CORRESPONDENCE

Plagiarism

To the Editor:

We wish to draw your attention to a serious case of plagiarism.

The article is titled "High Insulinogenic Nutrition—An Etiologic Factor for Obesity and the Metabolic Syndrome" by Wolfgang Kopp and was published in the July issue of *Metabolism*. It draws heavily on a similar hypothesis which we published in the journal *Diabetologia* in 1994. Yet, inexplicably, the latter paper is not cited at all.

I have enclosed both papers highlighting 21 separate instances of almost word-for-word plagiarism.

Moreover, the proposal that high glycemic index foods are involved in the etiology of the metabolic syndrome and obesity is one in which we have published extensively (see attached list of papers) and thus the idea is not novel.

We believe that this matter deserves an apology and a correction in the journal.

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Editor's Note

Drs Brand-Miller and Colagiuri submitted the above letter to *Metabolism* regarding the paper "High Insulinogenic Nutrition—An Etiologic Factor for Obesity and the Metabolic Syndrome" by Wolfgang Kopp, which was published in *Metabolism* 52:840–844, 2003. As is the custom when such letters are received, they are sent to the author of the paper in question with the request that a response be prepared and which would then be published in the "Letters to the Editor" section of the journal. The response from Dr Kopp is thus published with the letter from Drs Brand-Miller and Colagiuri. The paper by Drs Brand-Miller and Colagiuri, "The Carnivore Connection: Dietary Carbohydrate in the Evolution of NIDDM," was published in *Diabetologia* 37:1280–1286, 1994. Both of these papers postulated a critical role for the quantity and quality of dietary carbohydrate in the pathogenesis of obesity and insulin resistance in the paper by Dr Kopp and NIDDM in that of Brand-Miller and Colagiuri. While they both have relied on changes in nutrition going back in geologic time and human evolution, they differ in their postulation and interpretation of the mechanism which might be involved in explaining the current situation in regard to the prevalence of obesity, NIDDM, and the metabolic syndrome. Despite this difference, more than 20 of the statements and sentences in the paper by Dr Kopp are exactly or almost exactly the same as those in the paper by Brand-Miller and Colagiuri, but yet they were not attributed to that paper and it is not cited in the bibliography.

As Dr Kopp acknowledges in his reply, it would have been appropriate to include the paper by Brand-Miller and Colagiuri in the discussion of his paper since the subject matter was quite relevant. This omission is not as serious as the use of verbatim statements and sentences from a previous publication without proper citation. Although Dr Kopp indicates in his letter that he had planned to rephrase these sentences, in actual fact, he did not do it. Such plagiarism is not acceptable in the scientific literature. As a consequence, *Metabolism* will no longer consider any further manuscripts which are authored or coauthored by Dr Kopp.

James B. Field, MD
Editor-in-Chief

REPLY: High Insulinogenic Nutrition—An Etiologic Factor for Obesity and the Metabolic Syndrome

To the Editor:

When I received your letter last week and recognized the similarities of several sentences, I was so in shock that my first intention was to take the blame and get away from this awful situation. Now, after a couple of days, I finally was able to take a close look at the marked lines in both publications and I am able to answer your letter appropriately. The reason why I did not include the paper into my bibliography is this:

The pathomechanism proposed by Brand-Miller et al[1] is completely different from my hypothesis. Brand-Miller et al postulate that *insulin resistance is a genetic program* that has developed in our Paleolithic ancestors to cope with a shortage of dietary glucose. Brand-Miller et al propose that an insulin resistance genotype would have offered a survival advantage to specific populations consuming a low-carbohydrate, high-protein diet. They propose that the low carbohydrate nutrition of our Paleolithic ancestors has positively selected for individuals with insulin resistance. According to Brand-Miller et al, natural selection would therefore result in a high proportion of people with genetically determined insulin resistance.

In complete contrast to their hypothesis, I have proposed that insulin resistance (as well as obesity) develop as a result of high-carbohydrate nutrition: high-insulinogenic nutrition represents a chronic stimulus to the β cells that may induce an adaptive hypertrophy and a progressive dysregulation of the cells, resulting in postprandial hyperinsulinemia, and that *hyperinsulinemia promotes the development of insulin resistance* as well as of obesity. This definitely is in contrast to the hypothesis of Brand-Miller et al who have proposed a genetic selection for insulin resistance.