



The Neuroprotective Role of Purslane in a chronic model of Depression Induced By reserpine in mice: Prevention of Behavioral, Mitochondrial and Neuro Dysfunction.

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ABSTRACT

Portulaca oleracea is a universal species with a broad range of biological activities, including antioxidant and neuroprotective actions. The present study aimed to estimate the neuroprotective effect of purslane ethanolic extract (PEE) and aqueous extract (PAE) on depression model induced by reserpine. Methods: Seventy five male mice were divided into 5 groups (15 mice of each). 1st group control (C), 2nd group treated with reserpine 0.1 mg/kg (Res) as a single dose at the day 15th of the beginning of experiment, 3rd group treated with Escitalopram 1mg/kg for 15 days plus 0.1 mg/kg Res at a latest day of the treatment. 4th group treated with PEE 2 mg/kg for 15 days plus 0.1 mg/kg Res at a latest day of the treatment. 5th group treated with PAE 5 g/kg for 15 days plus 0.1 mg/kg Res at a latest day of the treatment. Forced swimming test (FST) was performed 1 hour later after Res treatment. Results: Revealed data showed that the Res treated group induced significant ($P>0.05$) decrease in FST, decrease of brain monoamines [norepinephrine (NE), dopamine (DA) and serotonin (5HT)], brain cell energy [adenosine triphosphate (ATP)] increase metabolic energy [adenosine diphosphate (ADP) and adenosine monophosphate (AMP)]. On the other hand other treatment groups showed significant amelioration in comparing with Res group and almost recovery in comparing with control group. Conclusion: Obtained data concluded that PEE more potent than PAE, ameliorate depression induced by reserpine and act as a neuroprotective, neuromodulator and stimulate monoamines secretion.

الدور الوقائي العصبي للرجلة في نموذج مزمن للاكتئاب الناجم عن ريزيربين في الفئران: الوقاية من الخلل السلوكي والميتوكوندريا والعصبي.

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الكلمات المفتاحية:

الوقاية العصبية
الرجلة
الريزيربين
الاسيتالوبرام
الفئران
الاكتئاب

المخلص

الرجلة هو نوع عالمي له مجموعة واسعة من الأنشطة البيولوجية، بما في ذلك مضادات الأكسدة والإجراءات الوقائية للأعصاب. هدفت الدراسة الحالية إلى تقدير التأثير الوقائي العصبي لمستخلص الرجلة الإيثانولي والمستخلص المائي على نموذج الاكتئاب الناجم عن الريزيربين. الطريقة: تم تقسيم الفئران إلى خمسة مجموعات تضم كل مجموعة خمسة عشر فأراً، المجموعة الأولى الضابطة أعطيت توين ايتي عن طريق الفم بجرعة (1 ملجم/100 جم) من وزن الجسم، المجموعة الثانية تم إعطاؤها مثل المجموعة الضابطة ثم حقنت بالريزيربين (0.1 ملجم/كجم)، حصلت المجموعة الثالثة على خلاصة العصارة المائية لنبات الرجلة 5 جم/كجم. المجموعة الرابعة تم إعطاؤها جرعة من خلاصة عصارة الكحول الإيثيلي لنبات الرجلة بمقدار 2 ملجم/كجم. المجموعة الخامسة أعطيت 1 ملجم/كجم من الاسيتالوبرام. أعطي كل من الرجلة والاسيتالوبرام كجرعة واحدة يومياً عن طريق الفم من بداية التجربة وحتى اليوم السادس عشر. استمرت هذه التجربة لمدة ستة عشر يوماً. حقنت فئران

