Prevalence and Determinants of Impaired Glucose Metabolism in Frail Elderly Patients: The Belgian Elderly Diabetes Survey (BEDS)

Michel P. Hermans, Thierry M. Peppersack, Lionel H. Godeaux, Ingo Beyer, and André P. Turc

Background. Although diabetes in elderly persons is generally type 2, the metabolic abnormalities associated with aging suggest that elderly persons may differ from younger persons with type 2 diabetes. In addition, nonobese elderly persons with type 2 diabetes show a marked impairment in insulin release accompanied by mild insulin resistance, whereas obese elderly persons have marked insulin resistance in the presence of “adequate” levels of insulin. Other factors that could adversely affect glucose tolerance in aging include drug use, associated disease, and other stressful conditions commonly encountered in geriatric inpatients units. The authors’ objectives in this study were 1) to prospectively assess the prevalence of glucose homeostasis abnormalities among elderly hospitalized patients and the degree to which it reflects abnormalities in insulin secretion or insulin sensitivity using homeostasis model assessment of fasting glucose, insulin, and C-peptide; and 2) to define the social, functional, pathologic, and nutritional characteristics of persons with impaired glucose tolerance or diabetes.

Methods. Ninety-eight patients underwent a comprehensive geriatric assessment. Determinants of glucose homeostasis were assessed using the homeostasis model assessment, which provides estimates of β-cell function (%B) and insulin sensitivity (%S).

Results. Twelve patients (12%) had fasting glucose concentrations greater than 110 mg/dl. Four patients had impaired fasting glucose levels greater than 110 mg/dl but less than 126 mg/dl (IFG group), and 8 patients had levels greater than 126 mg/dl (type 2 diabetes group). Except for a higher proportion of women in the IFG-diabetes group, the latter did not exhibit significant differences in functional, morbidity, or nutritional characteristics compared with the normal glucose tolerance group. The entire cohort (n = 98) presented with a mean (±SD) %B of 71% ± 47% and a mean %S of 208% ± 198%. Compared with the normal glucose tolerance group, the IFG-diabetes group had a fasting glycemia level of 142 ± 24 mg/dl (vs 92 ± 9 mg/dl), a %B of 43% ± 21% (vs 74% ± 45%), and a mean %S of 126% ± 113% (vs 219% ± 205%).

Conclusions. These data confirm the high prevalence of impaired glucose metabolism among elderly people, although the usual risk factors were not significantly increased. Marked β secretory defects seem to be the rule, whereas a significant degree of insulin resistance is unusual.

The prevalence of impaired glucose tolerance and diabetes mellitus increases with aging (1–3), and mechanisms of age-related glucose intolerance include decreased insulin sensitivity, decreased exercise level, increased adiposity, decreased β-cell function, and changes to dietary habits (3–12). Although diabetes in elderly persons is usually type 2, the metabolic abnormalities associated with aging suggest that elderly persons may differ from younger persons with type 2 diabetes (7,8). In addition, nonobese elderly persons with type 2 diabetes show a marked impairment in insulin release accompanied by mild insulin resistance, whereas obese elderly persons have marked insulin resistance in the presence of “adequate” levels of insulin (9,12). Other factors that could adversely affect glucose tolerance in aging include drug use, associated disease, and other stressful conditions commonly encountered in geriatric inpatients units.

The prevalence of diabetes varies widely in elderly persons. Detection of this condition in elderly populations has been insufficient. The prevalence in geriatric persons is even less well documented (13–17). Wingard and colleagues (18) reported a figure greater than 20% in participants aged 80 to 89 years from the Rancho-Bernardo survey, whereas an overall prevalence of 17.5% was observed in institutionalized participants (vs 12% in persons living in the community) (18,19). Conversely, diabetes tends to be overdiagnosed in geriatric populations when the diagnosis is based on admission fasting glucose level alone.

The aims of this study were: 1) to assess the prevalence of glucose metabolism impairment among hospitalized elderly patients from values obtained at admission and before discharge; 2) to precisely determine in those persons with abnormal glucose metabolism the degree of impairment in insulin secretion and insulin sensitivity, respectively, using the homeostasis model assessment (HOMA) model; and 3) to determine whether these abnormalities are associated with particular social, functional, pathologic, and nutritional characteristics.

