*Critical Public Health*
Vol. 22, No. 2, June 2012, 159–177



# Slaying sacred cows: is it time to pull the plug on water fluoridation?

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(*Received 19 March 2010; final version received 27 April 2011*)

Water fluoridation continues to be a contentious public health policy. Recent moves to introduce schemes in England raise important questions about the use of evidence in public policy. Of particular concern is how evidence is used for public health policy-making purposes. This article reviews some of the key debates about water fluoridation and examines the way evidence has been promoted and used. The background to water fluoridation is discussed and also key ideas about how evidence influences policy. While traditionally the problem of evidence is characterised as one where policy makers either accept or ignore evidence, a central concern of this article is where poor evidence is promoted by professionals and accepted by policy makers. The article then examines the evidence on the effects of water fluoridation. Drawing on the idea of the 'Gold Effect', the article shows how deeply held beliefs about public health actions shape not just policy but also the application of evidence itself by professionals and researchers.

**Keywords:** evidence; healthy public policy; population health; water fluoridation



In his first major speech at the National Health Service (NHS) Confederation Conference in June 2009, the then United Kingdom (UK) Secretary of State for Health, Andy Burnham MP, argued that 'We've been too timid at times on the public health agenda. Let's press ahead with fluoridation of water supplies, given the clear evidence that it can improve children's dental health'. While a welcome emphasis on public health, the choice of water fluoridation as an example is of particular interest as despite the Department of Health's long-standing commitment to extending water fluoridation,[1] there is no scientific consensus that water fluoridation is either safe or effective. Despite this uncertainty, the UK Government and the NHS in England, along with governments in Australia and the USA are committed to extending community water fluoridation. However, proposals for water fluoridation remain contentious and lead to strong opposition. This article explores why water fluoridation policy is controversial within the context of how evidence is used in public health policy making. This article reviews the evidence on water fluoridation and questions whether uncritical support for this public health policy should be continued. While issues of ethics and civil liberties are

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ISSN 0958–1596 print/ISSN 1469–3682 online

http://dx.doi.org/10.1080/09581596.2011.596818
http://www.tandfonline.com

also pertinent to debates about water fluoridation, these are not covered in this article. However, the concerns expressed here about the current evidence base and the way evidence is used are of direct and important relevance to such broader discussions about consent and individual liberty.

## Water fluoridation

Water fluoridation has been a controversial intervention since its introduction in the USA in the early post-war period. It involves adding a fluoride compound (usually hexafluorosilicic acid – a by-product of the phosphate industry) at 1 part per million (ppm) to public water supplies to prevent dental caries. The addition of 1 ppm is widely referred to as the optimum concentration in the literature on dental health, policy documents, NHS guidance and reports on water fluoridation. This 'optimum concentration' is the concentration of fluoride within water supplies that, it is argued, will provide maximum protection from dental decay without causing dental fluorosis. As is discussed later, there are key questions to be asked about the idea of an 'optimum concentration'. The claimed preventive effects of fluoride on dental caries were first observed at the turn of the twentieth century when mottled teeth, caused by exposure to fluorides naturally found in water supplies (calcium fluoride), were found to be associated with less dental caries (Black and McKay 1916, Dean and Elvove 1935, Dean 1938). These findings served as the basis for determining the optimal concentration of water fluoridation for preventing caries and minimising dental fluorosis, although Dean and colleagues were concerned about wider toxicological effects on human tissue beyond the observed problem of dental fluorosis and argued that further studies were needed. Based on these observational studies, a number of pilot schemes were introduced in the 1940s in North America to provide a long-term study of the effects of water fluoridation. However, the US Public Health Service adopted the 1 ppm 'optimal concentration'[2] and supported the widespread introduction of community water fluoridation schemes in 1950 before the results of the pilot community studies were known (Connett *et al*. 2010). More importantly, these early studies have been subject to severe criticism about their methodological quality and interpretation of the results (Sutton 1959, Ziegelbecker 1981, Connett *et al*. 2010).

Debates about water fluoridation are inherently shaped by history and by a system of values about the role of fluoride as a key preventive measure in oral health care. To some extent, both advocates and opponents of water fluoridation draw on the development of the practice of water fluoridation to substantiate their positions. Proponents support the 1 ppm 'optimal concentration' citing the widespread use of water fluoridation in countries such as the USA, Australia and Eire as examples of its perceived success in reducing dental caries, and highlight the fact that there have been no health problems observed in the West Midlands of the UK (see, e.g. Martin 1991, British Fluoridation Society 2003). Opponents refer to problems associated with fluoride as a toxic chemical waste, its associations with the nuclear and aluminium smelting industries, problems of fluoride poisoning and a wide range of health concerns including cancer, fluorosis and neurotoxicological problems. They also highlight the fact that water fluoridation is an enforced medication (see, e.g. Bryson 2004, Connett *et al*. 2010).

In the UK, schemes were introduced in the 1960s in the West Midlands and North East. Despite numerous proposals for new schemes (over 60 in the past 20 years), apart from a limited extension in the West Midlands there have been no new schemes despite strong government, dental profession and public health support. Reluctance by local authorities and water companies to instigate fluoridation schemes and the complexities of gaining multi-organisation agreement across local authorities and water companies has, from the proponent's perspective, frustrated the expansion of water fluoridation. In 2003, the government changed the law to give the NHS stronger powers to request water companies to add fluoride to water supplies and then, in 2005, regulations were introduced that removed water companies' veto and indemnified them against liabilities they may incur in complying with fluoridation arrangements. The regulations also set out rules for Strategic Health Authorities (SHAs) to consult local populations before water fluoridation schemes were introduced (Figure 1).