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المجموعات 2، 3، 4 و5 بجرعة واحدة من الرزيربين (0.1 ملجم/كجم) في اليوم الخامس عشر فقط من بداية التجربة. ثم أجري عليهم اختبار للسباحة بعد مرور ساعة من حقن الرزيربين. كانت هناك فترة راحة للفئران من اختبار السباحة في اليوم السادس عشر وفي النهاية كل المجموعات تم تشريحهم بالذبح في اليوم السابع عشر. النتائج: أظهرت البيانات التي تم الكشف عنها أن المجموعة المعالجة بـ الرزيربين تسببت في انخفاض معنوي ($P > 0.05$) في اختبار السباحة القصري، ونقص أحادي الأمين في الدماغ (النوربينفرين والدوبامين والسيروتونين)، و طاقة خلايا الدماغ [الأدينوزين ثلاثي الفوسفات. وزيادة الطاقة الأيضية (الأدينوزين ثنائي فوسفات والأدينوزين أحادي الفوسفات). من ناحية أخرى، أظهرت مجموعات العلاج الأخرى تحسناً ملحوظاً في المقارنة مع مجموعة الرزيربين وتقريباً الشفاء مقارنة بمجموعة التحكم. الخلاصة: خلصت البيانات التي تم الحصول عليها إلى أن المستخلص أليثانولي للدرجة أكثر فعالية من المستخلص المائي لهذا النبات، يخفف الاكتئاب الناجم عن الرزيربين ويعمل كواقي عصبي، ومعدّل عصبي، ويحفز إفراز أحادي الأمين.

Introduction

Depression is one of the most common neuropsychiatric disorders that affect approximately 20% of the world population [1]. It can be caused by one or more changes in the brain, which may or may not be directly related. These changes may include monoamine neurotransmitters depletion, cellular atrophy, neuronal death or decreased neurogenesis [2]. Also depression is a neuro-psychiatric illness that involves the whole body, mood and thoughts and affects the way a person eats, sleeps, feels about himself or herself and thinks about things. It is a mood disorder that causes a persistent feeling of sadness and loss of interest. Also called major depressive disorder or clinical depression, it can lead to a variety of emotional and physical problems [3].

Depression has been linked to problems or imbalances in the brain with regard to the Neurotransmitters Serotonin, Norepinephrine, and Dopamine. The evidence is somewhat indirect on these points because it is very difficult to actually measure the level of neurotransmitter in a brain. Antidepressant medications which used to treat depression are known to act upon these particular neurotransmitters and their receptors [4].

Reserpine appears naturally in the dried roots of plants such as *Rauwolfia Serpentina* and *Vomitoria*. It is an indole alkaloid antipsychotic and antihypertensive drug that has been used for the control of high blood pressure and for the relief of psychotic symptoms [5]. The antihypertensive actions of Reserpine are a result of its ability to deplete catecholamines from peripheral sympathetic nerve endings. These substances are normally involved in controlling heart rate, force of cardiac contraction and peripheral resistance [6]. After chronic administration of this drug some patients complained of major depression in 1950's [7]. On the other hand, Reserpine irreversibly blocks the intracellular vesicles monoamine transporters. This blockage overlap with the storage of monoamines into the intracellular vesicles, which effect in the depletion of catecholamines in nerve terminals and transient hypolocomotion and muscular rigidity [8].

Materials and methods:

Experimental animals:

Seventy five adult male albino mice were used weighing 20-25 g. purchased from National Research Centre (NRC, Cairo, Egypt). Upon arrival the animals were acclimatized for 7 days to a quiet colony room, with controlled ambient temperature (22 ± 2 °C) and a 12 hour natural light/dark cycle, fed a standard diet and water was provided ad libitum.

Preparation of extracts:

Preparation of aqueous extract of purslane:

The fresh *Portulaca oleracea* herb, mainly stem and leaves which are free of blemishes or obvious defects, an aqueous juice of the purslane herbs was prepared by mashing and left for about 24 h in a refrigerator. After mashing, the resulting crude extract was filtered and the filtrate was kept at -20°C for future use according to the method of [9].

Stem and leaves of *Portulaca oleracea* were extracted using ethanol, the method described by [10]. 2 kilogram of purslane was washed thoroughly and dried in oven 160°C . Dried samples became

(0.5 kg) were weighed and placed in 100 mL conical flasks and 80% (v/v) ethanol was added. The extract was concentrated under reduced pressure then centrifuged at 5000 rpm for 4 minutes. The mixture was refluxed for 2 h at 90°C and filtered through What man No. 1 filter paper (What man, UK). It is dried by evaporation of the filtrate under vacuum using a rotary evaporator (Buchi, Switzerland). The samples were weighed about 15 g and stored at -20°C .

