A
diponectin is a collagen-like protein secreted predomi-
nantly by adipose tissue. In humans, plasma adiponectin
concentrations are inversely related to measures
of insulin resistance, with reduced adiponectin concentra-
tions reported in obesity and patients with essential
hypertension, dyslipidaemia, diabetes, and coronary artery
disease (CAD).

Low serum adiponectin concentrations appear to be associated with an increased risk of myocardial
infarction, but it is not clear if this increased risk is related to
increased coronary disease burden.

People of South Asian descent have increased susceptibility
to glucose intolerance, dyslipidaemia, and CAD, the cause(s)
of which are complex and multiple. One small study
suggested lower adiponectin concentrations in South Asians
compared to body mass index (BMI) matched white people,
but ethnic differences in plasma adiponectin among patients
with CAD have not been reported. We hypothesised that
serum adiponectin is lower in CAD patients of South Asian
descent compared to white counterparts, and secondly, that
there was a relation between serum adiponectin and coronary
disease severity by angiography. To test these hypotheses, we
performed a cross sectional study of consecutive white and
South Asian patients undergoing cardiac catheterisation.

METHODS
Consecutive patients attending outpatient diagnostic cardiac
catheterisation for the investigation of CAD were recruited.
The South Asian patients attending our unit are almost
exclusively of Punjabi origin, and their ethnic group was
confirmed by direct enquiry. Patients with angiographically
normal coronary arteries, concurrent inflammatory or neo-
plastic disease, haemodynamically significant valvar heart
disease, prior revascularisation, or recent (≤3 months)
admission for coronary ischaemia were excluded. Data from
these patients were compared with healthy controls.

All patients underwent conventional coronary angiogra-
phy, with each angiogram reviewed independently by two
two experienced observers blinded to the clinical details for the patient; the inter- and intra-observer coefficient of variation was <5%. The coronary atheroma score (CAS) and coronary
stenosis score (CSS) were calculated for each patient, as
previously established. Left ventricular systolic function was
qualitatively assessed as normal or abnormal by visual
inspection and quantitative analysis of the ventriculogram
(defined as a calculated ejection fraction <40%).

Venous blood was centrifuged at 1000 g and 4°C for 20
minutes. Serum was aliquoted and stored at −70°C for batch
analysis. Serum adiponectin was measured by ELISA using
commercial kits and reagents (R&D Systems, Abingdon, UK).
Intra-assay coefficients of variation were <5% and inter-
assay variance <10%.

Analyses and power calculations were performed using
Minitab 13 (Minitab Inc, State College, Pennsylvania, USA).
A stepwise multiple regression analysis was performed to
determine independent predictors of serum adiponectin.

Variables that were significantly different between patient
groups or related on univariate analysis were included in the
analysis.

RESULTS
We recruited 139 patients (90 white, 49 South Asians) with
angiographically confirmed CAD and 31 comparable normal
healthy white controls (table 1). Systolic and diastolic blood
pressures and total cholesterol were highest in the white
controls, reflecting the use of lipid lowering therapy (statins)
in the patient groups. There were no significant differences in
CAS or CSS between patient groups.

Serum adiponectin was highest in the control group,
intermediate in the white patients, and lowest among South
Asian patients (Kruskal Wallis test, p < 0.001; Tukey’s post
hoc test for inter-group differences after log transformation, p < 0.05).

There was no difference in CAS (0.888 (0.483–1.650) vs
0.915 (0.270–1.900), p = 0.616) or CSS (0.867 (0.384–1.498)
vs 0.688 (0.158–1.415), p = 0.515) between the lowest and
the highest tertile of serum adiponectin. Similarly, there was
no difference in serum adiponectin concentrations between the
highest tertile and lowest tertiles of CAS (3.0 (2.0–4.4) vs
2.7 (2.1–3.5) μg/ml, p = 0.570) or CSS (3.0 (1.9–4.1) vs 2.5
(2.0–3.4) μg/ml, p = 0.391).