In 2008, the South Central SHA (SCSHA) was the first SHA to undertake such a consultation and in February 2009 gave the go-ahead for the introduction of the first major water fluoridation scheme in England for many years.[3] The decision was welcomed by the President of the Faculty of Public Health who called it a mould-breaking decision suggesting that in cases like Southampton, civil liberty arguments have to be set aside for the good of the majority. While there are undoubtedly civil

Regulation 5 of the Water Fluoridation (Consultation) (England)

… an SHA would not decide to conduct a consultation if it were not persuaded of the potential benefits of fluoridation, but SHAs are also corporately responsible for conducting a consultation as prescribed in the legislation including an objective assessment of the responses to that consultation. This responsibility should not prevent officers of the SHA promoting the case for fluoridation as the preferred option of the SHA.

Regulations 2005 ('the Consultation Regulations') prescribes the circumstances in which a SHA may proceed with a proposed 'step' regarding fluoridation arrangements, i.e.

- proposals for a new scheme;
- a variation in an existing scheme in the circumstances described at paragraphs 12 –14 of Appendix 2); or
- maintenance of a fluoridation scheme, ie replacement or upgrading of fluoridation plant required for the in the circumstances as described in paragraph 15 of Appendix 2)
- notice to a water undertaker to terminate fluoridation arrangements.

Regulation 5 of the Consultation Regulations describes the circumstances in which an SHA, after a consultation, may proceed with implementing it's proposals. The circumstances are that, 'having regard to the extent of support for the proposal and the cogency of the arguments advanced, the SHA is satisfied that the health arguments in favour of proceeding with the proposal outweigh all arguments against proceeding.'

Figure 1. Water Bill and guidance.

liberty issues that are pertinent in the case of water fluoridation, the key questions actually relate to its effectiveness in preventing dental caries and reducing inequalities, its adverse effects on health, its economic effectiveness and whether it is an ethical intervention (Cheng *et al*. 2007). However, the problem is that despite large numbers of studies, the evidence base is not strong and reviews of water fluoridation conclude that there are still fundamental questions to be answered about water fluoridation's effectiveness and safety - raising questions about the use of evidence in public health policy (McDonagh *et al*. 2000, National Research Council, NRC 2006, Nuffield Council on Bio-ethics 2007, Scientific Committee on Health and Environmental Risks, SCHER 2010).



### *Evidence and public health policy*

Evidence-based public health presents particular problems relating to the nature of the evidence and how this is applied in practice (Dobrow *et al*. 2004, Petticrew *et al*. 2004). Evidence is often viewed through particular frames and is by nature contestable. The question for public health policy makers is, therefore, what evidence to draw on? Medicine focuses on the individual patient and evidence is '... developed through systematic and methodologically rigorous clinical research, emphasising the use of science and de-emphasising the use of intuition, unsystematic clinical experience, patient and professional values, and patho-psychologic rationale' (Dobrow *et al*. 2004, p. 207). Critics of evidence-based medicine (EBM) argue that this approach is too narrow and excludes other forms of clinically relevant evidence (Miles *et al*. 2000). Proponents of EBM have responded by developing hierarchies of evidence based on methodological rigour but these remain value bound and are not, themselves, evidence based (Miles *et al*. 2000, Oxford Centre for Evidence-Based Medicine, 2001). Despite these debates EBM is increasingly influential in the health policy arena, focusing attention on evidence based health policy. Yet as Black (2001) has argued '... evidence based policy is not simply an extension of EBM: it is qualitatively different' (p. 277).

Public health is not focused on the individual but on populations, and, by its very nature, is a more political process than medicine as it deals with social processes. For example, the relative priority given to health inequalities is underpinned by ideological positions about the nature of inequality itself. Debates about the extent to which the state should intervene in individual lifestyles is not one that is open to a strict evidence-based approach, although evidence is often employed in arguments to sustain particular view points (Holland 2008). Public health policy involves not only decisions about the degree or distribution of *health* harm or benefit but also how to define those health harms and benefits and balance these against issues such as individual freedom. As Kenny and Giacomini (2005) have argued: 'The quintessential ethical problem of the public policy maker is how to define, identify, justify, and distribute inevitable benefits and harms, rather than simply striving to ensure benefit and avoid harm' (p. 254). It is because of the population-wide application of public health interventions that Skrabanek (1994) argued that the link between evidence and public health interventions should be *stronger* than that for other forms of medical interventions because public health deals with people who are not 'ill' nor have they approached a health care practitioner asking for medical assistance.

Water fluoridation is a universal measure that is justified in policy terms because, it is argued, it primarily addresses social inequalities and, while helping those children with the worst dental caries, it is believed that there are no health risks to the wider population (BFS 2003). However, this view is contestable as there is evidence that questions water fluoridation's effectiveness and safety (McDonagh *et al*. 2000, NRC 2006). But as Weed (2004) has suggested, public health is located at the crowded, and difficult, intersection between risk, health effects and prevention. To help practitioners and policy makers navigate their way through this contentious area there is an appeal to scientific evidence to support particular interventions and actions in public health.