Methods. During an 8-month period, 104 white persons were included from a randomly sampled cohort of 1445 participants...
in a prospective survey of glucose tolerance status and undiagnosed diabetes mellitus among an elderly hospitalized population. Admission to a geriatric ward was deemed necessary for a variety of acute medical conditions in these otherwise frail persons, who were found to be eligible for this study and studied prospectively and consecutively between January and August 1998, after stabilization of the acute condition that led to their being admitted to the geriatrics ward of one university and three general hospitals and before discharge. The Ethics Committee of Brugmann University Hospital (Free University of Brussels) approved the study protocol.

All participants had a fasting plasma glucose (FPG) measurement on admission, 4 participants with normal FPG values were lost to follow-up, and 2 participants were excluded because eventually we realized that they were known diabetics. The remaining participants (n = 98) had a repeated FPG measurement to confirm glucose tolerance status (20) and as part of a HOMA model designed to noninvasively measure their insulin sensitivity and β-cell function. The HOMA was performed on the evening before scheduled discharge in association with a comprehensive geriatric assessment including medical history and therapeutic, social, functional, and nutritional problem evaluation. These tests were performed an average of 20 days after admission. Glucose tolerance status was confirmed from both admission and the HOMA FPG levels, according to 1997 criteria from the American Diabetes Association (20,21).

The HOMA is a structural model of glucose–insulin interaction, with mathematical equations describing the functioning of the major organs involved. Assessment of the glucose and insulin concentrations in each person allows any combination of deficient β-cell function (as a percentage of normal, %β) and impaired insulin sensitivity (%S) to be evaluated. Basal (fasting) homeostasis is assessed by measuring FPG and insulin levels. The HOMA has been validated against independent measures of insulin sensitivity and β-cell function, including clamp-derived measures, and it is a practical, inexpensive, and minimally invasive test, and it has been tested in elderly persons (22–31).

For HOMA modeling, we used an antecubital cannula to sample venous blood, with the participant’s sampled arm wrapped in electric blankets to provide arterial blood. We obtained three fasting samples at 5-minute intervals (at 0, 5, and 10 minutes after insertion of the cannula) for radioimmunoassay insulin and glucose assays. We performed modeling of %B and %S with HOMA from the mean of these 3 FPG and fasting plasma insulin measurements.

We scored the severity of concomitant medical problems using a comorbidity index adapted from Greenfield and coworkers (32). We reviewed all concurrent medications that could interfere positively or negatively with glucose homeostasis (i.e., likely to increase or decrease plasma glucose, respectively), including angiotensin-converting enzyme inhibitors, estrogens, salicylic acid, thiazide diuretics, glucocorticoids, and beta-blockers.

The nutritional evaluation included the measurement of fasting serum albumin and a Mini Nutritional Assessment (33). The latter can be completed within 10 minutes by a well-trained person and is validated in an elderly population. It includes basic anthropometric measurements, a dietary questionnaire, and a global plus subjective assessment of the participants’ perception of their health and nutrition. Scoring of these allowed us to distinguish among elderly patients those who had “adequate” nutrition from others at risk for malnutrition or those who are frankly undernourished.

The functional evaluation assessed the activities of daily living with the help of the Katz scale, which involves the following tasks: bathing, dressing, transfer, toilet use, continence, and eating. With the Katz scale, each task is graded on a four-level scale (1 to 4), with the lower level representing absence of dependence, the intermediate (2 and 3) levels partial dependence, and the upper level representing maximal dependence to complete the task (34).

**Assays**

We measured plasma glucose levels using a conventional, in-house, enzymatic method kit. To decrease interparticipant variation in insulinemia levels, we simultaneously analyzed plasma samples for insulin assays in 1 central reference laboratory, and we assayed immunoreactive insulin using a bi-insulin immunoradiometric assay (Sanofi Diagnostics, Institut Pasteur, Marnes la Coquette, France), consisting of a sandwich assay with two monoclonal antibodies, one adsorbed on coated tubes and the other labeled with 125I insulin (radioimmunoassay insulin). The assay range was 2 to 17 mU/l.

**Statistical Analyses**

We collected the results in a database (Access, Microsoft, Redmond, WA), and we performed statistical analyses using Statistica 5 software (Microsoft). Results are presented as means (±1 SD). We used unpaired Student’s t test and nonparametric Welch’s t test to compare means between groups and Fisher’s exact test to assess group differences in proportion of variables.

