Thyroid autoantibodies, preterm birth, and miscarriage

The association is clear, but the effects of levothyroxine treatment are uncertain

RESEARCH, p 1065

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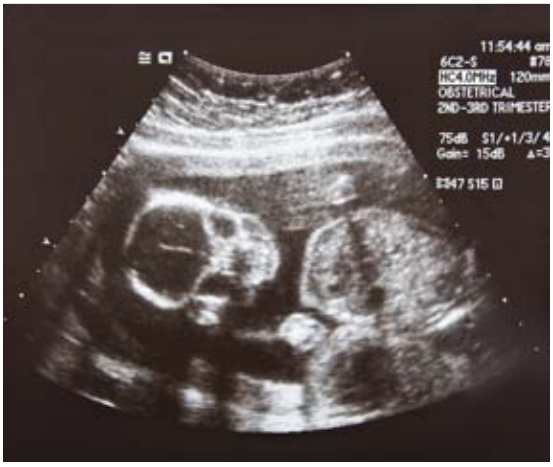
In the linked systematic review Thangaratinam and colleagues assess the association of thyroid autoantibodies with miscarriage and preterm birth in biochemically euthyroid women and the effect of levothyroxine treatment on pregnancy outcomes.[1]

An association between thyroid antibodies and miscarriage was first reported in 1990.[2] The finding was serendipitous—the prospective study was originally designed to evaluate the incidence and causes of postpartum thyroiditis. As the study progressed, the unexpected number of spontaneous miscarriages in euthyroid women with thyroid antibodies prompted the researchers to assess pregnancy outcome in the 552 women who were initially screened in the first trimester. The rate of miscarriage in thyroid antibody positive women was twice that seen in thyroid antibody negative women (17.0% *v* 8.4%; P=0.01). The increased rate of pregnancy loss was not related to differences in maternal age, cardiolipin antibody status, pregnancy history, or concentration of thyroid stimulating hormone. Similarly, the initial discovery of a significant association between thyroid antibodies and preterm delivery seems to have been by chance. In 1994 a prospective study of 87 pregnant women with thyroid antibodies reported a significantly higher rate of preterm delivery in euthyroid women with thyroid autoantibodies (16% *v* 8%; P<0.005). Further information was not provided.[3]

During the next 20 years numerous studies evaluated the relation between thyroid antibodies and both pregnancy loss and preterm delivery. Although research initially focused on pregnancy loss in unselected women, it soon began looking at recurrent abortion and pregnancy loss in women undergoing in vitro fertilisation. This is not surprising because 10-25% of all such pregnancies end in miscarriage, 0.5-1.0% of all women experience recurrent abortion, and in vitro fertilisation is expensive and invasive.[4 5] A smaller literature on preterm delivery has also developed, which again is not surprising because most neonatal deaths are related to preterm birth.

Studies so far have varied in terms of the number of participants, country of origin, study design, gestational age at study entry, and laboratory methods used. Although most studies of both miscarriage and preterm delivery have shown a significant correlation with thyroid autoantibodies, results are conflicting.

The systematic review and meta-analysis by Thangaratinam and colleagues is therefore a useful contribution to the literature. The review included 31 studies on miscarriage (12 126 women) and five studies on preterm delivery (12 566 women). Meta-analysis of both the cohort (n=19) and case-control studies (n=12) showed a positive association of thyroid antibodies with pregnancy loss (odds ratio 3.9, 95% confidence interval 2.48 to 6.12 (P<0.001) for cohort studies and 1.8, 1.25 to 2.6 (P<0.002) for case-control studies). A doubling in the odds ratio was also found for preterm birth in thyroid antibody positive women (2.07, 1.17 to 3.68). The mean concentration of thyroid stimulating hormone was significantly higher in thyroid antibody positive women with pregnancy loss than in the antibody negative group (by 0.51 mIU/L) P=0.007). Meta-analysis of the two randomised controlled trials of levothyroxine treatment showed a significant reduction in miscarriage (relative risk 0.48, 0.25 to 0.92). One trial also found a significant reduction in the rate of preterm birth (0.31, 0.11 to 0.90).[1]



Future studies need to identify the mechanism that links thyroid antibodies to preterm delivery