This raises two problems. The first is that evidence can be either philosophical-normative (independent of context) with an emphasis on the quality and criteria for evidence, or practical-operational (context-based) where evidence '... is characterised by its emergent and provisional nature, being inevitably incomplete and inconclusive' (Dobrow *et al*. 2004, p. 209). Such debates about the nature of evidence are not new. Douglas (1986) has argued that the way we make sense of the world, the categories of classification, are all socially constituted and socially reproduced. The second is that we must also examine how such evidence is used in the policy process. There are a number of models that purport to explain how evidence is used for policy making including: the rational (or linear model) which is the underlying theory behind many calls for evidence-based approaches; the enlightenment model where research and policy making take place alongside other social processes; the strategic model in which research is seen as 'ammunition' to support predetermined positions or to delay decisions, and the media 'conspiracy' model that confers a specific role to the media in transferring 'evidence' to policy makers (Buse *et al*. 2005). All these models suffer from significant limitations as they are predominantly descriptive and in particular do not say anything about *why* particular evidence is used in any given situation or about *when* evidence is used strategically, and they lack explanatory theories. Evidence from research has to compete with 'ordinary knowledge' which owes its origins to 'common sense, casual empiricism or thoughtful speculation and analysis'. The scientific evidence is generally seen as most definitive, even though it is sometimes ignored or overridden by other interests in the policy process (Petticrew *et al*. 2004, Nutley *et al*. 2007). Fundamentally, the development of policy is based upon values. The use of evidence is influenced by both ideological positions and what Sabatier has described as core beliefs, thus selective use of evidence is likely (Sabatier and Jenkins-Smith 1999) – a process described by Freeman (2007) as 'bricolage'.

However, these models of evidence use in policy do not explain why some areas of policy are promoted despite concerns regarding the type of evidence and sometimes in the face of substantial contradictory evidence. Well-known examples of policy flying in the face of evidence include the recent furore over drugs misuse policy in the UK and cholesterol screening and statins use (Gossop and Hall 2009, Hann and Peckham 2010). While appeals to ideas and political context affecting political policy making may explain the recent controversy over the classification of drugs in the UK (Lancet 2010), of particular interest are areas such as cholesterol screening and water fluoridation where there is substantial professional medical and public health support for such measures. This phenomenon is not restricted to medicine and similar debates are found in the physical sciences (Kundt 2008). The so-called 'Gold Effect' (named after its creator Professor T. Gold) was outlined by Lyttleton (1979) who describes a process by which an idea comes to be held as a generally accepted

truth. In short, the process begins with a small group of like-minded people who meet to discuss their idea. Next, those who have conceived the idea decide to have a conference or meeting and at this meeting there will be more people favouring the idea than against it. Then, a representative committee might be selected to publish a collection of papers and articles to foster interest in the idea. The resulting articles and papers will show an increasing consensus of agreement and the idea will gather pace and momentum. One of the aspects of the 'Gold Effect' which is important here is that the pace at which such an idea can grow is assisted if the idea links in with pre-existing beliefs or assumptions (Lyttleton 1979, Hann and Peckham 2010). For example, there is already a commonplace belief that fluoride is 'good for your teeth' and this view has attracted medical endorsement and media attention. Fluoride is in most toothpastes and mouth rinses, the inclusion of fluoride is used as a positive marketing ploy by commercial companies and they stress the endorsement of dental authorities. Thus there is a strong 'fluoride is good' discourse which reinforces the orthodox position both within the health care 'establishment', and, importantly, in the over-the-counter drugs industry and minds of the general public. The 'Gold Effect' ensures that these mutually reinforcing messages are rarely questioned or challenged. People who might have opposing views find it difficult to get their views published and are regarded as being misguided or just plain wrong. As Lyttleton (1979) remarked, by this stage 'the idea is well on its way to becoming something akin to a religion with a following of devout believers' (p. 189). This framework provides a useful way of reflecting on water fluoridation policy. The remainder of this article outlines some of the key central beliefs of water fluoridation and explores these in relation to the evidence.

There are thousands of articles examining the use of fluorides to prevent dental caries ranging from those examining the well-known toxic effects of fluoride to those promoting its use in oral hygiene. This article focuses, however, specifically on its use in water as a prevention measure for dental caries and how this is promoted as a safe and effective public health measure. Two aspects are considered: first, the idea of the 'optimum concentration' and second, its effectiveness in preventing caries and its safety. These aspects are then considered in the context of the use of evidence in public health policy.

### *The optimum concentration*

Promoters of water fluoridation refer consistently to the 'optimum concentration' – the level of fluoride in water that will maximise prevention of dental caries and minimise the effects of fluorosis. The 'optimum concentration' is generally regarded as being one part of fluoride to one million parts of water – often referred to as 1 ppm. During the 1930s and 1940s, Dean and colleagues conducted a number of studies that provided epidemiologic evidence linking what they referred to as 'dental fluorosis' or 'enamel fluorosis' to excessive fluoride in drinking water. However, they also observed that in communities in the USA where water was naturally fluoridated children had fewer decayed teeth than in communities without fluoride in their water (Dean and Elvove 1935, Dean 1938, Dean *et al.* 1942, 1950). They identified what appeared to be a tradeoff between the protective effect of fluoride concentration for dental caries and increasing levels of fluorosis and that the former were maximised, while the latter minimised at about a concentration of one part of fluoride to 1

million parts of water (1 ppm) (Nichols 1939). Dean's studies were undertaken long before the use of topical fluorides and the more widespread use of fluoride in oral health products and treatments and as a pesticide. In addition, there have been a number of criticisms of Dean's observations regarding the attributing of water fluoride concentrations to variations in dental caries and also of the research on the first pilot schemes suggesting other confounding factors such as diet and socio-economic differences were not fully considered and that data were used selectively (Sutton 1959, Ziegelbecker 1981). In fact, later analysis of the data demonstrated substantial methodological flaws in the collection of data, *ad hoc* selection of comparators and lack of control of confounders (Sutton 1959). More importantly Dean himself initially did not support the water fluoridation studies in the 1940s due to concerns about fluoride toxicity (Dean 1944).

