6 Lift Generation in Highly Compressible Porous Media
From Red Cells to Skiing to Soft Lubrication

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CONTENTS

In a recent study, Feng and Weinbaum (2000), hereafter referred to as F&W, laid the foundation for a new type of lubrication theory that is applicable for porous medium whose structure is so compressible that the normal forces generated by the elastic compression of the matrix are negligible compared to the pressure forces generated within the porous layer. This new theory shows that there is an unexpected and remarkable dynamic similarity between the motion of a red cell gliding at <20 μm/s on a compressed endothelial glycocalyx layer (EGL) of sulfated proteoglycans and glycoproteins that lines the endothelial cells (ECs) of our capillaries (Figures 6.1 and 6.2a) and a human skier or snowboarder skiing on soft snow powder (Figures 6.3 and 6.46), even though their difference in mass is of the order 10^{15}. F&W predicted that the excess pore pressure generated by a planing surface moving on any compressible porous medium scales as $\alpha^2 = h^2/K$, where h is the layer thickness and K is the Darcy permeability and α is of an order 10^2 or larger for both red blood cells gliding on the EGL as shown in Figure 6.2a and human skiing as shown in Figure 6.3. Thus, the lift forces generated can be four or more orders of magnitude greater than the classical lubrication theory. The huge enhancement in the lift arises from the fact that as the matrix compresses, there is a dramatic increase in the lubrication pressure because of the marked increase in the hydraulic resistance that the fluid encounters as it tries to escape from the confining boundaries through the thin compressed porous layer.

At velocities >20 μm/s, the red blood cell rises out of the EGL and there is an intervening thin fluid lubricating layer between the EGL and RBC membranes. The compression of the EGL is

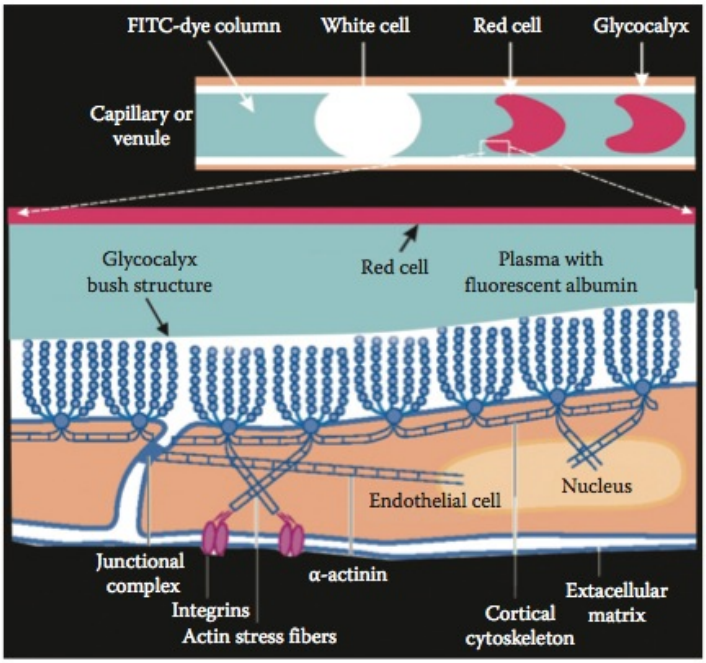

FIGURE 6.1 Schematic of the Vink and Duling (1996) experiment that shows the motion of blood cells over the bush-like EGL rooted from the underlying ACC of the endothelium.