**RESULTS**

Tables 1 and 2 list the participant characteristics. The mean duration of hospitalization was 21 days, and tests were thus performed an average of 20 days after admission (data not shown). The mean age was 84.7 ± 6 years, and the sex ratio (M:F) was 34:66. The mean body mass index was 22.2 ± 5 kg/m², a value within normal limits. As shown in Table 1, 8% and 54% of participants were using “negative” or “positive” drugs, respectively (i.e., drugs likely to negatively or positively affect glucose tolerance).

Twelve of 98 (12%) participants presented on admission and subsequently on HOMA day with FPG concentrations greater than 6.1 mmol/l. Among those, 4% had FPG concentrations greater than 6.1 mmol/l but less than 7 mmol/l (impaired fasting glucose [IFG] group). Eight participants (8% of the cohort) had FPG levels greater than 7 mmol/l (diabetes mellitus group), according to the revised criteria for defining diabetes (20). The FPG levels on admission were, on average, 1.56 mmol/l greater than fasting glycemia measured on HOMA day. This difference was 1.33 mmol/l in participants with normal glucose tolerance (NGT group;
In our geriatric cohort numbering 104 participants, the prevalence of IFG was thus 3.8%, and that of undiagnosed diabetes mellitus reached 7.7% (i.e., a total prevalence of 9.6% for known \( n = 2 \) plus undiagnosed \( n = 8 \) diabetes mellitus).

The subgroups with IFG or diabetes did not exhibit significant differences in functional characteristics compared with NGT participants (NGT group; Table 1). Nevertheless, the Greenfield score was higher in NGT participants, as was the Katz scale score, which could denote a higher degree of comorbidity and a lesser degree of functional ability. The Katz scale results confirm the geriatric profile of the studied population, because fewer than one half the participants could, on discharge, perform the activities of daily living without external assistance (Figure 1).

We found no significant differences between the NGT and IFG-diabetes groups regarding body mass index, serum albumin, or Mini Nutritional Assessment scores (Table 1).

With respect to glucose tolerance determinants, the combined participants (\( n = 98 \)) presented with a mean β-cell function (%B) of 71% ± 47% and a mean insulin sensitivity (%S) of 208% ± 198% (Table 2). By comparison, the IFG-diabetes subgroup (\( n = 12 \)) had a mean fasting glycemia level of 7.89 ± 1.33 mmol/l, a mean %B of 43% ± 21%, and a mean %S of 126% ± 113% (vs, respectively, 5.11 ± 0.50 mmol/l, 74% ± 45% [%B], and 219% ± 205% [%S] in the NGT subgroup; Table 2). Participants with IFG or diabetes had significantly decreased %B and %S compared with NGT participants (−42% and −43%, respectively). They also had the greatest difference in FPG between admission and HOMA day levels (Table 2).

Figure 2 shows individual insulin sensitivity (%S) and β-cell function (%B) values for all participants (\( n = 98 \)), ranked according to %B, and log-scaled on the Y axis. Most participants in the entire cohort and, to a lesser extent, in the IFG-diabetes subgroup, showed either normal (=100%) or greater than normal insulin sensitivity, together with normal or moderately decreased β-cell function. Participants with IFG (\( n = 4 \)) or diabetes (\( n = 8 \)) had significantly decreased %B and %S compared with NGT participants, although both abnormalities were not necessarily found in combination. Indeed, certain participants exhibited a single deficit in either of both parameters.

