Telmisartan and Obesity

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To the Editor I read with interest the case report “The insulin sparing effect of telmisartan in a case of type 2 diabetes mellitus associated with schizophrenia under treatment of risperidone”, recently published by Yamaguchi and Tsutsumi (1). A striking aspect of the case was the loss of 16 kg in 15 months without remarkable life-style modification. A recent experimental study provides a pathogenic mechanism that might be operating in this patient. He et al (2) have demonstrated that telmisartan prevents adipogenesis and weight gain through activation of PPARδ-dependent lipolytic pathways and energy uncoupling in several tissues. In this study, long-term administration of this angiotensin II receptor blocker significantly decreased visceral fat and prevented high-fat diet-induced obesity in wild-type mice and hypertensive rats but not in PPARδ knockout mice. Interestingly, in this experimental study, the authors showed that an increased expression of PPARδ in 3T3-L1 preadipocytes was not observed after the administration of other angiotensin II receptor blockers, such as losartan and candesartan. Consistent with this mechanism, in the case reported by Yamaguchi and Tsutsumi the reswitching to candesartan from telmisartan resulted in a worsening of the patient’s metabolic parameters. PPARδ, an isotype less well known than PPARα and PPARγ, enhances fatty acid catabolism and energy uncoupling in adipose tissue and muscle, and it suppresses macrophage-derived inflammation (3).

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References