INTERACTION OF NARDOSTACHYS JATAMANSI ROOT EXTRACT ON NICOTINE INDUCED OXIDATIVE STRESS IN THE SKELETAL MUSCLE TISSUE OF MALE ALBINO RAT

K. Chennaiah¹, M. Jayachandrudu¹, Kamakshamma ²
¹Department of Zoology, Sri Venkateswara University, Tirupati, Andhra Pradesh, India
²Department of Botany, Sri Venkateswara University, Tirupati, Andhra Pradesh, India

Correspondence should be addressed to K. Chennaiah

Received November 23, 2015; Accepted November 30, 2015; Published December 24, 2015;

Copyright: © 2015 K. Chennaiah et al. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Cite This Article: Chennaiah, K., Jayachandrudu, M., ., K.(2015). Interaction of nardostachys jatamansi root extract on nicotine induced oxidative stess in the skeletal muscle tissue of male albino rat. Pharmacology & Toxicology Research, 2(1).1-9

ABSTRACT

Nardostachys jatamansi (N.J) is a small, perennial, dwarf, hairy, rhizomatous, herbaceous, endangered and most primitive species within family Valerianaceae. Nicotine as most biologically active chemical in tobacco(Family; Solonacae) smoke. Nicotine has been reported to induce changes both in vivo and in vitro. Pathogen free, Wistar strain male albino rats were used in the present study. Rats were divided into 4 groups of six in each group i) Normal Control (NC) (Control rats received 0.9% saline) ; ii) Nicotine treated (Nt) (at a dose of 0.6 mg/ kg body weight by subcutaneous injection for a period of 2 months ); iii) Nardostachys Jatamansiextract treated (NJEt) (50mg/kg body weight) via orogastric tube for a period of 2 months); IV)) Nicotine + Nardostachys Jatamansiextract treated (Nt+NJEt), rats were received the nicotine at a dose as mentioned in Group II through subcutaneous injection and N Jatamansiextract as mentioned in Group III via orogastric tube for a period of 2 months. The animals were sacrificed after 24 hrs after the last treatment by cervical dislocation. Isolated the skeletal muscle tissue and measured the activity levels of Superoxidedismutase (SOD), Catalase (CAT), Glutathione (GSH) and Glutathioneperoxidase (GPx). The decrease was observed in nicotine treated rats and increase was observed in NJ rats over the control. . In the combination treatment (Nt+NJEt) up regulation was observed. This results stating that Nardostachys Jatamansiextracts are beneficial, especially for the nicotine subjects improve the antioxidants enzymes status.

KEY WORDS: Nicotine, Nardostachys Jatamansi extract, SOD, CAT, GSH, GPx, Skeletal muscle and Male albino rat

INTRODUCTION

Nardostachys jatamansicommonly known as muskroot, is indigenous to the Himalayan regions of India. Its a small, perennial, dwarf, hairy, rhizomatous, herbaceous, endangered and most primitive species within the family of Valerianaceae. India has been using its rich biodiversity in the healthcare segment for many years. Its rich traditional experience and wisdom is ensconced in the Ayurveda and Siddha systems of medicine. The uninterrupted use and the popularity of herbal formulations for thousands of years and the belief that Ayurvededic medica could be a viable resource for modern drug development has been a strong incentive for the Research and Development into the herbal wealth of Ayurveda. Traditional medicine is one of the common forms of therapy across the world. It is generally known that the consumption of a variety of local herbs and vegetables by man contributes significantly to the improvement of human health, in terms of prevention and or cure of diseases.
because plants have long served as a useful and natural source of the therapeutic agents. According to WHO, about three quarters of the world’s population use herbs and other forms of medicine in treatment of various ailments. Anand [1] reported it is estimated that approximately 1000 herbal formulations prepared from 750 plants are in regular use at the present time. With the shifting of attention from synthetic drugs to natural plant products, the use of plant extracts for enhancing growth performance in animals is now in the increase. Plants that were once considered of no value are now being investigated, evaluated and developed into drugs with little or no side effects [2].