### Table 1. Patient’s Characteristics (Social, Medical, Nutritional, and Functional)

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>All Subjects</th>
<th>NGT</th>
<th>IFG + Diabetes</th>
<th>( p )</th>
</tr>
</thead>
<tbody>
<tr>
<td>( N )</td>
<td>98</td>
<td>86</td>
<td>12</td>
<td></td>
</tr>
<tr>
<td>Age, y</td>
<td>84.7 (6.0)</td>
<td>84.9 (5.9)</td>
<td>82.7 (7.1)</td>
<td>NS</td>
</tr>
<tr>
<td>Sex ratio, F:M</td>
<td>66:34</td>
<td>64:36</td>
<td>83:17</td>
<td>NS</td>
</tr>
<tr>
<td>Body mass index, kg/m²</td>
<td>22.2 (5.0)</td>
<td>22.3 (5.2)</td>
<td>21.6 (3.5)</td>
<td>NS</td>
</tr>
<tr>
<td>Greenfield index</td>
<td>11.0 [8.0–15.0]</td>
<td>11.0 [8.0–15.0]</td>
<td>9.5 [7.8–12.3]</td>
<td>NS</td>
</tr>
<tr>
<td>“Negative” drugs (%)</td>
<td>8</td>
<td>9</td>
<td>0</td>
<td>NS</td>
</tr>
<tr>
<td>“Positive” drugs (%)</td>
<td>54</td>
<td>53</td>
<td>67</td>
<td>NS</td>
</tr>
<tr>
<td>Serum albumin, g/l</td>
<td>3.57 (0.64)</td>
<td>3.55 (0.63)</td>
<td>3.66 (0.71)</td>
<td>NS</td>
</tr>
<tr>
<td>Katz’s scale score</td>
<td>10.0 [7.0–15.0]</td>
<td>10.5 [7.0–15.0]</td>
<td>8.5 [6.0–16.3]</td>
<td>NS</td>
</tr>
</tbody>
</table>

**Note:** Values are means (1 SD) or medians [percentile 25–75]. Homocysteine was measured using the HOMA model, alongside a social, functional, pathologic, and nutritional survey. Our results are in keeping with the geriatric profile of the studied population: old and frail status (mean age, ≈85 years), use of multiple medications, concurrence of diseases, and high degree of disability.

We found a prevalence of IFG of approximately 4%, and of diabetes mellitus of approximately 10%, most of which (≈80%) was undiagnosed. To avoid overdiagnosing diabetes based on admission glycemia level alone, participants had a repeat FPG measurement, as part of the HOMA test on the eve of discharge, to confirm glucose tolerance status according to American Diabetes Association criteria (20,21).

The relevance of FPG as a screening tool for type 2 diabetes mellitus was analyzed in the upper-middle-class white community of Rancho Bernardo, California. Although specificity did not change, sensitivity decreased with age, with a poorer sensitivity of FPG with increasing age reflecting that the numerator of the sensitivity equation was not affected by age, whereas the denominator increased with age. From

### Table 2. Patient’s Metabolic Characteristics

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>All Subjects</th>
<th>NGT</th>
<th>IFG + Diabetes</th>
<th>( p )</th>
</tr>
</thead>
<tbody>
<tr>
<td>( N )</td>
<td>98</td>
<td>86</td>
<td>12</td>
<td></td>
</tr>
<tr>
<td>Fasting plasma glucose on admission (mmol/l)</td>
<td>7.00 (2.94)</td>
<td>6.44 (2.28)</td>
<td>10.83 (4.11)</td>
<td>&lt;.005</td>
</tr>
<tr>
<td>Fasting plasma glucose on HOMA day, mmol/l</td>
<td>5.44 (1.11)</td>
<td>5.11 (0.50)</td>
<td>7.89 (1.33)</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>Delta glucose (admission vs HOMA day, mmol/l)</td>
<td>0.75 [0.22–2.17]</td>
<td>0.71 [0.07–1.74]</td>
<td>2.34 [−0.01–4.93]</td>
<td>NS</td>
</tr>
<tr>
<td>Fasting insulinemia, pmol/l</td>
<td>45.6 (51.5)</td>
<td>42.7 (51.9)</td>
<td>66.3 (44.6)</td>
<td>NS</td>
</tr>
<tr>
<td>HOMA %B</td>
<td>71 (47)</td>
<td>74 (45)</td>
<td>43 (21)</td>
<td>&lt;.0005</td>
</tr>
<tr>
<td>HOMA %S</td>
<td>208 (198)</td>
<td>219 (205)</td>
<td>126 (113)</td>
<td>&lt;.05</td>
</tr>
</tbody>
</table>

**Note:** Values are means (1 SD) or medians [percentile 25–75]. Homeostasis model assessment (HOMA) %B and %S were calculated from the mean of three baseline samples of glucose and radioimmunoassay-insulin or C-peptide. \( p \) denotes significances of differences between normal glucose tolerance (NGT) and impaired fasting glucose (IFG) plus diabetes groups, assessed from either unpaired Student’s \( t \) tests (two tailed), Welch’s test (two tailed), or Fischer’s Exact test.
this study, age-specific screening criteria were considered not warranted for diabetes (35). Nevertheless, our results show that the extent of FPG decrement between admission and discharge values was far from negligible, particularly in participants with impaired glucose metabolism.

