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Muscle IGF-I levels in hemodialysis patients

To the Editor: We read with great interest Wang et al's paper [1] describing reduced skeletal muscle mRNA levels for insulin-like growth factor (IGF)-IEa, IGF-II, and the IGF type 1 receptor in hemodialysis (HD) patients. While the low levels of IGF-IEa mRNA relative to healthy controls is anticipated, the elevated muscle IGF-I protein level (mIGF-I) reported is at odds with the diminished levels, relative to healthy controls previously reported in our HD patients [2] and animal models of chronic renal failure (CRF) (e.g. [3]). An obvious difference between these studies is that in contrast to our patients and CRF rats, the HD patients recruited by Wang et al were not muscle wasted. This supports our contention that mIGF-I may play an important role in muscle atrophy in CRF populations.

Notwithstanding species and methodologic differences, we were also surprised by the huge disparity in mean mIGF-I levels reported by this group for HD patients and CRF rats [1, 3]. Assuming a protein content of 15.5% in wet skeletal muscle for nonobese humans [4], the 131 and 100 ng IGF-I/mg wet muscle values reported by Wang et al convert to 845 and 645 ng IGF-I/mg muscle protein for HD patients and controls, respectively. These values are 1 to 2×10^5 greater than those reported by Ding et al (0.0042 and 0.0069 ng/mg muscle protein for CRF and control rats, respectively). Are the units correctly stated in these papers? Additionally, the serum IGF values in Wang et al's paper need to be reported as ng/mL, not μg/mL, to be correct.

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Anti-proteinase 3 antibody binding to neutrophils as demonstrated by confocal microscopy

To the Editor: We read with great interest the article by Van Rossum et al [1] and wish to provide supplementary microscopic evidence of binding of anti-neutrophil cytoplasm antibodies (ANCA) to a subset of neutrophils.

Following the report by Abdel-Salam et al [2] of a failure of ANCA to bind to neutrophils, we investigated binding of anti-PR3 antibody positive serum from patients with systemic vasculitis to human neutrophils using indirect immunofluorescence and confocal microscopy, as detailed in Figure 1. The priming and staining procedure was virtually identical to that used by Van Rossum et al, with the addition of an incubation step with the neutrophil marker CD16. Our results support their findings, with membrane staining of a fraction of neutrophils incubated with anti-PR3–positive serum. Thus, the hypothesis that ANCA binding in vivo results in dysregulated degranulation of neutrophils [3] remains viable.