Table 1—*Prevalence of known diabetes, undiagnosed diabetes, and impaired fasting glucose in rural Andhra Pradesh, India, 2005 by age and sex groups*

	Overall	By age-group			
		30–39	40–49	50–59	60+
All					
Known	6.4 (5.6–7.2)	2.4 (1.3–3.5)	5.6 (4.1–7.2)	9.1 (7.1–11.1)	11.5 (9.5–13.6)
Undiagnosed	6.8 (5.9–7.6)	2.8 (1.7–3.9)	7.9 (6.0–9.8)	10.0 (7.9–12.2)	9.0 (7.3–10.7)
IFG	15.5 (16.8–14.0)	14.0 (11.6–16.3)	17.3 (14.6–19.9)	17.1 (14.3–19.8)	14.5 (12.4–16.6)
Male					
Known	6.8 (5.6–7.9)	2.6 (4.3–6.3)	6.3 (4.0–8.6)	10.1 (7.1–13.0)	11.5 (8.8–14.3)
Undiagnosed	7.5 (6.3–8.8)	3.1 (4.9–8.0)	8.0 (5.3–10.8)	11.5 (8.3–14.8)	10.7 (8.2–13.2)
IFG	16.6 (14.7–18.5)	16.4 (12.6–20.2)	18.1 (14.2–22.0)	18.7 (14.5–22.8)	13.4 (10.6–16.2)
Female					
Known	6.0 (5.0–7.1)	2.2 (0.8–3.5)	4.8 (2.9–6.9)	8.1 (5.5–10.7)	11.6 (8.6–14.5)
Undiagnosed	6.0 (4.9–7.0)	2.5 (1.2–3.9)	7.7 (5.1–10.2)	8.5 (5.7–11.3)	7.5 (5.2–9.7)
IFG	14.3 (12.7–15.9)	11.6 (8.8–14.4)	16.3 (12.7–19.8)	15.4 (11.8–19.0)	15.5 (12.3–18.6)

Data are percent (95% CI). IFG, impaired fasting glucose.

and treatment rates could produce substantial health benefits.

While these data are by no means representative of rural India as a whole, they do provide a reasonably precise and reliable estimate of diabetes and its treatment in the study area. Since much of rural India is likely to develop to a similar or greater extent as the Godavari region of Andhra Pradesh (2), the data provide an early indication of the likely huge burden of diabetes that will occur in rural India in the coming few decades. The generation of new evidence about detection and management strategies suited to resource poor setting is an urgent public health priority for India.

Additional data from this study can be viewed at: http://thegeorgeinstitute.org/

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COMMENTS AND RESPONSES

Validity of Glycemic Index Estimates in the Insulin Resistance Atherosclerosis Study

Response to Liese et al.

The article by Liese et al. (1) investigated the association of glycemic index, glycemic load, and total carbohydrate and fiber intake with direct measures of insulin sensitivity, insulin secretion, and adiposity in a cohort of 979 adults with normal and impaired glucose tolerance. The authors concluded that there were no significant associations of glycemic index, glycemic load, and carbohydrate intake with any measure of insulin sensitivity or secretion.

In their study, usual dietary intake was assessed via a 114-item food frequency questionnaire (FFQ) that had been previously "validated" in a subsample of the Insulin Resistance Atherosclerosis Study (IRAS) population. Unfortunately, the validation study showed that the FFQ did not confidently predict total carbohydrate intake as assessed by repeated 24-h recall of food intake. The Pearson correlation coefficient between the two methods was only 0.37 after adjustment for energy. Furthermore, the correlation coefficients for starch and sucrose were similarly low at $r = 0.33$ and 0.46, respectively (2).

Is a correlation coefficient between 0.3 and 0.4 adequate for the purposes of validation? Brunner et al. (3) have suggested that a value "of about 0.5 for most nutrients and 0.8 for alcohol between methods is good evidence that the FFQ has the ability to rank individuals . . . according to nutrient intake." Indeed, correlation coefficients in the order of 0.6–0.7 are more typical for energy-adjusted nutrients in FFQs (4). In the Nurses Health Study (5) and Health Professionals Follow-up Study (6), the correlation coefficient for energy-adjusted total carbohydrate was $r = 0.69$ (7).

Like most current FFQs, the IRAS questionnaire was not originally constructed for the purpose of measuring glycemic index, and glycemic index was not

included in the original validation study. Therefore, the validity of the calculated glycemic index values is essentially unknown. However, given the relatively low correlation coefficients for quantity of total carbohydrate, starches, and sucrose, it would seem unlikely that subtle differences in the quality (i.e., glycemic index) of that carbohydrate can be accurately assessed. The authors note that correlations for carbohydrate were higher in urban non-Hispanic white subjects (<20% of the sample) and that stratification for ethnicity did not alter their conclusions. However, due to the relatively small sample size, subanalysis may not have had sufficient statistical power to detect associations.

Interestingly, observational studies that found no association between glycemic index, glycemic load, and incidence of type 2 diabetes (8,9) also had validation correlation coefficients for total carbohydrate that were <0.5. In contrast, those studies finding glycemic index, glycemic load, or both to be predictive of diabetes (5,6,10) had *r* values >0.6.

Given the limitations of the IRAS study in relation to its ability to accurately assess total carbohydrate intake, starches, and sucrose, we believe that the conclusions drawn from the study of Liese et al., i.e., that glycemic index and glycemic load are not related to measures of insulin sensitivity, insulin secretion, and adiposity, should be interpreted with caution.

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A Single Factor Underlies the Metabolic Syndrome: A Confirmatory Factor Analysis

Response to Pladevall et al.

We were pleased to see the article by Pladevall et al. (1) in a recent issue of *Diabetes Care*, as it extends our prior results to Spanish and Mauritian samples. While the authors suggest that their findings differ from our results in the Normative Aging Study (2), we wish to clarify that we had previously demonstrated that a single factor underlies components of the metabolic syndrome using confirmatory factor analysis.

In our report, we showed that 10 risk factors associated with the metabolic syndrome were predicted by one primary factor, albeit through subfactors of insulin resistance, obesity, lipids, and blood pressure. The report of Pladevall et al. and the recently published review by Kahn et al. (3) refer to our model as a "correlated-factor model" or "four-factor model." However, our analyses went well beyond a model of correlated factors to establish that the four subfactors were not only correlated but indeed predicted by a single common factor (second-order factor) (Fig. 1 in ref. 2), which we labeled the metabolic syndrome. Furthermore, it should be noted that with a second-order factor, we have found that it is possible to include the full complement of components of the metabolic syndrome in confirmatory factor analysis. For example, it is possible to include both systolic and diastolic blood pressure, consistent with current metabolic syndrome criteria, rather than mean arterial pressure alone.

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