Dear Editor,

Recently, Lodi et al. described a deficiency of cardiac energetics in Friedreich’s ataxia (FRDA) patients in the absence of cardiac dysfunction and hypertrophy [1]. The authors concluded that cardiac metabolic dysfunction proceeds the development of myocardial hypertrophy.

In this manuscript left ventricular hypertrophy (LVH) was defined as a septal (IVS) or posterior (PW) wall thickness of $\geq 11$ mm. Most researchers, however, define hypertrophy when wall thickness is $\geq 13$ mm [2–4]. This is a well-established and widely used criterion for hypertrophy.

Also, wall thickness depends on height, weight and sex. Therefore, Vasan et al. suggested determining wall thickness in relation to these variables [5]. Hypertrophy could be then determined as a wall thickness larger than the 95th percentile of normal wall thickness as suggested by examination of the Framingham Heart Study population [5].

Lodi et al. did neither use the criterion of $\geq 13$ mm nor did they adjust wall thickness to height, weight, and sex. Therefore, the classification of patients only with IVS $\geq 11$ mm as hypertrophy may not be correct. Because of the limitations in defining LVH by Lodi et al. we cannot follow their conclusions. In particular, the similar degree of energy depletion in patients with and without LVH as classified by Lodi et al. may be purely the result of the arbitrary definition of myocardial hypertrophy. Thus, we would suggest recalculating the data with respect to accepted definitions of myocardial hypertrophy.

References