Anti-Depressant Potential of Banana Fruit

MILIND PARLE AND SUMAN MALIK

ABSTRACT

The prevalence of depression is alarming all over the world afflicting as many as 121 million individuals. Females in India are more susceptible than males owing to societal customs. The efficacy of anti-depressant medicines is limited. Therefore, there is an urgent need for safe, better-tolerated and more efficacious anti-depressants. Furthermore, depression is largely a hidden problem in India, which prompts the necessity of identifying the nutrients commonly available to Indian population for the management of depression. Recently, the search for novel drugs of vegetable origin for psychiatric illnesses has progressed significantly (Zhang, 2004).

Musa paradisiaca Linn or banana, a plant belonging to family Musaceae, is consumed all over the world as a popular fruit throughout the year. Traditionally, the fruits of banana have been used as a remedy for constipation, hangovers, anthelmintic and as a rich source of iron (Davey et al., 2009) and potassium (Rai et al., 2009). Banana contains very high levels of antioxidants, viz. vitamin-A, vitamin-C, carotenoids (Davey et al., 2009), thiamine, niacin, catechins and α-tocophenol (Enayde et al., 2006). Furthermore, banana contains hypoglycemic agents (Ghosal and Saini, 1984) such as sterylacyl glycoside (sitoindosides I and II) and diacylglycerols in addition to several other bioactive compounds.

However, there is no scientific evidence for therapeutic potential of banana in neuropsychiatric disorders. In the light of above, the present study was designed to determine the anti-depressant potential of banana fruit in mice.

Objectives:

The present study was undertaken to explore the anti-depressant potential of Musa paradisiaca paste (MPP) using forced swim test and tail suspension test. An attempt has also been made to determine the underlying mechanism of action of MPP by co-administration of agents modulating noradrenaline, serotonin, malonaldehyde and GABA activities.

MATERIALS AND METHODS

Plant material:

The fresh banana (Musa paradisiaca) fruit was purchased from local market of Hisar and got authenticated from Raw Materials Herbarium and Museum, National Institute of Science Communication and Information Resources, New Delhi. Different concentrations of Musa paradisiaca paste (5, 10, 20%, w/w) was fed to separate groups of mice along with their diet. This diet comprised of a mixture of Musa paradisiaca paste...
(MPP), wheat flour kneaded with water, a small amount of refined vegetable oil and a pinch of common salt. Each animal consumed approximately 3 gm/day of this specially prepared diet. Control animals received the same diet without the fruit paste.

**Animals:**
A total of 138 Swiss mice divided in 23 different groups were employed in the present study. Each group comprised of a minimum of 6 animals. Young (3-4 months old) mice weighing around 20-25 g were procured from the Disease-Free Small Animal House of C.C.S. Haryana Agricultural University, Hisar. The experimental protocol was approved by the Institutional Animals Ethics Committee and the care of laboratory animals was taken as per the guidelines of CPCSEA, Ministry of Forests and Environment, Government of India (registration number 0436).

**Statistical analysis:**
All the results were expressed as mean ± Standard Error (SEM). Data were analyzed by one-way ANOVA followed by Dunnett’s t-test.

**RESULTS AND DISCUSSION**

**Locomotor activity:**
*Musa paradisiaca* paste (10%, w/w) administered orally for 15 successive days did not show any significant change in the locomotor function of mice (713.667±27.782) as compared to the control group (733.33±29.095).

**Effects of Musa paradisiaca paste (MPP) on immobility periods in FST and TST:**
Forced swim test is a standard laboratory model for testing the despair behaviour of mice, which is considered equivalent to depression in human being. Despair behaviour/helplessness is correlated with the immobility exhibited by the mouse, when placed in a swimming chamber (Porsolt et al., 1978). On similar lines, tail suspension test served as another experimental model to measure the helplessness of animals (Steru et al., 1985). MPP (5, 10 and 20% w/w) per se administered for 15 successive days to mice decreased the duration of immobility significantly (p<0.01) in a dose-dependent manner in both the experimental models (FST and TST). The antidepressant efficacy of MPP was found to be comparable to that of fluoxetine (5-HT-reuptake inhibitor) and imipramine (Tricyclic anti-depressant) administered for 15 successive days (Table 1 and 2). The concentration of MPP (10% w/w) was selected for further studies.

