Response to Blood Pressure Control: A Facelift for Macrophages?

Schmaderer et al\(^1\) indicate that they had no success increasing blood pressure further in spontaneously hypertensive rats after giving them clodronate-laden liposomes. They studied spontaneously hypertensive rats and Wistar-Kyoto control rats with radiotelemetry. We cannot explain the findings of this carefully done study; however, we have no data on salt intake and interstitial sodium contents in these animals, no histology was shown, and no information on macrophage disposition was given.

We are not macrophage biologists. However, we were interested in the recent review by Benoit et al\(^2\) that discussed subclasses of macrophages. M1 macrophages lead to tissue injury when an appropriate program is polarized. So-called M2 macrophages instead play a critical role in the resolution of inflammation by producing anti-inflammatory mediators. M2 macrophages were further subdivided into M2a, M2b, and M2c subtypes that covered a continuum of functional states. Apparently we cannot generalize on what macrophages do. Our suggestion was that they do some totally unexpected things, such as osmosensing.\(^3,4\) Regulation of macrophage activation has also been reviewed.\(^5\) Macrophages are capable of making a wide array of secondary products depending on their local environment and the stimulus involved.

In our rat studies, we measured blood pressure by direct catheter measurement in anesthetized animals, which we admit is less precise than radiotelemetry in conscious animals. However, we do not believe that this methodological difference explains the findings. Spontaneously hypertensive rats can be relatively resistant to salt and also to target organ damage. We have tested clodronate-laden liposomes in double-transgenic rats harboring human renin and human angiotensinogen genes. These rats were also the model that responded well to dexamethasone and etanercept.\(^6\) The liposomes did not elevate blood pressure further in these rats (unpublished observations) that were given standard chow. However, target-organ damage was made worse rather than ameliorated with macrophage depletion. Evidently we have a lot to learn about macrophages, salt, and their interactions. We are pleased that Schmaderer et al\(^1\) have joined this line of investigation.

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Disclosures

None.

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