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Paul Connett's misrepresentation of maternal F exposure study debunked

Posted on [March 5, 2018](#) | [1 Comment](#)



Title slide for Paul Connett's presentation to parliament

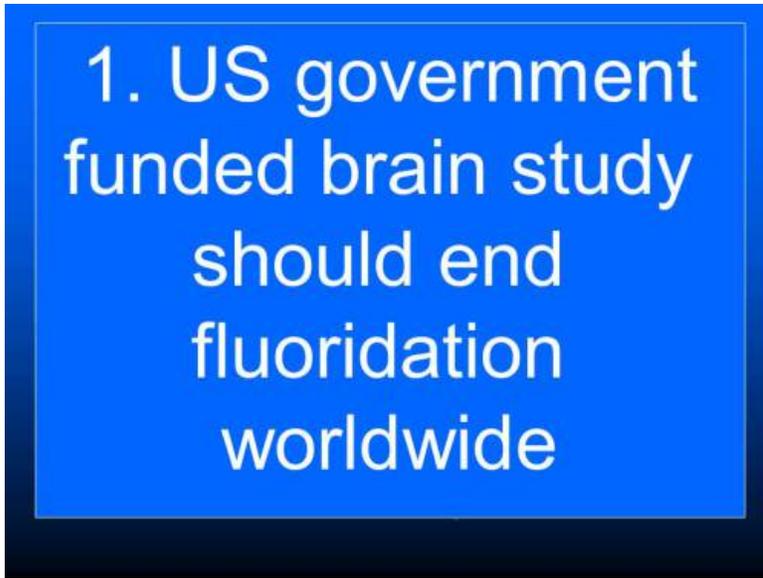
Anti-fluoride campaigners are misrepresenting a recent Mexican study claiming its findings should cause governments around the world to abandon community water fluoridation (CWF). Their claims are unwarranted because the study has a high degree of uncertainty. Activists are misrepresenting the accuracy of the studies findings. Because Mexico has areas of endemic fluorosis the study itself is not relevant to CWF.

Misrepresentation of the Mexican study was a central argument used by US anti-fluoride activist Paul Connett in his recent New Zealand speaking tour. This is shown in the Powerpoint presentation he prepared for his meeting at parliament buildings last month (see [Anti-fluoride activist commits "Death by PowerPoint"](#)).

It may have not been used in the end as only 3 MPs turned up. But, given his status in the anti-fluoride movement, this presentation will present the current strongest arguments against CWF. It is therefore worth critiquing his presentation whether it was given or not.

In this article, I will concentrate on Paul's presentation of the Mexican study and may deal with other arguments used in the presentation in later articles. The paper reporting the study is:

In Connett's mind, the study's results are so overwhelming we should immediately stop fluoridation throughout the world! This was the first and main argument he presented. His title slide and slide no. 10 introducing the study demonstrates the importance to him.



Slide No. 10 introducing Connett's presentation of the Bashash et al (2017) study.

I have critiqued this study in previous articles – readers can find them at:

[Fluoride, pregnancy and the IQ of offspring](#),
[Maternal urinary fluoride/IQ study – an update](#),
[Anti-fluoridation campaigners often use statistical significance to confirm bias](#) and
[Paul Connett “updates” NZ MPs about fluoride?](#)

Paul is clearly aware of these articles because he included a note in his presentation about them. I am honoured (it is the only comment in the presentation) and pleased he has made an effort to engage with my critique.

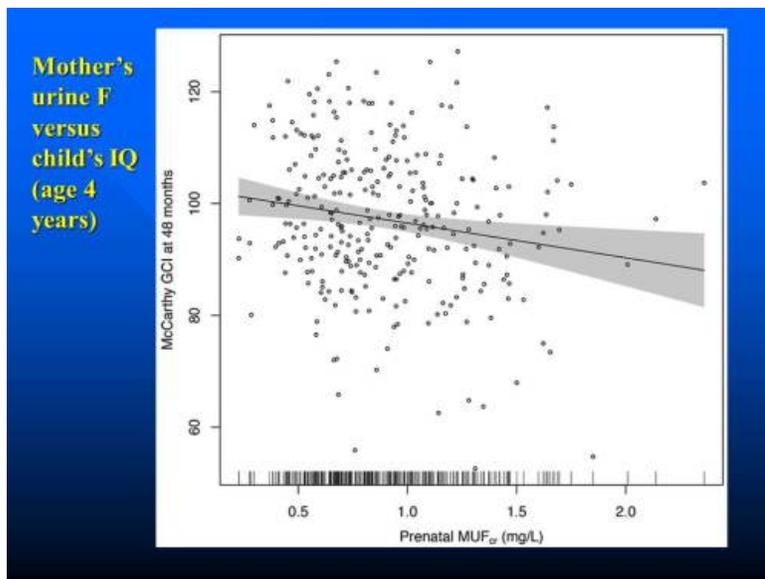
This is what he says:

“Ken Perrott and those who follow him will claim that the wide degree of scatter in the data means the findings of this study are unreliable. That is an incorrect interpretation of this graph and the study. The effect size is very large (decrease by 5-6 IQ points per 1 mg/L increase in urine F) and is highly statistically significant. The fact that urine F can only explain a small amount of the variation of IQ does not invalidate the finding. Rather, it is a reflection that there are many other factors that affect IQ, most of which are essentially random with respect to F exposure. For example, individual genetics plays a huge role in IQ (it explains 80% or more of variation in IQ), therefore it would not be possible for F to explain more than the small remaining portion of variation in IQ. Most studies of other developmental neurotoxins like Pb and Hg find very similar low correlation coefficients, yet there is no debate that their findings are valid.”