Experimental design:

Seventy five male mice were divided into 5 groups (15 mice of each). 1st group served as control (C), 2nd group treated with reserpine 0.1 mg/kg (Res) as a single dose at the day 15th of the beginning of experiment which is the best choose dose of the previous study that induced depression with minimal side effect and decrease mortality rate, 3rd group treated with escitalopram 1mg/kg for 15 days plus 0.1 mg/kg Res at a latest day of the treatment (ESC+Res). 4th group treated with purslane ethanolic extract (PEE) 50 mg/kg for 15 days plus 0.1 mg/kg Res at a latest day of the treatment (PEE+Res). 5th group treated with purslane aqueous extract (PAE) 50 mg/kg for 15 days plus 0.1 mg/kg Res at a latest day of the treatment (PAE+Res).

Forced Swimming Test (FST):

Each mice was placed for 6 minutes in a cylindrical water tank (40 cm high, 40 cm diameter) where, water level was about 25 cm and water temperature was maintained at 23°C to 25°C . The total duration of immobility time and the Jumbling numbers of each animal in the last 4 minutes was recorded [11].

Biochemical assays

Brain tissue was homogenized in 75% aqueous HPLC grade methanol (10% w/v). The homogenate was spun at 4000 r.p.m. for 15 minutes and the supernatant was isolated. The detection of Adenosines (AMP, ADP & ATP) by HPLC was done according to the method of [12]. Brain monoamines (5-HT, NE & DA) were detected by HPLC according to method described previously [13].

Statistical Analysis

Reported values represent means \pm SE. Statistical analysis was evaluated by one -way ANOVA. Once a significant F test was obtained, LSD comparisons were performed to assess the significance of differences among various treatment groups. Statistical package for social science "SPSS" for Windows software, Release 18.0 (SPSS, Chicago, IL) was used at p value ≤ 0.05 .

Results:

Results of forced swimming test of mice of different experimental groups are illustrated in Table 1 and Figure (1, 2). Data revealed a significant increase in immobility time in Res group as compared to the control group. In the group treated with escitalopram and injected with reserpine (ESC + Res group) when compared to the Res group. In addition ESC + Res treated mice exhibited a significant increase in immobility time in comparison with PAE + Res and PEE + Res treated mice at ($P < 0.05$).

Moreover, it was noticed a significant decrease in immobility time in the group treated with aqueous extract of purslane and injected with reserpine (PAE + Res group) and in the group

treated with ethanolic extract of purslane and injected with reserpine (PEE + Res group) there was a significant decrease in immobility time as compared to the Res group. While marked increase was shown in PAE + Res and PEE + Res mice in immobility time when compared with the control. Number of jumping mice during forced swimming test of different treated groups revealed a significant decrease in number and percentage of jumping mice in Res, ESC +

Res and PAE + Res groups as compared to the control group. Moreover, a significant increase in the number and percentage of jumping mice in ESC + Res and PAE + Res groups as compared to Res group. Also the PEE + Res group recorded a significant increase as compared with PAE + Res and showed non-significant change with the control group at (P <0.05).

Table 1: Prophylactic effects of PAE or PEE on behavioral tests in reserpine induce rat's depression model.

Groups	Immobility time	% of Change	No. jumping	Jumping	% of Change from control
Control	40.00 ± 0.03	--	7	18.75 ± 2.92	-
Res	205.75 ± 0.05 ^a	414.375%	2	1.00 ± 0.68 ^a	- 94.6%
ESC + Res	68.38 ± 0.02 ^{abcd}	70.95%	4	8.63 ± 3.30 ^{ab}	- 53.9%
PAE + Res	57.63 ± 0.04 ^{ab}	44.075%	3	3.50 ± 1.73 ^a	- 81.3%
PEE + Res	50.50 ± 0.04 ^{ab}	26.25%	6	14.63 ± 3.27 ^{bc}	- 21.9%

Data are expressed as Mean ± S.E.M for 15 mice /group.

a significant difference from control group at the same column with one way ANOVA at P < 0.05.

b significant difference from Res at the same column with one way ANOVA at P < 0.05.

c significant difference from ESC + Res at the same column with one way ANOVA at P < 0.05.

