Effect of selenium and melatonin on some parameters Related to metabolic syndrome induced by Acryl amide in male rats (Part I)

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Summary

This study was designed to investigate the ameliorative role of sodium selenite and melatonin on acryl amide induced metabolic syndrome in adult male rats. Twenty (20) adult male rats were randomly and equally divided into 4 groups (G1, G2, G3 and G4) and were treated orally for seven weeks G1, (control group) was given distilled water, G2 were given orally 1mg/kg /B.W of Acryl amide, G3 group received 1mg/kg /B.W of Acryl amide followed by 0.5 mg/Kg/ B.W. of sodium selenite orally. G4 group received 1 mg/kg /B.W of acryl amide (orally) followed by 8 mg/Kg/ B.W. melatonin (IP/ injection). Fasting blood samples were collected by cardiac puncture at (0, 7 weeks) of the experiment for measuring serum total cholesterol, triacylglycerol, high density lipoprotein-cholesterol and serum uric acid, in addition to measuring waist circumference .The results showed an occurrence of central obesity, hyper uricemia, dyslipidemia (hyper cholesterolemia, triacyleglycerolemia and lowered high density lipoprotein-cholesterol concentration) in acryl amide treated animals. The results also indicated the alleviation of the changes in the above- mentioned parameters related to metabolic syndrome by sodium selenite and melatonin through lowering central obesity, total cholesterol, triacylglycerol, elevation of serum high density lipoprotein-cholesterol and lowering in serum uric acid.

Keywords: Acryl amide, Metabolic syndrome, Melatonin, Sodium selenite, Dyslipidemia.

Introduction

Metabolic syndrome (MetS) is a state of chronic low grade inflammation as a consequence of complex interplay between genetic and environmental factors, central obesity, atherogenic dyslipidemia, hyperuricemia, insulin resistance and hypertension are major criteria of the syndrome (1 and 2). Acryl amide (ACR) is produced in starchy foods those are baked, roasted or fried at high temperature (3). Bread, crisps, coffee and fried potato are the most contaminated food with ACR (4). The primary source of human exposure to acryl amide is occupational; other sources include food, drinking water, and smoking (5). Acryl amide is neurotoxic to experimental animal and human (6) and has mutagenic and carcinogen effect (7). Selenium, which is nutritionally essential for humans, is a constituent of more than two dozen selenoproteins that play critical roles in reproduction, DNA synthesis, and protection from oxidative damage and infection (8). Depending on the fact pointed to the role of reactive oxygen species and oxidative stress in the pathogenesis of may diseased condition correlated with metabolic syndrome, this study aimed to evaluate the effect of sodium selenite and melatonin (the well-known antioxidants) on some parameters related to metabolic syndrome induced by Acryl amide in adult male rats.

Materials and Methods

Twenty adult male rats were randomly and equally divided into 4 groups (5/group) and treated orally (using gavage needle) for 7 weeks: G1: Rats of this group received distilled water and served as control, G2: Rats of this group received 1mg/kg B.W of Acryl amide, G3: Animals in the group received in addition to Acryl amide, 0.5 mg/Kg B.W. of sodium selenite, G4: Rats in this group received in addition to ACR, 8mg/Kg B.W. melatonin by interaperitoneal injection (IP). Fasting blood samples were collected at (0 and 7 weeks) of the experiment. Blood was drawn by cardiac puncture. Then serum samples were separated and frozen at -20˚C until analysis of the following parameters: Metabolic syndrome parameters: A-Waist circumference using tape measure according to savory *et al*., (9). B-Serum total cholesterol concentration using total cholesterol enzymatic kitlinear chemical,

(Barcelona, Spain) according to (10). D-Serum triacylglycerol concentration using TAG -C enzymatic kit (Merck company, U.K.) according to Fossati *et al* (11). E- Serum HDL-C concentration using HDL-C enzymatic kit (Bio system, Spain) according to Burstein *et al* (12). F- Serum uric acid concentration using uric enzymatic kit linear chemical, Barcelona (Spain) according to Fossati *et al* (13). Statistical analysis of data was performed on the basis of Two-Way Analysis of Variance (ANOVA) using a significant level of (P<0.05). Specific group differences were determined using least significant differences (LSD) as described by (14).