The prevalences of impaired glucose tolerance and diabetes mellitus increase with ageing (1–3), although published prevalence rates vary widely. Reports on the prevalences of diagnosed and undiagnosed diabetes among elderly persons suggest that detection (and care) of diabetes in elderly persons may be inadequate (13–15). Thus, the Coventry Diabetes Study found 66% and 45% of undiagnosed diabetes in European and South-Asian participants older than 65 years, respectively (14), whereas that proportion was 33% in Finnish persons older than 70 years (15). Wilson and colleagues (36) reported a 10% to 15% prevalence from the Framingham study, whereas Neil and coworkers (37) in Oxford reported a 3% prevalence in persons older than 60 years. Wingard and colleagues (18) reported age-adjusted rates of 16.5% in men and 13.1% in women aged 50 to 89 years from the Rancho-Bernardo community-based study.

The prevalence of diabetes in geriatric participants is even less well documented. Wingard and coworkers (18) reported a 21.8% prevalence in men and 23.7% in women aged 80 to 89 years from the community-based Rancho-Bernardo survey (18). Rockwood and associates (19) reported an overall prevalence of 17.5% in participants living in institutions (compared with 12% in community-dwelling participants). The prevalence figures reported in our survey are somewhat lower in our frail and rather lean population, although the proportion of undiagnosed conditions (IFG or diabetes) remains high.

We used the HOMA, a noninvasive structural model of glucose–insulin interaction, to assess in each participant β-cell function and insulin sensitivity. HOMA was validated against independent measures of insulin sensitivity and β-cell function (22–31,38). Our results show that most participants in the entire cohort and, to a lesser degree, in the IFG-diabetes subgroup, had either normal or higher than normal insulin sensitivity, together with normal or moderately decreased β-cell function, whereas participants with IFG or diabetes had significantly decreased β-cell function and insulin sensitivity, although both abnormalities were not necessarily found combined in persons with IFG homeostasis. Indeed, certain participants exhibited single deficits in either of these parameters.

The HOMA is generally used to assess insulin sensitivity and release in large epidemiologic studies or metabolic studies involving ambulatory or otherwise healthy persons. The model is also validated in hospitalized patients. Thus, a French team reported on the utility of HOMA as a predictor of insulin-requiring stage in hospitalized patients with type 2 diabetes. That team found that HOMA was both a simple and a clinically valid predictor of the two major metabolic parameters involved in glucose homeostasis, namely insulin sensitivity and β-cell function. These authors also found that HOMA modeling was superior to standard clinical indices.
used to predict insulin requirements (39). In another setting, Kishimoto and colleagues (40) also reported using HOMA in hospitalized persons. Our study included primarily nonobese persons with various degrees of glucose homeostasis (from normal [the majority] to impaired glucose tolerance and diabetes). Another study (41) found that HOMA-IR was highly correlated with clamp insulin resistance in a population of both normal weight and moderately obese type 2 diabetic and nondiabetic participants.

Another line of evidence suggests the validity of using HOMA in elderly persons. First, the physiologic assumptions of the model do not preclude its use beyond a certain age, although some care should be exercised when interpreting the results (this is actually the case for all methods aimed at measuring insulin sensitivity). For this reason, we insist on making relative assumptions in interpreting the respective abnormalities in β-cell function and insulin sensitivity. Katsuaki and associates (42) specifically addressed the topic of using HOMA as an indicator of insulin resistance in elderly patients with type 2 diabetes mellitus. They found that this noninvasive method was correlated with clamp measures as long as diabetes was not poorly controlled. In our study, type 2 diabetes was well controlled in the majority of patients.

Katsuaki and colleagues (43) also reported that HOMA modeling was appropriate for evaluating the clinical course of patients with type 2 diabetes when compared with the reference clamp method. They concluded that HOMA was a useful method not only for diagnosing insulin resistance but also for follow-up during treatment of patients with type 2 diabetes (43). In addition, evidence is mounting for the use of HOMA modeling in elderly people, as inferred from the following list of recent articles in which HOMA indices were specifically used in this age group (44–49).

Ferrara and Goldberg (50) have questioned the validity of using a HOMA-based index of insulin resistance based on limited correlation with a reference method in persons with impaired glucose tolerance. However, this comparison of performance was based on a calculated HOMA estimate rather than on the computer-based software, as recommended (30).

