FLUORIDE **Vol.5, No.2, Winter 1999**

WATERSHED

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Dobson's fluoride review said to have 'potential for criminal behaviour'

FRANK DOBSON ignored our call for a public inquiry, which would have examined the science and the legal and constitutional implications of water fluoridation. Instead, he commissioned an 'independent, systematic review' of the science alone. This review, funded by the NHS, is being carried out by the NHS Centre for Reviews and Dissemination at York University. The final report is expected in February. If the NHS CRD reports that water fluoridation is safe and effective, the Government intends to "legally oblige" water companies to fluoridate when requested.

The protocol for the review excludes all animal studies, all biochemistry, all mathematical models; it also excludes all studies on the effects of fluoride from any source other than water fluoridation. There are two panels - Advisory and Review (the latter is the 'business end'). Advisory panel members may suggest papers for inclusion. Both panels met in July to agree the original protocol and will meet to agree the draft final report. Advisory panel members include the director of the Cochrane Institute, an NHS official, dental professors, the editor of Evidence-based Dentistry (distributed with the British Dental Journal), the chief executive of Water UK, a professor of social medicine, a representative of Help for Health Trust and the President and two Vice Presidents of NPWA. (The last three attend in their private capacities; they are not mandated by NPWA to represent the Association).

The review panel includes five NHS CRD staff at York and two sub-contracted dental public health lecturers from Cardiff University. (One of the latter took part in a New Zealand 'review' in the early 1990s that found water fluoridation 'safe and effective'). A request that an international expert opposed to water fluoridation be appointed to the review panel was rejected.

An exchange of emails between Andreas Schuld of Parents of Fluoride Poisoned Children, Vancouver, Canada and the NHS CRD at York took place in September. Mr Schuld submitted more than 100 scientific references on the effects of fluoride on the thyroid gland. This resulted in a further narrowing of the already-narrow protocol and the exclusion of his references. Mr Schuld wrote three times to every member of both panels, finally informing them that all the correspondence between himself and York would be posted on his website so that people around the world could see how this UK review is being conducted.

Extracts from Parents of Fluoride Poisoned Children Newsletter, (reproduced on p.2 with their kind permission), clearly illustrate the need to address the dangers of fluoride ingestion from ALL sources - and not just from water fluoridation - as a matter of great urgency.

Please copy this Watershed to your local newspaper editors, radio stations, councillors, your MP, the Chairmen of your water company and your CHC, your GP and your dentist. Ask your MP to inform the new Health Secretary and the Minister for Public Health that, despite several authoritative warnings, before any attempt is made to increase public exposure, existing fluoride levels in people should be determined. The 'systematic review' excludes any consideration of fluoride ingestion from any source other than artificial water fluoridation.

NPWA asserts that this is a gross abuse of science, an affront to Parliament and the people and a serious misuse of public funds.

INTERNATIONAL INTEREST IN YORK FLUORIDE REVIEW

In September, Andreas Schuld, Head of Parents of Fluoride Poisoned Children wrote to the York review on the effects of fluoride on the thyroid gland. More than 100 references were submitted for consideration. The following is a series of emails between York and Andreas - the first is from Dr Matthew Bradley in response to the submission.

Dr Bradley:

Thanks for your comments Andreas. You have highlighted a number of important points that we had not taken into account. We will review the terminology in light of your comments and hopefully post a revised version on the web in the near future. Many thanks again. Matthew

Andreas:

Hello Matthew, Would this mean a widening of criteria, or a narrowing? Andreas

Dr Bradley:
Subject: Good question

Thanks for the message Andreas. **I am afraid that, as you may have guessed, it means a narrowing of the criteria.**

However, we have indicated that as such this review will require information from other sources before a policy decision should be reached. We are aware that some members of the Pure Water Association intend to collect this additional information and prepare it for submission at the time when our review will be released. Many thanks, Matthew

[*What* 'information from other sources'? This costly exercise, purporting to be a 'systematic [*"unchallengeable"*— Dobson] review' of the science, specifically excludes information vital to a proper outcome. - Ed.].

