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Acknowledgments— Supported by the Cystic Fibrosis Trust and the National Services Division of NHS (Scotland).

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Low Plasma Adiponectin Levels Are Associated With Increased Hepatic Lipase Activity In Vivo

Response to Schneider et al.

Recently, close attention has been given to the relationships between plasma adiponectin levels and lipolytic enzymes such as hepatic lipase and lipoprotein lipase activities in an effort to understand the possible mechanism of hyperlipidemia in low adiponectin. The cross-sectional study by Schneider et al. (1) has concluded that postheparin plasma hepatic lipase activity is inversely associated with plasma adiponectin levels, independent of insulin resistance represented by homeostasis model assessment of insulin resistance and inflammation. In a separate issue of *Diabetes Care,* the same group also concluded that postheparin plasma lipoprotein lipase activity is positively associated with plasma adiponectin levels, independent of insulin resistance and inflammation (2). We believe they have overgeneralized in terms of populations and race regarding the independency of the observed associations. In fact, in our study of Japanese hyperlipidemic men, we also found that postheparin plasma hepatic lipase activity is inversely and lipoprotein lipase activity positively associated with plasma adiponectin levels in univariate analysis, which is quite similar to their findings up to this point. However, in our study these associations did not persist after adjustment for age, BMI, and homeostasis model assessment of insulin resistance (3). Although we recognize that the sample size of our study subjects was smaller than theirs, the possible factors contributing to this inconsistency could be due to the different genetic background between Western and Japanese populations. Plus their study subjects had much higher BMI and fasting insulin levels in either nondiabetic or diabetic subjects than ours. We presume that their findings on the independency of the association of hepatic lipase or lipoprotein lipase activities to plasma adiponectin from insulin resistance and inflammation may be limited to certain populations. Further studies are needed to clarify this point in other populations.

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Low Plasma Adiponectin Levels Are Associated With Increased Hepatic Lipase Activity In Vivo

Response to Kobayashi et al.

We thank Kobayashi et al. (1) for their interest in our work on the relationship between adiponectin and human plasma lipases (2,3). These authors raise the question of whether differences in genetic background, BMI, or insulin levels of the studied populations could help to explain the differences with regard to statistical significance between their and our results.

Although we exclusively studied Caucasian subjects, other authors have recently reported an independent influence of adiponectin on hepatic lipase activity in Chinese and African-American populations (4,5). Therefore, the genetic background does not appear to play a major role in the association of adiponectin and hepatic lipase. However, we cannot rule out that the association may be different in a Japanese population. This could be due to the fact that the T-allele frequency of the functional −514C/T polymorphism in the hepatic lipase promoter has been reported to be much higher in Japanese than in Caucasian subjects (6). In