Alterations in Malondialdehyde Levels and Laboratory Parameters among Methamphetamine Abusers

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Objective: *To determine the concentrations of malondialdehyde, biochemical, and hematological parameters among methamphetamine abusers compared with a healthy control group and to evaluate the association between malondialdehyde and biochemical-hematological parameters.*
Material and Method: *The concentrations of malondialdehyde, lipids, liver enzymes, albumin, blood urea nitrogen, creatinine, and hematological measurements were determined in 60 methamphetamine abusers and 60 controls.*
Results: *Significantly higher levels of malondialdehyde were found in the methamphetamine abusers than the controls [2.45 (2.12-2.81) vs. 1.41 (1.15-2.08)]. The levels of alanine aminotransferase and alkaline phosphatase and white blood cell and platelet counts of the methamphetamine abusers were significantly elevated (p-value < 0.05) compared with the controls. Meanwhile, the levels of hemoglobin, hematocrit, albumin and body mass index were significantly lower among the methamphetamine-abusing group than the control group (p-value < 0.05). It was found that higher numbers of methamphetamine tablets per day were associated with higher malondialdehyde concentrations in methamphetamine abusers, and that malondialdehyde concentration inversely correlated with albumin level (r = -0.458, p-value < 0.05). Stepwise multiple regression analysis revealed that number of methamphetamine tablets per day, white blood cell count and albumin level were independent predictors of malondialdehyde level (p-value < 0.05).*
Conclusion: *Methamphetamine abuse is related to increased lipid peroxidation, changes in inflammatory marker level, increase in liver enzymes, and decrease in hemoglobin and hematocrit concentrations. These effects may be early signs of the development of diseases associated with methamphetamine abuse.*

Keywords: *Malondialdehyde, Methamphetamine, Hematological parameters, Biochemical parameters*

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The number of methamphetamine (MA) addicts in Thailand continues to rise[1]. MA abusers can develop many serious social problems and health problems, such as acute renal failure, cardiovascular disease, and neurotoxicity[2-4]. Evidence from animal experiments has shown that MA increases inflammation and oxidative stress[5]. MA-induced neurotoxicity may involve alterations in cellular redox status[6]. Oxidative stress plays an important role in dysfunctions of the respiratory, renal, cardiovascular and cerebral systems[7]. Oxidative stress may be defined as the disequilibrium between pro-oxidants and antioxidants in biological systems. When this imbalance appears, cellular macromolecules may be damaged by the predominant free radicals[8]. Malondialdehyde (MDA) is one of several low-molecular-weight end-products formed via the oxidative degradation of polyunsaturated fatty acids[9]. Serum MDA has been used as a biomarker for lipid peroxidation and has served as an indicator of free radical damage. However, knowledge of and information about the underlying biochemical mechanisms of many pathological conditions associated with MA abusers remain incompletely understood. The present study was conducted to investigate the concentrations of MDA and the biochemical and hematological

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parameters, of Thai MA abusers, compared with healthy control subjects, who did not use MA. It also sought to evaluate the relationships between MDA and biochemical-hematological parameters.

Material and Method

Subjects

The subjects comprised 120 adult males, among whom 60 with a history of MA abuse were recruited from Thanyarak Institute, Pathumthani. Sixty healthy subjects with no history of drug abuse were recruited for comparison. All subjects underwent physical examinations, and their medical histories were taken, to identify healthy controls from MA-abusing subjects. Based on the screening evaluation, methamphetamine users were enrolled only if they fulfilled the following criteria:

1. Age, 20-50 years
2. Regular methamphetamine use for at least 3 months, at least 5days/week

Subjects in both groups were excluded if they

1. Were seropositive for HIV
2. Had a history of substance dependence (other than methamphetamine, nicotine, or caffeine)
3. Had a history of any chronic medical, neurologic, or psychiatric illnesses (*e.g.*, seizure disorders, depression, hypertension, heart disease, diabetes, or liver disease).

All volunteers were interviewed by use of a questionnaire, regarding lifestyle pattern and MA-abuse characteristics, such as number of MA tablets per day, and duration of MA use (years). The study protocol was approved by the Ethics Committee of Rangsit University, Pathumthani, Thailand.

Serum was used to determine biochemical variables, *i.e.*, MDA, albumin, blood urea nitrogen (BUN), creatinine, total cholesterol, triglycerides, aspartate aminotransferase (AST), alanine amino-transferase (ALT), alkaline phosphatase (ALP), and uric acid. EDTA blood was used to determine hematological variables, *i.e.*, hemoglobin, hematocrit, platelet count, and white blood cell count. Anthropometric measurements (weight and height) were recorded. Body mass index (BMI) was expressed as weight (kg)/ height (m^2).

