

did either diabetic patients with documented normal GH secretion or an appropriately matched cohort of diabetic patients whose GH secretory status was undetermined"; this attempt to assert causation is faulty (1).

The stated objective of this study was to determine the role of GH in the development of diabetic retinopathy. Even if an association is proven (which apparently cannot be shown with the data presented), the leap of faith claiming causality is too broad to traverse.

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**Reference**

- Alzaid AA, Dinneen SF, Melton LJ III, Rizza RA: The role of growth hormone in the development of diabetic retinopathy. *Diabetes Care* 17:531-534, 1994

**Response to McGowan and Kiser**

**W**e thank Drs. McGowan and Kiser for their comments regarding our recently published paper examining the role of growth hormone in the development of diabetic retinopathy (1).

Our objective was not to determine the prevalence of growth hormone deficiency among diabetic people, nor did we claim that the growth hormone status observed in our patient sample necessarily represented that of the diabetic population at large. Rather, as stated in the paper, the study attempted to define the frequency of diabetic retinopathy

in a unique group of diabetic patients who were incidentally identified as being growth hormone-deficient during the conduct of a very large number of insulin tolerance tests.

The difference in the prevalence of retinopathy between the growth hormone-deficient (2 out of 16 patients) and -sufficient (5 out of 8 patients) is statistically significant using Pearson's  $\chi^2$  test ( $P = 0.011$ ) or Fisher's exact test ( $P = 0.021$ ). In fact, we think we know how Drs. McGowan and Kiser arrived at the  $P$  value of 0.099; they have analyzed the wrong data, as outlined in Tables 1 and 2.

Drs. McGowan and Kiser must surely recognize the difficulties and limitations inherent to all retrospective analysis studies. Though we would have liked more information on, for example, "lifetime" glycemic control, this was not possible; many of the patients in this study were seen in the era before the development of routine testing for glycosylated hemoglobin, and others had only a single or brief visitation to the Mayo Clinic. Furthermore, we very carefully alluded to this limitation, acknowledging that "...although the groups were matched for glycemic control, this by no means excludes chronic differences in blood glucose control. . ." (1).

Finally, we fully agree that evidence incriminating growth hormone in the pathogenesis of diabetic complications remains elusive. However, we believe our findings taken in conjunction

**Table 1—Correct layout of the data (Alzaid et al.)**

	Diabetic retinopathy		Total
	Yes	No	
Growth hormone			
Deficiency	2	14	16
Sufficiency	5	3	8

\* $P = 0.021$ .

**Table 2—Incorrect layout of the data (McGowan and Kiser)**

	Diabetic retinopathy		Total
	Yes	No	
Growth hormone			
Deficiency	2	16	18
Sufficiency	5	8	13

with previous reports implicate growth hormone in the development of diabetic microvascular complications. We look forward to future studies by Drs. McGowan and Kiser and others that either support or refute our findings.

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**Reference**

- Alzaid A, Dinneen SF, Melton LJ III, Rizza RA: The role of growth hormone in the development of diabetic retinopathy. *Diabetes Care* 17:531-534, 1994

**Atmospheric Pressure Effects on Glucose Monitoring Devices**

**C**hanges in altitude have been shown to affect the readings of blood glucose monitors (1,2). In particular, while handheld glucose monitors offer