Whether thyroid antibodies cause pregnancy loss and preterm delivery remains unanswered. It has been suggested that thyroid antibodies are an epiphenomenon indicative of an undefined autoimmune process; that thyroid antibodies reflect a subtle decrease of thyroid function; and that thyroid antibodies are causally related to pregnancy loss—perhaps through a mechanism at the placental level. Preliminary data on each of these hypotheses have been forthcoming. Studies in pregnant women have recently shown an increased rate of pregnancy loss in thyroid antibody negative women with thyroid stimulating hormone values in the first trimester of 2.5-5.0 mIU/L and an increase in child loss (defined as the combination of miscarriage, fetal death, and neonatal death) as concentrations increase within the normal range.[6 7] A prospective randomised trial showed a 69% decrease in the preterm delivery rate in euthyroid women with thyroid autoantibodies given levothyroxine.[8]

The systematic review by Thangaratinam and colleagues represents a turning point in the ongoing story of thyroid antibodies and adverse pregnancy outcomes. Their meta-analysis clearly shows a significant association between thyroid antibodies and both pregnancy loss and preterm delivery. Research should now focus on identifying the underlying mechanism that links thyroid antibodies to miscarriage and preterm delivery and appropriate treatment. Animal models need to be expanded and in-depth studies of immunological markers in thyroid antibody positive women who miscarry, as well as their placentas, are logical next steps. Further randomised controlled trials assessing the effect of giving levothyroxine to women with thyroid autoantibodies are needed, as currently only one exists.[8] The TABLET (Thyroid AntiBodies and LEvoThyroxine) trial—a multicentre, placebo controlled, double blind trial—is set to assess the impact of levothyroxine treatment on miscarriage and preterm delivery in euthyroid women with thyroid antibodies and investigate multiple immunological markers.

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Arsenic in drinking water

Increases deaths from cardiovascular disease

RESEARCH, p 1067

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Arsenic has more effects on health than any other toxicant, and the list continues to grow, along with evidence that exposure is widespread throughout the world. Ingestion of inorganic arsenic in drinking water causes cancer of the skin, bladder, lung, liver, and kidney.[1 2] Mounting evidence suggests that arsenic is also a cause of chronic respiratory disease,[3 4] and adverse effects on reproductive outcomes and child development have also been reported.[5 6] In the linked cohort study, Yu Chen and colleagues add to the evidence that arsenic in water increases mortality from cardiovascular disease with the findings of their prospective cohort study in Bangladesh.[7]

The first evidence of a link between cardiovascular disease and arsenic in drinking water came in 1980 from Antofagasta, Chile, with a report of 17 deaths from myocardial infarction in people under the age of 40.[8] Later, a comprehensive body of evidence from a series of studies in Taiwan starting in 1988 found that arsenic in water was associated with increased mortality from cardiovascular disease.[9] In 2007, cardiac effects including QT prolongation were shown to be associated with arsenic in drinking water in China.[10] In 2008, a 50 year study in Chile showed that mortality from acute myocardial infarction was the main initial cause of death attributable to arsenic in drinking water. This effect started about a year after exposure commenced and peaked at a mortality rate ratio of 1.48 for men (P<0.001) and 1.26 for women (P<0.001).[8] Increased mortality from myocardial infarction gradually decreased after exposure ceased, after which lung and bladder cancers became the main long term causes of death as a result of arsenic. In 2009, a prospective cohort study in Bangladesh of more than 115 000 people reported a clear dose-response trend between increased concentrations of arsenic in water and increased mortality from cardiovascular disease.[11] For drinking water concentrations under

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Arsenic poses higher health risks than any other known environmental exposure

10 µg/L, 10-49 µg/L, 50-149 µg/L, 150-299 µg/L, and 300 µg/L or more, the mortality rate ratios were 1, 1.03, 1.16, 1.23, and 1.37, respectively (test for trend P=0.026).

Chen and colleagues' study adds important new evidence that arsenic increases mortality from ischaemic heart disease. In a prospective cohort study of 11 746 people, they report a

clear dose-response trend for mortality between ischaemic heart disease and arsenic concentrations in water—rate ratios were 1 in the reference group, but increased to 1.22, 1.35, and 1.92 as water concentrations increased (test for trend P=0.002).[7] They found a similar association with urine arsenic concentrations, making this the first study to use this biological marker to confirm exposure. This is also the first study to find evidence of synergy between smoking and arsenic. Non-smokers in the group who had high arsenic exposure had a rate ratio of 1.53 for mortality from cardiovascular disease (an imprecise estimate because of the small numbers), but for smokers the rate ratio was significantly increased to 3.45 (95% confidence interval 1.32 to 8.98).

