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Comparison of the effects of acute and chronic psychological stress on metabolic features in rats*

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Abstract: This study was aimed to compare the effects of acute and chronic psychological stress on metabolic factors. Forty-two male Wistar rats were divided into control and stressed groups. Stress was applied by a communication box acutely (1 d) and chronically (15 and 30 d). Blood sampling was carried out by retro-orbital-puncture method. The plasma levels of glucose, cholesterol, triglyceride, insulin, and corticosterone were measured. In addition, feed and water intake, latency to eat and drink, adrenal and body weights were determined. Acute and chronic psychological stress did not significantly change basal plasma corticosterone levels. However, immediately (1 min) after acute exposure to stress, plasma corticosterone level increased compared to that before stress exposure. Acute stress increased plasma insulin levels significantly. Fifteen days of stress exposure resulted in plasma glucose increase. Chronic stress significantly increased feed intake, latency to eat, and adrenal weight compared to acute stress. The body weights of both control and stressed groups increased markedly during the experiment. Homeostasis model assessment of insulin resistance (HOMA-IR) index did not change significantly in the stressed group. In conclusion, application of acute and chronic psychological stress leads to different metabolic and/or behavioral changes but the metabolic changes resulting from acute exposure to stress seem to be more pronounced.

Key words: Psychological stress, Corticosterone, Insulin, Glucose, Cholesterol, Triglyceride
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1 Introduction

As Hans Selye believed, "stress is a general adaptation syndrome", i.e., a single stereotypic response elicited by any demand upon the body (Rosmond, 2005). The adaptation response to stress would be different according to the type, duration, intensity, and history of the stress (Jean Kant *et al*., 1985; Pitman *et al*., 1990; Ishikawa *et al*., 1992; Ricart-Jane *et al*., 2002; Rai *et al*., 2003). Psychological stress as a more frequent type of stress in humans evokes adaptive responses such as changes in plasma levels of some of the blood parameters and hormones and subsequent behavioral and metabolic alterations to survive. Acute restraint, shaking, and restraint plus shaking for 2 h each increased plasma corticosterone level significantly in male rats (Dhabhar and McEwen, 1997). In addition, acute water immersion stress (as a psychological stress) increased plasma noradrenaline, adrenaline, corticosterone, and glucose levels in male rats (de Boer *et al*., 1990). Moreover, acute noise stress (another example of psychological stress) decreased serum

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glucose and insulin concentrations (Armario *et al*., 1985) but enhanced plasma corticosterone levels (Armario *et al*., 1984). Acute immobilization stress that has a psychological component (van de Kar and Blair, 1999), increased plasma glucose and corticosterone levels during the stress session, whereas plasma insulin concentration was increased at the beginning of the stress session and returned to its normal value at the end (Yamada *et al*., 1993). On the other hand, the response to chronic stress is completely different from that to acute short-term stress as mentioned above. In this regard, chronic exposure to restraint, shaking and restraint plus shaking for 6 h (during 6 h the stressors were changed every hour) resulted in a marked reduction of the stress-induced increase in plasma corticosterone level (Dhabhar and McEwen, 1997). Moreover, chronic water immersion caused a smaller increase of plasma noradrenaline, adrenaline, corticosterone, and glucose levels in the rats as compared to acute stress exposure (de Boer *et al*., 1990). The chronic noise exposure caused no changes in the serum levels of insulin, glucose (Armario *et al*., 1985) and also basal plasma level of corticosterone (Armario *et al*., 1984). In addition, repeated immobilization increased plasma corticosterone, decreased plasma insulin, and had no effect on plasma glucose concentrations (Makino *et al*., 1999).

In human societies, psychological stress in acute and chronic forms is much too prevalent and by changing some metabolic and hormonal parameters, may be the basis of many disorders (Rosmond, 2005; Dong *et al*., 2011), but whether acute or chronic psychological stress induces more profound metabolic defects is still an unanswered question. In this regard, the first step is to choose animal models of psychological stress which are very close to human psychological stress, followed by the evaluation of the metabolic consequences of acute and chronic application of the stressors. Therefore, this study aimed to use a communication box (com-box) to induce a psychological stress without a physical component in rats, and to compare some metabolic (plasma glucose, triglyceride, and cholesterol), hormonal (insulin and corticosterone), and behavioral (feed and water intake, latency to eat and drink) parameters following acute and chronic exposure to the stress.