We accounted for the specificity of the insulin assay, which is a valid point. Many authors have not allowed for this confounding factor when they have used the HOMA mathematical model in its simple, noncomputerized equation version. We used the computer version of the HOMA model (as we recommended in our 1998 article [30], which allows for selection of the appropriate type of assay (radioimmunoassay vs specific) to correct for proinsulin in the calculations. We also used a central laboratory for all assays, and the assay method that we used throughout was highly specific for insulin.

The observed difference in fasting glucose over the sampling interval does not necessarily imply an intrinsic difference in the determinants of glucose metabolism over time, but rather, as we show, a difference in the HOMA product of insulin sensitivity and β-cell function. Using the oral glucose tolerance test as a measure of glucose metabolism instead of HOMA would not have provided greater insight into the respective abnormalities underlying the differences in fasting (and likely post-load) glycemia. Thus, glucose and insulin areas under the curve after an oral glucose tolerance test are under the same determinants as those underlying the assumptions of the HOMA model. This issue of fasting versus post-load homeostasis was specifically addressed in developing the CIGMA part of the mathematical development of the HOMA-CIGMA model. CIGMA was designed to explore the post-load state. Because we have shown previously that both HOMA and CIGMA are closely interrelated for assessing both β-cell function and insulin sensitivity in participants spanning the range of glucose tolerance from normal to diabetes (31), we preferred to use the less invasive HOMA rather than CIGMA part of the model, in keeping with most authors who have used this model. Conversely, unraveling the precise underlying mechanisms responsible for changes in insulin resistance and β-cell function in our elderly participants would have required highly complex metabolic investigations (such as isotopic clamps and biopsy) that were beyond the scope of this study.

The presence of combined β-cell secretion and insulin sensitivity deficits in diabetic persons is implicit and expected from what is known about type 2 diabetes physiopathology (23,24,26,38,51–62). Indeed, diabetes mellitus in persons older than 65 years is usually considered type 2. Invoked mechanisms of age-related glucose intolerance include: 1) decreased insulin sensitivity (i.e., increased insulin resistance), either from (post-) receptor abnormalities, decreased exercise level, or increased adipose tissue; 2) decreased β-cell function or insulin degradation or removal; and 3) altered dietary habits and decreased insulin-to-glucose ratio (3–12).

Compared with those commonly associated with obesity and type 2 diabetes, the metabolic abnormalities associated with aging suggest, however, that elderly persons differ from younger persons with type 2 diabetes. Thus, in addition to having FPG and insulin concentrations similar to those of younger persons, the glucose tolerance of elderly participants was markedly impaired. Basal hepatic glucose output was similar in young and elderly men, but hepatic glucose output suppression in response to insulin occurred later in elderly persons (7,8). In nonobese elderly persons with type 2 diabetes, a marked impairment in insulin release was observed, possibly attributable to β-cell glucotoxicity (9,12).

Mooradian and colleagues (63) compared the clinical features of frail nursing home diabetic patients with those of nondiabetic nursing home residents. They found that diabetic patients had a higher prevalence of renal failure, proteinuria, retinopathy, neuropathy, and infections. In their study, 21% of nursing home residents with diabetes had body mass indices that were more than 20% below average, suggesting that malnutrition is an important problem in diabetic patients in the nursing home setting (63).

Other factors that could adversely affect glucose tolerance in aging include drug use, interfering conditions from associated diseases, and other stressful conditions commonly encountered during acute hospitalization. In our survey, the subgroups with IFG or diabetes did not exhibit significant differences in functional characteristics compared with participants with NGT, although we did note a higher
comorbidity score in NGT participants and Katz scale scores that tended toward higher comorbidity and lesser functional ability. In addition, we found no significant differences between the NGT and IFG-diabetes groups for body mass index, serum albumin, and Mini Nutritional Assessment scores.

Conclusion
Our survey confirms the high prevalence of diagnosed and undiagnosed cases of impaired glucose metabolism among elderly persons. Impaired glucose homeostasis is characterized by a marked heterogeneity in the degree of β-cell function and insulin resistance, and by a high proportion of significant secretory deficit in the presence of normal or enhanced insulin sensitivity. The presence of IFG or diabetes was not associated with specific functional characteristics or with higher comorbidity ratings. Conventional risk factors for type 2 diabetes, as encountered in relatively younger persons, were not involved in elderly participants, especially regarding insulin resistance.

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