Results and Discussion

A Significant (P<0.05) reduction in waist circumference was observed in both groups which received sodium selenite (G3) or melatonin (G4) concurrently with ACR (G4) at the 3rd week of the experiment as compared with the values in ACR (G2) group (Table, 1). The results also showed that oral administration of sodium selenite or (I/P) melatonin concurrently with ACR caused gradual significant reduction (P<0.05) in waist circumference in groups G3, G4 along the last four weeks (4th, 5th, 6th and 7th) of the experiment compared to the values in ACR and control groups.

Herein study showed significant (P<0.05) increase in serum total cholesterol (Table, 2) and triacylglycerol (TAG) concentration (Table, 3) in rats received 1mg/kg B.W ACR for 7 weeks (G2) groups compared to the values in other treated groups and control. Oral intubation of sodium selenite (group G3) or melatonin (group G4) concurrently with acryl amide caused significant elevation in serum HDL-C concentration compared to the values in ACR (G2) treated groups (Table, 4). At the end of the experiment, significant (P<0.05) increase in serum uric acid concentration was observed in Acryl amide (G2) treated group compared to the values in the control, G3 and G4 groups .On the other hand significant (P<0.05) decrease in serum uric acid concentration was observed after oral intubation of sodium selenite, or IP/injection of melatonin compared to the values in ACR treated group (Table, 5).

Table, 1: Effect of sodium selenite and melatonin on waist circumference (cm) in acryl amide treated rats.

Groups Periods (Weeks)	G1 Control	G2 Acrylamide (1mg/kgB.W)	G3 Acrylamide(1mg/kg B.W) and sodium selenite (0.5mg/kg B.W)	G4 Acrylamide (1mg/kgB.W) and melatonin (8mg/kg B.W)
1	14.71 ± 0.18 A C	14.78 ±0.26 A c	14.71 ± 0.18 A a	14.71 ± 0.18 A a
2	14.71 ± 0.18 A C	15.04 ±0.29 A bc	14.50 ± 0.17 A bc	14.64±0.17 A a
3	15.08±0.18 A B b	15.54±0.24 A ab	14.28 ± 0.16 B abc	14.41 ± 0.17 B ab
4	15.27 ± 0.18 A b	15.75 ±0.27 A a	14.04 ± 0.17 B bcd	14.34 ± 0.18 B ab
5	15.55 ± 0.19 A b	15.95 ±0.28 A a	13.78 ± 0.18 B cd	14.20 ± 0.18 B cd
6	15.90 ± 0.20 A a	15.95 ±0.28 A a	13.78±0.18 B cd	14.20 ± 0.18 B ab
7	15.55 ± 0.20 A a	16.35 ±0.27 B ab	13.45±0.18 C d	13.91±0.18 C b

Values are expressed as mean ±SE, n=5. Different small letters represent significant difference within group (P<0.05) vs. zero time .Different capital letters represent a significant difference between groups (P<0.05) vs. control.

Table, 2: Effect of sodium selenite and melatonin on serum total cholesterol (TC) concentration (mg/dl) in acrylamide treated rats.

Groups Periods (Weeks)	G1 Control	G2 Acryl amide (1mg/Kg B.W)	G3 Acrylamide(1mg/Kg B.W) and sodium selenite (0.5 mg/Kg B.W)	G4 Acrylamide (1mg/Kg B.W) and melatonin (8mg/Kg B.W)
Zero	77.39 ± 1.40 A a	79.77 ± 0.93 A b	75.92 ± 0.73 A b	76.35 ± 0.67 A b
7	78.73 ± 1.43 D a	128.49 ± 2.54 A a	119.85 ± 0.73 B a	99.32± 5.33 C a

Values are expressed as mean ±SE, n=5. Different small letters represent significant difference within group (P<0.05) vs. zero time. Different capital letters represent a significant difference between groups (P<0.05) vs. control.

Table, 3: Effect of sodium selenite and melatonin on serum triacylglycerol (TAG) concentration (mg/dl) in acrylamide treated rats.

Groups Periods (weeks)	G1 Control	G2 Acrylamide (1mg/Kg B.W)	G3 Acrylamide(1mg/KgB.W) and sodium selenite (0.5 mg/Kg B.W)	G4 Acrylamide (1mg/KgB.W) and melatonin (8mg/Kg B.W)
Zero	152.91 ±1.46 A a	155.16 ±1.61 A b	153.13 ±1.61 A b	156.96 ±2.02 A b
7	152.14± 1.40 D a	225.22±2.31 A a	199.08 ±2.01 B a	190.69± 3.02 C a

Values are expressed as mean ±SE, n=5. Different small letters represent significant difference within group (P<0.05) vs. zero time. Different capital letters represent a significant difference between groups (P<0.05) vs. control.