<p>| Table 1 : Effects of <em>Musa paradisiaca</em> on immobility period in Tail Suspension Test |
|-----------------|-----------------|-----------------|-----------------|-----------------|</p>
<table>
<thead>
<tr>
<th>Group no.</th>
<th>Treatment (15 days)</th>
<th>Number of animals</th>
<th>Dose (kg⁻¹)</th>
<th>Immobility time (Sec) (mean ± SEM)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Control</td>
<td>6</td>
<td>Normal diet</td>
<td>180.166±6.284</td>
</tr>
<tr>
<td>II</td>
<td>Fluoxetine</td>
<td>6</td>
<td>20 mg (p.o.)</td>
<td>97.666±5.671**</td>
</tr>
<tr>
<td>III</td>
<td>Imipramine</td>
<td>6</td>
<td>15 mg (p.o.)</td>
<td>86.333±6.917**</td>
</tr>
<tr>
<td>IV</td>
<td>5% MPP</td>
<td>6</td>
<td>150 mg (p.o.)</td>
<td>125.333±9.766**</td>
</tr>
<tr>
<td>V</td>
<td>10% MPP</td>
<td>6</td>
<td>300 mg (p.o.)</td>
<td>98.166±4.498**</td>
</tr>
<tr>
<td>VI</td>
<td>20% MPP</td>
<td>6</td>
<td>600 mg (p.o.)</td>
<td>92.166±5.665**</td>
</tr>
</tbody>
</table>

Values are in Mean ± SEM. (n=6). ** denotes p<0.01 as compared to control group
MPP = *Musa paradisiaca* Paste.

<p>| Table 2 : Effects of <em>Musa paradisiaca</em> on immobility period in forced swim test |
|-----------------|-----------------|-----------------|-----------------|-----------------|</p>
<table>
<thead>
<tr>
<th>Group No.</th>
<th>Treatment (15 days)</th>
<th>Number of animals</th>
<th>Dose (kg⁻¹)</th>
<th>Immobility time (Sec) (mean ± SEM)</th>
</tr>
</thead>
<tbody>
<tr>
<td>VII</td>
<td>Control</td>
<td>6</td>
<td>Normal diet</td>
<td>146.5±8.895</td>
</tr>
<tr>
<td>VIII</td>
<td>Fluoxetine</td>
<td>6</td>
<td>20 mg (p.o.)</td>
<td>51.166±4.764**</td>
</tr>
<tr>
<td>IX</td>
<td>Imipramine</td>
<td>6</td>
<td>15 mg (p.o.)</td>
<td>58.5±8.269**</td>
</tr>
<tr>
<td>X</td>
<td>5% MPP</td>
<td>6</td>
<td>150 mg (p.o.)</td>
<td>99.5±8.078**</td>
</tr>
<tr>
<td>XI</td>
<td>10% MPP</td>
<td>6</td>
<td>300 mg (p.o.)</td>
<td>61.833±3.308**</td>
</tr>
<tr>
<td>XII</td>
<td>20% MPP</td>
<td>6</td>
<td>600 mg (p.o.)</td>
<td>58.333±5.308**</td>
</tr>
</tbody>
</table>

Values are in Mean ± SEM. (n=6). ** denotes p<0.01 as compared to control group
MPP = *Musa paradisiaca* Paste.
Effect of combination of Musa paradisiaca paste (10% w/w) with prazosin/baclofen/p-CPA on immobility periods using TST:

Prazosin (62.5 mg/kg i.p.), Baclofen (10 mg/kg, i.p.) and p-CPA (100 mg/kg, i.p.) per se increased significantly the immobility period of mice as compared to the control group. Pretreatment of mice with pCPA/prazosin/ baclofen significantly (p<0.01 and p<0.05) reversed the diminished immobility time observed with MPP (Fig.1) using TST.

