulant with a healthy individual experiencing a great ability to re-establish physiological balance (Xu, J. 2025). These findings are in line with recent literature which suggest that, moderate doses of caffeine consumption has limited and reversible effects on metabolic and cardiovascular function in healthy populations (Hamad, A. K. S. 2025).

CONCLUSIONS

The results of this study demonstrated that the consumption of caffeine produces highly significant physiological effects on metabolic and autonomic balance in the healthy subjects. Acute Caffeine intake causes a temporary increase in blood glucose levels, while resulting in an increase in insulin secretion indicating a rapid metabolic response directly associated with caffeine intake. The detected increase in insulin resistance index (HOMA-IR) shows a temporary decrease of insulin sensitivity, an alteration that is reversible in a short time, that seems to indicate that the caffeine's metabolic effect is transitory when there is no distinct physiological alteration.

Furthermore, the results confirm that caffeine is a stimulator in the central nervous system since the increase in heart rate, systolic and diastolic blood pressure and the marked increase in the LF/HF ratio through the analysis of heart rate variability are obtained. Collectively, these indicators are representative of transitory sympathetic dominance against a background of a slow recovery of autonomic equilibrium thus illustrating the efficiency of compensatory regulatory mechanisms

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in the autonomic nervous system of healthy individuals.

Comparison with the placebo group indicates that the changes in physiology are a direct result of caffeine intake rather than as a result of psychological or environmental factors. Accordingly, the present study establishes the fact that acute caffeine intake produces transient and reversible metabolic and autonomic responses. However, one cannot exclude that these effects have some clinical relevance in people with metabolic or cardiovascular disorders, and thus caution should be exercised in caffeine consumption in these populations.

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