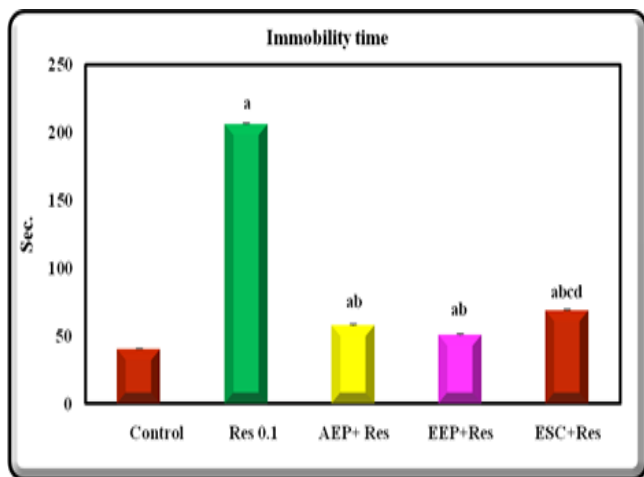


Figure (1): Showing immobility time (seconds) of different treated groups.

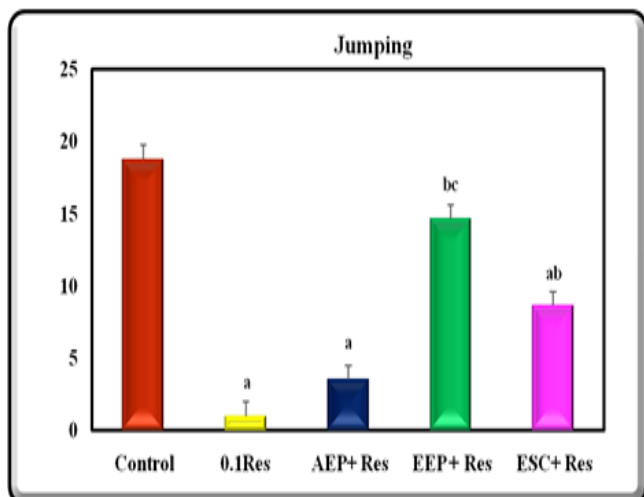


Figure (2): Showing number of Jumping of different treated groups.

The cortex and brain stem norepinephrine (NE), dopamine (DA) and serotonin (5-HT) of different experimental groups illustrated in Table 2 and Figures 3, 4. There was a significant decrease in cortex and brain stem NE contents in Res group as compared to the control group. Whereas, a significant decrease in cortex and brain stem NE

contents in ESC + Res group as compared to control group. At the same time, a significant increase was recorded when compared with Res group.

Moreover, AEP + Res group exhibited a significant decrease in cortex and brain stem NE contents in comparison to control group but was significantly increased as compared to the Res group. Also, there was a significant decrease in cortex NE in this group when comparison with ESC + Res group. In the EEP + Res group treated mice exhibited a significant increase in cortex and brain stem NE content when compared with Res and ESC + Res groups at P value ≤ 0.05.

Concerning cortex and brain stem DA contents, there was a significant decrease in Res group when compared to the control group. Whereas, there was a significant decrease in cortex and brain stem DA contents in ESC + Res group as compared to the control group, at the same time, there was a significant increase in comparison with Res group. Results of PAE + Res group revealed a significant decrease in cortex and brain stem DA as compared to the control and ESC + Res groups. While, mice of this group exhibited a significant increase in cortex and brain stem DA when compared to the Res group.

Also, EEP + Res group showed a significant increase in cortex DA content in comparison with the control. At the same time, in cortex and brain stem there was a significant increase as compared to the Res group. A significant increase in cortex and brain stem DA contents as compared to Res group. On the other hand, PEE + Res group showed a significant increase in DA contents in comparison with the control, Res and PAE + Res groups. However in brain stem there was a significant decrease as compared to the control group and at the same time there was a significant increase in comparison with Res group. The ESC + Res group exhibited a significant increase was shown in cortex and brain stem DA contents when comparison with reserpine group. In addition it showed a significant increase in cortex DA contents when compared with PAE + Res group and a significant increase in brain stem DA contents as compared with PEE + Res group at p value ≤ 0.05. in cortex NE contents in PEE + Res group as compared to PAE + Res group and Res group. While, brain stem NE contents of EEP + Res group showed a significant increase in comparison to Res group.

