

# PostScript

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### Paradoxical severe decrease in serum HDL-cholesterol after treatment with a fibrate: reply by the authors

We read the letter of Olukoga<sup>1</sup> (in response to our earlier paper<sup>2</sup>) with interest and feel that it covers the area of fibrate induced hypoalphalipoproteinaemia excellently. Unfortunately, we are not aware of a clear and unifying explanation as to why some patients exhibit a paradoxical decrease in serum high density lipoprotein (HDL)-cholesterol concentrations upon initiation of a fibrate drug. However, we accept that further studies are important to unravel this apparent paradoxical situation, particularly because a decline in HDL-cholesterol concentration would be expected to put the patient at increased cardiovascular risk. It would of course be useful to be able to predict those who might be susceptible to this phenomenon because they may benefit from alternative lipid modifying treatment, but as yet the extent and the cause of the problem are not known. This is particularly relevant because there are controversies

in the literature regarding the benefit of fibrates in reducing cardiovascular disease—for example, the VAHIT and BIP studies.<sup>3,4</sup>

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#### References

- 1 **Olukoga AO**. Paradoxical severe decrease in serum HDL-cholesterol after treatment with a fibrate [letter]. *J Clin Pathol* 2002;55:718.
- 2 **M Crook, J Lynas, R Wray**. A paradoxical severe decrease in serum HDL-cholesterol after treatment with a fibrate. *J Clin Pathol* 2000;53:796-7.
- 3 **Rubins HB, Robins SJ, Collins D, et al**. VAHIT. Gemfibrozil for the secondary prevention of coronary heart disease in men with low HDL-cholesterol. *N Engl J Med* 1999;341:410-18.
- 4 **BIP Study Group**. Secondary prevention by raising HDL-cholesterol and reducing triglycerides in patients with coronary heart disease. *Circulation* 2000;102:21-7.

### Artefactual hypoglycaemia: and audit

Hypoglycaemia is not an uncommon clinical presentation. However, it is important to distinguish spurious from genuine hypoglycaemia to prevent inappropriate further investigations and management. Many laboratories including ours frequently measure glucose in non-anticoagulated tubes. Delay in separation of serum from cells could result in artefactual hypoglycaemia from anaerobic glycolysis. This audit was undertaken to ensure our practice of measuring glucose in clotted samples does not result in artefactual hypoglycaemia.

We studied all inpatients with serum glucose of  $\leq 2.5$  mmol/litre over a two month period. Patients were categorised into the following:

(1) Genuine hypoglycaemia: when serum glucose, as determined in the laboratory, was  $\leq 2.5$  mmol/litre and there had been either a previous low ward blood glucose, as measured by glucose meters, or a repeat of laboratory glucose confirmed the presence of hypoglycaemia.

(2) "Unknown": if either there was no documentation of a previous low ward blood glucose or if repeat laboratory glucose was not performed.

(3) Artefactual hypoglycaemia: when repeat laboratory blood glucose was normal and there was no evidence that patients were treated for hypoglycaemia before this time.

Data were available on 48 patients with serum glucose  $\leq 2.5$  mmol/litre. The median age was 66 years (range, 1 day to 92 years) with a male : female ratio of 23 : 25.

Thirty two patients had genuine hypoglycaemia, four had artefactual hypoglycaemia, and 12 were classified as "unknown".

Our study provides evidence that artefactual hypoglycaemia could occur in hospital patients whose blood samples for glucose measurement are collected into non-anticoagulated tubes. In most patients, the time of specimen collection was not indicated on the request form but the results of other analytes, such as phosphate and potassium, suggest that there was some delay in sending these specimens and therefore in separating serum from cells. It is possible that some patients with hyperglycaemia were determined to have artefactual normoglycaemia, thereby missing a diagnosis of diabetes mellitus. Our study is consistent with that of Chan *et al*,<sup>1</sup> who showed a significant fall in glucose concentrations with time in samples collected in heparinised tubes compared with those treated with fluoride oxalate.

We suggest that other laboratories measuring glucose in serum samples and also in heparinised tubes should critically examine their methods to ensure that their glucose results are reliable.

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#### Reference

- 1 **Chan AY, Swaminathan R, Cockram CS**. Effectiveness of sodium fluoride as a preservative of glucose in blood. *Clin Chem* 1989;35:315-17.