In the UK the government and advocates of water fluoridation continue to promote 1 ppm as the 'optimum concentration' of fluoride despite the fact that in Canada it is 0.7 ppm, in Eire it is 0.8 ppm, in Hong Kong it is 0.5 ppm and in January 2011 the US department of Health and Human Services announced it was consulting on reducing the maximum level of fluoride that can be added to water to 0.7 ppm. The 'optimum concentration' of fluoride is only relevant in discussions about prevention of dental caries as there is no physiological requirement for fluoride in the human body. More importantly, there is no scientifically agreed safe limit of fluoride ingestion, although scientists generally agree that maximum limits are between 0.05 mg/kg per day and 0.07 mg/kg per day – i.e. measured by body weight and referred to as dosage – with substantially lower limits for small children and babies. It is argued that water fluoridation does not lead to levels in excess of these limits based on reference dose (achieved by using average weight for an adult or child), but as water fluoridation delivers fluoride by level of concentration in water, the individual dosage of fluoride received by the individual is not controlled and depends on how much water is used for bathing, drinking, cooking, etc., and also on individual weight. Also, water is not the only source of fluoride as we now use fluoridated toothpaste and other dental products, and fluoride is also found in tea, processed food (from fluoridated countries), pesticides (residues of which remain on food), etc. (SCHER 2011).

Increasingly, researchers are finding that adults and children are exceeding these agreed limits, contributing to a rapid rise in dental fluorosis – the first sign of fluoride toxicity (Levy *et al.* 1995, Marshall *et al.* 2004, Erdal and Buchanan 2005). In 1991, the *Centers for Disease Control* (CDC) in the USA measured fluoride levels and found that where water is fluoridated between 0.7 and 1.2 ppm overall fluoride, total fluoride intake for adults was between 1.58 and 6.6 mg per day while for children it was between 0.9 and 3.6 mg per day (Figure 2 and Table 1), and that there was at least a six-fold variation just from water consumption alone (US Public Health Services 1991). A recent analysis of fluoride exposure in the UK National Diet and Nutrition Survey suggests that we should be concerned about increasing fluoride levels with over 15% of people consuming more than 5 mg of fluoride a day (the figure that World Health Organisation (WHO 2001) considers adverse health effects can occur in adults). For children, the situation is more worrying. In the USA, a study in Iowa found that 90% of 3-month-olds consumed over their recommended upper limits, with some babies ingesting over 6 mg of fluoride daily, above what the Environmental Protection Agency and the WHO say is safe to avoid crippling skeletal fluorosis (Levy *et al.* 2001). Since the 1980s, numerous studies have

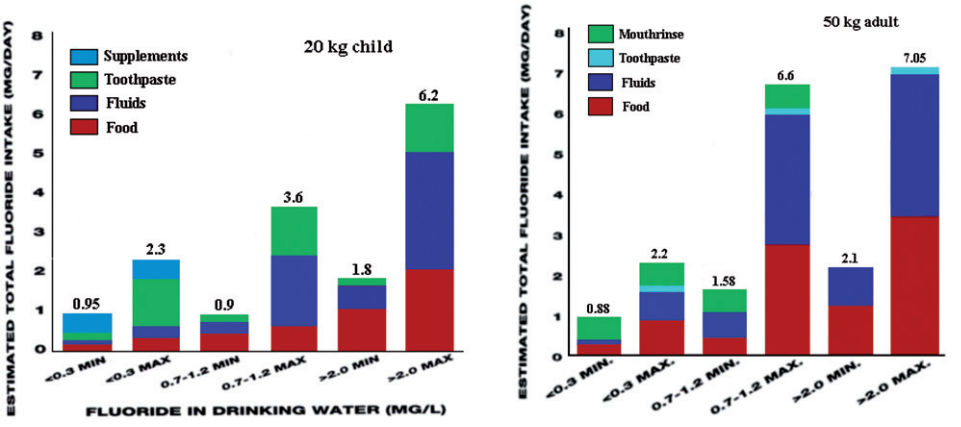


Figure 2. Estimated fluoride intake for children and adults in the USA.
Source: US Public Health Services (1991). Accessed from http://www.fluoridation.com/fluoride.htm on 12 Oct 2010.
Note: Water fluoride from $<0.3$ ppm to $>2.0$ ppm; equivalent to milligram per litre.

highlighted over-consumption of fluoride and recommend using fluoride-free water for reconstituting infant formula (Clarkson 2000, Bazalef *et al.* 2001, Siew *et al.* 2009) – advice also given by the American Paediatric Society and the American Dental Association. Advocates of water fluoridation often cite the fact that water fluoridation cannot give rise to acute fluoride poisoning as the concentration of fluoride is low and is therefore safe. However, fluoride accumulates in the body over time – approximately 45% of the fluoride ingested or absorbed is retained by the body – and fluoride is deposited in the bones and other tissues. It is this accumulation that gives rise to dental fluorosis.

