cular administration of adiponectin in mice decreased body weight by stimulating energy expenditure, adiponectin may also promote wasting.[5] Interestingly, high, but not low, molecular weight adiponectin has been associated with increased energy expenditure in CKD.[6] Because to date it is unknown if dialysis alters the proportion of the different adiponectin isoforms, we cannot speculate on the possibility of a uremic accumulation of high molecular weight adiponectin. In any case, in the context of CKD, elevated adiponectin may be confounded by, or alternatively, act as an inducer of protein-energy wasting. Thus, future epidemiological studies should take into account this potentially malevolent 'Mr Hyde side' of adiponectin.

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Response to 'Adiponectin in chronic kidney disease: Dr Jekyll and Mr Hyde'

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We thank Carrero *et al.*[1] for their thoughtful comments and for bringing up an important issue. Indeed, adiponectin's involvement in protein-energy wasting may be both possible and perhaps a constituent of this 'paradox'. However, we failed to observe differences in body mass index between those who progressed to renal function decline (<30 ml/min per 1.73 m^2) and those who did not progress among the 108 participants of the Epidemiology of Diabetes Complications Study with a diagnosis of type 1 diabetes and overt nephropathy at study entry. Thus, the elevation in adiponectin concentration in progressors throughout the 16-year follow-up period was not accompanied by differences in body mass index in this population. It should be noted here that these data were based on a small subsample of the Epidemiology of Diabetes Complications cohort and need to be replicated in larger samples. Nevertheless, they and Dr Carrero's observations add more complexity to an already intricate issue, reminding us that there is probably quite a bit more to be learned regarding this adipokine.

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