Laboratory techniques

Venous blood samples were taken without stasis after a 12-hour fast. Serum total cholesterol, triglycerides, BUN, creatinine, uric acid, AST, ALT and ALP, were measured by DADE Dimension® AR, using enzymatic methods. White blood cell count, platelet count, hemoglobin and hematocrit concentrations were determined (Coulter Counter). MDA, in terms of thiobarbituric acid reacting substances (TBARS), was measured by the modified method of Wong et al[10] and 1, 1, 3, 3-Tetraethoxypropane was used as the standard. The optical density of pink chromogen was read at 532 nm in a double-beam spectrophotometer (UV-610 Shimadzu). To minimize analytical variation further, the same technician performed all assays. The within-run and between-run precision for the MDA assays were analyzed in pooled sera. The analyses showed coefficients of variation (CV) of 6.4% for within-run precision assay, and 6.9% for between-run precision assay.

Statistical analysis

Statistical analysis was performed using SPSS for Windows version 17.0 (SPSS, Chicago, IL). The median and 95% confidence interval (CI) were calculated. The differences between the two groups were compared by Mann-Whitney U test. Spearman rank was used to calculate correlations among the variables. Stepwise multiple linear regression analysis was used to determine the independent predictors of MDA; A p-value < 0.05 was considered statistically significant.

Results

The average duration of MA abuse was about 3 years. The median and 95% confidence interval (% CI) for age, BMI and biochemical-hematological measurements in the MA-abuse group and the healthy control group are shown in Table 1. No significant differences in age, BUN, lipid and uric acid levels were observed between the two groups. The MDA concentrations, platelet counts and WBC counts of the MA-abuse group were clearly higher than the control group ($p < 0.001$). Meanwhile, BMI, albumin, hemoglobin, and hematocrit were significantly lower in the MA-abuse group than the control group. Significantly higher concentrations of creatinine and liver enzymes, including ALT and ALP, were observed in the MA-abuse group than the controls; AST levels tended to increase among the MA-abusing subjects.

The relationship between MDA and other parameters were also determined for the MA-abusing subjects (Table 2). MDA concentrations correlated positively with age ($r = 0.315$, $p < 0.05$), total cholesterol ($r = 0.294$, $p < 0.05$), triglycerides ($r = 0.404$, $p < 0.01$)

Table 1. Median and 95% confidence interval (CI) for age, BMI, and biochemical-hematological parameters in the MA-abuse group and the healthy control group

Parameters	Total				p-value
	MA abuse group (n = 60)		Healthy control group (n = 60)		
	Median	95% CI	Median	95% CI	
Age (years)	33	30-36	34	31-40	0.513
BMI (kg/m^2)	20.55	19.40-21.79	23.88	23.34-24.88	0.000*
BUN (mg/dL)	14.00	13.00-14.80	13.00	12.00-14.33	0.250
Creatinine (mg/dL)	1.10	1.00-1.20	1.05	0.90-1.10	0.023*
Total cholesterol (mg/dL)	220	211-230	219	209-240	0.562
Triglycerides (mg/dL)	139	124-155	137	120-151	0.342
AST (U/L)	32	28-34	29	27-30	0.076
ALT (U/L)	30	25-32	26	23-28	0.013*
ALP (U/L)	65	58-78	56	51-62	0.003*
Uric acid (mg/dL)	5.80	5.02-6.36	5.30	5.17-5.39	0.506
Albumin (g/dL)	4.30	4.20-4.49	4.63	4.39-4.70	0.003*
MDA (mmole/L)	2.45	2.12-2.81	1.41	1.15-2.08	0.000*
Hemoglobin (g/dL)	12.90	12.32-13.46	13.90	13.60-14.20	0.001*
Hematocrit (%)	38.90	37.94-40.08	43.00	42.30-4 4.63	0.000*
White blood cell count (10^9/L)	7.98	7.32-8.66	6.62	5.97-7.09	0.000*
Platelet count (10^9/L)	298	274-314	260	246-274	0.001*

Significant levels; * = $p < 0.05$ by using Mann-Whitney U test (Two-Tailed)
BMI = body mass index; BUN = blood urea nitrogen; AST = aspartate aminotransferase; ALT = alanine aminotransferase; ALP = alkaline phosphatase; MDA = malondialdehyde

and number of MA tablets per day ($r = 0.431$, $p < 0.01$), but MDA concentrations correlated inversely with albumin levels ($r = -0.458$, $p < 0.01$).