Proponents of water fluoridation repeatedly stress that the only negative effect of water fluoridation is dental fluorosis and that this is only of the very mild kind leading to white spots or whitening of the teeth and is essentially a cosmetic issue (BFS 2003, see also National Fluoride Information Centre and BDA websites). In addition, research on dental fluorosis has shown that the whitening of the teeth found in very mild fluorosis is considered as a cosmetic benefit by those who experience it (Browne *et al.* 2006). However, the World Health Organisation defines dental fluorosis as a health problem and it is widely accepted that dental fluorosis is a manifestation of systemic toxicity – including by the UK Government (Hayman 1999, US Agency for Toxic Substances and Disease Registry 2003, Cuhna-Cruz and Nadanovsky 2005). The York Review and Australian National Health and Medical Research Council (NHMRC) systematic reviews concur that fluorosis of aesthetic concern will be in the range 7–14% and that water fluoridation is significantly associated with high levels of dental fluorosis. In fluoridated areas, some 35% of children have dental fluorosis, with studies in Newcastle, UK and Hong Kong identifying levels of 54%, and studies have shown that between 2% and 7% of children have mild-to-severe brown staining (Tabari *et al.* 2000, Irish Forum on Fluoridation 2002, Levy *et al.* 2006, Lo and Wong 2006). Most recently, the US Department of Health and Human Services has proposed substantially reducing the maximum permissible level of fluoride from 1.2 to 0.7 ppm as a direct response to

research showing that 41% of 12–15 year-olds experience dental fluorosis reaching over 50% in fluoridated areas (Beltran and Barker 2007, Associated Press 2011). Interestingly, while reducing fluoride concentrations to 0.5 ppm in Hong Kong reduced the rate of fluorosis, it did not lead to any change in rates of dental decay (Lo and Wong 2006). The fact that moderate-to-severe fluorosis can lead to damage to the tooth enamel and contribute to dental caries has been known since the earliest studies of water fluoridation in the 1930s (Connett *et al*. 2010). While measurement of the degree of dental fluorosis is undertaken using a number of different classification methods, making comparison difficult, it is clear that in areas with substantial water fluoridation, fluorosis has become more widespread. More recent research suggests that for young people with brown staining, the condition is viewed as worse than having dental decay (Spencer *et al*. 1996, Williams *et al*. 2006, Lawson *et al*. 2008) and a study in Southern Ireland found that fluorosis gave rise to embarrassment among girls more than boys (Browne *et al*. 2006).

Dental fluorosis is a result of excessive fluoride consumption in early childhood and there is substantial research evidence to support that excess fluoride consumption is widespread in young children and babies in fluoridated areas (Clarkson 2000, Bazalef *et al*. 2001, Levy *et al*. 2001, Siew *et al*. 2009). In their recent review of water fluoridation, the EU (European Union) Scientific Committee on Health and Environmental Risks highlight that young children are likely to exceed the upper tolerable limits for fluoride consumption in areas with water fluoridation greater than 0.8 ppm and using fluoride toothpaste, although the estimates of ingestion are probably underestimated as they are based on ingestion from food and beverages in non-fluoridated areas (SCHER 2011). Warren *et al*. (2009) have highlighted the complexity of quantifying fluoride intake in areas where there is widespread water fluoridation and increased availability of fluoride-containing products. They argue that '...it is doubtful that parents or clinicians could adequately track children's fluoride intake and compare it with the recommended level, rendering the concept of an 'optimal' or target intake relatively moot' (p. 114). Their conclusion supports Burt and Eklund's (2005) view that the term optimal fluoride intake be dropped from common usage and Ismail and Hasson (2008) also argue that 'We believe that dentists should dismiss the misconception that there is a balance between dental caries and fluorosis, because patients can accrue the benefits of topical fluorides without developing fluorosis and without systemic intake' (p. 1465). The inability to control individual dose renders the notion of an 'optimum concentration' obsolete.

It is suggested, especially by opponents of water fluoridation, that long-term ingestion of fluoride can lead to other more long-term health problems, but here the evidence is also unclear (McDonagh *et al*. 2000, NRC 2006). Proponents of water fluoridation generally dismiss these concerns arguing that there is no evidence of harmful effects,[4] yet the picture is more complex than this and after over four years, the move to reduce levels of fluoride added to drinking water in the USA acknowledges the importance of concerns raised in this report. The National Research Council (NRC) review published in 2006 was a wide-ranging examination of over 1000 studies on the effects of fluoride in water. While the focus of the review was to examine the effects of water fluoridation at between 2 and 4 ppm, it identified a number of studies demonstrating health effects at levels lower than 2 ppm, including effects to the central nervous system and brain at 1.8 ppm, brittle bones at $<1.2$ ppm, thyroid gland at 1 ppm, and result in osteosarcoma in young boys at

1 ppm, and hypersensitivity reactions of 1% at 0.25 ppm (NRC 2006). The NRC review authors were particularly concerned about thyroid effects and there are strong clinical grounds that support this view as fluoride has been used to treat hyperthyroidism in the past at levels of 2.5 mg per day. Research studies have demonstrated long-term communal effects of low-dose fluoride affecting thyroid function and epidemiological studies and animal studies support the hypothesis that fluoride affects the thyroid gland (Susheela *et al*. 2005, Idris and Wihardja 2008). Osteosarcoma has also been highlighted as an area where there is evidence of problems requiring further research by SCHER based on analyses by Bassin *et al*. (2006).



### *Effectiveness and safety of water fluoridation*

In the UK, proponents continually compare fluoridated Birmingham with non-fluoridated Manchester, where the level of dental decay is higher (Table 1), and refer to the USA where over 70% of the population drink fluoridated water. This, it is claimed, shows fluoridation works and is safe. However, water fluoridation is not necessarily associated with low caries rates. If you compare fluoridated Birmingham (the 8th most deprived area in England) with unfluoridated Islington (the 3rd most deprived), it is the latter that has lower levels of dental decay (see Table 1). In 2000, the US Surgeon General called dental caries the number one childhood disease – despite 50 years of water fluoridation. Claims that reductions in dental decay in countries such as the USA and Ireland are the result of water fluoridation are not

Table 1. Comparing DMFT rates in non-fluoridated and fluoridated PCT areas.