PFPC wrote: Sept. 22,1999:

Hi Matthew; please let me know when your protocol has been updated. Thanks, Andreas.

Date: Fri. 24 Sep 1999 15:22:22
From: Fluoride Project To: PFPC -

Both the Protocol and database of relevant studies will be updated at the beginning of next month.

[The protocol was actually 'updated' - *narrowed* - on 27 September, as a result of Andreas' submissions. - Ed.].

Andreas wrote THREE open letters to every member of both panels, finally informing them that the entire correspondence would be posted on his website so that people around the world could see how the UK is conducting this "review".

The York NHS CRD website is on - www.york.ac.uk/inst/fluorid/crd.htm

Members who do not have personal access to the internet can visit this site via a computer at their public library. We regret that it is not possible to copy this lengthy material and references to individuals.

We are pleased to print the following from Parents of Fluoride Poisoned Children with their kind permission.

PFPC NEWSLETTER
No.l, November 1, 1999
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1) Welcome and Introduction.
2) "One a day".
3) How do fluorides interfere with thyroid hormones? (Brief overview).
4) History of fluoride-iodine antagonism,
5) What do thyroid hormones do?

1) WELCOME

Hello everyone. Welcome to the first edition of our newsletter. The main purpose of this newsletter is to shed light on issues surrounding fluoride and the effects on thyroid function and related disorders.

We have learned that the major iodine deficient areas of the world are identical to endemic fluorosis areas. We learned that in some endemic fluorosis areas hypothyroidism as a result of iodine deficiency affects over 50% of all children.

We learned that at least one child dies every minute due to complications associated with this "fluorine-induced" iodine deficiency, and that this is a problem affecting almost a third of the population, perhaps more.

Because ALL studies that we looked at, which measured both the fluorine and iodine levels, showed that iodine levels are reduced directly corresponding to increasing F- intake, the obvious conclusion must be that it is the excessive fluoride intake in those areas which is the primary cause of this iodine deficiency and all related consequences. **These findings clearly take this matter out of the dental domain and make it a health issue of great global concern, demanding all of our attention as a global community. The urgency cannot be over-emphasised.** Fluoride poisoning IS thyroid dysfunction.

Some news: as a result of the York submissions, enthusiasm has been created in our organisation, which hosts a few artists involved in all aspects of the entertainment industry. This is due to the quite obvious attempt by a renowned institution to approach a review from an entirely unscientific position.

Their highly visible attempt to reclassify a halogen (described in high school textbooks) as something new, and re-writing a protocol to avoid submitted information - in direct contradiction to stated minutes - and hiding behind a "review process", shows how utterly twisted things have become.

It's going to change. It must change. The truth has become self-evident. We are doing what we can with our means. We all must act. What can you do? The best to all. Andreas Schuld, Head, Parents of Fluoride Poisoned Children Vancouver. BC, Canada.

2) "ONE A DAY" by Bob Johannsen

Every day millions of Americans take a small pill to compensate for a lack of thyroid hormone (iodine deficiency) in their bloodstream, which causes a condition called hypothyroidism. Hypothyroidism is related to over 150 diseases.

The drug of choice for the treatment of this thyroid disorder is synthetic levothyroxine sodium. (Physicians and scientists may use different scientific names for levothyroxine, such as L-thyroxine, thyroxine, and T4; however, all of these terms refer to the same chemical.).

Checking the prescribed drugs in the United States during 1996, we found that 36,000,000 prescriptions of synthetic levothyroxine were filled. The majority of patients taking levothyroxine have a permanent form of hypothyroidism and will take one pill, every day, for the rest of their lives. In iodine-induced, as well as fluorine-induced hypothyroidism, thyroid function may revert to normal with iodine or fluoride restriction alone. Once on levothyroxine, the thyroid gets "lazy" and fails to function properly, causing life-long dependence on the "pill once a day",

At current fluoride intake levels - being much higher than the dose given once daily specifically to reduce thyroid activity - **the solution should be obvious.**

REDUCE ALL FLUORIDE INTAKE.

STOP WATER FLUORIDATION.