2 Materials and methods

2.1 Animals

Forty-two male Wistar rats (Pasteur Institute, Tehran, Iran) with an initial body weight of 170–250 g were used. The animals were housed three per plastic cage at a constant temperature ((22±2) °C) and a 12-h light cycle with free access to standard feed (standard pellets, pars production and distribution of animal feed company, Iran) and tap water. For fasting conditions, feed was removed for 16 h before the beginning of the trial. Rats were adjusted to the environment for one week prior to the experiments. Animals' body weight was measured on the first and last days of the experiments by a digital scale (FEW, Japan, sensitivity 0.1 g). All procedures were approved by the Animal Care and Use Committee of the Neuroscience Research Center, Shahid Beheshti University of Medical Sciences, Iran.

2.2 Stress procedure

A com-box (Borje Sanat, Iran) was used as a psychological stress stimulus devise. This device (48 cm×48 cm×50 cm) is divided into nine compartments (16 cm×16 cm×50 cm) by transparent plastic sheets. In each session, five rats were exposed to the electrical foot-shock (1 mA, 1 Hz) for a 10-s duration every 60 s (1 h/d) through the stainless steel grids. Four remaining animals in the other compartments were exposed to psychological stress by observing the animals under foot-shock stress (Endo *et al*., 2001). In two sessions, eight animals were psychologically stressed, seven of which were used in this study.

The animals of the stressed group were subdivided into acutely stressed (i.e., 1 d exposure to stress) and chronically stressed (i.e., 15 and 30 d exposure to stress) groups. The stressed groups were exposed to psychological stress (1 h/d) for 10 to 14 h. The animals of the control group had the same subgroups and served as the control Days 1, 15, and 30. These animals were placed in the box (1 h/d) without receiving any stress.

2.3 Blood sampling

Blood samples were obtained by orbital sinus puncture method (Hoff, 2000) under light isoflurane anesthesia. The samples were collected in an Eppendorf

tube containing 0.5% heparin and centrifuged at 3000×*g* for 5 min (Toleikis and Godin, 1995). The plasma was separated and kept at −80 °C until used.

2.4 Assessment of plasma glucose, insulin, triglyceride, cholesterol, and corticosterone levels

To evaluate plasma glucose, insulin, triglyceride, and cholesterol levels in both the control and stressed groups, blood sampling was performed 1 d after the last session of the trial (i.e., on Days 2, 16, and 31 of the experiment) at 8:00 to 8:30 AM.

To determine the basal plasma corticosterone concentration, blood samples were obtained before placing the animals in the com-box for the first time (basal-before, B-B) and 1 d after the last session of the trial (basal-after, B-A) at 8:00 to 8:30 AM. Moreover, blood sampling was performed on Days 1, 15, and 30 of the experiment immediately after removing the animals from the com-box (with or without stress).

Plasma samples were analyzed for insulin and corticosterone concentrations by a rat insulin enzyme-linked immunosorbent assay (ELISA) kit (Mercodia, Sweden) and corticosterone ELISA kit (DRG, Germany), respectively. Plasma glucose concentration was determined using the glucose oxidase method (Pars Azmoon, Iran). Plasma triglyceride and cholesterol levels were measured by colorimetric method (Pars Azmoon, Iran). The intra- and inter-assay coefficients of variations for insulin were 3.40% and 2.20%, for corticosterone 4.08% and 6.35%, for glucose 1.74% and 1.19%, for triglyceride 1.82% and 1.60%, and for cholesterol 1.62% and 1.22%.

2.5 Homeostasis model assessment of insulin resistance (HOMA-IR) index

Fasting plasma glucose and insulin were measured to determine HOMA-IR index, the formula is: HOMA-IR=(c_i×c_g)/22.5, where c_i is fasting insulin level (μU/ml) and c_g is fasting glucose level (mmol/L) (Farahani *et al*., 2010).

