is provided by many sources (e.g. staff, relatives, religious or community leaders). Such contributions are common on hospital wards, but their informal nature may not prompt entries in clinical notes. When discussing our data with staff, it was highly disputed, with nursing staff feeling that they often made provision for spiritual support, although agreeing that it was often ad hoc and inconsistent due, in part, to time constraints.

Conclusion

This study supports suggestions (both anecdotal and evidence-based) that palliative care offered to elderly patients dying from non-malignant conditions requires improvement. One factor limiting effective management may be lack of awareness of the relevance of palliative care in non-malignant diseases. This is being addressed by greater education of medical and nursing staff, by the utilisation of documents such as the LCP, and by a more comprehensive approach to counselling patients and relatives throughout the course of a disease from the time of initial diagnosis.

Senior clinicians have a role in supporting junior members of their team in making decisions regarding resuscitation, symptom management and withdrawal of interventions. Data obtained from this study has been fed back to relevant stakeholders with several positive outcomes. As a result of this work, the LCP has been adopted within our department. A 4-day course aimed at improving knowledge and standards in elderly care practice has been developed, including a session specifically addressing some of the practical issues surrounding palliative care in the elderly.

By increasing awareness of issues relevant to care of dying patients and introducing the LCP, many of the problems associated with communication, prescribing and ‘missing data’ are being addressed, thus improving care. With its careful monitoring of variance from pre-defined standards, clear entry criteria, and accompanying guidance, the LCP provides a valuable resource for improving and measuring patient care allowing us to ‘work smarter’.

Key points

- Only a minority of terminally ill patients receive formal palliative care input.
- Provision of palliative care support for the elderly with non-malignant diseases is often poor.
- Careful attention to symptom management is essential for non-malignant conditions (e.g. dementia).
- Further research is required into palliation of non-malignant diseases in the elderly.

Conflicts of interest

None.
studies have reported that older individuals are more insulin resistant than younger individuals. In addition, obesity plays a central role in insulin resistance, although most elderly Japanese subjects are not obese. On the other hand, the effect of age on beta-cell function has been a matter of debate [9–12]. Notably, Japanese subjects have lower beta-cell function compared with other ethnic groups [13]. In the present study, we investigated the relationship between insulin resistance and beta-cell dysfunction in older Japanese adults without diabetes.

Methods

Subjects

A total of 579 non-diabetic Japanese subjects aged 50 or older were included in this analysis. The study sample was drawn from a database of 954 individuals who had undergone a 75-g oral glucose tolerance test (OGTT) at our institution as part of an evaluation of glucose intolerance based on the presence of one or more risk factors: overweight, a first-degree relative with diabetes, past diagnosis of gestational diabetes mellitus, hypertension, dyslipidemia or a history of vascular disease. Exclusion criteria were known diabetes, individuals with fasting hyperglycemia ≥7.0 mmol/l or 2-h plasma glucose ≥11.1 mmol/l, incomplete data for calculating the insulin sensitivity index (ISI), or those with signs of serious liver diseases (clinically diagnosed chronic hepatitis or liver cirrhosis), renal failure [estimated glomerular filtration rate (GFR) ≥15 ml/min], chronic infectious diseases, endocrine diseases that affect insulin secretion or insulin sensitivity, cancer, or those with a prior gastrectomy. This study was performed in accordance with the Helsinki Declaration, and written informed consent was obtained from each participant.

Measures

A 75-g OGTT was performed after a 10-h overnight fast. Plasma glucose was determined using a glucose oxidase autoanalyzer. Plasma insulin was measured using an electrochemiluminescence immunoassay (ECL-IA, Roche-Diagnostic, Basel, Switzerland), which does not cross-react with pro-insulin. Insulin sensitivity was evaluated by the homeostasis model assessment of insulin resistance (HOMA-R) [14], quantitative insulin check index (QUICKI) [15] and the ISI-composite proposed by Matsuda and DeFronzo [16]. To assess insulin secretion, insulinogenic index was defined as the ratio of the increment of plasma insulin to that of plasma glucose 30 min after glucose loading [17]. Estimated GFR was calculated using the formula of the modification of diet in renal disease study [18].

Statistical analysis

All statistical analyses were performed using the SYSTAT statistical package (Systat Software, Inc., CA). One-way analysis of variance was used to compare variables among age tertiles (50–60, 61–68, and 69–90 years). P-values <0.05 were considered statistically significant.