ELIMINATE FLUORIDE IN TOOTHPASTE.

LABEL FOODS.

CONTROL INDUSTRY EMISSIONS.

Think about it.

3) HOW DO FLUORIDES INTERFERE WITH THYROID FUNCTION? (BRIEF TSH OVERVIEW).

For the purpose of this brief article we will concentrate on the thyroid-stimulating-hormone

(TSH), for here the mechanisms of fluoride's effects can be most easily identified and verified.

(The fact that fluorides have been used as treatment, specifically to reduce thyroid function, has been mentioned numerous times in the papers and articles sent to the York review, and can be verified with the references supplied. See also article No.4 below).

The process of producing thyroid hormones is controlled by a "feedback mechanism". The process starts in the hypothalamus, a portion of the brain. It releases TRH (thyrotropin releasing hormone) which, in turn, stimulates the pituitary gland, another part of the brain, to secrete the thyroid stimulating hormone (TSH). TSH stimulates the thyroid gland to produce the thyroid hormones T4 and T3, which are released into the bloodstream. (As the name implies. T4 carries 4 iodine molecules, T3 carries 3). T4 and T3 circulate in the blood to all the organs of the body, including teeth and brain. When the pituitary gland senses a drop in thyroid hormone levels in the blood, it releases more TSH to stimulate the thyroid gland, and the cycle of thyroid hormone production continues. If the pituitary gland detects too much thyroid hormone in the blood, it will decrease production of TSH, which then, in turn, reduces the production of thyroid hormone in the normal thyroid gland.

In addition to stimulating the thyroid gland to make thyroid hormone, TSH causes the thyroid gland to grow. As stated above, if there is not enough thyroid hormone in the body, the pituitary gland will increase production of TSH in an attempt to stimulate the thyroid gland to make more thyroid hormone. When patients are hypothyroid due to iodine deficiency, their thyroid gland cannot make enough thyroid hormones, and the pituitary gland continues to produce TSH. Because of this continued elevation of TSH, the thyroid gland may enlarge. This enlarged thyroid gland is called a goitre.

The leading cause of goitres worldwide is a lack of iodine in the system. Thyroid hormone contains molecules of iodine, and. when there is not enough iodine, goitre and/or hypothyroidism can develop. Iodine deficiency is rarely the cause of goitres in N. America because iodine is added to salt and other foods. The common cause of goitre/hypo-thyroidism in America is thought to be an increase in thyroid stimulating hormone (TSH) in response to a defect in normal hormone synthesis within the thyroid gland. TSH hormone is responsible for activating the many G proteins in the body. G proteins function essentially as "off" and "on" switches for

cellular signalling, with TSH as the power source, if you will. **Without TSH, G proteins are inactive.**

Fluorides behave like TSH in their ability to activate those off/on switches. **They are now known as universal G protein activators, which means they can activate all G proteins in the body.** However, signal production is not controlled by the normal thyroid feedback system. As a result, the thyroid gland function as well as feed-back mechanism are seriously disturbed. Not only can fluorides activate the TSH receptor to cause profound excitation or inhibition, they can cause desen-sitisation of the TSH receptor, thereby causing hypothyroidism, for now the signal is inhibited and thyroid hormone production depressed.

Some labs we have contacted have reported indication of hypo-thyroidism in 90% of blood tests. Many on/off switches are responsive to fluoride as activators and sometimes even prefer them to the natural activator, TSH.

Here are some links for more Info on G proteins:

G PROTEINS IN MEDICINE.

The New England Journal of Medicine, January 19, 1995 -Vol. 332, No.3, 186-187

http://www.neim.org/content/1995/0332/0003/0186.asp

FIGURE: HOW G PROTEINS WORK (On/Off)

http://www.neim.org/contenl/figs/1995/0332/0003/0186.asp?section=Fl

SOME DISEASES CAUSED BY G PROTEIN COUPLED RECEPTORS

http://www.gcrdb.uthscsa.edu/GCRs-disease.html

RECEPTOR LISTING:

http://www.le.ac.uk/csn/e56/GPCR-listing.html

G COUPLED RECEPTORS POINT MUTATIONS DATABASE

http://tinygrap.uit.no/GRAP/ligrecsub.html

LIGANDS OF G PROTEIN COUPLED RECEPTORS

http://www.gcrdb.uthscsa.edu/GCR_Ligand.html

SUSA REVIEW (1999)