There were no significant differences in serum adiponectin
between males and females (p = 0.243), and patients with
normal and abnormal left ventricular ejection fraction
(LVEF) (p = 0.360) and with or without statin treatment
(p = 0.380) (full data not shown).

On univariate analysis, serum adiponectin correlated
positively with age in both patient groups (Spearman
r = 0.225, p = 0.033, and r = 0.525, p < 0.001 in white
and South Asian respectively), but correlated positively with
high density lipoprotein (HDL) cholesterol (r = 0.379,
p < 0.001) and inversely with serum triglycerides
(r = −0.353, p = 0.011) in the white patients. Serum
adiponectin did not correlate with CAS or CSS in either
patient groups or the whole patient cohort (CAS:
 r = −0.054, p = 0.541; CSS: r = 0.018, p = 0.837). For
a multivariate analysis, we included variables that were
significantly different between white and South Asians and
variables which correlated with serum adiponectin on
univariate analysis (that is, age, systolic and diastolic blood
pressures, ethnicity, diabetes, statin use, serum triglycerides,
and HDL cholesterol). Ethnic group (p = 0.044) and HDL
cholesterol (p = 0.015) were independent predictors of
serum adiponectin in patients with CAD.
DISCUSSION

South Asians living in Britain suffer from an increased risk of coronary events and mortality compared to age matched white patients. Insulin resistance and associated metabolic abnormalities may be contributory factors. Serum adiponectin concentrations correlate well with measures of insulin resistance and accumulating data suggest that adiponectin may have anti-atherogenic properties. Hence, low adiponectin concentrations may be a link between increased prevalence of insulin resistance and coronary events among South Asians. One small study suggested lower serum valence of insulin resistance and coronary events among South Asians living in Britain suffer from an increased risk of insulin resistance and accumulating data suggest that adiponectin abnormalities may be contributory factors. Serum adiponectin and angiographic CAS or CSS, 5 in either ethnic group or the whole patient group. This is unlikely to be because of small sample size as our patient numbers provided us with the power to detect even a modest relation between serum adiponectin and CAS. Hence, our data suggest that the link between adiponectin and coronary events may not be mediated by angiographically quantified coronary disease severity. It is recognised that the majority of culprit lesions arise from haemodynamically insignificant coronary stenoses. Hence, the relation between adiponectin and perhaps the more qualitative changes of the atherosclerotic plaques (that is, the presence of CAD, and myocardial infarction) and the association with pathophysiological processes such as thrombogenesis and endothelial dysfunction should be studied.

In conclusion, serum adiponectin is lower in South Asian patients with CAD compared to their white counterparts, but was not significantly related to angiographic coronary disease. The lower adiponectin concentrations may contribute to the development of CAD, especially in the South Asian population.

Authors’ affiliations

H S Lim, M H Tayebjee, K T Tan, J V Patel, R J Macfadyen, G Y H Lip, Haemostasis Thrombosis and Vascular Biology Unit, University Department of Medicine, City Hospital, Birmingham, UK

Correspondence to: Professor Gregory Y H Lip, Haemostasis Thrombosis and Vascular Biology Unit, University Department of Medicine, City Hospital, Birmingham, B18 7QH, UK; g.y.h.lip@bham.ac.uk