Does all this matter? After all, the relative risk estimates reported here are moderate compared with those associated with outcomes such as bladder cancer and lung cancer, which often exceed 5. However, cardiovascular disease is the most common cause of death worldwide, so moderately increased relative risks mean very large numbers of excess cases. Also, exposure in utero and in children has a major effect on mortality in young adulthood,[6] including mortality from myocardial infarction.[8] More research is needed on the impact of early life exposure, and on the mechanisms that make arsenic so toxic.

In the meantime there is enough evidence to highlight a serious public health concern because exposure to groundwater containing arsenic is widespread throughout the world. Early research into the health effects of arsenic was largely limited to populations in Taiwan, Argentina, Mexico, and Chile and to areas with private wells in the United States. Many more countries can now be added to that list, which continues to grow. And arsenic poses far higher health risks than any other known environmental exposure, with about one in 10 people dying because of high concentrations of arsenic in water.[8]

Water contaminated with arsenic is tasteless, looks crystal clear, and boiling the water only concentrates the arsenic in it. In all parts of the world where groundwater is used for drinking, clinicians should therefore ask their patients where they obtain their drinking water. If it comes from a well, the next question should be whether the water has been tested for arsenic. If not, the patient should be urged to have it tested. It is too late to identify exposure after diseases caused by arsenic have been diagnosed, because many are fatal.

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Treatment with β blockers in people with COPD

May reduce respiratory symptoms and mortality independently of cardiovascular effects

RESEARCH, p 1068

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Treatment with β blockers reduces mortality associated with cardiovascular diseases. Unfortunately, doctors tend to avoid β blockers in patients who have concomitant obstructive lung disease. This stems from the fear of precipitating a catastrophic respiratory event—acute bronchospasm has been reported after administration of β blockers to patients with asthma and chronic obstructive pulmonary disease (COPD).[1-4] However, evidence suggests that β blockers reduce mortality in patients with COPD, which makes sense because patients often have concomitant cardiovascular disease.[5] A study of 462 patients with COPD undergoing major vascular surgery found a 27% reduction in perioperative mortality in those who were receiving cardioselective β blockers compared with those who were not.[6]

Surprisingly, in addition to reducing overall mortality and death from cardiovascular disease in patients with COPD, long term use of β blockers may also reduce respiratory events and morbidity. One study reported a 30% reduction in mortality and risk of exacerbations in patients with COPD who were taking β blockers regardless of whether they had cardiovascular disease.[7] The linked cohort study by Short and colleagues adds to this evidence by further defining the respiratory effects of β blockers in COPD, in addition to their cardiovascular benefits.[8]

Short and colleagues assembled relevant data from robust Scottish databases. They selected more than 5000 patients by the presence or absence of concomitant β blocker treatment and reported outcomes systematically according to the stepwise tier of treatment for COPD. The death rate in the entire cohort was 34% a year. As expected, for each level of COPD severity (as judged by prescribed drugs) patients taking β blockers had significantly lower mortality from cardiac


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disease than those not taking β blockers. This mortality benefit persisted even after adjustment for known cardiovascular risk factors and events. Deaths classified as related to COPD were lower in those taking β blockers too. Respiratory related hospital admissions and emergency oral steroid prescription were also significantly lower in people taking β blockers (hazard ratios ranged from 0.23 to 0.88).

It is tempting to dismiss these results as being related to improvements in cardiovascular disease (such as chronic heart failure) resulting in reduced shortness of breath, which is then interpreted as a reduction in COPD symptoms. However, these differences persisted after adjustment for cardiovascular disease, which suggests that β blockers have an independent effect on COPD itself. Is such a direct effect on COPD plausible?

Animal data suggest altered airway responsiveness and airway inflammation after chronic β blockade.[9 10] A recent small pilot study in humans supports this observation. Despite initial acute bronchospasm, chronic escalating doses of β blockers in patients with asthma reduced airway responsiveness.[11] Thus, chronic β adrenoceptor blockade in COPD may exert positive effects through bronchoprotective, anti-inflammatory, and mucus resolving effects.

Short and colleagues' study suggests that COPD related events were reduced in patients taking β blockers, irrespective of disease severity. Should clinicians therefore prescribe these drugs to treat people with COPD? Not yet. Although the data on mortality from cardiovascular disease clearly show that β blockers should not be withheld for cardiovascular indications, using them directly for COPD is premature. The study was a retrospective analysis of healthcare databases, so confounding by indication and post hoc adjustment by doctors treating the patients cannot be eliminated. However, together with other data, these results indicate that the use of β blockers may help reduce the morbidity associated with COPD. Prospective studies are therefore needed to ascertain any beneficial effect of β blockers on COPD related outcomes.

