Serum Leptin and Vascular Risk Factors in Obstructive Sleep Apnea*

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Study objectives: To define the metabolic profile relevant to vascular risks in obstructive sleep apnea (OSA) and the role of leptin resistance in this risk profile.

Design: Case control study.

Setting: Sleep Laboratory, Queen Mary Hospital, University of Hong Kong, China.

Methods: Thirty OSA subjects were matched with 30 non-OSA subjects for body mass index (BMI), age, sex, and menopausal status. Neck, waist, and hip girth, skinfold thickness, and fasting serum levels of lipids, glucose, insulin, and leptin were compared between these two groups.

Results: Compared with control subjects with a similar BMI but without OSA, the OSA group had a significantly more adverse vascular risk factor profile, including dyslipidemia, higher diastolic BP, insulin resistance, and greater adiposity reflected by skinfold thickness. OSA subjects also had higher circulating leptin levels (9.18 ± 4.24 ng/mL vs 6.54 ± 3.81 ng/mL, mean ± SD, p = 0.001). Serum leptin levels correlated positively with BMI, skinfold thickness, serum cholesterol, low-density lipoprotein cholesterol, insulin, insulin/glucose ratio, apnea-hypopnea index, and oxygen desaturation time; multiple stepwise regression analysis identified skinfold thickness, waist/hip ratio, serum low-density lipoprotein cholesterol, and diastolic BP as independent correlates, while only serum insulin and diastolic BP were independent correlates in OSA subjects. After treatment with nasal continuous positive airway pressure for 6 months, there was a significant decrease in circulating leptin (p = 0.01) and triglyceride levels (p = 0.02) without change in other parameters.

Conclusion: Despite controlling for BMI, OSA subjects showed distinct profiles with clustering of vascular risk factors. Hyperleptinemia was present in the OSA subjects, but it can be normalized by treatment with nasal continuous positive airway pressure, suggesting that increased leptin resistance was not the cause of OSA or its associated vascular risks. (CHEST 2000; 118:580–586)

Key words: leptin; metabolic variates; sleep apnea; vascular risks

Abbreviations: AHI = apnea-hypopnea index; BMI = body mass index; HDL = high-density lipoprotein; LDL = low-density lipoprotein; nCPAP = nasal continuous positive airway pressure; OSA = obstructive sleep apnea; TC = total cholesterol; WHR = waist-to-hip ratio

Obstructive sleep apnea (OSA) has been associated with increased cardiovascular and cerebrovascular morbidity and mortality rates.1–3 However, it remains controversial whether this association reflects comorbidity from confounding factors or from causal factors due to effects of recurrent sleep-disordered breathing. Epidemiologically and clinically, several features well known to predispose individuals to vascular risks4 have also been linked to OSA, including obesity, hypertension, and diabetes mellitus.5–8 Nevertheless, OSA itself, with the apnea/hypopnea-induced hemodynamic and sympathetic activation,9–11 certainly has the potential to interact with these risk factors and to aggravate vascular morbidity and mortality rates.

For editorial comment see page 569

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Based on clinical observation, OSA has been described as an additional vascular risk factor (syndrome Z)\(^{12}\) that interacts with the well-known cluster of central obesity, insulin resistance, dyslipidemia, and hypertension in syndrome X/abdominal obesity syndrome.\(^{13,14}\) It has been reported that men with OSA have excess fat, evidenced by skinfold thickness, in reference to their weight,\(^{7}\) suggesting preferential fat accumulation. The pathogenesis of obesity remains largely unknown, but studies have shown that, in obese humans, there is resistance to leptin, an adipocyte-derived hormone that regulates body weight through the control of appetite and energy expenditure.\(^{15-17}\) There is also increasing evidence that leptin affects several neuroendocrine mechanisms and regulates multiple hypothalamic-pituitary axes.\(^{17}\) Conversely, leptin expression and secretion may be regulated by many hormones, cytokines, and physiologic mechanisms.\(^{17}\) It has been demonstrated in animal studies that leptin could prevent respiratory depression in obesity, suggesting that a deficiency in CNS leptin levels or activity may induce hypoventilation in some obese subjects,\(^{18}\) while work with subjects with COPD has shown that circulating leptin levels correlated with body mass and fat mass, independent of the metabolic variates in a cohort of OSA subjects compared with controls without OSA, matched for sex and body mass index (BMI), to assess whether OSA was associated with an increase in cardiovascular risk and, if so, to determine the role of leptin therein. We also measured leptin levels in OSA subjects before and after treatment with nasal continuous positive airway pressure (nCPAP) to understand the relationship between leptin and sleep apneic activity.