Fig. 1 shows that the relationship between MDA concentration (Y) and albumin (X), which seemed to be linear ($Y = 9.099 - 1.486X$) ($p < 0.01$). The relationship between MDA concentration and number of MA tablets per day are shown in Fig. 2. The maximum doses of MA per day were five tablets and the minimum 0.5. The number of MA-abusing subjects who took < 1 tablets of MA per day, between 1 to 3 tablets per day, and > 3 tablets per day were 23, 26 and 11, respectively. Higher number of MA tablets per day was associated with higher MDA concentration among the MA abusers. Stepwise multiple regression analysis revealed that number of MA tablets per day, white blood cell count and albumin, were independent predictors of MDA levels for MA abusers (Table 3).

Table 2. Correlation coefficients of MDA with other parameters in the MA-abuse group (n = 60)

Variables	MDA	
	r	p-value
Age	0.315	0.016*
BMI	0.023	0.454
BUN	0.038	0.399
Creatinine	0.002	0.493
Total cholesterol	0.294	0.020*
Triglycerides	0.404	0.002*
AST	0.001	0.499
ALT	0.186	0.101
ALP	0.196	0.088
Uric acid	-0.074	0.307
Albumin	-0.458	0.001*
Hemoglobin	0.012	0.469
Hematocrit	0.015	0.459
White blood cell count	0.239	0.044*
Platelet count	0.147	0.157
Number of MA tablets per day	0.431	0.001*

* p-value < 0.05 was considered statistically significant
BMI = body mass index; BUN = blood urea nitrogen; AST = aspartate aminotransferase; ALT = alanine aminotransferase; ALP = alkaline phosphatase; MDA = malondialdehyde

Discussion

MA produces intoxication by potentiating the presynaptic neural terminal's release of neurotransmitters, such as catecholamine, norepinephrine and dopamine, causing stimulation of the postsynaptic

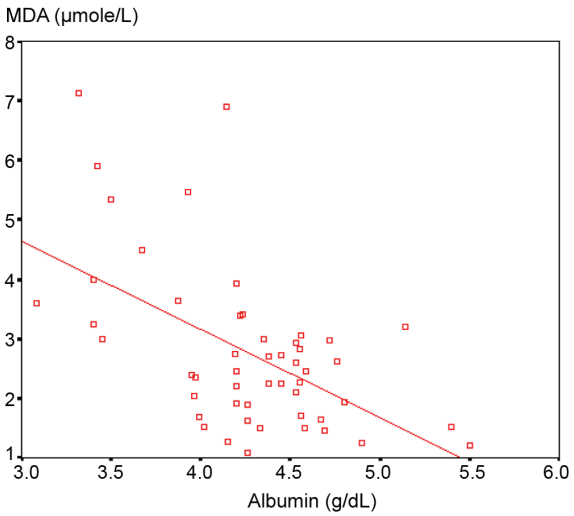

Fig. 1 Relationship between serum MDA concentration and albumin concentration among the MA-abuse group (n = 60)

