

Subject: RE: Debate

Date: Thursday, 21 May 2009 at 23:56:40 British Summer Time

From: Michael Mew

To: [REDACTED]

CC: [REDACTED]

Dear [REDACTED], [Chair of BOS]

Thank you very much for your well considered answer. I was not expecting this to become such a popular conversation. With great respect I must take issue with a number of points in your email. I will seek to respond to these points but please could you send this to the editor of the BDJ, as I would prefer an open discussion.

There are certainly some gaps in our understanding, and if we want the truth then we must start with the aetiology. We have to review what we know and see if there is some sense to it all. And a debate would be an excellent way to start that.

A debate should be scientific, interesting, educational and fun.

On the separate issue, could we possibly discuss placing an advertisement in the BOS asking for an opponent? Why not one of you?

Very best wishes,

Mike

-----Original Message-----

From: [REDACTED] [[mailto:\[REDACTED\]](mailto:[REDACTED])]

Sent: 19 May 2009 22:50

To: Michael Mew

Cc: [REDACTED]

Subject: Debate

Dear Mike

A debate is an interesting idea. One challenge with this particular debate is that I feel that the hypothesis that malocclusion is caused by a mixture of genes and environment is fairly uncontroversial. The mainstream view of the aetiology of malocclusion surely is that it is indeed a mixture. This is mainly a statistical deduction and the result of the well known observations on the Mary Rose skulls and the plague pit skulls. This is what I teach all the undergrads at Bristol and my predecessors before me. Of course very little light has been shed in research as to what the environmental factors are (although there are well known hypotheses relating to airways, pollen, diet, tooth wear etc) and even less light has been shed on any successful intervention with a possible environmental factor apart for the very small effect from Linder Aronsen and his

adenoidectomy advocacy of 20 years ago. Even there, he was not attacking the environmental factors which might cause adenoidal enlargement. So I feel your summary of aetiology in the editorial is very mainstream in its broad thrust and I am not sure anyone would argue against it, although the truth of these well-known more specific hypotheses about airway etc is unknown.

When going on to remedies which are put forward as influencing the environment, of course I realise that you feel that what your father has always referred to as orthotropics is put forward as potentially influencing the environmental factors but given a whole day to present on this in Manchester a few years ago, John was equally at a loss to suggest a line of experimentation that might shed light on our ability to identify or influence an environmental factor. I clearly recall that he felt that some analysis of 3/4 face photos was likely to be the best source of evidence about the effect of treatment. Also I recall that he was unable to offer a series of cases of his own or suggest another clinician who was practising orthotropics who might have some cases which could be prospectively followed. He did mention Harry Orton who had died several years previously as someone who had used his Mew 1 appliance and I remember using that myself when I first met and spoke at length with your father in 1979 when I was working with Harry. As you know, that appliance aligned the arch before using a functional appliance of your choice including your father's design. As with the majority of clinicians I remain keen on functional appliances, but am not aware of any of them influencing the proposed environmental factors such as diet, breathing, pollen etc. We do all of course get some very impressive results sometimes when growth turns out to be favourable, but we know that in those cases we may well have seen that growth in the absence of treatment which is why controlled trials are so informative. There is little doubt that arch expansion can favourably influence nasal airflow and this is undergoing a renewed popularity of investigation, but whether this change in airflow lasts or is more than a side effect of tooth movement or influences future malocclusion is at present debatable.

So the problem with a debate on genes and environment in aetiology is that it is likely to consist of agreement that both are important, then the putting forward of some hypotheses about environmental factors on which we have little fact to chew over and then an amount of shoulder shrugging.

Regarding the separate issue of the hypothesis that orthotropics effects environmental factors, there are two hurdles to be overcome. Firstly in the 30 years in which I have heard John refer to it on many occasions I have not gained a useful working knowledge of what exactly it is other than the use of functional appliances, arch expansion and possibly some imprecisely defined orofacial exercises. It is fair to say that this is an obstacle to its adoption by another clinician. Secondly, it is only those who practice a technique who can test that technique. Many techniques have been compared e.g. fixed vs removable functional appliances, early vs later treatment of class 2, functional vs fixed appliances for class 2, orthodontics vs surgery. Other novel and at first sight rather unlikely treatment approaches such as reverse pull headgear, RME, all sorts of applications of TADS, self-ligation, have all found enthusiasts and then increasingly good scrutiny and quantified assessment. Even uncomfortable, complex and difficult appliances such as the Frankel which I myself used on a good number of cases in the 1970s found a significant following for a while. A challenge with orthotropics is the lack of adopters and therefore of cases to match and compare.

So although I love debate, I am not sure that aetiology is a fruitful source of difference of opinion. Regarding orthotropics as a potential influence on the environment, I think a lack of users and of comprehension of the proposed distinctive elements hinders the prospects of a further debate being useful at this stage.

Regarding the lack of hard evidence on environmental factors, I suspect that a problem is that changing a proposed influence or number of influences over a long term is difficult in an individual or a society and may be difficult ethically in the absence of sufficient reason to support the intervention. At least we are in good company - we don't yet know much about what causes some people to get osteoarthritis, but we do at least now have excellent hip replacements.

best wishes

██████████ [Chair of BOS]

--On 19 May 2009 21:20 +0100 Michael Mew <mikemew@gmail.com> wrote:

Dear ██████████ [Chair of BOS]

I hope that you have received the letter that I sent to you via the BOS. In this I ask if you could, as the Chairman of the BOS, ask if one of your members would like to stand against me in a debate. The hypothesis to be tested would be "Malocclusion is caused by the environment and modified by the genes".

I have over the last few months received several emails from the BOS on subjects such as consultant positions or 2nd opinions for cases, and was wondering if an email could be sent to all the BOS members on this subject. If between us we could write a few lines and send them out, it would then help me to organise such an event. It would be best to meet you but I believe that you are not based in London so that would depend on our movements in the near future.

It really would be a pleasure to meet up with you or even to discuss this by email.

Very best wishes,

Mike

PS Attached is the letter that I sent you and the editorial

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Version: 8.5.283 / Virus Database: 270.12.29/2114 - Release Date: 05/19/09

06:21:00