### Materials and Methods

#### Subjects

Subjects were recruited from those who were referred for suspected sleep apnea to the Sleep Clinic, Department of Medicine, The University of Hong Kong, and from those who participated in a territory-wide study on prevalence of sleep apnea. The study was approved by the institutional ethics committee. All subjects underwent an overnight sleep study to document sleep-disordered breathing, and fasting morning venous blood samples were taken with informed consent. Adults with documented OSA, defined by apnea-hypopnea index (AHI) $\geq$ 5 and by symptoms of excessive daytime sleepiness, were compared with asymptomatic subjects with AHI < 5 who were matched for age, gender, and BMI (and menopausal status in females). Only OSA subjects for whom a matched control subject could be identified were included in the analysis.

The OSA subjects were advised on nCPAP therapy according to clinical protocol, and those who agreed to try nCPAP were further recruited for post-nCPAP assessment. To study the effect of overnight application of nCPAP, only those who underwent the 1-night nCPAP treatment within 2 weeks of the baseline study were evaluated, to avoid any change of body weight and sleep apneic activity over time. Those subjects who received regular treatment with nCPAP were recruited to the reassessment study at 6 months, and only those whose body weights were stable (< 5% change from baseline) were included in the evaluation.

#### Sleep Study

All subjects underwent an overnight sleep study, using a computerized polysomnogram system (Alice 3; Healthdyne; Respironics; Pittsburgh, PA) at the Sleep Laboratory, Queen Mary Hospital. Sixteen channels were used to document the following parameters: sleep stages (4-channel EEG, electro-oculogram, chin electromyogram), ECG, airflow at nose and mouth (thermistor), chest and abdominal respiratory movement (respiratory impedance), oxygen saturation (pulse oximetry), snoring (microphone), body position. Recordings were manually scored according to standard criteria.\(^{20}\) Apnea was defined as cessation of airflow for $> 10$ s, and hypopnea was defined as a discernible decrease in airflow from baseline associated with a fall in oxygen

### Table 1—Demographic, Anthropometric, and BP Measurements*

<table>
<thead>
<tr>
<th>Variables</th>
<th>OSA Subjects (n = 30)</th>
<th>Control Subjects (n = 30)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male/female, No.</td>
<td>27/3</td>
<td>27/3</td>
</tr>
<tr>
<td>Age, yr</td>
<td>43.6 ± 10.1</td>
<td>41.9 ± 7.4</td>
</tr>
<tr>
<td>BMI</td>
<td>27.0 ± 2.9</td>
<td>26.5 ± 2.1</td>
</tr>
<tr>
<td>Neck, cm</td>
<td>38.9 ± 7.4</td>
<td>38.5 ± 2.10</td>
</tr>
<tr>
<td>Waist, cm</td>
<td>91.9 ± 7.1</td>
<td>89.9 ± 5.8</td>
</tr>
<tr>
<td>Hip, cm</td>
<td>101.1 ± 6.6</td>
<td>100.3 ± 4.4</td>
</tr>
<tr>
<td>Waist/hip ratio</td>
<td>0.91 ± 0.03</td>
<td>0.90 ± 0.04</td>
</tr>
<tr>
<td>Sum of skinfold thickness cm†</td>
<td>86.5 ± 19.9</td>
<td>71.4 ± 22.6</td>
</tr>
<tr>
<td>BP, mm Hg</td>
<td>Diastolic†, 81 ± 12</td>
<td>73 ± 12</td>
</tr>
<tr>
<td>Systolic</td>
<td>129 ± 15</td>
<td>123 ± 15</td>
</tr>
</tbody>
</table>

*Values are given as mean ± SD.
†p = 0.021.
‡p = 0.036.
saturation of ≥ 4% from baseline. AHI was determined by taking the average of the number of apneic and hypopneic events per sleep hour.