"Heterotrimeric G proteins as fluoride targets in bone (Review)." Int J Mol Med 3(2): 115-126 (1999)

http://www.ncbi.nlm.nih.gov/htbinpost/Entrez/query?uid=9917518&form=6&db=m&Dopt=b

Strunecka, A; Patocka, J -

"Aluminofluoride complexes: new phosphate analogues for laboratory investigations and potential danger for living organisms."

http://www.cadvision.com/fluoride/brain3.htm

Thyrotropin-Receptor Mutations and Thyroid Dysfunction. The New England Journal of Medicine, January 19, 1995. Vol. 332, No. 3.

http://www.neim.org/content/1995/0332/0003/0183.asp

Defs: G-PROTEINS

http://www.graylab.ac.uk/cgi-bin/omd?G+proteins&action=Search+QMD

G-PROTEIN COUPLED RECEPTOR

http://www.graylab.ac.uk/cgi-bin/omd?G+proteincoupled+receptor

GTP BINDING RECEPTOR

http://www.graylab.ac.uk/cgi-bin/omd?GTP+binding+protein

4) BRIEF HISTORY OF FLUORIDE - IODINE

In 1854 Maumené feeds sodium fluoride to a dog and causes a goitre to appear. He is the first to consider fluorides as a cause of goitre.

Pighini (1923) is able to cause goitres in rats, dogs and chickens.

Goldemberg (1926, 1930) is the first to take advantage of the iodine-fluoride antagonism and begins to use fluorides to cure Basedow's disease (Graves' Disease).

May (1935, 1937) follows suit. In 1950 May publishes his findings that addition of thyroxine (T4) raised the iodine level in the blood, while the addition of fluorides lowered the iodine level in blood.

Litzka (1937) discusses the mode of action of fluorides in treating patients with Graves' disease: fluoride antagonises thyroid hormone effects on liver metabolism.

Wilson and DeEds (1940) report that dental fluorosis is a result of the synergistic action of fluoride and the thyroid. Results are "strikingly clear cut".

Wilson (1941) reports in the *Lancet* on his findings that mottling of teeth is prevalent in the same areas in the UK which had been previously prevalent with goitre.

The 1944 editorial in the **Journal of the American Dental Association** (JADA) states:

"We do know that the use of drinking water containing as little as 1.2 to 3ppm of fluorine will cause such developmental disturbances in bones as osteo-sclerosis, spondylosis and osteopetrosis, as well as goitre".

Steyn (1948, 1955) finds that fluoride has a thyrostatic effect. He investigates the incidence of endemic goitre (fluorine-induced) in die North Western Cape Province in South Africa and reports that his findings closely agree with the above JADA editorial.

Wespi (1954) finds mottled teeth (dental fluorosis) together with goitres in Italy.

Korrodi, Wegmann, Galetti and Held (1955) also verify a fluoride-iodine antagonism, **proclaiming that the fluoride ion pushes out the iodine in the thyroid gland.**

Galetti et al (1957) treats hyper-thyroid patients with fluoride, and documents a significant reduction in protein-bound iodine, as well as an overall reduction of iodine and a reduction of iodine uptake by the thyroid gland.

Jentzer (1959) further shows reduced iodine levels under the influence of fluorides.

In 1960 Gordinoff and Minder describe the results of experiments with radioactive iodine (1131) which show that fluorides remove an iodine atom. Effects were dose-responsive, meaning the higher the fluoride intake the lower the iodine measurements.

Steyn writes in 1962 that drinking water containing as little as 1 to 2 ppm of F- can cause serious disturbances of general health and especially in normal thyroid gland function and in the normal processes of calcium-phosphate metabolism (parathyroid function).