Regarding serotonin (5HT), there was a significant decrease in cortex and brain stem 5HT contents in all treated groups (Res, ESC + Res, PAE + Res and PEE + Res) except cortex PEE + Res which represented non-significant as compared to the control group. Also, a significant increase was recorded in cortex and brain stem 5HT contents in all treated groups (ESC + Res, PAE + Res and PEE + Res) as compared to the Res group at P value ≤ 0.05.

Table 2: Prophylactic effects of PAE or PEE on brain monoamines (NE, DA and 5HT) in reserpine induce rat's depression model.

Groups	Cortex			Brainstem		
	NE	DA	5HT	NE	DA	5HT
	µg/g tissue	µg/g tissue	µg/g tissue	µg/g tissue	µg/g tissue	µg/g tissue
Control	0.935	1.680	0.816	0.786	2.483	0.745
	±	±	±0.018	±	±	±
Res	0.032	0.022	0.251	0.030	0.069	0.028
	± 0.017 ^a	± 0.018 ^a	± 0.015 ^a	± 0.017 ^a	± 0.013 ^a	± 0.010 ^a
ESC + Res	0.803	1.343	0.665	0.574	2.145	0.617
	± 0.043 ^{ab}	± 0.042 ^{ab}	± 0.047 ^{ab}	± 0.029 ^{ab}	± 0.070 ^{ab}	± 0.027 ^{ab}
PAE + Res	0.507	1.030	0.619	0.603	1.666	0.601
	± 0.024 ^{abc}	± 0.020 ^{abc}	± 0.067 ^{ab}	± 0.035 ^{ab}	± 0.059 ^{abc}	± 0.035 ^{ab}
PEE + Res	0.864	1.426	0.758	0.649	1.941	0.646
	± 0.047 ^b	± 0.053 ^b	± 0.041 ^b	± 0.060 ^{bc}	± 0.084 ^{ab}	± 0.034 ^{ab}

Data are expressed as Mean ± S.E.M for 15 rats /group.
 a significant difference from control group at the same column with one way ANOVA at P < 0.05.
 b significant difference from Res at the same column with one way ANOVA at P < 0.05.
 c significant difference from ESC + Res at the same column with one way ANOVA at P < 0.05.

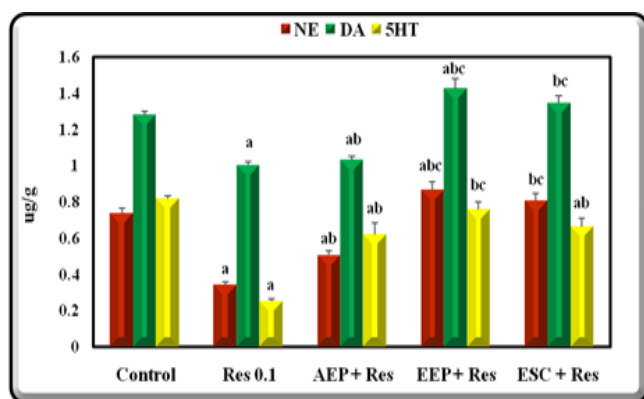


Figure (3): Showing cortex neurotransmitters (NE, DA and 5HT), (µg/g) of different treated groups.

