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LETTER TO THE EDITOR

"Spleen Contraction and Hemoconcentration" Regarding the Review "Hemoconcentration and Hemostasis During Acute Stress: Interacting and Independent Effects" by Austin et al. 2011

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Dear Editor,

We have read with great interest the well-written review "Hemoconcentration and Hemostasis During Acute Stress: Interacting and Independent Effects." In this comment, we would like to stress findings that challenge the mechanisms of hemoconcentration highlighted in the review, namely, the hemoconcentration following contraction of the spleen, which is not mentioned despite several publications on both animals and humans.

During exercise and hypoxic stress, many mammals can mobilize large numbers of erythrocytes from the spleen in order to improve oxygenation of metabolically active tissue [1]. Such "autotransfusion" of erythrocytes improves O_2-carrying capacity and increases both the aerobic performance in highly active terrestrial mammals and the diving capacity of, e.g., seals [1].

In adult humans, the splenic reservoir contains on the average 200–250 ml of blood [1], with more than twice the hematocrit of normal arterial blood [2]. The extent of splenic contraction after breath holding or exercise in humans has been reported to be 18–56 % [1, 3] resulting in a concomitant increase in hemoglobin concentration by typically 3–6 % [1, 3], a response not seen in splenectomized individuals [3]. The response is not due to hemoconcentration from extravasation of plasma [3], and is reversible within approximately 10 min after cessation of the initiating stimuli [3, 4], for example, hypoxia [5] and sympathetic stimulation of various origins [1].

Numerous animal studies implicate the importance of the sympathoadrenergic system for initiation of splenic contraction. In fact, all innervations of the spleen in the rat, mouse, dog, and human is sympathetic, and the spleen is among the most densely adrenergic innervated organs. Adrenoreceptors are located in the splenic capsule and parenchyma [1, 6], and the splenic nerve is composed of 98 % sympathetic nerve fibers [1]. Consequently, neurostimulation, epinephrine, and norephineprine all cause α -mediated contraction of the spleen [1, 6], and infusion by low-dose ephineprine also produced rapid spleen contraction in humans [7].

Based on the sympathetic pathway for initiation of spleen contraction and the substantial evidence showing spleen-induced increases in erythrocyte concentration, we propose that the understanding of "stress-" polycythemica and stress-hemoconcentration is not limited to the mechanisms of plasma volume shifts described in the review and that the contribution of the blood-boosting spleen contraction should be considered. Conceivably, a physiological function of spleen contraction in mammals is to enhance oxygenation under stressful conditions—while spleen storage of erythrocytes serves to minimize blood viscosity during periods of rest [1].

The authors suggested that the rapid normalization of hemoglobin to baseline following period of hemocontration after psychological stress is due to shifts in plasma volume. However, as the spleen and the concurrent hemoglobin increases are reversible within 10 min, we suggest that the "hemodilution" following stress could very likely be due to filtration of erythrocytes back into the spleen after its re-expansion. The authors concern of exacerbation of shear stress

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imposed by atherosclerotic plaques and plaque ruptures by increased blood viscosity indeed imply that the spleen's role in regulating hemoconcentration deserves attention in future psychohematology research.

Conflict of interest We hereby declare no conflict of interest regarding the letter to the editor, and that letter adheres to ethical standards in the Helsinki declaration.

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