In 1963 G**orlitzer von Mundy** reports on the [then] current knowledge gained from experiments with I131 as to how the effects of the enzyme responsible for the T4 to T3 conversion we're inhibited if a fluorine ion was absorbed before the conversion was supposed to happen.

In 1969 **Siddiqui** shows small visible goitres in persons 14 to 17 years of age in India to be connected directly to high fluoride concentrations in drinking water.

Willems et al (1972) document that sodium fluoride blocks thyroid hormone secretion.

Also in 1972 **Day and Powell-Jackson** study 648 people in 13 mountainous regions in Nepal where the iodine content in the water was low and find a close relationship between fluoride intake and the incidence of goitre.

Bobek and Kahl (1976) document that rats on 1.0 mg fluoride daily from drinking water had significantly lowered T4, T3, and free thyroxine index in plasma.

In 1978 **George Waldbott** writes that in most cases of poisoning from fluoridated water in which he had occasion to study the action of the thyroid gland, its function was low. He cites a case of a 33-year-old male who exhibited typical manifestations of pre-skeletal fluorosis and a basal metabolism rate of -22, indicative of hypothyroidism. Within three months after the man ceased consuming fluoridated water, the thyroid function had returned to normal (BMR=0) In addition, **Waldbott** writes that "simultaneously, other symptoms associated with low grade fluoride poisoning - including excessive thirst, headaches, blurred vision, arthritis in shoulders, elbows, knees, and gastrointestinal disturbances -also disappeared." [He did not know that many of the symptoms he ascribed to low-grade fluoride poisoning would be likewise con-sidered symptoms of hypo-thyroidism some 20 years later.].

Hillman et al (1979) find that cattle afflicted with fluorosis developed hypothyroidism. anaemia, and eosinophilia of leucocytes. [The latter two also now commonly associated with hypothyroidism. Anaemias are diagnosed in 20-60% of patients with hypothyroidism.]

Sidora (1983) finds iodine deficiency and adaptive amplification of the hypophyseal-thyroid system, not ensuring an absolute compensation in the citizens using drinking water with an "enhanced" fluorine content as compared to a "decreased" one, accompanied by an augmented incidence of functional disturbance.

Bachinskii et al (1985) document how fluorides at 2.3 ppm in water cause tension of function of the pituitary-thyroid system that is expressed in elevated TSH production, a decrease in the T3 concentration and more intense absorption of radioactive iodine by the thyroid. The results lead to a conclusion that excess of fluorine in drinking water was a risk factor of more rapid development of thyroid pathology.

Tokar' and others (1989), in a study on workers exposed to fluorides, write that changes in the pituitary-thyroid axis were seen without co-existing clinical manifestations of hypo- or hyper-thyroidism, and that those changes were caused by disorders of the regulatory chain and fluorine impact on thyroid hormones' metabolism **at the level of target cells.**

Lin Fa-Fu et al (1991) report that a low iodine intake coupled with "high" (0.88ppm) fluoride intake exacerbates the central nervous lesions and the somatic developmental disturbance of iodine deficiency.

Tezelmann et al (1994) report that fluoride, by increasing the intracellular cAMP concentration, causes desensitisation of the thyroid stimulating hormone receptor (TSHR). No specific thyroid factor(s) other than increased levels of cAMP arerequired for desensitisation.

Balabolkin et al (1995) study the thyroid and immune statuses in workers continuously exposed to fluorine. The examinees with euthyroid condition had immune disorders with an allergic tendency (increased number of B-lymphocytes, immuno-globulins A). In workers with sub-clinical hypothyrosis (T3 reduced in 51%), the immune alterations were more evident, T-lymphocytes count rose, but their functional activity declined, indicating impaired co-operation of immunocytes as a result of imperfect control under low concentrations of T3.

Zhao et al (1998) does an extensive study on mice receiving several fluoride-iodine combinations in addition to basal diet. He finds that iodine and fluorine have mutually inter-acting effects on both goitre and fluorosis in the experimental mice.

Jooste et al (1999) show that goitre occurrence in two iodine-sufficient areas in Africa seem to be due to high fluoride water levels.

