among patients with type 1 diabetes, but the reason for this is unclear.[7,8] It may be due to a genetic polymorphism in the gene encoding β-defensin 1.[8] However, there is no evidence that this genetic difference leads to an immunocompromised state allowing invasive fungal disease to occur. There have been case reports of patients with type 1 diabetes and diabetic ketoacidosis in whom severe opportunistic infections have developed.[9] The increased susceptibility may be attributed to the short-term acidic environment of diabetic ketoacidosis, which is ideal for certain opportunistic pathogens.

In summary, there is insufficient evidence to conclude that children with type 1 diabetes mellitus are immunocompromised. The evidence indicates that an immunocompromised state occurs only in the context of poor glycemic control with severe complications such as diabetic ketoacidosis or in adults with vasculopathy and peripheral neuropathy. Fortunately, with modern standards of care and education of families to manage intercurrent illness in their children with type 1 diabetes mellitus, hospital admission for diabetic ketoacidosis is now rare.

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Effect of thiazolidinediones on lipid profile

In their review of oral hypoglycemic therapy in type 2 diabetes mellitus, Alice Cheng and George Fantus[1] mention the effect of thiazolidinediones on high-density lipoprotein (HDL) cholesterol; I would like to add some comments about the effects of these agents on low-density lipoprotein (LDL) cholesterol and triglycerides.

In fact, the effect of thiazolidinediones on serum lipids and lipoproteins varies with the agent used (pioglitazone or rosiglitazone). As noted by Cheng and Fantus, HDL levels increase with either of these 2 drugs.[2–7] However, LDL cholesterol levels remain unchanged with pioglitazone monotherapy or a combination of pioglitazone with other oral hypoglycemic agents or insulin.[2–4] In contrast, LDL cholesterol levels increase with rosiglitazone monotherapy or combination therapy.[5–7] Although pioglitazone has been associated with a decrease in triglyceride levels,[2–4] the effects of rosiglitazone on triglycerides have been variable, ranging from a 2% increase to a 19% decrease.[6,7]

Studies directly comparing the 2 agents are scant, and the cause of this variation in lipid levels is unknown.

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[The authors respond:]

We appreciate Pankaj Madan's supplementary information to our article on oral antihyperglycemic therapy.[1] As Madan has correctly outlined, the studies comparing pioglitazone (monotherapy or combination therapy) with placebo have demonstrated no changes in LDL cholesterol,[2–4] whereas studies comparing rosiglitazone (monotherapy or combination therapy) with placebo have demonstrated an increase, ranging from 8% to 19%, in LDL cholesterol.[5–7] In clinical practice, this elevation may have a small impact, if any, for patients with diabetes mellitus using lipid-lowering therapy (statins) to achieve target LDL levels.[8,9]

The lack of direct-comparison studies makes it difficult to draw definitive conclusions regarding the lipid differences between the 2 medications. Simi-