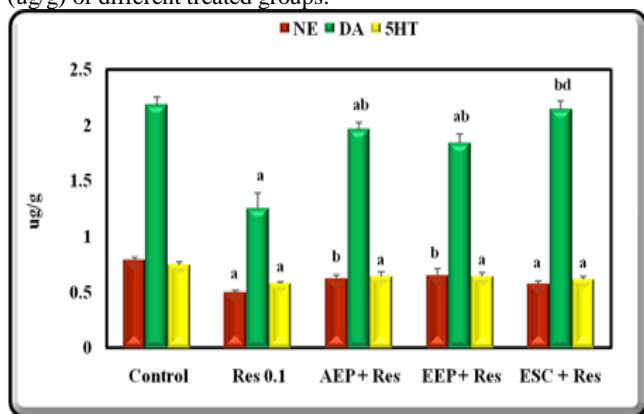


Table 3: Prophylactic effects of PAE or PEE on brain cell energy (ATP, ADP and AMP) in reserpine induce mice depression model.

Groups	Cortex			Brain stem		
	ATP	ADP	AMP	ATP	ADP	AMP
	µg/g tissue	µg/g tissue	µg/g tissue	µg/g tissue	µg/g tissue	µg/g tissue
Control	30.3 ± 0.96	10.4 ± 0.13	7.0 ± 0.446	30.3 ± 0.89	17.9 ± 1.12	9.0 ± 0.12
Res	16.7 ± 0.35 ^a	17.7 ± 0.37 ^a	6.8 ± 0.255	19.8 ± 1.01 ^a	25.3 ± 1.51 ^a	10.3 ± 0.53
ESC + Res	25.6 ± 0.80 ^{ab}	9.1 ± 0.50 ^b	7.8 ± 0.337	25.6 ± 0.99 ^{ab}	15.7 ± 1.38 ^b	11.8 ± 0.63
PAE + Res	16.8 ± 0.47 ^a	11.2 ± 0.33 ^b	6.4 ± 0.316	23.9 ± 1.13 ^{ab}	17.1 ± 1.28 ^b	10.3 ± 0.53
PEE + Res	26.8 ± 0.65 ^{ab}	8.8 ± 0.36 ^b	6.7 ± 0.594	27.7 ± 1.47 ^b	15.6 ± 1.49 ^b	11.1 ± 0.99

Data are expressed as Mean ± S.E.M for 15 rats /group.
 a significant difference from control group at the same column with one way ANOVA at P < 0.05.
 b significant difference from Res at the same column with one way ANOVA at P < 0.05.
 c significant difference from ESC + Res at the same column with one way ANOVA at P < 0.05.

Figure (4): Showing brain stem neurotransmitters (NE, DA and 5HT), (µg/g) of different treated groups.

The ATP, ADP and AMP content of the cortex and brain stem in different experimental groups illustrated in Table 3 and Figures 5, 6. In cortex and brain stem ATP contents, there was a significant decrease in Res group as compared to the control group. On the other hand, there was a significant decrease in cortex and brain stem ATP contents in ESC + Res group as compared to the control group, at the same time, there was a significant increase in comparison with Res group. Results of PAE + Res group revealed a significant decrease in cortex and brain stem ATP as compared to the control group. Moreover, mice of this group exhibited a significant increase in brain stem ATP when compared to the Res group at P value ≤ 0.05.

Also, EEP + Res group showed a significant decrease in cortex ATP content in comparison with the control group. However, in cortex and brain stem in this group there was a significant increase as compared to the Res group. Data revealed a significant increase in ADP in cortex and brain stem in Res group as compared to the control group. Moreover, cortex and brain stem ATP contents showed a significant decrease in all treated groups (ESC + Res, PAE + Res and PEE + Res) as compared to the Res group.

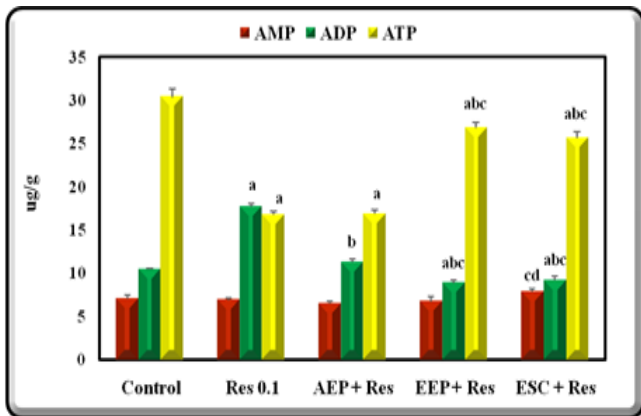


Figure (5): Showing cortex AMP, ADP and ATP (ug/g) of different treated groups.

