WHIDMT: Rossouw and Howard blatantly miss the point

To the Editor: Rossouw and Howard’s response to my article confirms that it is they and not I who miss the point.

My key focus was not whether the Women’s Health Initiative Dietary Modification Trial (WHIDMT) supports the use of carbohydrate-restricted diets. Nor did my key points focus on subgroup findings rather than the robust overall findings of the study. By introducing these arguments, Rossouw and Howard neatly sidestep the single most important question I raised.

I wished to understand why these authors have yet to communicate the sole significant finding of the WHIDMT, which is that women with established heart disease at the start of the trial fared worse if they changed to the low-fat ‘prudent’ diet than did those equally ill women who continued to eat a supposedly unhealthy diet. I also showed that the key finding in their Fig. 3 is unintelligible because an essential line of text is missing, and furthermore that no reference is made to Fig. 3 in their response.

Instead they dismiss the only significant finding in their study as ‘likely to be a chance finding’ because ‘there is no biologic basis for expecting a different outcome in this [ill] subgroup, as shown in cholesterol-lowering trials of women with prior disease’. An inconvenient outcome is therefore ignored because of their certainty that this adverse result has no (currently known) biological basis.
There are a number of reasons why this explanation is scientifically unacceptable.

First, the meta-analysis of the ‘cholesterol-lowering trials’ Rossouw and Howard cite was published in 2012, 6 years after their paper was published in 2006.

Second, ‘cholesterol-lowering trials’ use medications, not diet, to lower blood cholesterol concentrations and hence provide an invalid comparison. Their inability to find a single study showing that dietary-induced cholesterol lowering improves long-term outcomes is the conclusive admission by these experts that no such evidence exists.

Third, the meta-analysis that Rossouw and Howard cite has been subject to independent re-analysis. The new conclusion is that drug-induced cholesterol lowering improves long-term outcomes is the single study showing that dietary-induced cholesterol lowering improves long-term outcomes is the conclusion by these experts that no such evidence exists.

It is important to note that the WHIDMT prudent diet reduced the mean blood low-density lipoprotein cholesterol concentration by just 0.18 mmol/l at 3 years, proving that the most expensive low-fat dietary intervention yet undertaken was essentially ineffective in reversing hypercholesterolaemia.

Fourth, they make no reference to the Estrogenic Replacement and Atherosclerosis (ERA) Trial, which found that coronary atherosclerosis progressed significantly more rapidly over a 3-year period in postmenopausal women eating the equivalent of the WHIDMT low-fat prudent diet than in those eating a diet high in saturated fats and low in carbohydrates and polyunsaturated fats.

Rossouw and Howard concede that the WHIDMT was not designed as a trial of the diet-heart hypothesis. This is obvious from the experimental design in which the intervention group also received an ‘intensive behavioural modification program’ comprising 18 group sessions in the first year followed by quarterly maintenance sessions for the next 7 years. The control group received only a copy of Dietary Guidelines for Americans. This renders mute any conclusions that any positive outcomes can be ascribed purely to dietary change. Yet they are not discouraged from continuing to conclude that ‘the lower fat diet ... led to less weight gain, improved insulin resistance (at 1 year), and no increased risk of diabetes risk compared with the control diet’.

But once more Rossouw and Howard are economical with the truth, because at the finish of the 8-year trial, there were no biologically important differences between groups in body weight (~500 g) (Fig. 2[7]), in blood glucose or insulin concentrations, or in other measures of insulin resistance including homeostatic model assessment of insulin resistance (HOMA-IR) and the quantitative insulin sensitivity check index (QUICKI) (Table 2[8]). In fact, so disappointing were these findings that the authors were forced to conclude not that the dietary intervention produced positive outcomes, but that there were no significant adverse effects (my italics) on insulin sensitivity, a quite different conclusion from that which they aim to project in their letter.

Similarly, the only ‘robust’ conclusion of the total study was that a ‘low fat dietary pattern ... showed no evidence of reducing diabetes risk after 8.1 years’. In fact, as early as within the first year of the trial, glucose control worsened significantly in those postmenopausal women with type 2 diabetes mellitus who reduced their fat intake on the high-carbohydrate prudent diet (Table 4[9]).

Now is perhaps the time for its two senior authors finally to concede that the WHIDMT proved that a low-fat diet with or without an ‘intensive behavioural modification program’ is likely to be detrimental to the health of postmenopausal women with established heart disease or type 2 diabetes, and that mechanisms well described in the literature can readily explain these adverse outcomes.

These findings have important implications for dietary advice to women with either established coronary artery disease or type 2 diabetes mellitus, and could have wider connotations.

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