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Response "Re: Microparticles: markers and mediators of sepsis-induced microvascular dysfunction, immunosuppression, and acute kidney injury "

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We attempted to highlight progress in microparticle research and bring some clarity to a rapidly changing field that is still defining three major categories of extracellular vesicles: exosomes (smaller), microvesicles, and microparticles (larger). We thank Burger *et al*[1] for pointing out the difficulties in describing what is present and what is active in a given system. The majority of microvesicles described by Cantaluppi *et al*[2] were 60-160 nm, outside of our microparticle size range, 200-2000 nm. The key point by Burger *et al*[1] is that Cantaluppi *et al*[2] used a mixture of extracellular vesicles. These extracellular vesicle subtypes are typically distinguished by size, centrifugation method, and/or composition (including cell surface markers, lipids, and miRNA). The distinction between microvesicles and microparticles is subtle; they differ in size, but they are formed from the same blebbing of the plasma membrane, as confirmed microscopically by Cantaluppi *et al*.[2] The size of the protrusions and accompanying vesicles corroborate their Nanosight sizing data; however, their flow cytometry data are consistent with larger microparticles (>200 nm). Both microvesicles and microparticles were likely present in their preparation. Such ambiguities can be clarified by using complementary methods, such as sizing larger particles by flow cytometry[3] or characterizing smaller particles using fluorescent Nanosight detection. Unfortunately, their data cannot pinpoint whether the biological effect came from exosomes, microvesicles, or a small number of microparticles. miR126 can come from apoptotic bodies[3], exosomes[4], or microvesicles,[2] making the context of other extracellular vesicle contents important for functional impact. Emerging techniques will enable these distinctions to be made more routinely.

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