In 1999, as a result of research into molecular biology ("the art of cloning") **there are hundreds upon hundreds of studies available documenting the actions of fluorides upon G proteins,** the 'On' and 'Off switches involved in cellular signal transmission.

Fluorides become known as the universal G-protein activator. Although there have been numerous studies before showing that fluorides mimic TSH, the thyroid stimulating hormone, it can now be documented in deep detail, for it is known that G proteins are normally abso-lutely dependent on TSH and are inactive without it. Too much TSH (or fluoride) in the system will cause hypothyroidism and other severe thyroid disorders. Because high TSH levels in the system are indicative of hypothyroidism, TSH tests at birth are implemented on a global basis, to avoid severe brain damage and stunted growth resulting from congenital hypothyroidism.

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5) WHAT DO THYROID HORMONES DO?

Bodily mechanism — effects of thyroid hormone.

CARBOHYDRATE METABOLISM:
increases up-take of glucose by the cells, enhances glycolysis, gluconeo-genesis, increases rate of absorp-tion from the gastrointestinal tract, increases insulin secretion.

FAT METABOLISM:
increases lipid mobilisation from the fat tissue, increases free fatty acid concentrations in the plasma, accelerates the oxidation of free fatty acids.

PLASMA AND LIVER FATS:
decreases the quantity of cholesterol, phospholipids, and triglycerides in the plasma, increases the concentrations of cholesterol, phospholipids, and triglycerides in the liver.

VITAMIN METABOLISM:
increases the need for vitamins.

BASAL METABOLIC RATE:
increases basal metabolic rate to 60-100% of normal or, if thyroid hormone is not produced, basal metabolic rate falls to half of normal.

BODY WEIGHT:
increased thyroid hormone decreases body weight; decreased production Increases weight.

CARDIOVASCULAR SYSTEM:
Blood flow and cardiac output – increased metabolism in the tissues causes greater than normal quantities of metabolic end products to be released from tissues causing vasodilation (state of increased calibre of the blood vessels) and increased blood flow, cardiac output increases as a consequence of increased blood flow.

HEART RATE:
affects excitability of the heart which increases heart rate

Strength of Heartbeat **increases due to increased enzy-matic activity; when thyroid hormone is produced in excess heart muscle strength becomes depressed because of excessive protein catabolism.**

BLOOD VOLUME:
increases secondary to vasodilation.

RESPIRATION:
increases utilisation of oxygen and formation of carbon dioxide

GASTROINTESTINAL TRACT:
increases the rate of secretion of digestive juices and motility of the GI tract.

MUSCLES:
slight increases in thyroid hormone make muscles react with vigour, although excessive hormone production causes muscle weakness due to protein catabolism

OTHER ENDOCRINE GLANDS:
increases the rate of secretion of most other endocrine glands (pancreas, parathyroid, adrenals), increases metabolic activities related to bone.

So, you can see how comprehensively body functions are affected if the thyroid gland does not work properly.

FLUORIDE STOPS IT FROM WORKING PROPERLY.

Parents of Fluoride Poisoned Children,
Vancouver, BC. Canada

Extracts from the THIRD OPEN LETTER TO REVIEW AND ADVISORY PANEL MEMBERS from ANDREAS SCHULD, October 12,1999.

To: Review Panel Members: Dr Matthew Bradley, Marijke Van Gestel, Prof. Jos Kleijnen, Kate Misso, Penny Whiting, Dr. Ivor Chestnutt, Dr Elizabeth Treasure. Advisory Panel Members: Chair: Prof. Trevor Sheldon. The Earl Baldwin of Bewdley, Dr. lain Chalmers, Dr. Sheila Gibson, Ms. Sarah Gorin, Prof. MA Lennon, Dr. Peter Mansfield, Prof. JJ Murray, Mr. Jerry Read, Dr. Derek Richards, Prof. George Davey Smith, Ms. Pamela Taylor.

Dear Panel members: Today I glanced over the new document list, as posted on the website. I would like to ask you the following questions:

1) The study by Jooste (1998) has been included, showing higher goitre prevalence in two towns (28% and 29%) with **higher** than "optimal" levels of fluoride in the water. The areas were iodine sufficient. This is welcomed. However, I have supplied the Review with the complete paper by Lin et al documenting dental fluorosis rates of 16% in 7-14 year old children, at merely 0.34 ppm in the drinking water, when iodine intake was deficient. Why has this study not been included?