Accepted 24 February 2005

REFERENCES


Table 1 Characteristics of study participants

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Controls (n = 31)</th>
<th>White CAD (n = 90)</th>
<th>South Asian CAD (n = 49)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>65 (10)</td>
<td>62 (6)</td>
<td>61 (9)</td>
<td>0.068</td>
</tr>
<tr>
<td>Males (%)</td>
<td>15</td>
<td>61</td>
<td>34</td>
<td>0.108</td>
</tr>
<tr>
<td>SBP (mm Hg)</td>
<td>131 (12)</td>
<td>142 (22)</td>
<td>130 (17)</td>
<td>0.001*</td>
</tr>
<tr>
<td>DBP (mm Hg)</td>
<td>76 (10)</td>
<td>79 (10)</td>
<td>74 (10)</td>
<td>0.022†</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>29 (28–31)</td>
<td>29 (26–34)</td>
<td>28 (25–30)</td>
<td>0.444</td>
</tr>
<tr>
<td>Cholesterol (mmol/l)</td>
<td>5.5 (1.1)</td>
<td>4.5 (1.1)</td>
<td>4.5 (1.0)</td>
<td>0.001†</td>
</tr>
<tr>
<td>Triglycerides (mmol/l)</td>
<td>1.3 (0.8–2.0)</td>
<td>1.7 (1.2–2.6)</td>
<td>1.5 (1.1–2.4)</td>
<td>0.128</td>
</tr>
<tr>
<td>HDL (mmol/l)</td>
<td>1.55 (0.35)</td>
<td>1.28 (0.36)</td>
<td>1.25 (0.25)</td>
<td>0.001†</td>
</tr>
<tr>
<td>Smokers (%)</td>
<td>13</td>
<td>3</td>
<td>3</td>
<td>0.142</td>
</tr>
<tr>
<td>Previous MI (%)</td>
<td>36</td>
<td>22</td>
<td>22</td>
<td>0.576</td>
</tr>
<tr>
<td>LV dysfunction (%)</td>
<td>25</td>
<td>13</td>
<td>13</td>
<td>0.582</td>
</tr>
<tr>
<td>Patients with diabetes (%)</td>
<td>23</td>
<td>22</td>
<td>22</td>
<td>0.020</td>
</tr>
<tr>
<td>Statin treatment (%)</td>
<td>78</td>
<td>33</td>
<td>33</td>
<td>0.007†</td>
</tr>
<tr>
<td>ACE inhibitor (%)</td>
<td>35</td>
<td>21</td>
<td>21</td>
<td>0.649</td>
</tr>
<tr>
<td>CAS</td>
<td>1.04 (0.50–1.80)</td>
<td>0.82 (0.43–1.62)</td>
<td>0.481†</td>
<td></td>
</tr>
<tr>
<td>CSS</td>
<td>0.90 (0.38–1.61)</td>
<td>0.77 (0.20–1.32)</td>
<td>0.396†</td>
<td></td>
</tr>
<tr>
<td>Adiponectin (µg/ml)</td>
<td>5.2 (3.7–6.2)</td>
<td>3.0 (2.0–4.2)</td>
<td>2.3 (1.8–3.0)</td>
<td>&lt;0.001†</td>
</tr>
</tbody>
</table>

*White CAD v South Asians CAD and controls; †White CAD v South Asian CAD; ‡Controls v CAD patients; §Comparisons between patient groups only; ¶Controls v White CAD v South Asian CAD.

ACE, angiotensin converting enzyme; BMI, body mass index; CAD, coronary artery disease; CAS, coronary atheroma score; CSS, coronary stenosis score; DBP, diastolic blood pressure; HDL, high density lipoprotein; LV, left ventricular; MI, myocardial infarction; SBP, systolic blood pressure.
Serum adiponectin in coronary heart disease: ethnic differences and relation to coronary artery disease severity
H S Lim, M H Tayebjee, K T Tan, J V Patel, R J Macfadyen and G Y H Lip

Heart 2005 91: 1605-1606
doi: 10.1136/hrt.2004.047803

Updated information and services can be found at:
http://heart.bmj.com/content/91/12/1605

These include:

References
This article cites 5 articles, 3 of which you can access for free at:
http://heart.bmj.com/content/91/12/1605#BIBL

Email alerting service
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Topic Collections
Articles on similar topics can be found in the following collections
- Drugs: cardiovascular system (8842)
- Hypertension (3006)
- Clinical diagnostic tests (4779)
- Acute coronary syndromes (2742)
- Diabetes (842)
- Epidemiology (3752)
- Metabolic disorders (1030)
- Venous thromboembolism (495)

Notes

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/