Effect of Musa paradisiaca paste (10% w/w) on brain monoamine oxidase (MAO) and malondialdehyde (MDA) activities:

MPP (10% w/w) administered to mice for 15 successive days, significantly (p<0.01) reduced the brain MAO-A (22.16 ± 1.77 U/g protein) and MAO-B (33.666 ± 2.24 U/g protein) levels as compared to the control group. MPP, when administered for 15 successive days produced significant (p<0.01) decrease in brain MDA level (69.33 ± 2.714 nmol/g tissue) as compared to the control mice (108.5 ± 4.877 nmol/ g tissue) (Fig 2, 3 and 4).

The present study investigates the putative anti-depressant potential of Musa paradisiaca in mice. Swiss mice were administered the banana fruit paste at various concentrations ranging from 5-20% w/w once daily for 15 successive days. The antidepressant activity was measured using forced swim test (FST) and tail suspension test (TST). The results showed that the banana paste significantly reduced the immobility time of mice in both FST and TST. Banana paste per se neither had any significant influence on locomotor activity of mice nor showed any sedative effect. Immobility observed in forced swim test or tail suspension test has been correlated with helplessness or despair behaviour equivalent to depression in human beings. In these experimental models,
increased immobility indicates depression, whereas diminished immobility corresponds to anti-depressant effect. Since banana paste dose dependently diminished the immobility time of mice in both the experimental models, banana can be looked upon as a promising anti-depressant medicine.

The pathophysiology of depression highlights decreased activity of the neurotransmitters norepinephrine and serotonin in brain. Imipramine, which raises the level of norepinephrine by down regulation of presynaptic α2-adrenergic receptors is an established anti-depressant medicine (Manji et al., 2001). Since Musa paradisiaca paste produced its anti-depressant effects comparable to Imipramine, it appears that Musa paradisiaca paste also in some manner enhances the levels of norepinephrine. Furthermore, the depressant action of prazosin (a α1-adrenoceptor antagonist) as signified by increased immobility time was reversed successfully by banana fruit paste in the present study. This observation suggests that abundant availability of norepinephrine might have competitively displaced prazosin from postsynaptic α1-adrenoceptors. Since p-CPA (a serotonin synthesis inhibitor) showed increased immobility time in tail suspension test, the validity of this test model and the involvement of serotonin in depression is confirmed. The possibility that banana fruit paste facilitates serotonergic transmission in some manner cannot be ruled out, since banana fruit paste attenuated the depressant action of p-CPA in the present study. It is worthwhile to note that inhibition of monoamine oxidase enzyme by banana paste might further be elevating the levels of norepinephrine and serotonin. GABA-ergic system may also be involved in the mechanism of banana paste, since the effect of specific GABAB agonist (baclofen) was reversed by banana fruit paste. In the present study, Musa paradisiaca paste reduced the levels of malondialdehyde as well, thereby indicating decreased generation of free radicals. This antioxidant effect of banana fruit may be protecting the brain from oxidative damage.

Thus, banana fruit may be looked upon as a promising anti-depressant by virtue of its antioxidant, pro-serotonergic, pro-adrenergic and monoamine oxidase inhibitory activities.

**Conclusion:**

In the present study, banana fruit paste showed anti-depressant potential in two experimental models at the concentrations of 5% to 20% w/w in mice. The banana being commonly available to Indian population can serve as a useful nutrient-cum-medicine to counteract stressful lifestyle and depressive episodes occurring at different phases of our life by virtue of its anti-oxidant, MAO inhibitory and pro-serotonergic activities.

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**REFERENCES**