**Anthropometric Measurements**

BMI was calculated from body weight and height measured by a scale (Detecto Scale; Webb City, MO). Neck circumference and waist and hip girth were measured using a tape measure. Neck circumference was taken at the level of the cricothyroid membrane, 21 waist circumference at the level of umbilicus, 22 and hip circumference at the maximal girth level. Caliper readings for skinfold thickness were measured at the biceps, triceps, subscapular sites, and abdominal sites by one technician throughout the study.

**Determination of Circulating Metabolic Parameters**

All subjects had fasting blood samples taken between 8:00 AM and 9:00 AM. Blood samples were immediately sent to the hospital laboratory for estimation of glucose and lipids, while a specimen of clotted blood was centrifuged at 3000g for 10 min for serum, which was stored at −70°C in aliquots until analysis. Serum leptin concentrations were measured in duplicates with a highly sensitive radioimmunoassay (Linco Res; St. Louis, MO). Serum insulin concentrations were measured by immunoassay using commercially available kits (IM System; Abbott Laboratories; Tokyo, Japan), according to the manufacturer’s description.

**Statistical Analysis**

Results were expressed as mean ± SD. Comparison of data between two groups was made by application of Mann-Whitney U test for nonparametric variables. Correlation between serum leptin concentration and other parameters was evaluated with the Spearman rank correlation test. Significance was determined at the 5% level. Multiple regression analysis was applied to define factors that correlated with serum leptin levels. Statistical analysis was performed with the SPSS statistical software package (SPSS Inc; Chicago, IL). 25

**Results**

Thirty subjects with documented OSA syndrome (AHI = 35.7 ± 18) were compared with 30 subjects without OSA (AHI = 1.8 ± 1.9), matched for sex, age, BMI, and in female subjects, for menopausal status. Their demographic and anthropometric characteristics are shown in Table 1. There were no significant differences between the two groups in their body habitus measurements of neck, waist, and hip circumference, but there was significantly more subcutaneous fat in the OSA group, as indicated by skinfold thickness. Diastolic BP was also significantly higher in the OSA group than in the control group, but there were no differences between the two groups in the systolic BP levels (Table 1).

The results for fasting serum leptin, glucose, insulin, and lipids of the two groups are shown in Table 2. Serum leptin levels (Fig 1) were significantly higher in the subjects with OSA (Fig 2, A). Levels of fasting serum insulin, insulin/glucose ratio, and triglyceride were also significantly higher in the OSA group. Levels of fasting glucose and cholesterol showed only a higher trend, but the percentage of OSA subjects with elevated total cholesterol (TC)/high-density lipoprotein (HDL) cholesterol ratio of ≥ 5 was significantly higher than in the control group.

At baseline in the entire study group, there were positive correlations between serum leptin levels and other anthropometric and metabolic parameters as follows: BMI, r = 0.285, p = 0.027; skinfold thickness, r = 0.501, p < 0.001; serum cholesterol, r = 0.347, p = 0.007; serum low-density lipoprotein (LDL) cholesterol, r = 0.317, p = 0.01; serum insulin, r = 0.518, p < 0.001; and insulin/glucose ratio, r = 0.493, p < 0.001. Serum leptin levels also correlated positively with the degree of sleep-disordered breathing as recorded by AHI (r = 0.390, p = 0.002) and sleep time with oxygen desaturation 90% (r = 0.428, p = 0.001). Multiple stepwise regression analysis identified the following factors as significant independent correlates of serum leptin in

