Surfactant protein D: not just for the lung anymore

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IN THEIR ARTICLE, Snyder et al. (5) provide compelling data that arterial smooth muscle cells of the vessel wall express surfactant protein D (SP-D) and that the expression of SP-D attenuates the release of IL-8 stimulated by LPS and TNF-α. Although most often associated with innate immune protection of the lung, the authors show clearly that arterial smooth muscle cells express SP-D and that SP-D increases the uptake of Chlamydia pneumoniae bodies while attenuating IL-8 expression. These data indicate that SP-D does not abandon its long-standing role in innate immunity simply because it is expressed by a different cell type. Rather, it seems to find ways to help the vasculature rid itself of a foreign antigen, a duty usually relegated to macrophages and dendritic cells.

As IL-8 increases the recruitment of monocytes (4) and neutrophils (3), their data show how vascular smooth muscle cell expression of SP-D may play a role in protecting the vessel wall by attenuating IL-8-mediated inflammation. Thus the findings in their report (5) show for the first time how SP-D protects the vessel wall by a mechanism that has long been recognized to protect the lung. This information could be important in the design of new treatment modalities that increase SP-D expression to decrease vascular inflammation and possibly atherosclerosis.

The exact signaling mechanisms by which SP-D decreases vascular inflammation remain unknown. What is known from the study of Snyder et al. (5), however, is that SP-D blocks both TNF-α- and LPS-mediated inflammation. Other studies indicate that SP-D binds to G-P340 (2), signal inhibitory regulatory peptide-α, as well as the calreticulin/CDF91 complex (1). Accordingly, new opportunities exist for examining a number of promising pathways of activation that could lead to new therapies to block inflammation. Alternatively, SP-D may act by blocking a common mechanism of gene activation such as NF-κB. Thus not only could one design new therapies aimed at increasing vascular SP-D expression to protect the vessel wall, but therapies could be designed to inhibit the pathways targeted by SP-D. Clearly, a continued examination of the role of SP-D in cardiovascular disease should lead to the discovery of new pathways and additional opportunities for inhibiting inflammation in the vessel wall.

REFERENCES