How should clinicians start a patient with COPD on a β blocker for cardiac indications? It is advisable to use a cardioselective β blocker and to observe the patient during the administration of the first dose. In the rare case that bronchospasm does occur, anticholinergic agents should probably be used first. Of interest, the data from asthma suggest that β adrenoceptor agonists may still be effective in reversing bronchospasm in the presence of the low doses of β blockers used in asthma pilot studies.[12]

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The health impacts of cold homes and fuel poverty

Three reasons to act: the health burden, inequity, and mitigation

NEWS, p 1046

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On 12 May Michael Marmot and his team published their report, "The heath impacts of cold homes and fuel poverty," commissioned by Friends of the Earth.[1] The report highlights an obvious, well known, and largely ignored fact—that cold homes waste energy and harm their occupants—and identifies an opportunity for simultaneous gains on three fronts. By improving the thermal efficiency of British homes the government would reduce carbon dioxide ("greenhouse") emissions, avoid a major burden of ill health, and reduce health inequity, which—as the report shows—maps closely with social and economic disadvantage. The report delivers three messages. Firstly, improving the energy efficiency of the housing stock—to spread "affordable warmth"—would bring multiple health gains, directly and through improved home finances. Secondly, fuel poverty as a result of poor housing stock causes avoidable health inequality and is unjust. Thirdly, reduced fuel use would bring environmental gains, in the short term through reduced air pollution and in the longer term in helping to mitigate climate change.

The same is true of Australia, which is perhaps often envied by inhabitants of northern Europe as a land of sand, sunshine, and seasonal tropical monsoons that bring welcome warm rains (albeit sometimes to excess). The reality is that even in the subtropical city of Brisbane (population two million) deaths as a result of extremes of winter cold are roughly equal to those attributable to extremes of summer heat.[2] This fact matches the finding in Europe that "higher rates [of excess winter deaths] are found in countries with less severe, milder winter climates."[3] The explanation is that building standards have been raised in colder countries such as Finland and

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Sweden, but not in countries with a milder climate such as the United Kingdom. The report estimates that in the UK, about 5500 more deaths a year occur in the coldest quarter of houses than would occur if those houses were warm. Of note, this substantial burden of mortality was shown only by careful accumulation and analysis of national statistics. Might measures of housing quality be added to the international health statistics website, gapminder.org? The software at this site (created by Hans Rosling) allows graphical cross referencing of many national statistics over time, but housing quality is not currently represented among the variables available.[4]

Living in a cold house can affect health at any age, not just in old age, for a variety of reasons. Although the extra deaths in elderly people are caused mainly by cardiovascular and respiratory disease, far greater numbers have minor ailments that lead to a huge burden of disease, costs to the health system, and misery. Compared with those who live in a warmer house, respiratory problems are roughly doubled in children, arthritis and rheumatism increase, and mental health can be impaired at any age. As the report notes, adolescents who live in a cold house have a fivefold increased risk of multiple mental health problems.[1]

The report also presents evidence that living in a cold house has indirect effects, some of which persist throughout life. In many such households, educational attainment is affected, emotional resilience is impaired, and the financial burden of heating a poorly insulated house takes food off the table, risking malnutrition.[1]

The action proposed in the report connects well with the important concept of "health co-benefits," wherein health benefits accrue directly within communities that undertake an intervention that is aimed primarily at mitigating climate change, such as insulating houses to reduce energy use.[5] The "win-win" aspect of co-benefits is often overlooked. For example, in Australia a government funded programme of home insulation was undertaken in 2009 as an economic stimulus measure. It was, commendably, aimed at mitigating climate change and the public health benefits of the programme were not much stressed (on this occasion, however, the public health community should probably be glad of its low profile: four deaths and many house fires associated with faulty installations led to early cancellation of the programme).

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Thermogram of a poorly insulated house: heat is escaping from the roof, chimney and windows

The Marmot report takes the same approach in reverse—an environmental benefit (reduced greenhouse emissions) will accrue from an intervention aimed primarily at protecting health. In addition to this double benefit, the social equity argument provides yet a third motivation.

We should not assume that because the planet is warming dangerously, cold temperatures will become a thing of the past. Climate scientists anticipate that warming will be accompanied by increased variability.[6] Furthermore, warming will not be globally uniform. In particular, northern Europe might become much colder later this century if the meridional overturning circulation is weakened by inflows of fresh water from a melting Greenland ice sheet (the geological record shows that such things have happened before).[7]

The world community is struggling to curb greenhouse gas emissions. The concentration of atmospheric carbon dioxide is not merely continuing to rise when it should be starting to fall, but its rise is accelerating.[8] The essence of the problem is our apparent unwillingness—as people, populations, and politicians—to put moral obligations above short term economic interests. So, when measures are identified that have negligible net cost and that will bring benefits on many fronts, including reducing health inequalities, they should be enthusiastically and promptly embraced and implemented.