2.6 Weight of adrenal glands

Rats were anesthetized with isoflurane and decapitated, and the abdomen was opened. The adrenal glands were carefully isolated from adhering adipose tissue and weighed immediately (Hoeflich *et al*., 2002) by a digital scale (Sartorius, Germany, resolution 0.1 mg).

2.7 Feed and water intake and latency to eat and drink

Feed and water intakes were measured once a week throughout the experiment by measuring the difference between the amount of feed or the volume of water put in the cage and the remaining amount after 24 h. Moreover, after each session of an animal being in the com-box, the time lag between the removal and starting to consume feed and water was recorded and the mean values of Days 7 to 30 were compared with the value of Day 1 (Macht *et al*., 2001).

2.8 Statistical analysis

Results are presented as mean±standard error of the mean (SEM). A mixed analysis of variance (ANOVA) with repeated measures within the stressed and control groups (day was considered as a repeated factor) and independent measures between the two groups (stress was considered as an independent factor) was performed by SPSS Version 9.0 program package. $P<0.05$ was considered statistically significant.

3 Results

3.1 Effects of acute and chronic stress on plasma corticosterone concentration and adrenal gland weight

Acute (1 d) and chronic (15 and 30 d) exposure to psychological stress had no significant effects on basal (both B-B and B-A) plasma corticosterone concentrations. However, immediately after acute (Day 1) exposure to stress, plasma corticosterone concentration was increased as compared to that before stress exposure ($P<0.001$). The chronically stressed animals showed a reduction in plasma corticosterone level immediately after stress exposure on Days 15 and 30 as compared to the same group on Day 1; however, only on Day 15 the reduction was significant ($P<0.01$) (Table 1).

Acute stress had no significant effect on the adrenal gland weight, whereas in the stressed group, chronic stress (Day 30) caused a significant increase

in the weight of adrenal glands as compared to Day 1 of the control and stressed groups (P<0.001) and Day 15 of the control (P<0.01) and stressed (P<0.05) groups (Table 1).

3.2 Effects of acute and chronic stress on plasma glucose and insulin concentrations and HOMA-IR index

Acute (1 d) exposure to psychological stress had no significant effects on plasma glucose concentration, whereas chronic stress (15 d) significantly increased plasma level of glucose as compared to Day 1 (P<0.01). However, 30 d exposure to stress had no significant effect on plasma glucose concentration (Table 2).

Table 1 Plasma corticosterone concentrations and adrenal gland weights of control and stressed rats

Group	Corticosterone concentration (nmol/ml)			Adrenal gland weight (mg)
	B-B[1]	B-A[2]	After[3]	
Control				
Day 1	0.32±0.07	0.33±0.05	0.36±0.06	29.30±1.82
Day 15	0.31±0.03	0.31±0.07	0.32±0.04	32.39±1.84
Day 30	0.31±0.04	0.24±0.05	0.29±0.05	36.20±2.89
Stressed				
Day 1	0.29±0.05	0.40±0.06	0.55±0.03[m]	30.91±0.97
Day 15	0.34±0.06	0.31±0.04	0.30±0.08[c]	35.07±1.98
Day 30	0.30±0.04	0.27±0.05	0.36±0.07	42.61±1.79[abde]

Values are expressed as mean±SEM of 7 rats. [1] Basal-before stress exposure; [2] Basal-after stress exposure; [3] Immediately after removing from com-box. [a] P<0.001 significant difference versus Day 1 of the control group; [b] P<0.001, [c] P<0.01 significant difference versus Day 1 in the same group; [d] P<0.05 significant difference versus Day 15 in the same group; [e] P<0.01 significant difference versus Day 15 in the control group; [m] P<0.001 significant difference versus B-B in the same group. Letters a to e show differences in column and letter m shows difference in row

Psychological stress caused significant elevation of plasma insulin levels on the first day of the experiment as compared to the respective control group (P<0.01). Acute and chronic psychological stress did not change fasting plasma glucose and insulin levels (Table 2).