Table 2—Metabolic Parameters in the OSA Group and Control Group*

<table>
<thead>
<tr>
<th>Serum Concentrations</th>
<th>OSA Subjects (n = 30)</th>
<th>Control Subjects (n = 30)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Leptin, ng/mL</td>
<td>9.18 ± 4.24</td>
<td>6.54 ± 3.81</td>
<td>0.001</td>
</tr>
<tr>
<td>Glucose, mmol/L</td>
<td>5.7 ± 1.7</td>
<td>5.3 ± 0.7</td>
<td>NS</td>
</tr>
<tr>
<td>Insulin, ng/mL</td>
<td>0.39 ± 0.17</td>
<td>0.26 ± 0.13</td>
<td>0.003</td>
</tr>
<tr>
<td>Insulin/glucose ratio</td>
<td>0.07:0.04</td>
<td>0.05:0.03</td>
<td>0.01</td>
</tr>
<tr>
<td>Cholesterol, mmol/L</td>
<td>5.2 ± 0.9</td>
<td>4.8 ± 1.0</td>
<td>NS</td>
</tr>
<tr>
<td>≥ 5.2 mmol/L</td>
<td>14 (47)</td>
<td>11 (37)</td>
<td>NS</td>
</tr>
<tr>
<td>LDL cholesterol, mmol/L</td>
<td>3.4 ± 0.7</td>
<td>3.1 ± 0.9</td>
<td>NS</td>
</tr>
<tr>
<td>≥ 3.4 mmol/L</td>
<td>14 (47)</td>
<td>10 (33)</td>
<td>NS</td>
</tr>
<tr>
<td>HDL cholesterol, mmol/L</td>
<td>1.10 ± 0.33</td>
<td>1.12 ± 0.29</td>
<td>NS</td>
</tr>
<tr>
<td>≥ 0.9 mmol/L</td>
<td>19 (63)</td>
<td>22 (73)</td>
<td>NS</td>
</tr>
<tr>
<td>TC/HDL cholesterol ratio</td>
<td>5.1:1.6</td>
<td>4.5:1.3</td>
<td>NS</td>
</tr>
<tr>
<td>≥ 5.0</td>
<td>15 (50)</td>
<td>7 (23)</td>
<td>0.032</td>
</tr>
<tr>
<td>Triglyceride, mmol/L</td>
<td>1.6 ± 0.6</td>
<td>1.2 ± 0.6</td>
<td>0.028</td>
</tr>
<tr>
<td>≥ 2 mmol/L</td>
<td>10 (33)</td>
<td>2 (7)</td>
<td>0.01</td>
</tr>
</tbody>
</table>

*Values are given as mean ± SD or No. (%) unless otherwise indicated. NS = not significant.
all subjects: skinfold thickness, waist/hip ratio, serum LDL cholesterol, and diastolic BP. Within the OSA group, the significant independent correlates of serum leptin were serum insulin and diastolic BP.

After one overnight application of nCPAP, there were no significant differences in morning fasting leptin levels (n = 7; level before = 10.32 ± 6.15 ng/mL; level after = 10.5 ± 6.51 ng/mL; p = 0.31). After 6 months of nCPAP treatment, there were significant decreases in the levels of serum leptin despite similar BMIs (n = 9; level before = 10.45 ± 4.55 ng/mL; level after = 6.97 ± 1.82 ng/mL; p = 0.012; BMI before = 27.1 ± 2.43 ng/mL; BMI after = 26.8 ± 2.24 ng/mL) (Fig 2, right, B).

Discussion

OSA is now known to affect at least 2 to 4% of the middle-aged population in white 26-28 as well as some Asian populations. 29 The major concerns about the adverse health effects of OSA, apart from symptoms of excessive daytime sleepiness, are its association with cardiovascular and cerebrovascular morbidity and mortality from coronary heart disease and stroke. 1-3 While controversy and research are ongoing as to whether this association is comorbid or causal, it is well appreciated that independent risk factors may interact and exacerbate any adverse sequelae, either as additional risk factors or possibly through synergistic effects. 4

Obesity is known to be an important pathogenetic factor in OSA, and BMI and other indexes of obesity have been predictive factors for OSA in several population studies. 26-29 It is believed that increased deposition of fat or soft tissue in the neck and upper-airway region predisposes the subject to upper airway collapse and apnea during sleep; a larger neck circumference, probably reflecting greater fat or soft tissue deposition, is more significantly associated with sleep apnea. 21 Greater adiposity in the region near the upper airway has also been shown with MRI in relatively nonobese subjects with OSA compared with their BMI-matched counterparts without OSA. 30 Furthermore, other studies have reported that central obesity, reflected by waist-to-hip ratio (WHR), was a similar or better predictor of OSA than was BMI, 5,26 and the correlation between waist circumference and neck circumference was very strong, 5 supporting the idea that OSA is closely linked to central obesity. Central obesity is associated with diabetes mellitus, 13 as well as being an indicator of coronary heart disease. 14 The presence of central obesity in OSA may confer pathogenetic risks as a result of the mechanical effect on the upper airway, in addition to the vascular implications of central obesity. Since obesity may be an overwhelming confounding factor in the vascular risk profile, the control group was matched to the OSA group for BMI, the most widely applied surrogate marker of obesity. The subjects in this study, similar to most subjects with OSA encountered in clinical practice in our community, 31 had BMIs in the overweight range for the reference population values in the local Chinese community, 32 with a mean BMI significantly above that of the average age- and sex-matched population (men, mean BMI 24.4 for age 40 to 44 years; women, mean BMI 25.1 for age 50 to 54 years). 32 These study subjects also had features of central obesity with mean WHR of 0.91 (men, 0.92; women, 0.86) and 0.89 (men, 0.90; women, 0.86) in OSA and control groups, respectively, when the local population mean WHR was 0.88 for men and 0.81 for women. 32 Interestingly, despite similar BMIs in OSA and control groups, the OSA subjects had comparatively greater subcutaneous adiposity, reflected by the higher skinfold thickness indexes,