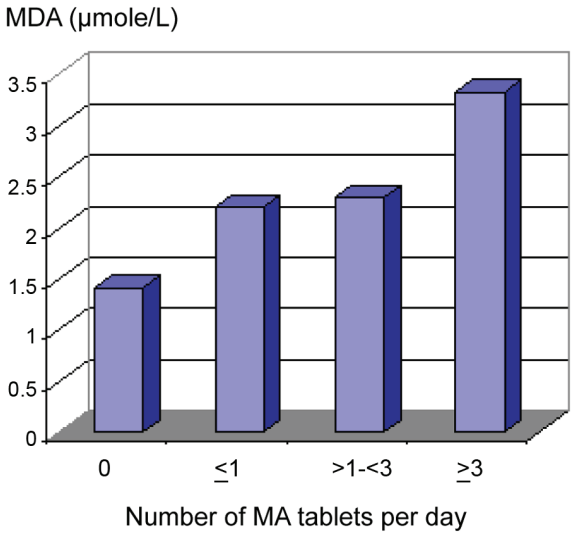

Fig. 2 Relationship between MDA concentration and number of MA tablets per day

receptor and by inhibition of the reuptake of these neurotransmitters[11,12]. High levels of these neurotransmitters are responsible for the production of reactive oxygen species (ROS) and oxidative stress, by either auto-oxidation or degradation by monoamine oxygenase[5,6,13,14]. Studies with animals provide evidence of the effects of MA on pro-oxidant processes and on the production of ROS[12,15,16]. ROS can react with macromolecules, such as lipid, protein, and DNA, which lead to cell dysfunction. The reaction of ROS with lipid can cause lipid peroxidation; MDA is the end-product of lipid peroxydation. In the present study, serum lipid peroxidation was estimated by thiobarbituric acid (TBA) reactivity[10]. The present study revealed that the MDA levels in the MA-abuse group were clearly higher than the healthy control group. Moreover, albumin, which has antioxidant properties, was inversely correlated with a marker of the end-products of lipid peroxidation. The present study suggested that elevated MDA concentrations might be due to MA-induced change in oxidative stress status. Many studies have shown that MA-induced disturbances in cellular redox status and activation of transcription factors can play a critical role in the signaling pathways leading to the up-regulation of inflammatory genes, such as gene encoding for tumor necrosis factor-α (TNF-α)[17,18]. Oxidative stress can result in damage to many biomolecules in the body, and may be one of many factors involved in the inflammation process and the pathogenesis of diseases[5,19,20]. However, the results of the present study indicated that MA abusers had significantly changed inflammatory markers, including white blood cells, platelets, and albumin, compared with the controls. In animal experiments, MA also altered serum biochemical values, such as albumin, creatinine and blood cell counts[21]. White blood cell count may be a marker of exposure to oxidants, the inflammatory response and/or host susceptibility to inflammatory

Table 3. Stepwise multivariate regression analysis when MDA was used as a dependent variable, and albumin, white blood cell count, number of MA tablets per day, and duration of MA use, were used as independent variables

Variables	Unstandardized coefficients		p-value
	β	Std. error	
Constant	4.043	1.790	0.029*
Albumin	-0.964	0.325	0.005*
White blood cell count	0.213	0.075	0.007*
Number of MA tablets per day	0.639	0.196	0.002*
Duration of MA use	0.012	0.049	0.811

* p-value < 0.05 was considered statistically significant

stimuli[22]. Platelets represent an important linkage between inflammation, thrombosis, and atherogenesis, as they are able to interact with white blood cells and endothelial cells and promote the release of prothrombotic and pro-inflammatory factors[23]. Albumin is a well-known "negative" acute-phase protein; it acts as a marker for inflammation and has antioxidant properties[24,25]. Oxidative stress and inflammation have been implicated in MA toxicity[26]. Therefore, the alteration of inflammatory markers, including white blood cell count, platelet and albumin, may be influenced by MA. However, the mechanistic role of MA-induced inflammation has not been fully elucidated. Yamaguchi et al reported that MA caused increases in the levels of interleukin-1β, pro-inflammatory cytokine[27]. Flora et al showed that MA-induced disturbances in cellular redox status and activation of activator protein-1 (AP-1) could play a critical role in signaling pathways leading to up-regulation of inflammatory genes *in vivo*[6]. Therefore, the present study confirms that MA abuse is associated with increased oxidative stress and impacts inflammatory markers, which is important for multi-systemic adverse health consequences.

MA is metabolized by cytochrome P450s in liver microsomes; the elimination half-life of MA ranges from 10-12 hours[28,29]. Studies have reported that liver damage may be a complication of MA abuse and clinical presentations of MA hepatotoxicity may range from mild acute hepatitis with prompt recovery, to fulminate hepatic failure[30,31]. The postulated mechanisms for this hepatotoxicity include direct toxic effects, necrotizing angiitis, and lipid peroxidation[32]. The present study confirmed that MA abuse may induce the elevation of liver enzymes. Furthermore, MA is a stimulant with side effects that include a significant reduction in appetite, which may result in weight loss and eventual malnutrition[33]. The present study of Werb et al reported that MA was significantly associated with risk of malnutrition[34]. The present study found that MA abusers had lower BMI, hemoglobin and hematocrit levels than healthy controls. Decreased levels of hemoglobin and hematocrit might increase the risk of anemia for MA abusers, because anemia involves many factors, such as nutrition deficiency (*e.g.* iron, vitamin B12, folate, and protein) or ineffective red cell formation (*e.g.* chronic inflammation and hemoglobinopathy)[35]. Therefore, the impact and effects of MA abuse may be both direct and indirect, including food intake and nutrition-related health outcomes.