The HOMA-IR index was not significantly different between the control and stressed groups (Table 2).

3.3 Plasma cholesterol and triglyceride changes

No significant difference was observed between plasma cholesterol levels of the control and stressed groups throughout the trial (Table 2).

Acute and chronic psychological stress caused no significant changes in the plasma triglyceride concentration. However, a non-significant reduction of plasma triglyceride was observed following 30 d stress exposure compared to the control Day 30 (Table 2).

3.4 Feed and water intake and latency to eat and drink

The animals which were chronically exposed to stress showed a significant increase in feed intake on Day 21 of the experiment as compared to Day 1 (P<0.01) and on Day 30 of the experiment as compared to Day 1 and the control animals on Day 30 (P<0.01), as shown in Table 3. Furthermore, the latency to eat following chronic stress exposure (Days 7–30) was increased compared to acute exposure to stress (Day 1), as shown in Fig. 1. Acute and chronic psychological stress had no significant effects on water intake (Table 3) or on the latency to drink (Fig. 2).

Table 2 Effects of acute and chronic psychological stress on plasma insulin, glucose, triglyceride, and cholesterol concentrations and HOMA-IR index

Group	Insulin (μg/L)	Glucose (mg/dl)	Triglyceride (mg/dl)	Cholesterol (mg/dl)	Fasting glucose (mg/dl)	Fasting insulin (μg/L)	HOMA-IR index
Control							
Day 1	0.77±0.10	116.84±6.91	179.10±6.47	107.37±4.16	98.39±5.40	0.76±0.15	4.47±0.82
Day 15	1.10±0.14	124.06±3.64	180.54±5.38	118.03±5.79	104.39±5.02	0.62±0.18	4.13±1.29
Day 30	1.17±0.13	120.68±3.85	191.04±6.53	108.68±7.60	114.29±4.87	0.99±0.43	4.07±1.14
Stressed							
Day 1	1.61±0.24[**]	110.07±3.48	184.13±7.42	113.61±5.10	87.82±1.18	0.71±0.06	3.86±0.29
Day 15	1.05±0.14	124.53±4.71[##]	194.07±12.37	108.84±3.06	104.18±3.85	0.99±0.37	3.73±1.71
Day 30	1.43±0.28	115.39±3.40	175.49±3.08	122.88±2.96	103.19±4.88	0.69±0.17	4.49±1.16

Values are expressed as mean±SEM of 7 rats. [**] P<0.01 significant difference versus the same day of the control group; [##] P<0.01 significant difference versus Day 1 in the same group

Table 3 Effects of acute and chronic psychological stress on feed and water intake

Group	Feed intake each rat (g/d)	Water intake each rat (ml/d)
Control		
Day 1	18.93±2.15	28.43±3.10
Day 7	17.22±0.82	33.71±3.47
Day 15	18.07±0.33	42.71±6.59
Day 21	21.20±1.27	44.40±3.92
Day 30	17.87±0.89	41.73±5.55
Stressed		
Day 1	16.54±1.42	33.00±1.89
Day 7	18.80±0.61	29.74±1.45
Day 15	20.21±1.21	37.14±1.82
Day 21	23.69±2.88$^{\#\#}$	36.86±3.69
Day 30	24.24±1.98$^{**\#\#}$	37.71±1.89

Values are expressed as mean±SEM of 7 rats. $^{**}P<0.01$ significant difference versus the same day of the control group; $^{\#\#}P<0.01$ significant difference versus Day 1 of the same group

