Discussionwith generated from membrane phospholipids by the action

Literature, Play

DiscussionWith effective ethnopharmacological value, P. bengalensis plant is widely used by different tribal as an herbal medicine for different disease remedies. Literatures survey has revealed that leaves are being traditionally used for the treatment of a headache and fever (Manandhar, 1989, Ghimire et al., 2009). Therefore, the present study was designed to scientifically validate the traditional claims for the different proposed activities.

The previous findings have indicated that the useful activities of plants could be attributed to the presence of bioactive compounds (Patel et al., 2014). The preliminary phytochemical evaluation was done to evaluate the presence of bioactive compounds, which validated the presence of tannins, flavonoids, alkaloids, glycosides, phenols, steroids, saponins and terpenoidsPhytochemicals including phenolic and flavonoid compounds have been extensively researched due to their beneficial properties and natural abundance in many herbs, fruit, vegetables, and grains (Lee, 2014). Different scientific investigations have indicated the role of these phytochemicals as antioxidant, analgesic, antipyretic and anti-inflammatory properties, (Saeed et al.

, 2010, Kreiner et al., 2017). The abundant of phenolic and flavonoid compounds could be a major factor for pharmacological activities shown by P. bengalensis leaves extracts. The research papers reported extraction of phenolic and flavonoid compounds was influenced by the solubility, the degree of polymerization, the interaction between plant constituents present and the formation of insoluble complexes (Gálvez et al., 2005) (Medini et al.

, 2014). In this study, methanol showed superior extraction of polyphenols and flavonoids compared to chloroform, which is supported by polar functional groups present in phenolic and flavonoid compounds. Plants rich in polyphenols and flavonoids had antioxidant activity due to their redox properties attributed by structural features (Mansouri et al., 2005, Baba et al., 2015) (Valcarcel et al.

, 2015). Structurally, these are rich in hydroxyl groups which can donate hydrogen atoms or electrons, and terminate radical chain reaction by converting free radicals to more stable products (Amarowicz et al., 2004, Dhalwal et al., 2008). DPPH radical scavenging is considered as a milestone in evaluating the antioxidant property, which is widely accepted because of the ease of the reaction (Khan et al., 2013). Our finding showed DPPH radicals' inhibition by methanol extract was higher than chloroform extract. It correlated the free radicals scavenging activity to the higher quantity of phenolic and flavonoids in the methanol extract.

Thermal nociceptive pain in the paw of rat is considered a highly sensitive model to evaluate the central analgesic effects of the therapeutic agents. During the inflammation pain process arachidonic acid is generated from membrane phospholipids by the action of phospholipases, later on degraded by cyclooxygenase (COX) to powerful inflammatory mediators; prostaglandins (Ricciotti et al., 2011).

Despite inhibition inflammatory mediators, central pain can be reduced by molecules acting as opioids agonists (Iwaszkiewicz et al., 2013). In this study

dose-dependent elevation in pain reaction threshold (latency time) was exhibited by both extracts and tramadol during the hot plate test.

Generally, latency time is related with the response on nociceptors, which are downregulated by opioids agonist (Al-Hasani et al., 2011) and stimulated by prostaglandins stimulated peptides (Juan et al., 1984).

The profound analgesic activity might be due to the presence active phytochemicals in the extracts which interference the pain mediators. Further study on of specific biochemical mechanism either opioids or prostaglandins may be beneficial to determine the specific path where the extract exerts its analgesic activity. The subcutaneous injection of Brewer’s yeast suspension markedly elevated the rectal temperature due to the release of prostaglandins which considered as the pathogenic cause of pyrexia. Antipyretic agents reduce the rectal temperature can be mediated by inhibition of prostaglandins by blockade of cyclooxygenase enzyme activity (Hullatti et al.

, 2007, Eldeen et al., 2008). Dose-dependent reduced in rectal temperature was demonstrated significantly (P <0. 001) by PBME, PBCE and standard (paracetamol) compared to control probably by inhibition of prostaglandins synthesis. The observed effects might be due to the presence of biologically active compounds that might interfere with the release of prostaglandins. However, it must be noted that the numbers of biological phenomena occur eventually to produce prostaglandins.

Investigation of the biochemical pathway may be worthwhile to determine the specific mechanism where the extracts exert its antipyretic effect. Carrageenan-induced hind paw edema assay has been widely used to estimate the anti-inflammatory potential of new pharmaceutical agents (Afsar et al., 2015). Development of edema with carrageenan is a biphasic process, the initial phase (0 – 1 h) of edema is attributed by the release of histamine, 5-hydroxytryptamine, and bradykinin. Whereas, cyclooxygenase (COX-2) plays a vital role in the progression of later phase (1 – 6 h) by converting arachidonic acid into prostaglandins, increasing the size of edema and swelling.(Di Rosa et al.

, 1971, Khan et al., 2009). This enzyme is considered to be recognized target for a variety of NSAIDs, such as aspirin and indomethacin, which inhibit rat paw edema at the later phase following carrageenan injection. Therefore, for comparison of both extract was performed with indomethacin. The plant extracts inhibited paw edema in the later phase in a pattern similar to indomethacin, whose mechanism is the reduction in prostaglandins synthesis.

Even though the definite mechanism of action is not known, it is possible that presence of active constituents in the extracts displayed the anti-inflammatory activity which could be attributed to the inhibition of the synthesis, release or action of inflammatory mediators.