Traditionally the plants have been used from many years for nervous headache, excitement, menopausal symptoms, flatulence, epilepsy and intestinal colic. Nadkarni [3] reported the root is of bitter taste and used as aromatic, antispasmodic, diuretic, emmenagogue and nerve sedative in the Indian system of medicine. In Ayurveda, roots and rhizomes of *N. jatamansi* are used to treat hysteria, epilepsy, and convulsions [4]. The decoction of the drug is also used in neurological disorders, insomnia and disorders of cardiovascular system [5]. According Nishateswar [6] and Sharma [7], the bitter tonic obtained from the rhizomes of the Jatamansi, also called Bhutajata, Jathilaa, Thapaswini can be used as a neuroprotective, sunscreen, stimulant, antispasmodic, repellent, antipyretic, antioxidant, as well as to treat herpes infection, leprosy, various neuropsychiatric illnesses, and excessive thirst. Sukhdev [8] reported it also has other Ayurvedic applications such as in complexion, strength, kidney stones, jaundice, removes blood impurities, spasmodic hysteria and other nervous convulsive ailments; heart palpitations, nervous headache, flatulence, anemia, convulsions, respiratory and digestive diseases, skin diseases, typhoid, gastric disorders, and seminal debility.

Nicotine is a naturally occurring alkaloid found primarily in the members of the Solanaceae family, which includes tobacco, potato, tomato, green pepper, and eggplant. Nicotine was first isolated and determined to be the major constituent of tobacco in 1828 [9]. In commercial tobaccos, the major alkaloid is nicotine, amounting for about 95% of the total alkaloid content [10]. Tobacco use is the leading cause of death in the world today. With 4.9 million tobacco-related deaths per year, no other consumer product is as dangerous or kills as many people as tobacco [11].

Nicotine, as most biologically active chemical in tobacco smoke, has been the subject of intense scientific scrutiny. Among the most well-characterized chemicals found in tobacco and tobacco smoke, are polycyclic aromatic hydrocarbons [PAHs] and the highly addictive alkaloid, nicotine and its metabolites [12]. To further complicate the picture, nicotine is converted, during the production of cigarette and chewing tobacco, into two highly mutagenic nitrosamine, *N*-nitroso nicotine [NNN] and 4-[methylnitrosamine]-1-[3-pyridyl]-1-hexanone [NNK] and is metabolized into cotinine. These chemicals derivatives also exhibit a wide spectrum of biological activity as compared to parent compound [12]. Nicotine has been reported to induce oxidative stress both in vivo and in vitro [13]. The mechanism of generation of free radicals by nicotine is not clear. But oxidative stress occurs when there are excess free radicals and/or low antioxidant defense, and result in chemical alteration of biomolecules causing structural and functional modification. Oxygen free radicals [OFR] production has been directly linked to oxidation of cellular macromolecules, which may induce a variety of cellular responses through generation of secondary metabolic reactive species [14].

Skeletal muscle is a form of striated muscle tissue which is under the voluntary control of the somatic nervous system. It is one of three major muscle types, the others being cardiac muscle and smooth muscle. Most skeletal muscles are attached to bones by bundles of collagen fibers known as tendons. Skeletal muscle is made up of individual muscle cells or myositis, known as muscle fibers. They are formed from the fusion of developmental myoblasts [a type of embryonic progenitor cell that gives rise to a muscle cell] in a process known as myogenesis. Muscle fibers are cylindrical, and multinucleated. Therefore, it is not only important for investigators studying skeletal muscle to have an appreciation for the diverse characteristics of muscle fibers but also to know the fiber composition of specific muscles, muscle groups, or the entire body musculature. This study was designed to investigate the effects of nardostachys Jatamansie extract on nicotine induced oxidative stress in the skeletal muscle tissue of male albino rat.

**RESEARCH MEHTODS**

**MATERIALS AND METHODS**

**Animals**

Male pathogenic free wistar albino rats were obtained from the Department of Zoology, Animal House, S.V. University, Tirupati and Andhra Pradesh, India. The animals were housed six to each polypropylene cage and provided with food and water ad libitum. The animals were maintained under standard conditions of temperature and humidity with an alternating 12hr light/dark. Animals were fed standard pellet diet [Agro Corporation Pvt. Ltd., Bangalore, India] and maintained in accordance with the guidelines of the National Institute of Nutrition and Indian Council of Medical Research, Hyderabad, India.

