

Platelet aggregation inducing activity of *Ficus racemosa* stem bark extracts

Sir,

Blood platelet activation and aggregation play an important physiological role in the hemostasis and it is well established that many cardiovascular disorders are linked to an abnormal excessive activation of platelets. It is reported that patients with hypertension/coronary heart disease tend to have increased platelet reactivity.^[1] Inhibition of platelet hyperactivation is therefore an important approach for treating cardiovascular disorders and studies indicate that phenolic compounds may protect against cardiovascular diseases by inhibiting platelet aggregation.

Ficus racemosa Linn. (Moraceae) commonly known as 'cluster fig' is an excellent source of phenolic compounds and flavonoids exhibiting significant antioxidant activity in terms of radical scavenging, reducing power, and antilipidperoxidative activity.^[2] With this background, since phenolic compounds are particularly attributed for the antiplatelet aggregation activity of plant extracts, effect of standardized aqueous extracts of *F. racemosa* stem bark on isolated human platelets was studied.

Ficus racemosa stem bark was collected from Mukkadahally, Chamara Nagar district of Karnataka, India (BOT-001/2008). A cold aqueous extract (FRC) was prepared by extracting powdered *F. racemosa* bark (FRB) with distilled water (1:8 w/v) at room temperature on a mechanical shaker for 24 h, while the hot aqueous extract (FRAE) was prepared by extracting FRB with distilled water at 70°C on a mechanical shaker for 24 h, filtered, and freeze dried. Earlier, we reported that the cold aqueous extract contained bergenin as a major component, while the hot aqueous extract contained ferulic acid, kaempferol, and coumarin in addition to bergenin.^[2]

Blood collected from volunteers who were selected based on the criteria that they were healthy, non-smokers, had not taken any medications, including aspirin, within the last 2 weeks and had not taken any food within the last 8 h was used for the preparation of platelet-rich plasma (PRP) and platelet poor plasma (PPP). Platelet aggregation was studied by adding FRC and FRAE at two concentrations (50 and 100 µg ml⁻¹) dissolved in 25 µl PBS to 450 µl aliquots of PRP. The final volume was made up to 0.5 ml with PBS, and aggregation was recorded over 10 min by the change in light transmission as a function of time using a dual-channel lumi-aggregometer in triplicates.

The effect of FRC and FRAE on human platelets is presented in Figure 1. Both the extracts induced aggregation of platelets to an extent of 3–51% compared to control. Further, the aggregatory activity of FRC and FRAE was dose-dependent and no significant differences were observed between the platelet aggregatory activity of FRC and FRAE. However, the extent of aggregation induced by FRC and FRAE were significantly lower ($P \leq 0.05$) than those induced by collagen (2 µg ml⁻¹), adenosine diphosphate (10 µM) and epinephrine (10 µM), respectively [Figure 2].

There are many compounds present in the diet such as phenolics, vitamins, and carotenoids^[3] that affect platelet function. Studies have shown the inhibitory effects of these compounds on platelet function leads to a lower rate of heart diseases and support the need for development of new antiplatelet and antithrombotic agents of the natural origin.^[4] In our study, although anti-platelet property of the various plants is attributed to polyphenols, flavonoids, and coumarins, which significantly inhibit platelet adhesion, aggregation, and secretion,^[5,6] it was of interest to

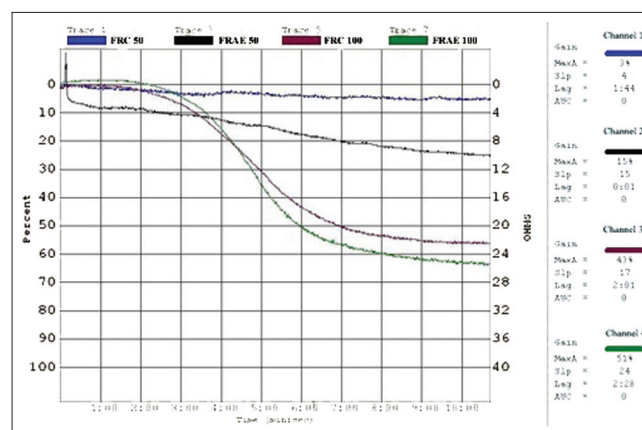


Figure 1: Effect of *F. racemosa* extracts on human platelets. FRC50: cold aqueous extract of *F. racemosa* bark (50 µg ml⁻¹). FRC100: cold aqueous extract of *F. racemosa* bark (100 µg ml⁻¹). FRAE50: hot aqueous extract of *F. racemosa* bark (50 µg ml⁻¹). FRAE100: hot aqueous extract of *F. racemosa* bark (100 µg ml⁻¹)

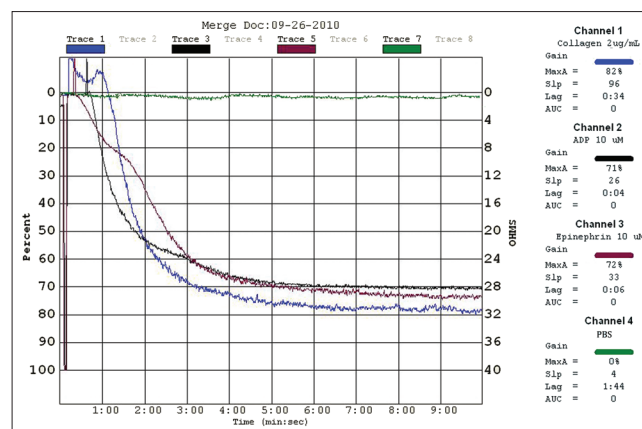


Figure 2: Aggregation induced by various agonists in comparison with control

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note that *F. racemosa* extracts induced platelet aggregation, despite being a rich source of phenolics, flavonoids, and isocoumarins. This particular observation indicates that some phytoconstituents present in FRB overshadow the anti-platelet activity of polyphenols and the flavonoids.

It is inferred that platelet aggregation inducing activity of FRB extracts is a limiting factor for its utilization despite having proven therapeutic potential. Thus, detailed and more intensive studies are warranted in order to elucidate the mechanism of platelet aggregatory action of *Ficus* extracts. The study also warrants isolation and characterization of the specific compound(s) responsible for the platelet aggregatory activity of the *Ficus* extracts for its optimum utilization as a therapeutic agent.

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10.4103/0976-500X.103692