Britain, like some of its former colonies, is saddled with obsolete housing stock many decades, if not centuries, old. These inadequate homes are a waste of energy, a health hazard, and (given today's levels of national wealth) a shameful relic for their part in fostering persistent, avoidable, social inequity. For many reasons—economic, ethical, environmental, and epidemiological—governments should heed the call in this timely report.

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Food price crises and health

Policies on trade, grain reserves, and biofuel subsidies all need to change

OBSERVATIONS, p 1060

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Listen to the interview with David Nabarro, who describes why and how we should control food price increases

The world's poor are experiencing the second drastic increase in food prices in four years. This time it will probably have an even worse effect on health and nutrition, because the first price shock in 2008 combined with the global economic recession undermined the meagre asset base of the poor and gave them no time to rebuild it. Narrowly conceptualised economic assessments of the "costs" of food price crises underestimate the true burden, which includes their impact on health.

The most relevant price for poor people is that of grain—wheat, maize, and rice in particular. On the international markets, maize prices increased by 105% and wheat by 102% between March 2010 and March 2011. There is little international trade in rice, and its price has increased less than that of wheat. Such increases mean that in many developing countries a kilogram of wheat costs about $0.30 (£0.18; €0.21) instead of $0.15—a crucial difference for people living on $1 a day, as about one billion people do worldwide. The increased price of grain has meant that people have to cut back on other food and non-food expenditures to maintain food energy consumption.

Consequently, the quality of people's diet and lives has suffered. The absolute number of undernourished people in developing countries has increased from 823 million in 1990 to about one billion today. The increase in dietary deficiencies and related lifelong physical and mental health effects are less well known.[1] Roughly two billion people have micronutrient deficiencies.[2] The prices of non-staple foods such as vegetables and pulses have risen even more than the price of grains, which has added to these deficiencies, especially in South Asia.

Even before the price crisis, undernutrition was a major underlying cause of 53% of deaths in children under 5 years worldwide.[3] Drastic increases in food prices cause ill health through multiple pathways. Firstly, at the household level, cutting back on energy consumption and a decrease in the quality of the diet has a direct effect on health. Secondly, cutting back on necessary food consumption leads to undernutrition, and undernourished people are more vulnerable to diseases (such as diarrhoea and malaria). Thirdly, the capacity for people to insure themselves and to buy services is affected: because high prices lower people's purchasing power, reduced resources are available for spending on health, and at the community level the capacity for people to help each other is reduced. Finally, public services including health services lose funding when food import bills increase. These combined and interacting pathways lead to millions of the poorest people in the world becoming trapped in a vicious cycle of food and nutrition insecurity.

Today's food price crisis is fundamentally the consequence of years of neglected public investment and low support for innovation in agriculture in many developing countries, neglect of agriculture in development aid, and energy subsidisation policies in industrialised countries

PANOS

The increased price of grain means that people's diet and quality of life suffers

that favour fuel over food. The 2008 crisis was triggered by adverse weather events and was exacerbated by export restrictions, lack of regulation of commodity trade, and speculation about food prices by financial organisations.[4]

Climate change will add to future risks in the global food system. Financial markets have become closely linked to food markets and these links between food and finance pose added risks and uncertainties for the poor. The 2008 food price crisis ended because demand contracted during the global recession, but this cannot be expected in 2011. Policy actions are needed to mitigate the price spike.

Extreme price volatility is an international problem that requires coordinated international action, such as changes in trade policy and an end to current subsidies for biofuels, which burden the poor. National self sufficiency policies—attempts to grow all food domestically—are economically wasteful because opportunities to gain income are foregone. The response to the food crisis must include innovation in production and technology, trade policies, and grain reserves policies. The opportunities for small farmers to increase their earnings as a result of higher prices need policy support, so that they can get better seeds and fertiliser and reach markets. We also need to reduce waste in consumption and food processing.

Policy actions in three areas are needed to protect health explicitly: income transfer payments in times of need and child nutrition to improve the diet of those who are most vulnerable; mitigation of short term risks (including cash transfers, pension systems, and employment programmes); and preventive health and nutrition interventions, such as access to clean water and improved infant feeding, to avoid long term negative consequences.

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