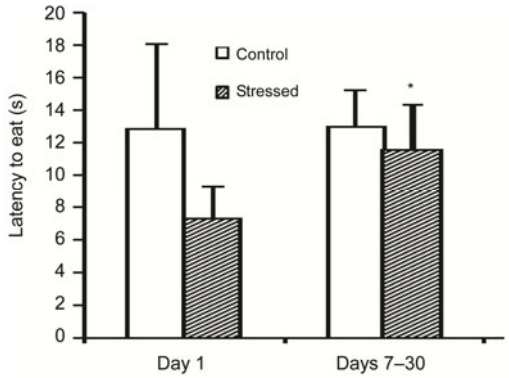

Fig. 1 Effects of acute and chronic psychological stress on latency to eat
The latency to eat following chronic stress exposure (Days 7–30) was increased compared to acute stress exposure. Each column represents mean±SEM of 7 rats. $^{*}P<0.05$ significant difference versus Day 1 of the same group

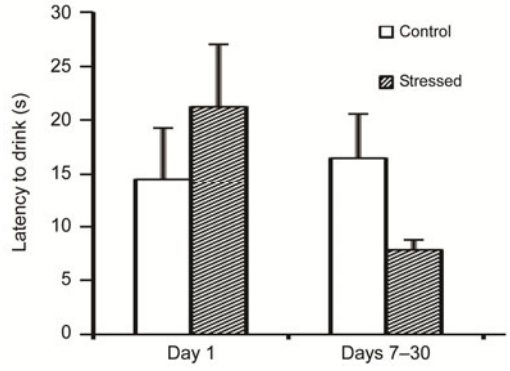

Fig. 2 Effects of acute and chronic psychological stress on latency to drink
Acute and chronic psychological stress had no significant effects on latency to drink. Each column represents mean±SEM of 7 rats

3.5 Body weight variations

The body weights of both control and stressed rats had a rising trend. The weights of control and stressed rats showed a significant ($P<0.001$) increase on Day 30 as compared to Days 1 and 15. In addition, the weight of stressed rats on Day 15 had a significant increase as compared to Day 1 ($P<0.01$) (Fig. 3).

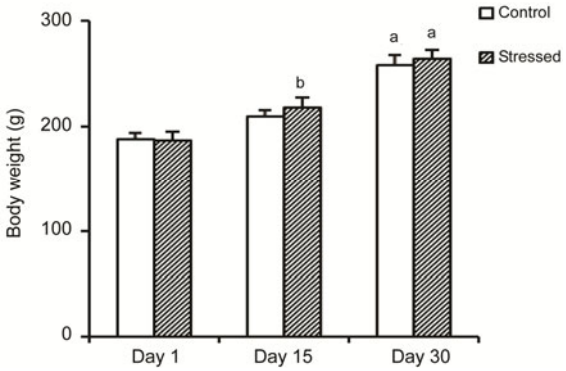

Fig. 3 Body weights of the control and stressed groups
Each column represents mean±SEM of 7 rats. $^{a}P<0.001$ significant difference versus Days 1 and 15, $^{b}P<0.01$ significant difference versus Day 1 of the same group

4 Discussion

In the present study, acute stress elevated plasma insulin concentration but did not change plasma glucose level. The plasma concentrations of glucose and insulin remained unchanged following chronic (30 d) stress exposure. In addition, exposure to stress for the first time (Day 1) significantly increased plasma corticosterone concentration immediately after exposure, whereas by chronic exposure to stress plasma corticosterone level decreased while the adrenal weight increased. Moreover, chronic exposure to stress significantly increased feed intake and latency to eat compared to acute stress. As a whole, the results of the present study showed a relatively typical metabolic response to acute and chronic stress.

Activation of the sympathoadrenal system leads to catecholamine release from nerves and the adrenal medulla. Activation of hypothalamic-pituitary-adrenal (HPA) axis in turn results in corticotrophin secretion from adenohypophysis and finally corticosterone, as the main glucocorticoid in rodents, is released from the adrenal cortex (Teague *et al.*, 2007).

In this regard, in the present study the up-regulation of gluconeogenesis, glycogenolysis, and glucose transport probably induced by the short-term increase of plasma corticosterone level immediately after the first stress exposure (acute stress) may keep the plasma glucose level to be unchanged (Andrews and Walker, 1999). In some other studies, acute exposure to immobilization (Ricart-Jane *et al*., 2002; Rai *et al.*, 2003) increased plasma glucose and insulin concentrations. Since stress can increase plasma catecholamine beside corticosterone levels and the secretion of the catecholamines is a part of the "fight or flight" response that stimulates glycogenolysis and increases the basal metabolic rates and productions of glucose and insulin as well (Teague *et al.*, 2007), the different observed results may reflect different stress level threshold responses, differential rates of glucose and insulin production, or other intrinsic metabolic differences between animals used in the various studies.

