metformin is absorbed intestinally, passes via the portal circulation into the liver, and exerts some hepatic effect that may underlie its mechanism of action (3,6). The cause for the apparent hepatotoxicity of metformin in this patient, which could not be confirmed on rechallenge, is unclear. A laboratory error is unlikely due to the consistent elevation of alkaline phosphatase and the stability of other chemistry tests. The patient and his wife denied symptoms suggestive of a concurrent subclinical viral illness (the episode took place in winter), binge drinking, or use of other medications. Other potential explanations for the apparent hepatotoxicity include desensitization to metformin so that rechallenge did not provoke the same inflammatory response as the initial challenge or failure of the patient to take the drug on rechallenge out of fear. Both the patient and his wife were emphatic that he had taken the metformin on rechallenge. One explanation for the transient hepatitis, given the ultrasound picture of gallstones, is the passage of a gallstone into the bile ducts. An alternate explanation is a subclinical viral illness. Neither the possibility of an effect of lovastatin or diltiazem nor a possible failure of the patient to take the metformin rechallenge can be excluded, despite our direction and his assurances. While it is disappointing not to be able to confirm the etiology of the apparent metformin-induced hepatotoxicity, it is reassuring that the drug did not appear to be implicated (on the basis of rechallenge). This observation also confirms the importance of careful ascertainment of adverse drug reactions, including observed rechallenge, to document the role of the putative toxin.

Confirmation of an adverse reaction is difficult and is usually dependent on rechallenge (7–9). In addition, diagnostic criteria and the assiduousness of surveillance differ at the national, institutional, and personal levels. In recognition of this, a European conference on pharmacovigilance recently proposed standard designations of drug-induced liver disorders and criteria for causality assessment (10). Applying these standards here, we can classify this case as demonstrating hepatocellular liver injury with a negative response to readministration of the drug.

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Errata

Hofmann MA, Kohl B, Zumbach MS, Borcea V, Bierhaus A, Henkels M, Amiral J, Fiehn W, Ziegler R, Wahl P, Nawroth PP: Hyperhomocyst(e)inemia and endothelial dysfunction in IDDM. *Diabetes Care* 20:1880–1886, 1997

The wrong version of this article was printed. Extensive and substantive revisions of this article were requested during the review process. However, the electronic file that was provided with the revised, accepted manuscript corresponded to the original version. The unrevised version was typeset, and proofs were approved by the corresponding author. The version of the article listed above is withdrawn, and the correct version will be printed in an upcoming issue of *Diabetes Care*.

Antonucci T, Whitcomb R, Norris RM, McLain R, Lockwood D: Impaired glucose tolerance is normalized by treatment with the thiazolidinedione troglitazone. *Diabetes Care* 20:188–193, 1997

Rebecca M. Norris, MD, has been added to the list of authors, as shown above. Dr. Norris was employed by the Parke-Davis Research Divsion of Warner Lambert at the time the research reported in this article was conducted.