nancy in type 2 diabetic women. Maternal weight gain and the rate of caesarean deliveries were lower in type 2 diabetes. Gestational age at birth was significantly higher and the rate of large infants for gestational age lower in infants of women with type 2 diabetes. The rates of perinatal mortality and major congenital malformations were comparable in both groups. First-trimester A1C in type 2 and type 1 diabetic mothers with perinatal mortality was 9.9 and 8.1 ± 1.2%, respectively. Among pregnancies complicated by major congenital malformations, first-trimester A1C was >7% in 84% of women with type 1 diabetes and only in one woman (16.7%) with type 2 diabetes \( (P = 0.006) \). Neonatal distress respiratory syndrome was more frequent in infants of mothers with type 1 diabetes.

In our study, pregnancy outcomes in type 2 diabetic women were, if anything, similar to those with type 1 diabetes. In fact, women with type 2 diabetes had lower rates of large infants for gestational age, neonatal respiratory distress syndrome, and caesarean delivery.

As in some of the studies available, we found no significant differences in perinatal mortality or major congenital malformations between women with type 2 and type 1 diabetes (1–2). However, the results of five recent publications (3–7) suggest that type 2 diabetes could even represent a higher risk of perinatal mortality or congenital malformations than that conferred by type 1 diabetes. Similar rates of preconceptional care in women with type 1 and type 2 diabetes in our study could explain this discrepancy, as could the fact that gestational age at first visit to the clinic was comparable in both type 1 and type 2 diabetic women who did not undergo preconceptional care.

In our study, congenital malformations in type 2 diabetes were not related to poor first-trimester metabolic control in most cases. The concurrence in women with type 2 diabetes of factors other than glycemic control, such as obesity and older age, may account for this finding (8).

In conclusion, our study shows that pregnancy outcomes in type 2 diabetes are better than in type 1 diabetes when type 2 diabetic women receive as much intensified medical treatment during preconception and pregnancy as that given to type 1 diabetic women.

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We thank Gentilucci et al. (1) for their comments on our articles (2,3) regarding the pathogenic mechanisms of diabetes in patients with hepatitis C virus (HCV) infection. The authors question why an increased T-helper (Th)1 immune response can be simultaneously the major determinant of insulin resistance and responsible for a poor response to antiviral treatment. This question is based on the statement that Th1 immune response favors HCV clearance. However, although a vigorous Th1 response could play an essential role in spontaneous viral clearance, this is not so evident after interferon treatment. It should be noted that in sustained responders, pretreatment intrahepatic mRNA levels of γ-interferon and tumor necrosis factor-α were lower than in non-sustained responders (4). In addition, a lower Th2 response during antiviral treatment (specifically a decrease in interleukin [IL]-10 rather than an increase of Th1) has been associated with a long-term virological response (5,6). Tsai et al. (7) and Eckels et al. (8) demonstrated that in vitro cytokine responses to recombinant hepatitis C virus-specific T-cell reactivity during interferon and ribavirin treatment in chronic hepatitis C. Altogether, one can depict a complex scenario in which Th1 response is only one more of the actors. Future studies are needed to not only confirm that insulin resistance and type 2 diabetes are poor response predictors of antiviral treatment but also to unravel the mechanisms involved.

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