Results

Subjects were divided into three groups based on age tertiles (Table 1). A higher proportion of women was found in older subjects. Body mass index (BMI) and waist circumference decreased in parallel with ageing. Of note, only 25 to 35% of study subjects were overweight as defined by BMI ≥25 kg/m². Significant differences among age tertiles were detected in diastolic blood pressure (DBP), fasting insulin, total cholesterol and triglycerides. In addition, estimated GFR decreased significantly as age advanced. No difference among age tertiles was found in other variables. Table 2 shows the various markers of insulin resistance and beta-cell function among the age tertiles after adjustment for confounding factors (gender, BMI, waist circumference, DBP, total cholesterol, triglycerides and estimated GFR). HOMA-R and QUICKI were comparable among the age tertiles, whereas ISI-composite decreased significantly as age advanced. Insulinogenic index did not differ among age tertiles.

Discussion

This study focused on Japanese aged 50 or older without diabetes. Of the risk factors for insulin resistance in older people, obesity appears to be most common in Caucasian populations. Importantly, the degree of obesity seen in older Japanese is lower than that seen in Western countries [19]. Indeed, in our older subjects, only 25–35% of study subjects were considered overweight as defined by BMI ≥25 kg/m². In addition, BMI and waist circumference decreased in parallel with advancing age.

To our knowledge, the present study provides the first precise information on insulin resistance and insulin secretion in older Japanese adults without diabetes. We considered the effect of kidney function because it has recently been suggested that impaired kidney function may affect insulin resistance [20]. After adjustment for confounding factors, including kidney function, our data demonstrated a decline in insulin sensitivity (obtained by ISI-composite) as age advances.

Age-related beta-cell dysfunction may contribute to the high rate of glucose tolerance in the older population [12]. Importantly, it has been reported that Japanese have lower beta-cell function compared with other ethnic groups [13]. In the present study, insulinogenic index did not differ among the age tertiles. These findings could have several explanations. An insulin response to oral glucose load with advancing age may be abolished after adjustment for obesity-related insulin resistance [21], although our older subjects were not obese. Another possibility is that insulin clearance may be changed in the elderly [22]. Decreased insulin clearance as a result of impaired renal function potentially may affect plasma insulin levels, but we believe this to be unlikely. In our data, fasting insulin levels were lower in older people despite decreased kidney function with ageing.
Research letters

Table 1. Clinical characteristics of study subjects classified by age tertiles

<table>
<thead>
<tr>
<th></th>
<th>Lower tertile</th>
<th>Middle tertile</th>
<th>Higher tertile</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years) [range]</td>
<td>55.3 ± 0.2 [50–60]</td>
<td>64.9 ± 0.2 [61–68]</td>
<td>74.4 ± 0.2 [69–90]</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Female (%)</td>
<td>24.0</td>
<td>36.5</td>
<td>37.2</td>
<td>0.007</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>24.2 ± 0.2</td>
<td>23.8 ± 0.2</td>
<td>23.1 ± 0.2</td>
<td>0.006</td>
</tr>
<tr>
<td>BMI ≥ 25 (%)</td>
<td>35.4</td>
<td>31.8</td>
<td>25.1</td>
<td>0.084</td>
</tr>
<tr>
<td>Waist (cm)</td>
<td>86.0 ± 0.8</td>
<td>82.6 ± 0.8</td>
<td>79.0 ± 0.8</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>SBP (mm Hg)</td>
<td>130.3 ± 1.3</td>
<td>126.1 ± 1.3</td>
<td>131.7 ± 1.3</td>
<td>0.159</td>
</tr>
<tr>
<td>DBP (mm Hg)</td>
<td>76.3 ± 0.9</td>
<td>73.3 ± 0.9</td>
<td>72.9 ± 0.9</td>
<td>0.012</td>
</tr>
<tr>
<td>Fasting glucose (mmol/l)</td>
<td>5.33 ± 0.04</td>
<td>5.26 ± 0.04</td>
<td>5.29 ± 0.04</td>
<td>0.503</td>
</tr>
<tr>
<td>Fasting insulin (pmol/l)</td>
<td>7.27 ± 0.27</td>
<td>7.32 ± 0.27</td>
<td>6.31 ± 0.27</td>
<td>0.013</td>
</tr>
<tr>
<td>Total cholesterol (mmol/l)</td>
<td>5.34 ± 0.08</td>
<td>5.35 ± 0.08</td>
<td>4.85 ± 0.08</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Triglycerides (mmol/l)</td>
<td>1.68 ± 0.06</td>
<td>1.42 ± 0.06</td>
<td>1.16 ± 0.06</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>HDL cholesterol (mmol/l)</td>
<td>1.31 ± 0.03</td>
<td>1.36 ± 0.03</td>
<td>1.26 ± 0.03</td>
<td>0.141</td>
</tr>
<tr>
<td>Estimated GFR (ml/min)</td>
<td>86.2 ± 1.5</td>
<td>80.6 ± 1.5</td>
<td>75.7 ± 1.5</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Data are mean ± SE or %.

BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; HDL-C, high-density lipoprotein cholesterol; GFR, glomerular filtration rate.