Conclusion

It was found that increased oxidative stress caused increased lipid peroxidation, measured as serum MDA concentration. MA abusers had significantly higher MDA concentrations than the healthy controls, and MDA level was associated with number of MA tablets per day. MA abuse affected changes in inflammatory marker levels, increases in liver enzymes, and decreases in hemoglobin and hematocrit concentrations. These observations may provide some insight into the biological mechanisms underlying the pathology associated with MA abuse among Thais, and help promote the MA withdraw campaign in Thailand.

Ethical Considerations

The authors declare that we have no conflict of interest associated with this work and all ethical issues (such as plagiarism, double submission, etc.) have been considered carefully.

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Potential conflicts of interest

None.

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การเปลี่ยนแปลงของระดับ malondialdehyde และตัวแปรทางห้องปฏิบัติการในผู้เสพยาบ้า

กาญจนา สุริยะพรหม, รสสุคนธ์ ธนธีระบรรจง, อัญชลี ตั้งตรงจิตร, รังสรรค์ ตั้งตรงจิตร

วัตถุประสงค์: *เพื่อตรวจวัดระดับความเข้มข้นของ malondialdehyde ตัวแปรต่างๆ ทางชีวเคมีและโลหิตวิทยาในผู้ที่เสพยาบ้าเปรียบเทียบกับกลุ่มควบคุมที่มีสุขภาพดี และเพื่อประเมินความสัมพันธ์ระหว่าง malondialdehyde และตัวแปรต่าง ๆ ทางชีวเคมีและโลหิตวิทยา*

วัสดุและวิธีการ: *ตรวจวัดระดับความเข้มข้นของ malondialdehyde ไขมันต่าง ๆ เอนไซม์ตับ อัลบูมิน สารยูเรียในเลือด ครีเอตินิน และค่าทางโลหิตวิทยา ในผู้ที่เสพยาบ้าจำนวน 60 ราย และในกลุ่มควบคุมจำนวน 60 ราย*

ผลการศึกษา: *ระดับ malondialdehyde พบว่าในผู้ที่เสพยาบ้าจะมีระดับสูงมากกว่าในกลุ่มควบคุมอย่างมีนัยสำคัญทางสถิติ [2.45 (2.12-2.81) vs. 1.41 (1.15-2.08)] ระดับเอนไซม์ alanine aminotransferase เอนไซม์ alkaline phosphatase เม็ดเลือดขาวและเกล็ดเลือดในผู้ที่เสพยาบ้าพบว่ามีระดับสูงขึ้นอย่างมีนัยสำคัญทางสถิติ เมื่อเทียบกับกลุ่มควบคุม (p-value < 0.05) ขณะที่ระดับฮีโมโกลบิน ปริมาตรอัดแน่นของเม็ดเลือดแดง อัลบูมิน และดัชนีมวลกายพบว่าในกลุ่มผู้ที่เสพยาบ้าจะมีระดับต่ำอย่างมีนัยสำคัญทางสถิติเมื่อเทียบกับกลุ่มควบคุม (p-value < 0.05) ในกลุ่มผู้ที่เสพยาบ้าพบความสัมพันธ์ระหว่างปริมาณเม็ดยาบ้าที่เสพต่อวันกับระดับของ malondialdehyde โดยพบว่าการเพิ่มขึ้นของปริมาณเม็ดยาบ้าที่เสพต่อวันมีความสัมพันธ์กับการเพิ่มขึ้นของระดับ malondialdehyde นอกจากนี้ระดับ malondialdehyde พบว่ามีความสัมพันธ์ในทางตรงกันข้ามกับระดับ อัลบูมิน (r = -0.458, p-value < 0.05) การวิเคราะห์แบบการถดถอยพหุคุณตามลำดับความสำคัญของการนำเข้าตัวแปรพบว่าปริมาณเม็ดยาบ้าที่เสพต่อวัน เม็ดเลือดขาวและอัลบูมินสามารถที่จะใช้เป็นตัวแปรอิสระในการทำนายระดับ malondialdehyde (p-value < 0.05)*

สรุป: *การเสพยาบ้าพบว่ามีความสัมพันธ์กับการเพิ่มขึ้นของกระบวนการ lipid peroxidation การเปลี่ยนแปลงของระดับตัวบ่งชี้ภาวการณ์อักเสบ การเพิ่มขึ้นของระดับเอนไซม์ตับ และการลดลงของระดับฮีโมโกลบิน และปริมาตรอัดแน่นของเม็ดเลือดแดง ซึ่งผลกระทบที่เกิดขึ้นนี้อาจจะนำไปสู่อาการเบื้องต้นของการพัฒนาไปสู่ระยะต่าง ๆ ของโรคที่เกี่ยวข้องกับการเสพยาบ้า*