**Chemicals:**

Nicotine and other fine chemical were obtained from Sigma chemical company, St. Louis, USA. All other chemicals and reagent used were of analytical grade.

**Preparation of the [N.J] extract and mode of administration**

100 grams of Jatamansi root powder [Indian Remedies, India] in 90 % ethanol [11] at 50°C to 60°C in a Soxhlet extractor for 72 hours. The cooled liquid extract was concentrated by evaporating its liquid contents in rotary evaporator, with an approximate yield of 20%. The dried jatamansi ethanol extract was suspended in distilled water, and used for the present study.
EXPERIMENTAL DESIGN

Age matched rats were divided into 4 groups of six in each groups.

**Group I – Normal Control**

The rats were treated with normal saline [0.9%] orally via orogastric tube for for a period of 2 months.

**Group II – Nicotine treatment [Nt]:**

Rats were received the nicotine at a dose of 0.6 mg/kg body weight [0.5ml] by subcutaneous injection for a period of 2 months.

**Group III – Nardostachys Jatamansi extract treated [NJEt]:**

Rats were received N. jatamansi extract 50mg/kg body weight via orogastric tube for a period of 2 months.

**Group IV – Nicotine + Nardostachys Jatamansi extract treated [Nt+NJEt]:**

These Rats were received both nicotine [at a dose of 0.6 mg/kg body weight [0.5ml] by subcutaneous injection] and N. jatamansi extract was 50mg/kg body weight via or gastric tube for a period of 2 months.

The animals were sacrificed after 24 hrs after the last treatment session by cervical dislocation and the kidney tissue were isolated at -4º, washed with ice-cold saline, immediately immersed in liquid nitrogen and stored at -80º for enzymatic assays. Before assay, the tissues were thawed, sliced and homogenized under ice-cold conditions. Selected parameters were estimated by employing standard methods.

**BIOCHEMICAL INVESTIGATIONS**

**Superoxide dismutase** [SOD – EC: 1.15.1.6]

Superoxide dismutase activity was determined according to the method of Misra and Fridovich [15] at room temperature. The Skeletal muscle tissue was homogenized in ice cold 50 mM phosphate buffer [pH 7.0] containing 0.1 mM EDTA to give 5% homogenate [w/v]. The homogenates were centrifuged at 10,000 rpm for 10 min at 0ºC in cold centrifuge. The resulting supernatant was used as enzyme source. The reaction mixture consisted of 500 µl of phosphate buffer, 100 µl of 0.01 M GSH [reduced form], 100 µl of 1.5 mM NADPH and 100 µl of GR [0.24 units]. The 100 µl of tissue extract was added to the reaction mixture and incubated at 37ºC for 10 min. Then 50 µl of 12 mM t-butyl hydroperoxide was added to 450 µl of tissue reaction mixture and measured at 340 nm for 60 s in a UV spectrophotometer. The molar extinction coefficient of 6.22 X 10³ M cm⁻¹ was used to determine CAT activity. One unit of activity is equal to the moles of H₂O₂ degraded / mg protein / min.

**Catalase[CAT – EC: 1.11.1.6]:**

Catalase activity was measured by a slightly modified version of Aebi [16] at room temperature. The Skeletal muscle tissue was homogenized in ice cold 50 mM phosphate buffer [pH 7.0] containing 0.1 mM EDTA to give 5% homogenate [w/v]. The homogenates were centrifuged at 10,000 rpm for 10 min at 0ºC in cold centrifuge. The resulting supernatant was used as enzyme source. 10 µl of 100% EtOH was added to 100 µl of tissue extract and then placed in an ice bath for 30 min. After 30 min the tubes were kept at room temperature followed by the addition of 10 µl of Triton X-100 RS. In a cuvette containing 200 µl of phosphate buffer and 50 µl of tissue extract was added 250 µl of 0.066 M H₂O₂ [in phosphate buffer] and decreases in optical density measured at 240 nm for 60 s in a UV spectrophotometer. The molar extinction coefficient of 43.6 M cm⁻¹ was used to determine CAT activity. The results were expressed in µ moles of NADPH oxidized / mg protein / min.

