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Letter to the editor:

POMEGRANATE PEEL ATTENUATES HYPERGLYCEMIC EFFECTS OF ALLOXAN-INDUCED DIABETIC RATS

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Dear Editor,

Diabetes is a major epidemic chronic metabolic ailment worldwide (WHO, 2008). It is characterized by insufficiency of insulin secretion and/or action, insulin resistance, and abnormal metabolism of glucose, lipid and protein (WHO, 2008; Middha et al., 2011). Today, diabetes has become a pandemic affecting approximately 5 % of people in both developed and developing countries. Globally, diabetes causes high mortality and is the second most common cause of death after cancer (Middha et al., 2012). According to the diabetes atlas, in 2025 India, China and the United States would gain region-wise emphasis as top three countries with people suffering from diabetes (Allgot et al., 2003). In 2011, type 2 diabetes mellitus (T2DM) was detected in about 370 million people and accounted for approximately 4.6 million deaths annually, causing substantial medical and economic burden worldwide.

Diabetic research also comprises herbal medicines (Middha et al., 2013a; Usha et al., 2013; Kiran et al., 2013). Pomegranate (*Punica granatum*), a fruit of promise, is considered as holy fruit for its therapeutic purpose since antiquity and is used as an alternative medicine in Ayurveda and other traditional medicines worldwide (Jurenka, 2008; Hajimahmoodi et al., 2008; Middha et al., 2013b). The probable scientific validation of the herbal drug extracted from pomegranate peel from Kumauni region was evaluated in an alloxan-induced diabetes model. The effects of methanolic extract of pomegranate peel (PGPE) (two diverse oral doses: LP=75 mg/kg body weight and HP=150 mg/kg body weight) on fasting blood glucose (FBG), lipid peroxidation (malondialdehyde [MDA]), antioxidant enzymes (superoxide dismutase [SOD] and glutathione peroxidase [GPx]) were tested and compared with standard drugs for 6 weeks.

Both doses of PGPE increased the plasma insulin levels by one and five folds and augmented the levels of the following antioxidants ($P<0.05$): SOD by 39.68 % and 75.03 %, GPx by 20.07 % and 67.60 % in plasma, SOD by 44 % and 66 %, GPx by 50 % and 80 % in kidney, respectively. Although PGPE did not decrease the plasma MDA level when compared to diabetic controls, a significant reduction in MDA levels was observed in the kidney (LP; 16.8 and HP; 52.08 %; $P<0.001$) (unpublished data). Histopathological studies validated our findings which were in accordance with that observed by Parmer and Kar (2008). Increase in the level of insulin following administration of PGPE in experimental animals indicates the restoration of pancreatic β-cells and demonstrates the anti-hyperglycemic and antioxidant properties of PGPE.

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