2) How will you deal with many other studies showing endemic fluorosis in iodine deficient areas, at levels way below 1ppm, mostly below 0.5ppm? I have not seen them included in your literature list, although searches with your published keywords should have easily brought them up.[Three examples given].

3) How will you address the fact that dental fluorosis is found at high levels (20%) even in cities having NO fluoride in the water supply, with even higher occurrence in children of high socio-economic level? [Examples].

4) How will you deal with those studies which do not identify the gender or ethnic origin of the children studied, other factors also greatly influencing caries occurrence, as well as hormone function and activity? *For example, in the group of Caucasian and Asian 12-14 year-olds there's usually the highest difference in caries incidence between girls and boys (girls having the higher DMF-value). So, if in a group of 100 "children" you have 80 girls and 20 boys there are more "carious teeth per 100 **children**" than in a group of 20 girls and 80 boys. [Examples].The greater the dose of fluorine that is taken up by the organism in the period of development, the longer is the retardation of dental age. The later the tooth erupts, the more severe is the fluorosis. How will all this knowledge be incorporated in the Review?

5) How will you deal with the different DMF indices used over the years? In older surveys you count all teeth that are decayed or missing or filled, [but] a tooth filled and again decayed counts **twice**. If in later surveys a tooth counts only once, no matter if it is already filled and decayed again, you'll find an "obvious" reduction in caries incidence. (Investigations into definitions of the DMF-Index will bring to light that it has changed several times over the years)

6) You have included one study involving parathyroid glands, by Spira: 560. Spira L. Fluorosis – toxicity: incidence of dystrophies in organs regulated by parathyroid glands. J Hyg 1944;43(Sept):402-408. Why are no other studies included? It is well known that, a) hyperparathy-roidism is closely associated with skeletal fluorosis and b) when parathyroid glands are removed in subjects, same mineral effects occur as can be observed in dental fluorosis patients. (Avecedo, 1996; Chardinetal, 1998). Hyperthyroidism is closely associated with chronic hypothyroidism, in rats (Paloyan et al,1997) as well as humans. Hyperparathyroidism is ten times more frequent in thyroid patients than expected in a general medical population and is especially prevalent in patients with **goitre**. (Stoffer, 1982).

7) You have included a study from San Luis Patosi, an area with endemic fluorosis. [Named]. You have also been notified that the same area is endemic with iodine deficiency, at the approx. same percentage (>60%) as endemic fluorosis. Knowing that dental fluorosis could not occur without thyroid hormones, why is low iodine intake not considered a risk factor for developing dental fluorosis in this area, or any other area, for that matter?

10) While there are many studies included in the Review list investigating fluorosis in fluoridated areas, we could only find 6 included relating to thyroid disease. Two points are to be made: a) There are many more studies available. We hereby include 10 more (1-10), including the work by Steyn (1948, 1955) who investigated the incidence of endemic goitre (fluorine - induced) in the North Western Cape Province in South Africa and reported that his findings closely agree with the editorial published in the J. of the American Dental Association (JADA) in 1944 [JADA 31 (1944) pp. 1360-63]: "We do know that the use of drinking water containing as little as 1.2 to 3 ppm of fluorine will cause such developmental disturbances in bones as osteosclerosis, spondylosis and osteopetrosis, as well as **goitre**". What happened to that "knowledge"?

Again and again dentists and scientists with plenty of PhDs behind their names proclaim that, since water fluo-ridation has been used by tens of millions of people for over 50 years, any significant health risk would have become obvious, but none have appeared. This is clearly a fraudulent statement, **for these people are not looking for any other effects than dental fluorosis. If they were truly looking at thyroid disease, it could be observed all too easily.**

The number below my signature is the estimated number of children that have died as a result of fluorine-induced hypothyroidism, since we have first notified you of our research. When will you, as human beings, take steps to address this issue properly?