**Glutathione content**

Glutathione content was determined according to the method of Theodorus et. al.[17]. The Skeletal muscle tissue was homogenized in 0.1M ice cold phosphate buffer [pH 7.0] containing 0.001M EDTA and protein is precipitated with 1 ml of 5% sulfosalicylic acid [w/v] and the contents were centrifuged at 5000 g for 15 min at 4ºC. The resulting supernatant was used as the enzyme source. The reaction mixture in a total volume of 2.5 ml contained 2.0 ml of 0.1M potassium phosphate buffer, 0.005 ml of NADPH [4 mg / ml of 0.5% NaHCO₃], 0.02 ml of DTNB [1.5 mg / ml], 0.02 ml of glutathione reductase [6 units / ml] and require amount of tissue source. The reaction was initiating by adding 0.41 ml of enzyme source and change in absorbance was recorded at 425 nm against the reagent blank. The glutathione content was expressed in nano moles / gram wet weight of the tissue.

**Glutathione peroxidase** [GSH-Px – EC: 1.11.1.9]:

Glutathione peroxidase [GSH-Px] was determined by a modified version of Flohe and Gunzler [18] at 37ºC. 5% [w/v] of Skeletal muscle tissue homogenate was prepared in 50 mM phosphate buffer [pH 7.0] containing 0.1 mM EDTA. The homogenates were centrifuged at 10,000 rpm for 10 min at 0ºC in cold centrifuge. The resulting supernatant was used as enzyme source. The reaction mixture consisted of 500 µl of phosphate buffer, 100 µl of 0.01 M GSH [reduced form], 100 µl of 1.5 mM NADPH and 100 µl of GR [0.24 units]. The 100 µl of tissue extract was added to the reaction mixture and incubated at 37ºC for 10 min. Then 50 µl of 12 mM t-butyl hydroperoxide was added to 450 µl of tissue reaction mixture and measured at 340 nm for 180 s. The molar extinction coefficient of 6.22 X 10³ M cm⁻¹ was used to determine the activity. One unit of activity is equal to the mM of NADPH oxidized / mg protein / min. The enzyme activity was expressed in µ moles of NADPH oxidized / mg protein / min.

**STATISTICAL ANALYSIS**

Statistical analysis has been carried out using INSTAT software. The data was analyzed for the significance; the results were presented with the P-values.
RESULTS

In the present study the Superoxide dismutase activity was decreased [-27.51%] in nicotine (Nt) treated rats when compared to control rats. In NJEt [Nardostachys jatamansi extract treated] rats an increase [+6.30%] was observed when compared to their respective control rats. In the combination treatment (Nt+NJEt) upregulation [+2.00%] was observed when compared to control rats. [Table-1].

In our study the Catalase activity was decreased [-33.85%] in nicotine treated rats when compared to control rats. In NJEt rats an increase [+15.78%] was observed when compared to the control rats. In the combination treatment (Nt+NJEt) slightly increase [+11.49%] was observed when compared to control rats [Table-2].

The Glutathione content was decreased [-13.18%] in nicotine treated rats when compared to control rats. In NJEt rats an increase [+5.81%] was observed when compared to the control rats. In the combination treatment (Nt+NJEt) slightly increase [+1.21%] was observed when compared to control rats [Table-3].

In the present study glutathione peroxidase activity was decreased [-38.88%] in nicotine treated rats when compared to control rats, in NJEt rats an increase [+8.56%] was observed. In the combination treatment (Nt+NJEt) slightly increase [+2.91%] was observed when compared to control rats [Table-4].