Table, 4: Effect of sodium selenite and melatonin on serum high density lipoprotein (HDL-C) concentration (mg/dl) in acrylamide treated rats.

Groups Periods (weeks)	G1 Control	G2 Acrylamide 1mg/Kg	G3 Acrylamide(1mg/Kg B.W) and sodium selenite(0.5mg/Kg B.W)	G4 Acrylamide (1mg/KgB.W) and melatonin (8mg/KgB.W)
Zero	47.39 ± 0.00 A a	44.80 ±1.55 B a	45.29 ± 0.74 AB a	44.68 ± 0.83 B a
7	45.72 ± 0.47 A a	23.46 ±1.48 C b	34.57 ± 1.10 B b	32.45 ± 0.71 B b

Values are expressed as mean ±SE, n=5. Different small letters represent significant difference within group (P<0.05) vs. zero time. Different capital letters represent a significant difference between groups (P<0.05) vs. control.

Table, 5: Effect of sodium selenite on serum uric acid concentration (mg/dl) in acrylamide treated rats.

Groups periods (weeks)	G1 Control	G2 Acrylamide (1mg/Kg B.W)	G3 Acrylamide(1mg/KgB.W) and sodium selenite (0.5mg/Kg B.W)	G4 Acrylamide (1mg/KgB.W) and melatonin (8mg/Kg B.W)
Zero	1.47± 0.15 A a	1.41± 0.04 A b	1.46 ± 0.10 A b	1.43 ± 0.11 A b
7	1.50 ± 0.10 C a	3.41 ± 0.10 A a	2.50 ± 0.04 B a	2.36 ± 0.25 B a

Values are expressed as mean ±SE, n=5. Different small letters represent significant difference within group (P<0.05) vs. zero time .Different capital letters represent a significant difference between groups (P<0.05) vs. control.

Similar results concerning dyslipidemia (elevation in TAG, TC and decrease in HDL-C) accompanied ACR treatment was observed by (15 and 16). Such results could be attributed to the increment in the synthesis of plasma lipoprotein and high mobilization of lipid from liver (17). Insulin resistance induced after ACR exposure (18 and 19) may

lead to atherogenic dyslipidemia through suppression of lipolysis in adipocyte due to impairment in insulin signaling (20), elevated levels of free fatty acids (FFA) in the liver that serves as a substrate for the synthesis of TAG leading to triacyglycerolemia. Visceral obesity, recognized by increased waist circumference, is the considered to be the first sign associated with metabolic syndrome (21). Visceral fat appears to be the most detrimental contributor to development of lipotoxicity in peripheral tissue by adipocytokines secretion (22). Adipocytokines secretion integrate autocrine and paracrine signal that mediate insulin sensitivity (23) and oxidative stress (24). The two criteria contribute to ACR toxicity and may claimed to be responsible for visceral obesity accompanied ACR. On the other hand, positive correlation was found between adiponectin and HDL-C level (25), therefore, it can be concluded that IR accompanied ACR treatment may lead to decrease in adiponectine which coincide with the increase in visceral obesity as well as depression in HDL-C concentration.

It is speculated that in situations where free radical levels are high (e.g., obesity-related oxidant stress), Selenium supplementation will reduce free radical production and serum lipid composition (26), and thus visceral obesity. Selenium supplementation caused an increase in low-density lipoprotein (LDL) receptor activity and decrease the 3-OH-methyl-glutaryl CoA reductase expression (27) leading to decreased plasma LDL cholesterol and total cholesterol levels (28). This may be explaining the hypolipidemic effect of selenium. The result also showed significant increase in serum HDL-C concentration after sodium selenite supplementation indicating protective role against cardiovascular disease. Sodium selenite supplementation significantly increased the protein and MRNA expressions of apoA-I causing to elevation in HDL-C concentration (29).

The postulated anti-obesogenic effect of melatonin is, in part, a result of its regulatory role on the balance of energy, acting mainly on the regulation of the energy flux to and from the stores and in energy expenditure (30). Additionally, it was demonstrated that even with an intact pineal production of melatonin, melatonin supplementation therapy in young animals reduces the size of the visceral fat deposits (by 50%) (31). Long-term administration of melatonin to humans and experimental animals reduces blood and liver cholesterol and LDL-cholesterol levels (32) and increased HDL-cholesterol in peri- and postmenopausal women (33). It has been proved that melatonin presence in the gall bladder enhanced conversion of cholesterol to bile, preventing oxidative stress causing hypocholesterolemia (34). Melatonin also decrease the amount of cholesterol produced in the gall bladder by regulating the cholesterol that passes through the intestinal wall.

