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Modulating the cytoprotective potential of *Bacopa monnieri* by altering *in vitro* propagation

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Oxidative stress induced neuronal cell deaths are one of the major cause of neurodegenerative diseases. Often such stress can instigate, Reactive Oxygen Species (ROS) mediated neuronal cell apoptosis and necrosis. Previous studies have been focused towards identification of potential neuroprotective agents that can prevent oxidative stress-induced neuronal cell damage. However, availability of various chemotherapeutic agents for alleviating devastating disorder which provide symptomatic relief and may turn ineffective or show adverse drug effect after prolonged use. Alternative source for controlling the progression of such diseases could be plant derived metabolites (phytochemicals) that regulate neuronal malfunction, with slow sustained effect and no side-effects would appear to have significant neuroprotective effects. Several medicinal plants, designated as "Rasayan Drugs" in Ayurveda are reported to have significant influence on brain functions. Bacopa monnieri, a well-documented nootropic plant, is extensively used in herbal formulations for neurological disorder. *Bacopa monnieri* plant has learning and memory antidepressant activity, anticonvulsive action and antioxidant activity. Therapeutic activities of plant are attributed due to the presence of active phytocompounds viz. saponins, flavonoids and alkaloids. However, variations in active phyto-pharmaceutically important compounds in such formulations often affect their therapeutic efficacy and market acceptance. Current study highlights the comparative cytoprotective potential of field acclimatized and *in vitro* propagated *Bacopa monnieri* extracts of different locations on neuronal cell line (HT-22/N2a).

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Antidiabetic effect of *Carissa carandas* in rats and the mechanism of its insulin secretagogues activity in isolated pancreatic islets

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Carissa Carandas (CC) has been documented as a traditional treatment for diabetes. In the present study, the CC fruit aqueous, methanol, chloroform and ethyl acetate extracts were examined for hypoglycemic activity in healthy Wistar rats. Aqueous extract of CC (AECC) showed highest fall of 67.08% in fasting blood glucose from 0 to 1h in Glucose Tolerance Test (GTT). The ED50 of AECC was 300 mg/kgbw in streptozotocin induced diabetic rats. Treatment of diabetic rats with ED50 of AECC for 28 days significantly reduced post prandial glucose (PPG) by 33.65% ($p<0.01$), glycosylated hemoglobin (HbA1c) by 45.79% ($p<0.01$) and increased insulin level by 69.7% ($p<0.05$). The increase in insulin secretion may be partly responsible for antidiabetic effect of AECC. To assess the mechanism of secretagogues activity, AECC was incubated with isolated pancreatic islets of Wistar rats at basal (3.3 mM) and high (16.7 mM) level of glucose in presence or absence of diazoxide (K-ATP channel opener), nimodipine (Ca2+ Channel blocker) and calphostin-C (PKC inhibitor). AECC induced insulin secretion at 16.7 mM of glucose was significantly ($p<0.01$) reduced by diazoxide and nimodipine, but non-significantly ($p>0.05$) by calphostin-C. The study indicated that the phytochemicals present in AECC may be inducing insulin secretion by closing K-ATP channels in β-cells of pancreatic islets.

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