| PCT | Fluoridation status | DMFT in 5-year-olds[a] | DMFT in 12-year-olds[b] | Index of multiple deprivation[c] |
|---|---|---|---|---|
| City and Hackney | Non-fluoridated | 1.27 | 0.46 | 44.91 (3rd most deprived) |
| Manchester | Non-fluoridated | 2.39 | 0.68[d] | 44.50 (4th most deprived) |
| Heart of Birmingham | Fluoridated | 1.67 | 0.36 | 38.67 (10th most deprived) |
| Islington | Non-fluoridated | 1.52 | 0.41 | 38.96 (8th most deprived) |
| Blackpool | Non-fluoridated | 1.47 | 1.27 | 37.66 (12th most deprived) |
| Newcastle | Fluoridated | 1.43 | 0.87 | 31.36 (30th most deprived) |
| Gateshead | Fluoridated | 1.13 | 0.90 | 29.0 (41st most deprived |
| Coventry | Fluoridated | 1.15 | 0.42 | 27.85 (47th most deprived) |
| Bristol | Non-fluoridated | 1.45 | 0.77 | 27.76 (65th most deprived) |
| Peterborough | Fluoridated | 1.6 | 0.34 | 24.29 (69th most deprived) |
| Southampton | Non-fluoridated | 1.13 | 0.69 | 24.31 (71st most deprived) |
| Medway | Non-fluoridated | 0.63 | 0.32 | 19.95 (93rd most deprived) |
| Solihull | Fluoridated | 0.72 | 0.67 | 16.16 (114th most deprived) |

Notes: Average for Bristol North and Bristol South and West PCTs.
[a]NHS Dental Epidemiology Programme for England Oral Health Survey of 5-year-old Children 2007/2008.
[b]BASCD Survey Report 2004/2005, *The dental caries experience of 11-year-old children in Great Britain (2004/2005)*.
[c]Local Authority Index of Multiple Deprivation 2007.
[d]Central Manchester PCT.

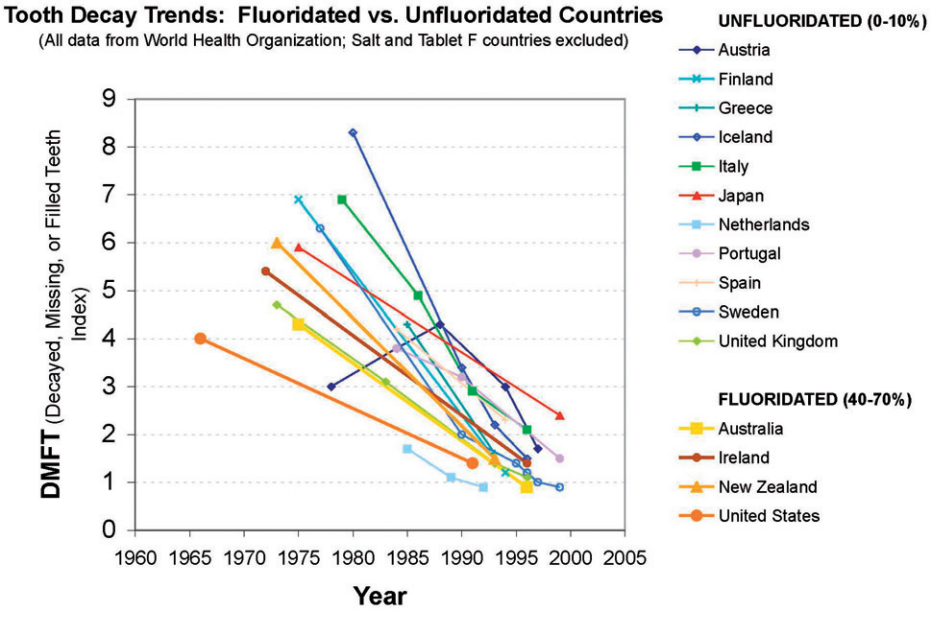


Figure 3. Comparison of decline in dental caries in fluoridated and non-fluoridated countries. Source: Neurath, C. (personal communication) amended from Neurath C., 2005. Tooth decay trends for 12 year olds in non-fluoridated and fluoridated countries. *Fluoride*, 38 (4), 324–325.

supported by the evidence. Comparing countries with fluoridated water and those without (Figure 3) shows little variation in the rate of reduction or level of dental caries.

There have been two major systematic reviews of water fluoridation. The most important of these is the York Review, commissioned by the UK Government in 1999 to provide a comprehensive review of the evidence on water fluoridation but which only served to create further controversy (McDonagh *et al*. 2000, Cheng *et al*. 2007). Proponents of water fluoridation point to two conclusions of the review – that the average reduction in tooth decay in the studies examined was 2.25 decayed, missing or filled teeth (DMFT) and that an average 14.6% more children would be caries-free. In fact, the range of DMFT was from 0.5 to 4.4 and in terms of the extent of children with caries, it varied from an increase of 5% to a decrease of 64% (14.6% being the mean) (McDonagh *et al*. 2000). Proponents also claim that the York Review found that water fluoridation reduced inequalities in dental caries for 5- and 12-year-olds (BFS 2003). Opponents of fluoridation stress the fact that the York Review found no evidence of safety or effectiveness, although they generally highlight the fact that the review team found that studies of the effect of water fluoridation on dental caries were of only moderate quality and of limited quantity. The review authors concluded that, in fact, the studies examined did not give clear evidence of caries reduction and that 'The research evidence is of insufficient quality to allow confident statements about other potential harms or whether there is an impact on social inequalities' (p. xiv). The review found that the research evidence on the effect of water fluoridation on dental caries was rather mixed, with many studies being old (undertaken when dental decay rates were substantially higher), observational, or insufficiently controlling for confounding factors (such as the use

of fluoridated toothpaste), and concluded that there was a need for good quality studies to assess the effect of and safety of fluoridation. While a subsequent review by a committee established by the UK Medical Research Council (MRC 2002) on behalf of the government to examine the recommendations of the York Review suggested that the authors had been too cautious, the findings of the York Review were corroborated by a later systematic review in Australia (NHMRC 2007). The UK MRC committee did reiterate the urgent need for more research to examine the effects of water fluoridation, especially on children (MRC 2002). Similarly the NRC review, while not systematic, provides a thorough analysis of the impact of fluorides in water (including animal and human studies and toxicological research) highlighting many problem areas. It draws similar conclusions to the York Review – that there are probable detrimental health impacts from fluorides, that not enough is yet known about the wider health impacts of fluoridation and further research is needed (NRC 2006).