DISCUSSION

Superoxide dismutase [SOD] is the key and primary antioxidant enzyme in the cell. Cellular defense against superoxide radicals is provided by the enzyme superoxide dismutase. Among other antioxidant enzymes, SOD considered as front line of defense against the potentially cytotoxic free radical cause oxidative stress. The superoxide dismutase catalyzes the dismutation of two superoxide radicals $[O_2^-]_2$ into hydrogen peroxide $[H_2O_2]$ and oxygen. These enzymes obey first order reaction kinetics and the forward rate constants are almost diffusion limited. This results in steady state concentration of superoxide radicals in tissues that may be vary directly with the rate of superoxide generation and inversely with the tissue concentration of scavenging enzymes [19 and 20]. It is well known that SOD is involved in destroying the superoxide radical and exists in several isoforms different in both cellular location and the metal Co-factor bound to its active site.

In the present study decrease was absorbed in SOD activity in the skeletal muscle tissue due to nicotine treatment. The present results in the current investigation are in consistence with the previous findings. Among the generated free radicals due to nicotine metabolism, superoxide anion is the first derived free radical from nicotine. Thus, increased generation of superoxide radicals caused oxidative stress and damages the skeletal muscle cells. The decrease in SOD activity due to nicotine consumption may impairs the other antioxidant enzyme activities like catalase and glutathione peroxidase. Because the superoxide radicals that are produced in the skeletal muscle tissue during nicotine metabolism are quickly scavenged to $H_2O_2$ by the enzyme superoxide dismutase.

Similar studies have been reported by several authors. Chennaiah [21] reported due to nicotine treatment SOD activity was decrease in the muscle tissue. The depletion of SOD activity was may be due to dispose of the free radical, produced by the nicotine toxicity. Helen et al., [22] reported the decreased SOD activity in brain tissue of rat due to nicotine toxicity. E.L Sokkary et al., [23] reported chronic administration of nicotine the SOD activity was decreased in ovary tissue. N. jatamansi extract treated produce a beneficial effect by decreasing the levels of oxidative stress in the mitochondria of skeletal muscle tissue. In the combination treatment (Nt+NJEt) upregulation of SOD activity was observed, decrease in oxidative stress and increased activity of mitochondrial electron transfer enzymes, are logically related.

Catalase is one of the most important antioxidant enzyme. Catalase is widely distributed in the body compartments, tissues and cell. In many cases the enzyme is located in subcellular organelles such as, peroxisomes and cytosol of liver [25 and 26]. Mitochondria contain little amount of catalase. Catalase is a tetrameric peroxidative enzyme which converts the hydrogen peroxide to water and molecular oxygen and whose gene expression is regulated by $H_2O_2$. Catalase plays an important role in ROS metabolism and in adaptation to ‘oxidant stress [27 and 28]. Catalase catalysis the destruction of hydrogen peroxide into water and oxygen.

In the present we found that the CAT activity was decreased in the skeletal muscle tissue due to nicotine then the control. Related to the nicotine similar studies have been reported by several authors in different tissues . Chennaiah [1] reported due to nicotine treatment CAT activity was decreased in the muscle tissue. Helen et al., [22] reported the decreased CAT activity in brain tissue of rat due to nicotine toxicity. Avati et al., [29] reported chronic administration of nicotine the CAT activity was decreased in the rat kidney, liver and lung. The depletion of CAT activity was may be due to dispose of the freeradical, produced by the nicotine toxicity.

In the present study, in the skeletal muscle tissue of NJEt rats the CAT activity was increased. The increased catalase activity indicates its active involvement in the decomposition of hydrogen peroxide during nardostachhs jatamansi extract treated. A change in the binding characteristics of enzyme to membrane or their release from peroxisomes has been proposed as a possible mechanism for the increased activity levels of CAT [30]. CAT and SOD are considered to be indispensable for the survival of the cell against deleterious effects of hydroperoxides. The combination of SOD and CAT provide an efficient mechanism for removal of free radicals from the cell [31 and 32]. In addition to their antioxidant activity, polyphenols also possess many different biological properties. Normally phenolic compounds act by
scavenging free radicals and quenching the lipid peroxidative side chain. It has been proposed that hydroxyl and hydroperoxy radicals initiate hydrogen abstraction from a free phenolic substrate to form phenoxy radicals that can rearrange to quinone methide radical intermediates which is excreted via bile [33].

