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Molecular Basis of Ankaferd-Induced Hemostasis in the Management of Sternal Bleeding

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LETTER TO THE EDITOR

We have read with great interest the paper by Ergenoglu and coworkers [Ergenoglu 2010] on the successful hemostatic effects of Ankaferd Blood Stopper (ABS) on the control of sternal bleeding during cardiac surgery. They also comprehensively discussed the histopathologic background of ABS with respect to the vascular tissues. We would like to add some molecular background regarding ABS-induced hemostasis relevant to the authors' findings, which are noteworthy in several respects.

In their report, the authors discussed the role of ABS within the context of the classic hemostasis model. They also pointed out the unique hemostatic effect that ABS has by rapidly promoting a protein network that anchors erythrocyte aggregation without directly affecting coagulation factors or platelets [Ergenoglu 2010]. Moreover, the molecular basis of the ABS-induced hemostatic alterations on vascular tissue must be further considered so that the concepts underlying this novel hemostatic remedy can be better understood.

ABS-induced formation of the protein network along with vital erythroid aggregation covers the entire physiological hemostatic process. There are several essential components of the ABS-induced protein network. Vital erythroid aggregation takes place in conjunction with the spectrin and ankyrin receptors on the membranes of red blood cells. Essential erythroid proteins (ankyrin recurrent and FYVE bundle–containing protein 1, spectrin α, actin-depolymerization factor, NADH dehydrogenase [ubiquinone] 1 α subcomplex, mitochondrial NADP+-dependent malic enzyme 3) and the required ATP bioenergy are included in the protein library of ABS. ABS also up-regulates the GATA/FOG transcription system affecting erythroid functions and urotensin II [Beyazit 2010]. Urotensin II is also a crucial component of the effects of ABS, and it acts as a link between injured vascular endothelium, adhesion proteins, and active erythroid cells. These concepts have been developed via MALDI-TOF mass spectrometry proteomic molecular analyses, cytometric arrays, transcription analysis, and ultrastructural examinations via scanning electron microscopy, as well as numerous investigations involving interactions in research settings in vitro and in vivo [Haznedaroglu 2009; Beyazit 2010; Demiralp 2010]. Furthermore, ABS affects the levels of transcription factors: activating protein 2 (AP2), androgen receptor (AR), cyclic AMP response element or activating transcription factor 1 (CRE-ATF1), E2F transcription factors 1 through 5 (E2F1-5), E2F6, interferon-stimulated response element (ISRE), Myc-Max, nuclear factor 1 (NF1), protein 53 or tumor protein 53 (p53), peroxisome proliferator-activated receptor (PPAR), and Yin Yang-1 (YY1) [Haznedaroglu 2009]. These regulator molecules affect distinct steps in the cellular-proliferation process, such as cell vascular hemostasis, angiogenesis, signal transduction, apoptosis, inflammation, acute-phase reaction, and several metabolic molecular pathways [Demiralp 2009; Beyazit 2010].

Therefore, the ability of ABS to induce formation of a protein network not only makes it an effective hemostatic agent but also confers biological properties of the extract that affect cellular responses that have been assessed in analyses focusing on proteomics and transcriptomics [Haznedaroglu 2009; Ergenoglu 2010].

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