In the chronically stressed rats of this study, an increase of plasma glucose concentration was observed on Day 15, but not on Day 30 as compared to Day 1, while plasma corticosterone showed no significant change. This increase of plasma glucose may be related to the possible increase of plasma adrenaline and noradrenaline (not measured in this study) following stress exposure. However, by increasing the days of exposure to stress the catecholamines' response may be adapted and therefore may cause the plasma glucose level on Day 30 not to change significantly (de Boer *et al*., 1990). It is noteworthy that the patterns of corticosterone and catecholamine of sympathoadrenal response and adaptation to intermittent stress are different (de Boer *et al*., 1989).

According to the results of the present study, HOMA-IR index, which is a method to quantify insulin resistance, also did not change significantly following acute and chronic stress. Thus it seems that this kind of stress in acute or chronic form did not have a significant effect on insulin resistance. In agreement with our results, it was shown that inducing a moderate hypercortisolism by hydrocortisone infusion (for 3 h) in human subjects, which is equivalent to that observed in response to a mild stress, did not change HOMA-IR index (Darmon *et al*., 2006). On the other hand, chronic exposure of normal chow fed male Wistar rats to electric footshock assisted with noise for 10 weeks (Fu *et al*., 2009) increased HOMA-IR index. The differences between results may be due to the variation in the type, duration, and intensity of the applied stressor.

Consistent with the present study, previous experiments have shown that different types of stress including immobilization (30 min) (Ricart-Jane *et al*., 2002), restraint, shaking, and restraint plus shaking (2 h) (Dhabhar and McEwen, 1997) in rats significantly increased plasma corticosterone level when applied acutely, whereas chronic ethanol injection (Spencer and McEwen, 1990), noise (Armario *et al*., 1984), and electroconvulsive shock (Thiagarajan*et al*., 1989) in rats decreased plasma corticosterone concentration compared to the first stress exposure. Moreover, in the chronically stressed groups of the present study the plasma corticosterone levels immediately or one day after the last stress exposure decreased to the levels of the control groups, even though the weight of the adrenal glands was increased in those animals. In general, the plasma corticosterone level decreased as the animal adapted to the stressor (Teague *et al.*, 2007). Thus the increased adrenal weight together with a return of corticosterone to control values in the chronically stressed rats could suggest a possible late adaptation following initial mass increases in early coping attempts or exhaustion of the adrenal gland, although the involvement of the negative feedback mechanism cannot be ruled out (Teague *et al.*, 2007). It is also possible that a certain adaptation at the cellular level may be contributing, though this remains to be elucidated. In this regard, several studies showed that following chronic exposure to some kinds of stressors (with lower intensity or having psychological aspect) the adaptation may occur (Zelena *et al*., 2003). Moreover, in previous studies, it has also been shown that despite adaptation to stress, the weight of the adrenal glands was increased, which in turn may indicate the demand on the ability of the adrenals to secrete even higher amounts of corticosterone.

In the present experiment plasma triglyceride concentration showed a non-significant reduction following chronic stress exposure whereas plasma cholesterol remained unchanged. Chronic immobilization for 2 periods of 5 and 4 consecutive days, separated by 2 d without stress in rats and chronic stress combining acoustic and restraint stress in mice

also decreased plasma triglyceride but increased plasma cholesterol concentrations (Ricart-Jane *et al*., 2002; Depke *et al*., 2008). Acute stress combining acoustic and restraint stress in mice had no significant effect on any of the mentioned parameters (Depke *et al*., 2008). Acute foot-shock (Ghalami *et al*., 2011) and immobilization stress in 24-h fasted rats (with chow diet) (Hershock and Vogel, 1989) decreased plasma triglyceride but did not change plasma cholesterol. The variations in the results may be attributed to the differences in type, intensity, and duration of stress and also feeding state. The reduction in the plasma triglyceride may be due to enhanced activity of lipoprotein lipase (Starzec *et al*., 1981; Ricart-Jane *et al*., 2002) or decrease in endogenous triglyceride production (Robertson and Smith, 1976).