![Figure 1. Serum leptin levels in 30 OSA subjects compared with 30 matched control subjects. ■ = women; ◆ = men.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=data/journals/chest/21952/)

![Figure 2. Serum leptin levels in OSA: (A) comparison with matched controls; (B) effect of nCPAP treatment for 6 months.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=data/journals/chest/21952/)
consistent with previous hypotheses that OSA subjects may have preferential fat accumulation.5-7

Insulin resistance or clinically apparent diabetes mellitus has been reported to be common in OSA subjects. Some studies have found that the relationship between insulin resistance and sleep-disordered breathing could be completely explained by the differences in body mass after adjusting for confounding variables.5-6 However, studies on the effect of nCPAP treatment have yielded conflicting results, with some, but not all, investigators finding an improved insulin sensitivity without a concomitant change in body weight, which suggested that OSA has an independent effect on insulin resistance.8,34

In this study, having controlled for body mass, the OSA group still had significantly higher fasting insulin levels with a trend toward higher fasting glucose, suggesting the presence of insulin resistance. This could be explained by a subtle confounding effect of greater adiposity in the OSA subjects, or by an independent effect due to OSA. In our study, the latter has not been confirmed as there was no significant decrease in serum insulin after nasal CPAP treatment.

Hyperlipidemia is an established independent risk factor for coronary heart disease and cerebrovascular disease. Lipid profiles in OSA subjects have not been widely studied, although clinical observation suggests that dyslipidemia is common in subjects with OSA. In a previous case series of 45 OSA subjects, the prevalence of hypercholesterolemia and elevated LDL cholesterol was not increased.6 In the present study, the OSA group had significantly more subjects who had dyslipidemia with elevated triglyceride and TC/HDL cholesterol ratio ≥ 5, an indicator of increased cardiovascular risk.

It is now believed that leptin is essential to the normal regulation of body weight and energy expenditure in human subjects. Obesity and body fat are the major factors regulating circulating leptin,15-17 which is also influenced by gender and age.35-36 Levels of circulating leptin correlate with indexes of adiposity, including BMI, body fat mass, and percentage body fat.15-17,35,36 Consistent with being overweight, the serum leptin levels of both OSA and control subjects were higher than those of lean healthy subjects in our population; men, n = 7, BMI = 23, leptin = 4.1 ± 0.81 ng/mL; premenopausal women, n = 6, BMI = 21.5, leptin = 7.5 ± 5.7 ng/mL; postmenopausal women, n = 3, BMI = 20.3, leptin = 5.5 ng/mL (M. Ip, MD; unpublished data; October 1999). However, despite being matched for the established determinants of serum leptin levels including BMI, sex, age, and menopausal status, compared with subjects who had no evidence of sleep apnea, increased degrees of hyperleptinemia were present in OSA subjects. Since we demonstrated greater skinfold thickness in the OSA subjects, which also correlated strongly with serum leptin levels, our finding suggests that at least part of the elevation of leptin was related to increased fat mass in these OSA subjects. The higher leptin levels may reflect either an increased leptin resistance, contributing to preferential fat deposition and thus predisposing the subjects to development of OSA, or the existence of increased fat stores in subjects who also have a tendency to develop OSA. However, the increased hyperleptinemia could be abrogated after treatment with nCPAP with no significant change in the anthropometric parameters measured, suggesting that other mechanisms apart from fat mass could have contributed to the increased leptin levels in OSA subjects.