If the animals take regularly the N. jatamansi the activity of CAT would increase. NJEt may capture the induced hydrogen peroxides before escaping it from the cell and breakdown them to water and oxygen. In this way NJEt can maintain the ample catalase activity in the skeletal muscle tissue under the induced oxidative stress condition. The upregulation in CAT activity was found with response of combination [Nt+NJEt] in the skeletal muscle tissue rats. The combination treatment augmented CAT activity in the skeletal muscle, suggesting that NJEt may help to develop a resistance in the skeletal muscle tissue to cope with nicotine induced oxidative injury and maintains the antioxidant system.

Glutathione [GSH] is the most abundant intracellular thiol based antioxidant present in milli molar concentrations in all living aerobic cells, but there is a wide variability in glutathione content across organs depending on their basal levels of free radical production [34 and 35]. Glutathione serves as a sensitive marker of oxidative stress and it plays an important role in maintaining the integrity of the cell system [36]. GSH is involved in several reactions in the body and is one of the most prominent non-enzymatic antioxidant [37] that detoxifies reactive oxygen species. It functions as a substrate for glutathione peroxidases and scavenges free radicals, oxy-radicals and singlet oxygen produced during stress conditions. These radicals also it should be convert the reduced GSH to oxidized GSH [GSSG], thereby increasing the levels of GSSG [38].

In the present study we found that the administration of nicotine showing the decreased in GSH activity in the skeletal muscle tissue. Literature is not available related nicotine and skeletal muscle. However similar studies have been reported by several authors in different tissues. E.L Sokkary et al.,[23] reported chronic administration of nicotine the GSH activity was decreased in the rat kidney, liver and lung. Sener et al.,[39] reported chronic administration of nicotine the GSH activity was decreased in the rat tissues. Nicotine is oxidized primarily into its metabolite cotinine in the liver [40], generates free radicals/ROS in tissues [41], and induces oxidative tissue injury [42 and 43]. The decrease in GSH concentration in mitochondria would thus be highly responsible for ROS generation and the structural and functional damage in this organelle [44].

In the present study the GSH activity was increased in supplemented with NJEt in the skeletal muscle tissue of rats. Moreover, the percent elevation of GSH was more pronounced in the skeletal muscle tissue in the combination treatment compared to control rats. Increased GSH content with NJEt may also due to the increase in the synthesis of precursors for GSH formation and increase the γ-Glutamyl- Cysteinlyglycin enzyme, which is very essential for the GSH. The synthesis and degradation of GSH is referred as the γ-Glutamyl cycle. This cycle small responsible for the enhanced GSH concentration in the skeletal muscle tissue with NJ extract treatment. Glutathione peroxidase [GSH-Px] is a well-known first line defense of the cell against oxidative challenge, which interum requires glutathione as a co-substrate. The enzyme contains selenium as a co-factor, most probably at the active site, but no other prosthetic group such as heme, flavine or other metal constituents. Selenium is involved in the protection of biological system, especially membrane lipids against peroxidation [45].

The present study reveals that the activity of glutathione peroxidase was decreased in nicotine treatment rats. The decreased GSH-Px activity in the current investigation may disturb the glutathione [GSH] homeostasis in the muscle cell and ultimately it leads to the damage of muscle. Similar studies, Santanukar Mahapatra et al., [46] reported smoking decreases the glutathione peroxidase in the serum of mans. GPX works nonspecifically to scavenge and decompose excess hydro peroxides including H2O2, which may prevalent under oxidative stress. In this study, decreased GPX activity seems to indicate the nicotine induced oxidative stress. The decreased level of GSH and activity of GSH- dependent enzymes i.e. GPX, GR.

This result reveals that NJ extract enhanced skeletal muscle tissue glutathione peroxidase activity when compared to their respective controls. The elevation of glutathione peroxidase activity due to nardostachys jatamansi[NJE] suggests an increased capacity to handle hydroperoxides in the skeletal musc tissue. It appears that nardostachys jatamansi provide the required substrate for a high increase in the GSH-Px activity.
Table 1: Changes in Superoxidismutase (SOD) activity due to Nicotine treatment (Nt), Nardostachys jatamansi extract treated (NJEt) and interaction of the both (Nt+NJEt) for a period of 2 months over the control in the Skeletal Muscle tissue of male albino rats. Values in parentheses denote percent change over respective control.