The effectiveness of water fluoridation is linked to how fluoride is thought to improve the resistance of the tooth enamel to caries. Initially, it was thought that fluoride primarily worked by strengthening the enamel of the developing tooth and that ingestion of fluoride was necessary. However, it is now widely accepted that fluoride helps prevent dental decay by topical means – direct action on the teeth predominantly after eruption (CDC 1999, Warren and Levy 2003). Fluoride contributes to, but is not necessary for, the remineralisation process in the enamel of the tooth surface and high levels of fluoride kill the bacteria that create the acids that damage the tooth enamel. While many years ago much caries appeared on the flat surfaces of the teeth, today most dental caries occurs mainly in the pits and fissures in the molars where fluoride has little preventive effect (White 1993, Pinkham 1999). Examination of children in the Iowa fluoride study supports the view that water fluoridation has no impact on pit and fissure lesions. In the cohort study, an examination of non-cavitated smooth surface and non-cavitated pit and fissure lesions and their progression to cavitated lesions was not associated with fluoride, socio-economic status or beverages. Generally, smooth surface lesions rarely progressed to cavitated lesions, while pit and fissure lesions led to frank decay or fillings (Warren *et al*. 2006). In a study comparing tooth enamel erosion in a number of locations, it was found that dentine exposure (where the enamel had been breached) was higher in fluoridated Birmingham than in non-fluoridated Liverpool and London and that socio-economic status was strongly associated with levels of dental erosion (Al-Dlaigan *et al*. 2001). The evidence suggests that most early childhood caries are related to feeding habits such as 'bottle caries' or high-levels of streptococcus mutans – neither of which are helped by water fluoridation, and that sugar remains the most direct cause of dental caries in older children (Cogulu *et al*. 2008). As Pizzo *et al*. (2007) have concluded, water fluoridation is only '. . . a relevant public health measure in populations where oral hygiene conditions are poor, lifestyle results in a high caries incidence, and access to a well-functioning oral health care system is limited' (p. 192).

## Using evidence: scientific concerns versus blinkered belief?

The Department of Health in the UK (with the support of Dental and Public Health Professions) continues to promote water fluoridation as a safe and effective solution

to tackling dental caries. Opponents continue to challenge all proposals for extending water fluoridation – not just in the UK but also the USA, Australia, New Zealand and Canada claiming that fluoridation is not effective, will lead to substantial health problems and subject people to enforced medication. Yet, many researchers have also raised doubts about the relevance of water fluoridation as an effective intervention. The York Review authors were very cautious about drawing conclusions about the contribution of water fluoridation above and beyond other factors that lead to improved oral health. There are also growing concerns about fluoride ingestion and the long-term effects of fluoride in the body. The two major systematic reviews and the NRC report raised several questions about potential health problems that have not yet been sufficiently researched and despite consistent calls for further research, this has not been done. As Professor John Doull (NRC chair) notes '... many of these questions [about the effects of fluoride] are unsettled and we have much less information than we should, considering how long this has been going on' (Quoted in Fagin 2007, pp. 80–81).

The findings of the York Review remain contentious and the government, British Dental Association and the BFS, all use the review to state that water fluoridation is effective, safe and reduces inequalities. In 2008, the Chief Dental Officer (CDO) issued guidance to NHS authorities and stated that the York Review confirmed 'that there were benefits to oral health from fluoridation and that was no evidence of any risk to health' (para 5). A British Dental Association briefing to MPs in 2003 (before the debate on water fluoridation amendments to the Water Bill) stated that 'All robust scientific research shows that fluoridation is still considered safe and effective'. More recently, in summing up a review of the evidence for the consultation on water fluoridation for SCSHA, the Director of Public Health stated '... that water fluoridation prevents dental disease and is likely to reduce inequalities in dental health. There is no reason to believe that water fluoridation causes any harm to health' (SCSHA 2009, para 50). Conversely, anti-fluoridation groups use the York Review to state that fluoridation is unsafe and ineffective. These statements are at odds with those made by the chair of the Review committee, Professor Trevor Sheldon, who wrote an open letter (2001) to specifically refute the misuse of the York Review findings by both pro- and anti-fluoridation groups. Cheng *et al*. (2007) argue that studies that have highlighted safety concerns cannot simply be discounted by the government and NHS.

The key problem is that there is a lack of good quality evidence of both effectiveness and harm. This is a point consistently raised in reviews of water fluoridation (McDonagh *et al*. 2000, NRC 2006, NHMRC 2007, SCHER 2011). However, both proponents and opponents of water fluoridation continue to selectively draw on the evidence to support a view that water fluoridation is effective and safe or that it is harmful. A more balanced reading of the evidence is that water fluoridation has little effect, is a poor delivery mechanism, causes dental fluorosis and may have other long-term harmful health effects. It certainly does not meet Skrabanek's (1994) standard for evidential proof of benefit. Fluoride is effective when applied topically but is potentially harmful when ingested (SCHER 2011).