Apart from its role in regulation of fat metabolism, leptin may also regulate insulin release as part of an adipoinsular feedback.37 Conversely, insulin resistance has been shown to lead to increased secretion of leptin independent of body mass.38,39 Obesity with intra-abdominal fat accumulation is particularly associated with insulin resistance and with noninsulin-dependent diabetes mellitus,13,14 but a previous study has demonstrated that serum leptin levels are not associated with abdominal obesity or elevated serum insulin and serum lipoproteins independently, after adjusting for percentage body fat, gender, and age.36 In our study, serum insulin was a significant independent correlate of serum leptin in the OSA group. However, the decrease in serum leptin after nCPAP treatment was not accompanied by a similar fall in serum insulin. This finding was consistent with a recent report in which serum leptin, but not serum insulin, decreased after nCPAP treatment.34

The baseline levels of leptin correlated with two parameters of sleep-disordered breathing, the AHI and sleep time with oxygen desaturation, and there was a significant decrease of leptin levels in subjects who received nCPAP for about 6 months despite similar BMIs, suggesting either that sleep-disordered breathing contributed to elevated leptin levels or that nCPAP itself could modify leptin expression. The finding of decreases in circulating leptin levels in OSA patients after treatment is consistent with a recently published study in which circulating leptin levels were demonstrated to decrease significantly after 3 or 4 days and after several months of nCPAP treatment in OSA subjects whose body weight was maintained.44 One interpretation of the elevated leptin levels in OSA patients would be the mediation of sleep-disordered breathing by leptin via the common factor of obesity. This is unlikely to be the entire explanation, since the leptin levels decreased after treatment of OSA with no change in BMI. However, nCPAP treatment decreases visceral fat without
changing BMI, and this possibility has not been firmly excluded in our subjects, although we have seen no change in the anthropometric index of WHR. Alternatively, the AHI and oxygen desaturation may be considered indicators of the degree of physiologic stress due to sleep-disordered breathing. Stress may lead to increased leptin levels either through an increase in leptin secretion or indirectly. It has been reported that increased muscle sympathetic nerve activity was associated with increased plasma leptin levels. In OSA patients, there is evidence of cerebral arousal as well as increased sympathetic nerve discharge induced by nocturnal events of asphyxiation, and abolition of the neuroexcitation with nCPAP treatment may result in decrease in leptin expression. An increase in HDL cholesterol has previously been found in OSA after 6 months of nCPAP treatment, suggesting that treatment of OSA may result in an improvement in dyslipidemia. In agreement with this observation, a reduction in serum triglyceride was observed in our OSA subjects treated with nCPAP for 6 months. It is possible, therefore, that sleep-disordered breathing and its heightened neuroexcitation and sympathetic activity may lead to alteration in leptin expression as well as to adverse cardiovascular risk profiles. Furthermore, recent work with animal (mouse) models of obesity demonstrated that leptin could prevent respiratory depression. Hence, the elevated serum leptin levels in OSA subjects could represent a homeostatic response to the hypoventilation induced by OSA, which decreased with the control of OSA by nCPAP therapy.

In summary, our findings confirm definitively a distinct profile of excess adiposity not adequately reflected by BMI, dyslipidemia, insulin resistance, and elevated BP in subjects with OSA, representing a clustering of multiple cardiovascular risk factors in these subjects. Hyperleptinemia is present in subjects with OSA and cannot be explained entirely by increased adiposity. The reversibility of hyperleptinemia with nCPAP treatment in our patients agrees with the observation in a recent report, and suggests that the increased hyperleptinemia is a marker of OSA. The exact mechanisms by which nCPAP treatment for OSA decreased leptin levels require further delineation.

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REFERENCES
1 Partinen M, Guilleminault C. Daytime sleepiness and vascular morbidity at seven-year follow-up in obstructive sleep apnea patients. Chest 1999; 97:27–32
26 Ma Z, Gingerich RL, Santiago JV, et al. Radioimmunoassay of
32 Janus ED. Hong Kong cardiovascular risk factor prevalence study 1995–1996. Hong Kong: Queen Mary Hospital, 1997

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