<table>
<thead>
<tr>
<th>S.No</th>
<th>Name of the tissue</th>
<th>Control</th>
<th>Nt</th>
<th>NJEt</th>
<th>Nt+ NJEt</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Skeletal Muscle</td>
<td>130.02±15.75</td>
<td>94.24±6.11</td>
<td>138.06±9.31</td>
<td>132.63±2.91</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(-27.51)</td>
<td>(+6.30)</td>
<td>(+2.00)</td>
<td></td>
</tr>
</tbody>
</table>

± SD of six individual observations.
Values in parentheses denote percent change over respective control.
** Values are significant at P < 0.01
@ Values are non-significant at P < 0.05

Table 2: Changes in Catalase (CAT) activity due to Nicotine treatment (Nt), Nardostachys jatamansi extract treated (NJEt) and interaction of the both (Nt+NJEt) for a period of 2 months over the control in the Skeletal Muscle tissue of male albino rats. Values in parentheses denote percent change over respective control.

<table>
<thead>
<tr>
<th>S.No</th>
<th>Name of the tissue</th>
<th>Control</th>
<th>Nt</th>
<th>NJEt</th>
<th>Nt+ NJEt</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Skeletal Muscle</td>
<td>104.86±11.21</td>
<td>69.36±8.07</td>
<td>121.41±12.41</td>
<td>116.91±8.65</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(-33.85)</td>
<td>(+15.78)</td>
<td>(+11.49)</td>
<td></td>
</tr>
</tbody>
</table>

± SD of six individual observations.
Values in parentheses denote percent change over respective control.
** Values are significant at P < 0.01
@ Values are non-significant at P < 0.05.
Table 3: Changes in Glutathione (GSH) activity due to Nicotine treatment (Nt), Nardostachys jatamansi extract treated (NJEt) and interaction of the both (Nt+NJEt) for a period of 2 months over the control in the Skeletal Muscle tissue of male albino rats.

<table>
<thead>
<tr>
<th>S.No</th>
<th>Name of the tissue</th>
<th>Control</th>
<th>Nt</th>
<th>NJEt</th>
<th>Nt+ NJEt</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>± 11.24</td>
<td>±12.10</td>
<td>±10.63</td>
<td>±13.84</td>
</tr>
<tr>
<td>1.</td>
<td>Skeletal Muscle</td>
<td>153.43</td>
<td>133.20**</td>
<td>162.35**</td>
<td>155.29@</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(-13.18)</td>
<td>(+5.81)</td>
<td>(+1.21)</td>
<td></td>
</tr>
</tbody>
</table>

± SD of six individual observations.
Values in parentheses denote percent change over respective control.
** Values are significant at P < 0.01
@ Values are non significant at P < 0.05

Table 4: Changes in Glutathioneperoxidase (GPx) activity due to Nicotine treatment (Nt), Nardostachys jatamansi extract treated (NJEt) and interaction of the both (Nt+NJEt) for a period of 2 months over the control in the Skeletal Muscle tissue of male albino rats.

<table>
<thead>
<tr>
<th>S.No</th>
<th>Name of the tissue</th>
<th>Control</th>
<th>Nt</th>
<th>NJEt</th>
<th>Nt+ NJEt</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>±7.59</td>
<td>±10.38</td>
<td>±11.89</td>
<td>±9.27</td>
</tr>
<tr>
<td>1.</td>
<td>Skeletal Muscle</td>
<td>85.63</td>
<td>52.33**</td>
<td>92.96**</td>
<td>88.13@</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(-38.88)</td>
<td>(+8.56)</td>
<td>(+2.91)</td>
<td></td>
</tr>
</tbody>
</table>

± SD of six individual observations.
Values in parentheses denote percent change over respective control.
** Values are significant at P < 0.01
@ Values are non significant at P < 0.05
CONCLUSION

This investigation draws a conclusion stating that this much of nardostachys jatamansi extracts to the skeletal muscle tissue of male subjects may be beneficial, especially for the nicotine subjects to improve the health status and life span.

REFERENCES