Therefore, what guides public health policy making on water fluoridation? Is it simply enough to say that water fluoridation is undertaken in other countries and is supported by many health organisations (e.g. US Public Health Service, US Centers for Disease Control, American Dental Association, British Dental Association) and therefore should be extended? The Gold Effect Framework highlights how belief

and conviction can structure both the application and interpretation of evidence and certainly the US Public Health Service endorsement came long before research evidence was available. This is also illustrated by the fact that in many of the studies highlighting excess fluoride consumption in very young children, cited above, authors (who are mainly dental public health specialists) focus on the fact that if fluoride concentration levels are reduced due to avoiding excess fluoride intake, children may not receive the 'optimum concentration' for protecting teeth (e.g. Siew *et al*. 2009). Acceptance that 1 ppm of fluoride is necessary for caries prevention is rarely questioned in the dental and public health literature. Even where levels of fluoride have been reduced this is always couched in terms of the important benefits of fluoridation (Associated Press 2011). Proponents appear to be working within a predetermined mindset as suggested by the 'Gold Effect'. One problem may be that as fluoride has clear topical preventive benefits and widespread use in oral health and hygiene products, it makes it more difficult for any criticism of fluoride to be aired. Such a view would tend to have a reinforcing effect on policy promoting fluoridation. In addition, in the history of water fluoridation, key commercial stakeholders have tried to influence the debate. Sugar corporations funded research to find a solution to tooth decay which did not lead to reductions in sugar intake. Similarly the phosphate industry in the USA found the switch from the use of sodium fluoride to hexafluorosilicic acid provided a useful way of disposing of dangerous fluoride waste following changes to laws regulating air pollution in 1975 (Connett *et al*. 2010).

With further expansion of water fluoridation planned in England it is time, however, to seriously question whether we should continue a policy where fundamental questions about efficacy and safety are unresolved. We need to stop '*believing*' that water fluoridation works and apply good evidence in policy and practice (Wilson and Sheldon 2006, Cheng *et al*. 2007). Public health policy in this area seems to be captured by history and a central belief that 1 ppm of fluoride is considered a standard with more concern registered about fluoride deficiency. However, as there are alternative and more effective ways of delivering fluoride topically, policy makers need to be more critical in their application and interpretation of the evidence. One major concern is that there has been no systematic recording of the effects of water fluoridation in the UK or other countries – research that SCHER is currently proposing should be adopted EU wide (SCHER 2011). For example, it is not possible to determine the extent of dental fluorosis in fluoridated areas in the UK as these data are not collected, despite the fact that the UK undertakes annual national oral health surveys in children. More detailed recording of other problems is not done either. As Cheng *et al*. (2007) note, the identification of additional cases of osteosarcoma, for example, cannot be done based on standard data collection as the numbers are so low.

Current processes for implementation leave decisions about effectiveness and safety very much within the purview of the NHS and, as the guidance currently stands, NHS views about safety and effectiveness remain the key determinants of whether to implement schemes, irrespective of the outcome of local consultations. Repeated claims in Department of Health guidance, ministerial statements and in NHS consultation and policy documents that there is 'no evidence' of harm and that water fluoridation is safe and effective and reduces dental health inequalities is clearly not an accurate assessment of the current evidence (CDO 2008, SCSHA 2009). There is no doubt that when applied topically to the surface of the tooth, fluoride is beneficial in preventing dental caries (Marinho *et al*. 2003). However,

there are substantial questions of safety and effect when fluoride is ingested. The causes of dental caries are varied and depend on individual behaviour, diet and access to good oral health care. Dental caries also affects specific groups of children and in many places in the UK, such as Southampton, the majority of children have no dental caries. Given the unknown balance between benefit and harm in relation to water fluoridation, we should perhaps be more cautious in pushing forward with further schemes and focus more on developing good oral health strategies that target support for those children and their families who experience worst dental health. There is good evidence that when delivered well such schemes are highly effective (Marinho *et al*. 2003). Changing key long-standing public health policies is difficult but we should heed Professor Gold himself who suggested the key problem is that 'Every complex problem has at least one simple, intuitive, and well presented wrong solution' (quoted in Kundt 2008).



## Notes

1. There are current proposals to introduce water fluoridation in a number of areas in England including Southampton, Bristol, parts of Yorkshire and the North West. In Southampton, 72% of those consulted opposed the scheme but the SCSHA still voted to go ahead with the scheme on the basis that they felt health benefits outweighed all other considerations. Recent proposals by the new coalition government about substantial NHS reorganisation and the abolition of SHAs and local PCTs and far-reaching changes to the organisation and delivery of public health services in England mean that the current legislation will need to be changed (Secretary of State for Health 2010).
2. Proponents of water fluoridation refer to the 'optimum dose'. However, technically this is not a dose as would normally be understood as a dose of medicine as it refers to levels of fluoride in water. Therefore in this paper it is referred to as the 'opitmal concentration'.
3. There are a number of different measurement scales for assessing the degree of dental fluorosis. The first was developed by Dean which provides six levels – no fluorosis, questionable, very mild, mild, moderate and severe fluorosis and is based on assessment of the front two teeth. The Dean index is widely used and supported by WHO. In addition, there are other classification and description methods of assessment including the TF index based on histologically identified changes in enamel, the Tooth Surface Index of Fluorosis and Young's description of location, colour and hypoplasia. See Browne *et al*. (2005) for a full discussion.
4. Guidance to the NHS by the Chief Dental Officer in 2008, statements by Ministers in Parliament and papers produced by SCSHA for the consultation consistently state that there is *no evidence* of harmful effects.

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