Data regarding the effect of ACR on serum uric acid concentration (Hyperuricemia) in the current study is in accordance with other studies (19 and 35). A case of dyslipidemia referred to hyper cholesterolemia, elevation in serum TAG and reduction in HDL-C concentration was found to be correlated with hyperuricemia and metabolic syndrome by some investigators (36). In the current study serum lipid profile was similarly affected by ACR which may explain its mechanism in hyperuricemia. Significant elevation in the reactive oxygen species and lipid peroxidation inducing oxidative stress by ACR accompanied by depletion in the antioxidant level of kidney (37) could impaired renal function leading to hyperuricemia.

Sodium selenite supplementation caused reduction in serum creatinine, urea and uric acid in rats exposed to acryl amide suggesting the protective effect of selenium on renal function (35 and 38). The antioxidant properties of selenium could be attributed to its renoprotective effect. Oxidative stress induced complications of diabetes may include stroke, neuropathy, retinopathy and nephropathy (39) and thus improving oxidative stress by selenium will lead to renoprotection and decrease in uric acid. Hyporuricemia induced after melatonin treatment could be attributed to its antioxidant effect that restored renal function. Melatonin stimulates synthesis of antioxidant enzymes, increased activity of others and protect

antioxidant enzyme from damage (40 and 41). Serum creatinine and urea nitrogen concentration was significantly restored to their normal values after co-treatment with melatonin in different types of nephrotoxicity (42) indicating its renoprotective effect.

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تأثيـــر السيلينيوم والميلاتونين في بعض المعاييرالمتعلقة بمتلازمة الأيض المستحدث بالاكريلامايد في ذكور الجرذان (المبحث الأول)

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الخلاصة

صُممت هذه الدراسة لمعرفة الدورالتحسيني لسيلينات الصوديوم والميلاتونين في بعض المعايير المتعلقة بمتلازمة الأيض في ذكور الجرذان المستحدثة باستعمال الاكريلامايد. استعملت (20) من ذكور الجرذان البالغة قسمت عشوائيا على اربع مجاميع (4) متساوية وعوملت لمدة (7) اسابيع: المجموعة الاول (G1) اعطيت الماء مقطر وعدت مجموعة سيطرة, في حين جرعت جرذان المجموعة الثانية (G2) فمويا"1ملغم/ كغم من وزن الجسم من الاكريلامايد، وأعطيت حيوانات المجموعة الثالثة (G3) فضلاً عن الاكريلامايد (1ملغم/كغم من وزن الجسم)، 0.5 ملغم/كغم من وزن الجسم من سيلينايت الصوديوم وأما حيوانات المجموعة الرابعة (G4) فقد حقنت عبر الغشاء البريتوني 8 ملغم/كغم من وزن الجسم من الميلاتونين بعد تجريعها فموياً 1ملغم/كغم من وزن الجسم. جمعت عينات الدم في بداية التجربة وبعد 7 اسابيع من التجربة بطريقة الوخز القلبي لغرض قياس تركيز الكولسترول الكلي الدهون الثلاثية، الكولسترول في الشحوم البروتينية عالية الكثافة وحامض اليوريك فضـلاً عـن قياس الخصر للدلالة على السمنة المركزية. اظهرت النتائج حدوث متلازمة الأيض بالاكريلامايد تمثلت بالسمنة المركزية واضطراب في ايض الدهون من خلال أرتفاع في تركيز الكولسترول والدهون الثلاثية مع انخفاض تركيز الكولسترول في الشحوم البروتينية عالية الكثافة وأرتفاع في تركيز حامض اليوريك. واظهرت النتائج أيضاً الدور الايجابي للسيلينات الصوديوم والميلاتونين عن تقليل حدة المعايير السابقة متمثلة بحدوث انخفاض في السمنة المركزية وتركيز الكولسترول والدهون الثلاثية وارتفاع في تركيز الكولسترول في الشحوم البروتينية عالية الكثافة مع أنخفاض في تركيز حامض اليوريك.

الكلمات المفتاحية: الأكريلاميد، متلازمة التمثيل الغذائي، الميلاتونين، الصوديوم سيلينات، دسليبيدميا.