Levels of 'good' cholesterol less relevant to cardiovascular risk once 'bad' cholesterol has been reduced

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In the general population, the more 'good' cholesterol that a person has, the less likely they are to suffer a cardiovascular event. But new research shows that if a person has their levels of 'bad' cholesterol substantially lowered with high-dose statin treatment, then levels of 'good' cholesterol in that person may no longer bear any relation to their remaining cardiovascular risk. The findings, based on the JUPITER study, are reported in an Article Online First and in an upcoming *Lancet*. The Article is by Professor Paul Ridker, Center for Cardiovascular Disease Prevention, Brigham and Women's Hospital, Boston, MA, USA, and colleagues.

In the JUPITER trial, patients with average to low levels of bad cholesterol received the potent statin 'Rosuvastatin' at a dose of 20 mg per day, which lowered their concentrations of bad cholesterol, in many cases substantially, to those typically seen in Aboriginal populations but rarely seen in Western patients. After a median follow-up of 1•9 years in JUPITER (maximum 5 years), treatment with rosuvastatin was associated with a 54% reduction in myocardial infarction, a 48% reduction in stroke, a 46% reduction in revascularisation, a 43% reduction in venous thromboembolism, and a 20% reduction in total mortality. In this new work, the authors assessed whether residual risk after initiation of high-dose statin treatment was related to baseline or on-treatment concentrations of good cholesterol.

For the patients in the JUPITER trial who were given placebo, concentrations of good cholesterol remained predictive of cardiovascular risk, with the patients in the highest 25% of levels of good cholesterol at around half the risk of suffering a cardiovascular event as those in the lowest 25%. By contrast, among the JUPITER patients given rosuvastatin, no significant relationships were noted between concentrations of good cholesterol and residual cardiovascular risk.

The authors conclude: "Although measurement of HDL-cholesterol concentration is useful as part of initial cardiovascular risk assessment, HDL-cholesterol concentrations are not predictive of residual vascular risk among patients treated with potent statin therapy who attain very low concentrations of LDL cholesterol."

However, the authors add that finding out whether or not raising HDL cholesterol improves cardiac outcomes after taking statin therapy remains a very important issue that can only be addressed by randomised trials of potent, efficacious cholesterol raising agents. Such studies would assess whether substantially raising HDL cholesterol levels would provide additional cardiovascular benefit beyond statin therapy.

In an accompanying Comment, Dr Derek Hausenloy, The Hatter Cardiovascular Insititute, University College London, UK, and colleagues say: "With the advent of more potent drugs, the issue of whether raising 'good' cholesterol concentrations reduces cardiovascular risk in patients with very low 'bad' cholesterol concentrations needs to be tested in clinical studies."

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