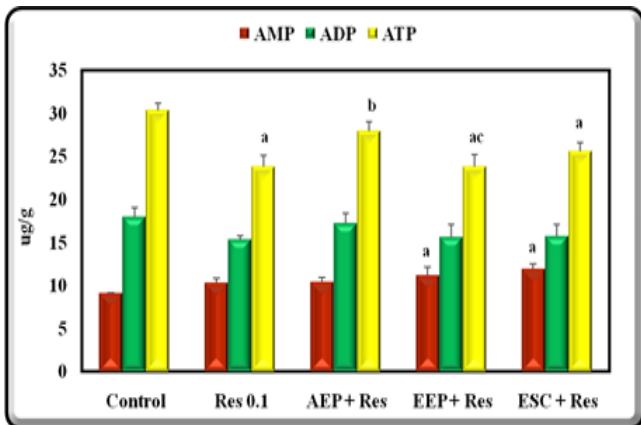


Figure (6): Showing brain stem AMP, ADP and ATP (ug/g) of different treated groups.

Discussion:

In the present study, results of forced swimming test for reserpine group revealed a significant increase in immobility time and decrease in number of jumping as compared to control group due to reserpine induced depression associated with despair and giving up and leave themselves to drowning. The result is in agreement with [14] and [15]. They noticed that the increase in immobility durations in the FST along with other physiological changes represented by prolonged monoamines depletion and oxidative stress. Additionally, the FST has been shown to share some of the factors that are influenced or altered by depression in humans including changes in food consumption, sleep abnormalities and drug withdrawal induced anhedonia. Also, in agreement with the present results [16], who found that the increase in immobility time shown by mice when subjected to unavoidable stress such as forced swimming test is thought to reflect a state of despair or lowered mood, which is thought to reflect depressive disorders in humans. The increase in immobility time was attributed to reserpine induced depression, this related to the mood state of the animal and brain levels of noradrenaline, dopamine, serotonin and cholinergic neurotransmitters. These neurotransmitters are involved in the pathophysiology of some types of depression and play a permissive role in the antidepressant activities. The mechanism by which reserpine induce depression is related to depletion of CNS bioamine activity.

In addition, the immobility time is reduced by treatment with antidepressant drugs. From the current study it was noticed that the groups (ESC + Res group, AEP + Res group and EEP + Res group), there was a significant decrease in immobility time and increase in number of jumping as compared to the reserpine group. The purslane exerted antidepressant activity which decreased neurobehavioral dysfunction [17]. In the same line, results of [18] showed that the enriched C-glycosyl flavonoids fraction of *Cecropia pachystachya* explained antidepressant-like effects in the forced swimming and decrease in immobility time. Moreover, purslane extends antidepressant activity because it contains flavonoids, which raised

the level of movement, it contain omega-3, omega-6 and phenolic compounds as antioxidants is also a significant neuronal defense mechanism against apoptosis and thereby preventing atrophy of brain regions in stress-induced depression, This effect Help to overcome the oxidative stress and its effects harmful on proteins, lipids, nuclear material (i.e., DNA), mitochondrial dysfunction and ATP level.

The mechanism of antidepressant action of escitalopram which is the most selective compound of the presently available SSRIs is related to selective activity at the rise of the serotonergic system in the CNS by inhibiting the reuptake of serotonin (5-HT). This mechanism, because of its highly selective nature, helps preclude the direct influence of other neurotransmitters, such as norepinephrine (NE) or dopamine (DA). It is worth remembering that behavioral effects spotted in the FST were not associated with the increase in spontaneous locomotor activity of animals [19]. Escitalopram most likely exerts its antioxidant effect by inhibiting the reuptake of serotonin by means of selective inhibition of presynaptic 5- hydroxytryptamine (5-HT) uptake site.

