The controversial results in human studies (Tybjaerg-Hansen et al., 1993; Helio et al., 1994; Shoulders et al., 1996; Wijsman et al., 1998) and also in animal models (Furukawa et al., 1998; Gotoda et al., 1999; Pravenec et al., 1999a) indicate that the encountered differences between these studies include genetic heterogeneity. Thus, it seems useful to attempt to determine genes responsible for disturbances in carbohydrate and lipid metabolism in this newly defined insulin resistant PD/Cub strain.

PD/Cub is highly inbred, and therefore all measured metabolic values are safely reproducible. The maintenance of the genetic homogeneity of the PD/Cub strain is also ensured by a morphologic marker – polydactyly, which being autosomal recessive on the Wistar background prevents genetic contamination. Significantly increased triglyceride levels, hyperinsulinemia, elevated blood pressure and tissue resistance to insulin action qualify the PD/Cub rats for further detailed analysis of genetic determinants of metabolic disturbances associated with insulin resistance.

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