This comment provides me with a basis for a more detailed discussion of his use of the study.

The small amount of variance explained

Connett acknowledges my point that the observed relationship with urinary fluoride can explain only a very small amount of the variation in IQ – only 3%. A bit hard to deny considering the high degree of scatter in the data which is obvious even in the slides Connett uses:



Slide 20 where Connett reproduces Fig 2 from the Bashash et al. paper.

But he claims that this:

“does not invalidate the finding. Rather, it is a reflection that there are many other factors that affect IQ, most of which are essentially random with respect to F exposure.”

Here he is, of course, referring to his own “finding” or conclusion – not the authors.

Notice his assumptions:

- Other factors will be “*essentially random with respect to F exposure,*” and
- The observed relationship will not be changed by the inclusion of these other factors.

Those are huge assumptions. And they are wrong.

Here is a relevant example illustrating the danger of such assumptions – the association between ADHD prevalence and extent of fluoridation observed by [Malin & Till \(2015\)](#). Their association was able to explain between 22% and 31% of the variance in ADHD, depending on the specific data used. Far more than the 3% for the Bashash et al., (2017) study.

Yet, when other risk-modifying factors were included, in this case, mainly altitude, the significant association with fluoridation disappeared. A model including altitude, but not fluoridation, explained 46% of the variability in ADHD (see [Perrott 2017](#) and a number of [articles](#) in this blog).

In this case, the incidence of fluoridation was correlated with altitude – fluoridation was simply acting as a proxy for altitude in the Malin & Till (2015) association. So much for Connett’s assurance that other factors “*are essentially random with respect to F exposure.*”

Other studies have found an association between symptoms of fluorosis and cognitive deficiencies. [Choi et al., \(2015\)](#), for example, reported an association of child cognitive deficits with severe dental fluorosis, but not with water F concentration. But there is a relationship between fluoride exposure and fluorosis prevalence – ie. fluorosis is not random with respect to F exposure. If the health effects resulting from fluorosis are the prime cause of the cognitive deficiency, the inclusion of fluorosis incidence in the multiple regression could produce a model where there is a statistically significant association with fluorosis but not with fluoride exposure. That is, the urinary fluoride values could be simply acting as a proxy for fluorosis incidence.

A similar non-random association of premature births and low birth weight could occur because these problems do occur in areas of endemic fluorosis. These could be two of the health issues related to fluorosis but fluoride intake may not be the prime cause (see [Premature births a factor in cognitive deficits observed in areas of endemic fluorosis?](#)).

Connett is completely wrong to assume that other risk-modifying factors not considered in the Bashash study would necessarily be random with respect to fluoride exposure. And he is wrong to assume that inclusion of these factors would not change the association of child IQ with mothers’ urinary fluoride reported in the paper.

Notably, the Bashash et al (2017) study did not include any measure of fluorosis as a risk-modifying factor – despite the fact that Mexico has areas of endemic fluorosis. I believe its consideration of gestation period <39 weeks or >39 weeks was inadequate (the normal average period is 40 weeks). The cutoff point for birth weight (3.5 kg) was also high.

The size of the IQ effect

We only have the data in the Bashash et al., (2017) study to go with here and the associations they report are valid for that data. But what about the calculations Connett makes from the reported association.

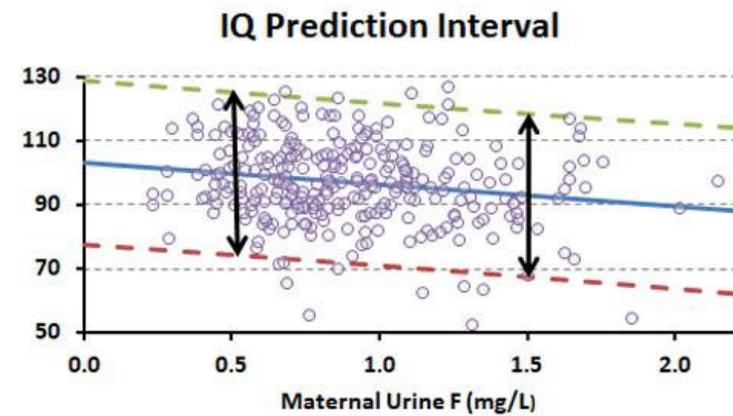
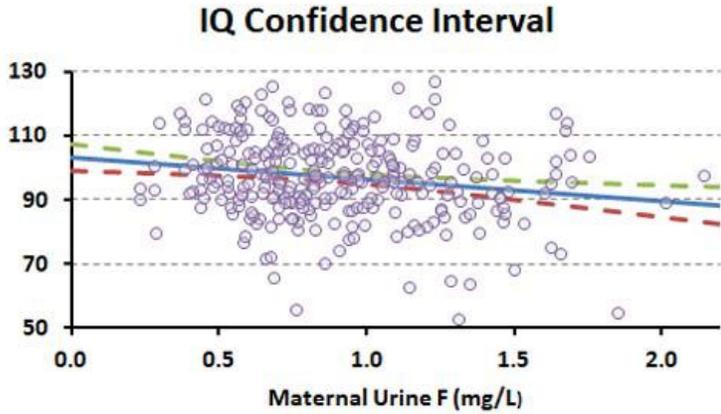
For example, Connett declares:

” The effect size is very large (decrease by 5-6 IQ points per 1 mg/L increase in urine F) and is highly statistically significant.”

Let’s test this claim – using the association represented in Fig 2 from Bashash, which is the figure Connett and other anti-fluoride activists are using (his slide 20 above).

Firstly, we need to calculate prediction intervals from the data (see [Confidence and prediction intervals for forecasted values](#)). The shaded region in the figure used by Connett (Fig 2 in Bashash et al., 2017) represents **the confidence interval – the region where there is a 95% probability that a best-fit line for the data lies**. The region for the **prediction intervals** is much larger and Connett may be confused because he has interpreted the confidence interval wrongly. Yet, the prediction intervals are the important measure when considering the effect size.

Here are my graphs for the confidence interval and the prediction interval using data I digitally extracted from the paper (see [Maternal urinary fluoride/IQ study – an update](#)).



Let’s consider the predicted values of “child IQ” for urinary F

concentrations of 0.5 and 1.5 mg/L.

Urine F (mg/L)	Predicted value	Lower	Higher
0.5	99.8	74.4	125.2
1.5	93.0	67.5	118.4

The prediction intervals are very large. This means the real value for “child IQ” at a urine F value of 0.5 mg/L has a 95% probability of being in the range 74.4 – 125.2. The corresponding range for a urine F concentration of 1.5 mg/L is 67.5 – 118.4. When Connett claims that an increase of 1 mg/mL in mother’s urinary F produces a drop of 5 – 6 IQ points he actually means **a drop of 5 – 6 ± 26 IQ points** which is not statistically significantly different to zero.

The best-fit line for the data may be statistically significant – but Connett is wrong to say this about his predicted effect of urinary F on child IQ. In fact, over the whole range of urinary F measured there is a 95% probability that IQ remains at 100.

Connett’s claim of a “highly statistically significant” effect size is completely false. If he had simply and objectively looked at the scatter in the data points he would not have made that mistake.

Comparing maternal urinary F levels to other countries

Connett makes an issue of the similarity of maternal urinary F levels found in this Mexcian study to levels found elsewhere. One is tempted to say – so what? After all, I showed above that his claim of a “highly statistically significant” drop in child IQ with increases in maternal urinary F is completely

wrong.

He does compare the urine F levels reported by Bashash et al., (2017) with some New Zealand data (Brough et al., 2015) and finds them to be very similar. Interestingly, Brough et al., (2015) reported their urinary F values as indicating fluoride intakes were inadequate for the women concerned. They certainly did not indicate toxicity.

The comparison does highlight for me one of the inadequacies in the Bashash (2017) paper – the inadequate measurements of urinary F. Whereas Brough et al., (2015) used the recommended 24-hr urine collection technique, the data used by Bashash et al (2017) relied on spot rather than 24 hr measurements. These spot measurements were only made once or twice during the pregnancy of these women.

Yes, these were the only F exposure measurements Bashash et al., (2017) had to work with but they are far from adequate.

Conclusions

Paul Connett, as a leader of the anti-fluoridation movement, is completely wrong about the Bashash et al., (2017) study. It will not lead to the end of community water fluoridation throughout the world – nor should it.

He has attempted to ignore, or downplay, the high scatter in the data and the low explanatory power of the relationship between children's IQ and maternal F exposure found in the study (only 3%). His denial that this relationship may disappear when other more important risk-modifying factors are included is also wrong – as other examples clearly show.

Connett's presentation of a size effect (5-6 IQ points with a 1 mg/L increase in F exposure) as "*highly statistically significant*" is also completely wrong. In fact, this size effect is more like **5 – 6 ± 26 IQ points** which is not significantly different to zero.

The misrepresentation of this study by Paul Connett and other anti-fluoridation activists demonstrates, once again, that their claims should never be accepted uncritically. This is just one more example of the way their ideological and commercial interests drive them to misrepresent scientific finding.

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One response to “Paul Connett’s misrepresentation of maternal F exposure study debunked”

1.  Steve Slott | [March 5, 2018 at 8:35 am](#) |

Connett misrepresents science?? Oh, come on....next you'll be telling us that the world isn't flat.

Steven D. Slott, DDS

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