prevalence and clinical features of atrial fibrillation in the community.

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Concentrations of von Willebrand factor in diabetes

Did authors measure serum or plasma concentrations?

EDITOR,—J-W Chen and colleagues' short report on concentrations of von Willebrand factor in diabetic patients contains a glaring error that could invalidate all their findings: it states that the authors measured serum, not plasma, concentrations of von Willebrand factor.¹ "Serum concentrations of von Willebrand factor" are mentioned seven times. The authors use this term even when describing the assay of von Willebrand factor, which was used by its originator to measure plasma concentrations.² The authors also cite an important paper that related plasma concentrations of von Willebrand factor to the risk of further myocardial events in people with angina pectoris;³ again they state that serum von Willebrand factor was measured.

As a haematologist I am often surprised at how many non-haematologists seem to have forgotten the difference between plasma and serum. The terms are not interchangeable. Plasma contains the anticoagulated residue of blood after the cellular components have been removed, and thus preserves concentrations of haemostatic factors. Serum is what remains after blood forms a clot. As von Willebrand factor is used in forming a clot, serum concentrations of von Willebrand factor have little meaning and so have not been studied academically.

Did the authors actually measure plasma concentrations? Did the referees realise the error? Or did the technical editor not understand and change all the comment on "plasma" to "serum." Perhaps the article was designed to give haematologists sleepless nights worrying about standards.

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Author's reply

EDITOR,—Beverley J Hunt states, "As von Willebrand factor is used in forming a clot, serum concentrations of von Willebrand factor have little meaning and so have not been studied

academically." It is true that nearly all previous studies have measured plasma concentrations of the factor. However, during our research into von Willebrand factor over the past eight years my colleagues and I have measured serum and plasma concentrations simultaneously in samples obtained from the same insulin dependent diabetic patients (n=24).¹ Our published study showed a mean (SE) difference between serum and plasma concentrations of von Willebrand factor of 0.04 (0.02) U/ml and a correlation coefficient of $r=0.97$ ($P<0.00005$).

The serum concentrations reported by us agree well with previous findings regarding plasma von Willebrand factor in patients with non-insulin dependent diabetes with and without albuminuria.² Von Willebrand factor antigen was determined by a microenzyme linked immunoabsorbent assay that used microtitre plates coated with a rabbit von Willebrand factor antibody and with a von Willebrand factor antibody-horseradish peroxidase conjugate as second antibody (Dako, Copenhagen, Denmark).³ A von Willebrand factor calibrator was obtained from the World Health Organisation's International Laboratory for Biological Standards in London. This calibrator gives results in U/ml. The coefficient of variation was below 10%. The comparison mentioned above clearly showed that no measurable consumption of von Willebrand factor occurs during in vitro clot formation (serum) and consequently that measurements of serum concentrations of von Willebrand factor are as valid as measurements of plasma concentrations.

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Don't rely on drinking wine to prevent traveller's diarrhoea

EDITOR,—Martin E Weisse and colleagues report that, in vitro, wine has greater antimicrobial activity against salmonella, shigella, and *Escherichia coli* than bismuth salicylate.¹ To determine whether there is epidemiological support for these findings we reviewed reports of investigations of outbreaks conducted by the Centers for Disease Control and Prevention (CDC) from January 1983 to the end of December 1995. We also conducted a Medline search of literature published since 1966.

We identified five CDC reports of outbreaks that listed shigella, salmonella, or *E coli* as the causative agent and that included an assessment of the effect

of consumption of alcoholic beverages (table 1). Drinking wine was found not to be protective in two (unpublished) reports of outbreaks of infection with *Shigella flexneri* and enterotoxigenic *E coli*. Three investigations did not ask specifically about wine but asked about "alcoholic beverages." One of these reports stated that no association between severity of illness and drinking alcoholic beverages was observed.² In the two remaining reports univariate analysis suggested that drinking alcoholic beverages at the time of exposure reduced the risk of becoming ill with *Sh sonnei* infection (unpublished report) or of becoming "severely ill" with *Salmonella heidelberg* infection,³ but in both instances the protective effect of alcoholic beverages was not significant after differences in exposure to the contaminated food item were controlled for. Our literature review yielded no epidemiological studies in which wine was protective against infection with salmonella, shigella, or *E coli*.

Extrapolations of in vitro data are always problematic ("there's many a slip 'twixt the cup and the lip"). While the findings of Weisse and colleagues suggest that wine is preferable to bismuth salicylate as a marinade for contaminated foods, it would be foolish to suggest that travellers to developing countries should drink wine as a means of preventing traveller's diarrhoea. The antibacterial activity of wine in vivo decreases rapidly with dilution,⁴ which suggests that a traveller would need to ingest wine with each meal, snack, or beverage. Any potential benefits would be outweighed by the ill effects that this level of consumption has for pregnant women, people with liver disease, and anyone operating a device more complicated than a deckchair. Furthermore, travellers who overestimate wine's protective effect might disregard the main recommendation to prevent traveller's diarrhoea: "boil it, cook it, peel it, or forget it."⁴ Although people find many reasons for drinking wine, the prevention of diarrhoeal disease should not be one of them.

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Table 1—Summary of CDC's reports of outbreaks of infection with salmonella, shigella, and *E coli* that assessed effect of consumption of alcoholic beverages on risk or severity of illness, 1983-95 inclusive

Alcohol type	Year	Pathogen	Vehicle	No exposed to vehicle	Relative risk associated with alcohol (95% confidence interval)	CDC Epi-Aid No
Wine	1995	Enterotoxigenic <i>E coli</i>	Courgettes	111	1.1 (0.8 to 1.5)	95-28
Wine	1994	<i>Sh flexneri</i>	Multiple items from buffet	380	1.3 (1.0 to 1.7)	94-89
Alcoholic beverages	1989	<i>Sh flexneri</i>	Potato salad	38	"No association"	90-08*
Alcoholic beverages	1988	<i>S heidelberg</i>	Roast beef	25	0.41* (0.28 to 0.81)	8*
Alcoholic beverages	1987	<i>Sh sonnei</i>	Chicken salad	38	0.5 (0.3 to 1.0)	88-09

*Association between consumption of alcoholic beverages and "severe" disease (≥ 8 stools/24 h and illness for ≥ 5 days).