The present results are coincide with [20] the most of serotonergic, noradrenergic and dopaminergic neurons are located in midbrain and brainstem nuclei and project to large areas of the entire brain. This anatomy suggests that monoaminergic systems are involved in the regulation of a broad range of brain functions, including mood, attention, reward processing, sleep, appetite, and cognition. Almost every compound that inhibits monoamine reuptake, leading to an increased concentration of monoamines in the synaptic cleft, has been proven to be a clinically effective antidepressant. Inhibiting the enzyme monoamine oxidase, which induces an increased availability of monoamines in presynaptic neurons, also has antidepressant effects. These observations led to the pharmacologically most relevant theory of depression, referred to as the monoamine deficiency hypothesis. Reserpine is a vesicular monoamines reuptake blocker which depletes monoamines in the brain, and produces depression-like syndrome in animals. Experimentally reduced central serotonin has been associated with mood congruent memory bias, altered reward-related behaviors, and disruption of inhibitory affective processing [21]. purslane increased the levels of monoamines neurotransmitters (serotone, dopamine and norepinephrine, acetylcholinesterase activity) of the rats brain and attenuate the effect of reserpine better than the escitalopram. This increase was due to its content of melatonin, omega-3 fatty acid, flavonoids and phenolic compounds and other active ingredients nutrients including an enough intake of micronutrients, essential fatty acids, amino acids and antioxidants, is essential for both physical and mental which are particularly important for brain development and prevent degenerative changes in neurons. one of the antidepressant mechanisms of purslane thought to involve flavonoid glycosides, which reach the brain tissues through the metabolizing process, protecting brain function from CNS disturbance, and consequently, exerting an antidepressant effect [22].

The present study demonstrated that the cortex and brain stem content of the adenyly system compounds (AMP, ADP and ATP) of group given reserpine showed a significant decrease when compared to the control group. In supporting to the present study, [23] decrease ATP level may be an indicator for neurodegenerative disorder results in suppression of energy in the neurons. Another interesting issue showed by [24] who observed that depression is a cellular energy problem involving the whole person. The cells and essential components of the cell, the mitochondria, are not receiving the requisite nutrients. The mitochondria of the cells are the biological energy factories. With individuals reporting depression, physical exercise is usually minimal. Lack of exercise contributes to the decreased energy in the mitochondria. Also, mitochondrial dysfunction indicated by declined complex I activity and adenosine triphosphate (ATP) level and increased apoptosis. The present study revealed that results of ATP-cortex and brain stem in mice treated with ethanolic extract of purslane and reserpine recorded a significant increase in comparison to reserpine group but still significantly lower than control group. In agreement with these results, [25] stated that *Portulaca oleracea* are characterized by highest amount of protein, ash, phenyls, flavonoids and carotenoids. This rich source of energy,

antioxidants and minerals reflects its benefit as remedial materials, in improving cortex and brain stem ATP contents in human. Escitalopram treatment significantly attenuated the dysfunctional mitochondrial enzyme complex. Inhibition of mitochondria permeability transition and extracellular catabolism of ATP can be brought about by antidepressants. Therefore, it is possible to suggest that changes induced by antidepressants on mitochondrial complexes may be due to modulating ATP levels in the mitochondria of brain [26].

Conclusion:

In conclusion the results of this study collectively emphasized that reserpine as a drug rarely used today due to its numerous side-effects. Reserpine causes subsequent depression in humans by depletion of monoamine neurotransmitters in the synapses. It is also concluded that purslane is a promising natural product, which could be useful for the prevention of depression, neurodegenerative and other chronic diseases caused by oxidative stress.

Escitalopram gives good results as a drug for treating from depression. Whereas, the purslane gives the best results than escitalopram. Consequently it was clearly noticed that ethanolic extract of purslane had more potent antidepressant activity than aqueous extract of purslane. In general, purslane provided a new dimension in herbal remedy of depression and neurological disorders.

Abbreviations and Acronyms:

PEE= purslane ethanolic extract.

PAE = purslane aqueous extract.

RES = reserpine.

ESC = Escitalopram.

NE = norepinephrine.

DA = dopamine.

5HT = serotonin.

ATP = adenosine triphosphate.

ADP = adenosine diphosphate.

AMP = adenosine monophosphate.

FST = Forced swimming test.

CNS= central nervous system

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