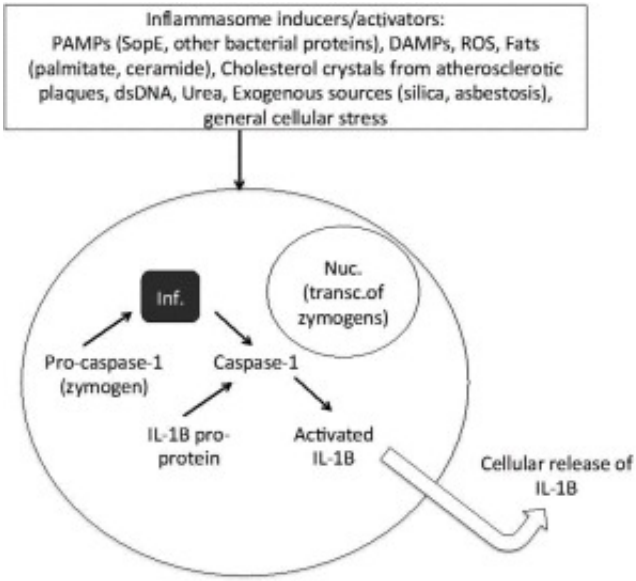
Researchers describe “imm-unifying theory” capable of explaining multiple medical problems

About a year ago, investigators at the Shock Trauma Center in Baltimore, MD found that patients who had organ transplants fared quite well after traumatic events. The authors of the manuscript, which was published in the Journal of Trauma and Acute Care Surgery, hypothesized that the reason for the better-than-expected outcome was a blunted “inflammatory” response. The investigators surmised that this “blunting” was the result of special medicines required for transplant patients, which suppress the immune system.

Blunted immunologic responses to trauma with medications for transplant? It might seem like an unimportant question to ponder, but the implications turn out to be somewhat dramatic.



Based on these data, researchers at the University of Wisconsin and the University of Maryland scoured scientific publications to identify the mechanisms responsible for the most common human diseases. After a rigorous review of the most nuanced literature, it became clear that most human maladies, including atherosclerosis, cirrhosis of the liver, diabetes and the metabolic syndrome, the body’s response to trauma, and even some psychiatric diseases are – at their root – quite similar.

When asked about the project, Dr. Joseph R. Scalea, the lead investigator stated “you know, there are thousands of similar observations made by scientists in different fields every day, but we infrequently sit down and try and put them all together.” Dr. Scalea mentions that, “at the end of the day, inflammatory mechanisms really underlie most human pathologies.” Dr. Scalea and his colleagues have attempted to synthesize the highest-end mechanistic data available and to provide a new construct for how we view human disease.

Naturally, the next question is: what do we do with this new construct for disease? Well, it's a double edged sword. While it is true that a small set of mechanisms are responsible for many human diseases, it is also true that those mechanisms are quite important to cellular function. As a result, simply shutting these mechanisms down in a switch-like fashion might have unanticipated consequences. Dr. Scalea and his colleagues highlight in their latest publication the various genetic and proteomic investigations taking place which exploit the very mechanisms described. In particular the authors highlight the unfolding story of the inflammasome (Fig. 1). These large intracellular proteins are now appreciated to help choreagraph the inflammatory response. With more knowledge of the important inflammatory mediators patient-directed therapies may become a reality.

Although therapies based on the presented findings may be many years away, perhaps it is time to reconsider how we teach (and learn) about pathophysiology – perhaps we should stop focusing on the differences between each disease and starting appreciating diseases for what they are – different locations of the same inflammatory processes.

Publication

Mechanistic similarities between trauma, atherosclerosis, and other inflammatory processes.
Scalea JR, Bromberg J, Bartlett ST, Scalea TM
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