Sincerely,

Andreas Schuld.

18,720.

Councils angered by 'biased review'

On 27 September, the Steering Committee of North West Councils Against Fluoridation met to discuss the review being carried out at York. After examining the information posted on the NHS CRD website the members expressed serious misgivings, concluding unanimously that the review appears to have a built-in bias. They asked the Chairman, Cllr Noel Spendlove, to write to the Secretary of State for Health, accordingly. **The text of Cllr Spendlove's letter reads, in part**:

"Tessa Jowell says that the review is to determine if water fluoridation is safe and effective. We understand that the protocol set down is:

1) Primary studies (no reviews);

2) Use human subjects (no animal or mathematical models);

3) Consider fluoridation of public drinking water (no other sources).

"Medical evidence shows that the cumulative effects of fluoride cause damage to health. The World Health Organisation warned, in 1994, 'Dental and public health administrators should be aware of the total fluoride exposure in the population before introducing any additional fluoride programme for caries prevention' yet this is being excluded from evidence considered.

"We understood that the review was to be independent but looking at the Review Panel, we see that four members are NHS employees, plus two dentists… We understand a proposal by people opposed to fluoridation that statistician Rudolf Ziegelbecker be invited to join the panel was refused. The Steering Committee were unanimous that the results of this review cannot be considered unbiased."

NPWA PUBLIC APPEAL FOR £60K LEGAL FUND

The narrow protocol of the York review has resulted in the exclusion of hundreds of scientific studies from the "once and for all systematic review". This has deeply disturbed many of our members as well as scientists whose own work has been excluded. If the York NHS CRD reports that water fluoridation is "safe and effective'" the Government intends to "legally oblige" water companies to fluoridate.

Next year NPWA will be FORTY YEARS OLD.

For FOUR DECADES members have funded the campaign to protect UK drinking water supplies from fluoride pollution while successive Governments continue to give massive annual sums to the British Fluoridation Society to promote it! We have repeatedly written to the DoH expressing concerns about fluoride ingestion from all sources - to no avail.

In October, one of our barristers said that because the York review is publicly funded a judicial review should now be considered. This will examine whether the protocol for the 'systematic review' is adequately designed to provide full and proper advice to enable Government to protect the public from the adverse effects of fluoride ingestion. We assert that the protocol should take account of the total fluoride exposure of the population, as recommended in the Report of the Ministry of Health Mission to the US in the 1950s, the World Health Organisation in 1994 - and evidenced by the scientific literature.

The NPWA is launching a PUBLIC APPEAL for funds to take whatever legal action our legal advisers recommend and to increase the level of campaign activity. This may include some targeted advertising with information about the Campaign.

We agree with *PFPC* that: CHILDREN ARE BEING POISONED.

THIS MUST STOP.

All UK legislation permitting artificial water fluoridation MUST BE REPEALED.

If you can, PLEASE donate -and ask your family, friends and colleagues if they will, too. As always, Campaign expenditure will be kept to a minimum. The greater part of all receipts WILL GO INTO THE LEGAL FUND.

Thank you!

The million dollar question -
A few members, all of whom acknowledged their worries about the York review, asked: "What if the review finds that water fluoridation is a) ineffective and/or b) unsafe?"

Well, we'll have the Mother of all Celebrations, that's for sure!!

Such a decision will be an admission that nearly six million people have been (a) needlessly and/or (b) dangerously treated with fluoride for 35 years. This potentially opens the flood-gates to approximately six million lawsuits against the Government. Is such a decision likely, when the protocol for the review **excludes** addressing the total fluoride exposure of the population from all sources? Because of this exclusion, is it likely that the drive to fluoridate school milk will end? Will 'target' children still be dosed with F drops and tablets? Will it curb sales of fluoridated toothpaste? Will the DoH (and the BDA) admit that fluoride causes a great deal of ill health? Will lab facilities be set up to monitor fluoride levels in people? Will stricter monitoring of fluoride emissions result?

Because of the demonstrably inappropriate protocol, is proper public health policy on artificial water fluoridation a *likely* outcome?