

Are fish oil supplements recommended during pregnancy?

Dear bpac,

I have to say I do love your publication. The articles are well written and researched and of practical use in General Practice.

I have a question for Dr Lisa Houghton on nutrition and supplements during pregnancy.

Does she recommend fish oil during pregnancy? There seems to be a growing mass of evidence to suggest that it is useful to decrease allergies in the infant and helps with learning and mood etc.

Susanna Kent, GP, Wellington

Fish oil supplements, which contain the omega-3 fatty acid docosahexaenoic acid (DHA), are increasingly being marketed to pregnant and lactating women to enhance cognitive development, visual acuity, nervous system maturity and immune function in the developing infant. At present, the potential benefits to the foetus of maternal DHA supplementation are suggested largely by randomised controlled studies in which supplements were given to the infants after birth.¹ Few clinical studies of DHA supplementation of pregnant women have been conducted. However the number of observational studies linking higher DHA status to favourable infant developmental outcomes has highlighted the need for more quality dose-response clinical trials.

Recent recommendations from the Perinatal Lipid Intake Working Group has suggested that pregnant and lactating women consume ≥ 200 mg/day of long chain polyunsaturated fatty acids, - DHA and EPA

(eicosapentaenoic acid),² while the Canadian and American Dietetic Associations recommend an intake of 500 mg/day.³ Both groups, however, emphasise consuming seafood. Evidence suggests that pregnant women consume low levels of DHA due to avoidance of specific sources of seafood because of their mercury content. Oily fish such as canned tuna, sardines, salmon, mackerel, eel, warehou and kahawai are an excellent source of DHA with little concern over the amounts eaten.

Dr Lisa Houghton, Dietitian, University of Otago

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2. Koletzko B, Cetin I, Brenna JT. Dietary intakes for pregnant women and lactating women *BR J Nutr* 2007;98:873-7.
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CVD and cholesterol

Dear bpac,

In BPJ 17 and 18 there was some good information on cardiovascular risk assessment and lifestyle interventions. I am finding that more and more of my patients, probably in light of some recent publications like the "Cholesterol Myth", are becoming rather suspicious of the pharmaceutical industry and of medicine as a whole.

I have been having trouble tracking down the original studies, that showed that saturated fat causes increased heart disease, and that identified dyslipidaemia as a

cause of heart disease and therefore needing treating rather than just an indicator of risk. I understand that recent study has shown statins mechanism in reducing second heart attacks comes more from its influence on vascular smooth muscle rather than its cholesterol lowering ability.

I am currently feeling a little high and dry on evidence to support my recommendations to patients to lower their cholesterol and reduce the saturated fat in their diet. I would be grateful if you could provide some references to this effect and would be interested in your comment.

Mark Edmond, GP, Christchurch

The so-called "Cholesterol Myth" is the real myth. It has been promulgated by journalists and other writers seeking controversy where there is none, in order to sell newspapers and books. The "false flames" of controversy have probably also been fanned by industries with vested interests in saturated fat production and these are powerful industries with huge propaganda resources. Unfortunately there is little money to be made from the truth, which is that almost everything new that has been learnt about saturated fat, blood cholesterol and congestive heart failure (CHD) in the last 30 years supports what we already knew back then, which is simply that "a diet high in saturated fat causes an increase in blood cholesterol which causes an increase in CHD".

With regards to local data, Jim Mann and colleagues undertook a great little trial randomising people to butter or margarine and showed clear evidence of worsening lipid profiles in those given butter which reversed when they were changed to margarine.¹ This evidence was unsurprisingly consistent with a huge body of international trial evidence from many decades of

research demonstrating that saturated fat consumption increases blood cholesterol levels.

The New Zealand diet has changed significantly over the last 30 years and in particular there has been a significant reduction in saturated fat consumption. For example butter consumption – which alone accounts for one-fifth of our total saturated fat consumption – has fallen from a high of almost 20 kg/head in the late 1950s to about 10 kg/head this decade (www.fao.org). Also the increasing range of low fat milk and other dairy products has had an important impact on our saturated fat consumption. Most of these products were not available until the 1970s and 1980s.

This change in diet has been associated with a substantial reduction in blood cholesterol levels. Since the early 1980s a fall in blood cholesterol of about 0.5 mmol/L on average has been documented. During the same period CHD mortality has fallen by 2–3% per year in New Zealand – more than two-thirds reduction in CHD mortality in New Zealand since the late 1960s! It has been estimated that the decline in blood cholesterol levels between the early 1980s and early 2000s account for about one-third of this decline in CHD mortality.²

However the best evidence comes from systematic reviews of the literature that avoid the peculiarities and random error in single studies. An international meta-analysis of cohort studies published in the Lancet in 2007 described a ten year follow-up of almost one million people and demonstrated beyond doubt that blood lipids are strongly associated with CHD mortality.³

Almost every trial of statins, of which there are now many, support the cohort data described above. Moreover it exposes the other cholesterol myth that statins work primarily by their influence on smooth muscle rather than

their cholesterol-lowering ability. While statins may well work indirectly on smooth muscle, it is almost certainly secondary to their cholesterol-lowering ability because the different declines observed in CHD risk in the different statin trials, can be explained by their effect on lipid levels. This has been demonstrated in another international meta-analysis of almost 100,000 people in randomised trials of statins versus placebos, published in the Lancet in 2005.⁴ The meta-analysis clearly shows a strong and consistent relationship between changes in blood lipids and changes in CHD risk.

*The cholesterol myths are generated by people who cherry pick individual studies or parts of studies to support their need for controversy. The boring headline one never gets to read, based on a more systematic approach to the huge range of evidence, is that: **“We continue to confirm with almost every new study, that what we knew 30 years ago about saturated fat, cholesterol and CHD is still true – they are strongly related and the effects are reversible.”** The proof is also in the (low saturated fat) pudding. In New Zealand saturated fat consumption has been falling for over 30 years, blood cholesterol levels have been falling over the same period (it started before statins were invented), and so has CHD.*

Professor Rod Jackson, School of Population Health,
Faculty of Medical and Health Sciences, Auckland
University

References:

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3. Prospective Studies Collaboration. Blood cholesterol and vascular mortality by age, sex, and blood pressure: a meta-analysis of individual data from 61 prospective studies with 55 000 vascular deaths. *Lancet* 2007; 370: 1829–39
4. Cholesterol Treatment Trialists' (CTT) Collaborators Efficacy and safety of cholesterol-lowering treatment: prospective meta-analysis of data from 90 056 participants in 14 randomised trials of statins. *Lancet*. 2005; 366: 1267–78.



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