Chronic exposure to psychological stress, in this study, did not change water intake but increased feed intake as well as body weight compared to the first day. This result may indicate that a balance between feed intake and energy consumption was maintained by the possible activation of the sympathetic nervous system following stress exposure, which stimulates thermogenesis and metabolism (Armario *et al*., 1986; Dorfman *et al*., 2009). However, no significant difference was observed compared to the control group. According to the previous studies, chronic exposure to stress led to different results in relation with the feed intake and weight gain. For instance, repeated immobilization in male Wistar rats (2 h daily, for 2 periods of 5 and 4 consecutive days, separated by 2 d of rest) (Ricart-Jane *et al*., 2002) and in male Sprague-Dawley rats (2 h/d, for 15 d) (Martí *et al*., 1993) decreased feed intake and weight gain, whereas intermittent shaker stress for 2-min periods (150 cycles/min), 45 times/d for 7 d in male C57BL6 mice increased water intake, induced no change in feed intake, and significantly decreased body weight (Bernatova *et al*., 2002). Repeated social stress (for 6 d) increased feed intake during the light period but decreased weight gain in male Sprague-Dawley rats (Bhatnagar *et al*., 2006). At last, a chronic social defeat increased both feed intake and body weight in male Syrian hamsters as compared to the controls (Foster *et al*., 2006).

In this experiment, latency to eat in the chronic stressed animals (Days 7–30) increased significantly as compared to Day 1 (acute stress), whereas latency to drink in the animals remained unchanged. Acute restraint stress (30 min in a plexiglass tube) increased latency to eat and decreased feed intake (Tabarin *et al*., 2007). Twenty-one days of chronic unpredictable stress in mice (Koo *et al*., 2010) and also social defeat stress in Wistar rats (Vidal *et al*., 2011) increased latency to drink. In a study on Sprague-Dawley rats, noise stress (95 dB white noise) increased latency to eat and also feed intake in hypophagic rats, but in normophagic rats the feed intake was not changed (Macht *et al*., 2001). The reasons for the stress-induced alterations in latency to eat and drink are not fully understood. Overall, the discrepancy in the results may be related to the differences in race, type, intensity, and duration of stress. Therefore, further investigation is necessary to be carried out. The combination of increased latency to eat and feed intake observed in this study is a somewhat different result compared to human studies. In general, from the studies on human subjects it seems that high arousal or intense emotions (such as fear, anger, or negative mood) suppress feed-intake and motivation to eat, whereas low to moderate emotions may increase feed-intake and motivation to eat (Macht, 2008).

On the basis of the "stress spectrum hypothesis" introduced by Dhabhar and McEwen (1997), the present study can propose that acute psychological stress used in this research might lead to an immediate physiological stress response which was stopped rapidly according to basal B-A value of the plasma corticosterone level (Table 1). The short-term increase of plasma corticosterone level immediately after stress exposure was beneficial for the stressed rats to promote adaptation and survival (Teague *et al*., 2007) which are the characteristics of a "eustress spectrum". By progression to chronic stress (Days 15 and 30), it seems that the animals' response to stress arrived into the middle part of the spectrum (i.e., "resilience"), which helps survival of the animals for longer periods under increasingly demanding conditions. If the stress continued for a longer duration it would be possible that the stress response of the animals get into the other end of the stress spectrum (i.e., "distress") in which apparent precipitation or exacerbation of metabolic impairments may occur (Dhabhar and McEwen, 1997).

From the results of this experiment it can be concluded that acute exposure to psychological stress may lead to more profound metabolic changes than chronic stress. This profound effect of acute stress should be considered in any intervention of stress managements.

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