Table 2. Insulin resistance and beta-cell function of study subjects classified by age tertiles

<table>
<thead>
<tr>
<th></th>
<th>Lower tertile</th>
<th>Middle tertile</th>
<th>Higher tertile</th>
<th>Adjusted (P^a)</th>
</tr>
</thead>
<tbody>
<tr>
<td>HOMA-R</td>
<td>1.63 ± 0.02</td>
<td>1.62 ± 0.02</td>
<td>1.65 ± 0.10</td>
<td>0.191</td>
</tr>
<tr>
<td>QUICKI</td>
<td>0.300 ± 0.002</td>
<td>0.302 ± 0.001</td>
<td>0.295 ± 0.002</td>
<td>0.104</td>
</tr>
<tr>
<td>ISI-composite</td>
<td>6.56 ± 0.14</td>
<td>6.13 ± 0.14</td>
<td>5.84 ± 0.14</td>
<td>0.003</td>
</tr>
<tr>
<td>Insulinogenic index</td>
<td>0.676 ± 0.064</td>
<td>0.686 ± 0.084</td>
<td>0.661 ± 0.041</td>
<td>0.916</td>
</tr>
</tbody>
</table>

Data are mean ± SE.

\(^a\) Adjusted for gender, body mass index, waist, diastolic blood pressure, fasting insulin, total cholesterol, triglycerides, and estimated GFR.

HOMA-R, homeostasis model assessment of insulin resistance; QUICKI, quantitative insulin check index; ISI-composite, insulin sensitivity index composite.

This study had several limitations. First, the validity of surrogate indices of insulin sensitivity must be considered. To date, clinical research has used HOMA-R or QUICKI as surrogate measures of insulin resistance. However, HOMA-R and QUICKI have obvious limitations, as they measure insulin sensitivity using only fasting values of glucose and insulin. Fasting glucose concentrations primarily depend on hepatic glucose production, and the ability of fasting insulin levels to predict insulin resistance is relatively modest. The concordance between compensatory fasting hyperinsulinemia and peripheral insulin resistance is not strong [23]. In addition, fasting hyperinsulinemia is not common, especially in lean Japanese subjects. In this context, we used a more elaborate OGTT-based method (ISI-composite) because this index was thought to be better for assessing insulin sensitivity than HOMA-R or QUICKI. Secondly, we did not measure other confounding factors. Age-related changes in body fat composition, decline in physical activity, and decreases in sex-hormone levels have been hypothesised as being among the main causes of insulin resistance [5]. Finally, as a cross-sectional study, the present analysis is limited in its ability to elucidate causal relationships between advancing age and insulin resistance. The participants of the current study were from a single institution and may not represent the overall elderly population in Japan.

In conclusion, in Japanese adults without diabetes, insulin sensitivity decreased significantly with advancing age after adjustment of other confounding factors, whereas beta-cell function did not decline as age advanced.

Key points

- Older Japanese adults without diabetes are not obese; only 25–35% of the study subjects were overweight as defined by BMI ≥25 kg/m².
- Insulin sensitivity decreased significantly with advancing age after adjustment for other confounding factors.
- Beta-cell function did not decline with advancing age.

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Conflicts of interest

None.

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References


Hearing loss and cortical atrophy in a population-based study on non-demented women

SIR—Age-related hearing loss (ARHL) is common and may cause difficulties in perception and understanding speech. ARHL is influenced by ageing, genetic factors, noise and other environmental factors [1]. Age-related brain atrophy is related to reductions in the number of neurons, and decreased cortical synaptic density. Brain atrophy may potentially damage hearing capacity through the impairment of primary and secondary auditory centres which are located in the temporal lobe. To our knowledge, no study has examined the relationship between brain atrophy and ARHL.

In a population-based sample of non-demented women, we examined whether there is an association between ARHL and brain atrophy, and whether this relationship is influenced by cognitive function.

Methods

Population

This study is part of the representative Gerontological and Geriatric Population Study in Gothenburg [2], and the Prospective Population Study of Women [3]. In 1992–93, 70-year-old women were examined (n = 299). Pure tone audiometry was performed in 163 randomly selected participants [4], of whom 80 had a brain computerised tomography (CT) scan. In 2000–01, 70-year-old women were examined (n = 523). Pure tone audiometry was performed in 42 randomly selected women, of whom 31 had a brain CT scan. Since there were no differences of hearing capacity and brain atrophy between these two groups, they were pooled. One hundred and eleven in the study population were comprised of 70-year-old women. None were demented according to criteria of the Diagnostic and Statistical Manual of Mental Disorders, Revised Third Edition [5].

Five right ears with pronounced hearing impairment of peripheral origin, not related to ageing, were excluded from the study. Three had conductive hearing loss (two chronic otitis media, and one otosclerosis), and two had severe, unilateral sensorineural hearing loss (one Ménière’s disease,