From: "Boris Nikolic (bgC3)" <[redacted]>
To: Jeffrey Epstein <jeevacation@gmail.com>
Subject: RE: FW: Autism and real science
Date: Sun, 06 Feb 2011 22:26:17 +0000

Agree 100% with you.
This time is irrelevant – next time might be something stupid that you never said. I do not want that this gets associated with you.

She is doing it to impress you and him.

The only way to stop is that you tell her to stop doing it.
You might say that you would like to focus your discussion with him on currency and that would prefer that the other topics do not distract.

Working like a dog here.

B

From: Jeffrey Epstein [mailto:jeevacation@gmail.com]
Sent: Sunday, February 06, 2011 3:25 AM
To: Boris Nikolic (bgC3)
Subject: Fwd: FW: Autism and real science

[redacted] forwarded this to me, As you know I think most of her thinking is goofy, The fact that she merely tells me about it , does not mean I have any interest, respect, agreement. she then sends a e mail to bill that she and i are having a conversation about it, I found strange. no need to mention me

---------- Forwarded message ----------
From: [redacted]>
Date: Sat, Feb 5, 2011 at 11:42 PM
Subject: FW: Autism and real science
To: "jeevacation@gmail.com" <jeevacation@gmail.com>

FYI

From: [redacted]
Sent: Saturday, February 05, 2011 8:39 PM
To: [redacted]
Cc: Boris Nikolic (bgC3)
Subject: Autism and real science

I know that autism has been coming up quite a lot lately, and I agree that it would be very unlikely that there is a relationship between vaccine administrations and any sort of static encephalopathy or regression. This is a line of conversation that JEE and I have been having recently, and since it might be of some interest to you – I will try to summarize some very creative and evidence-based thinking in this area.

Specifically, and most interestingly:

- **Influenza?** Maternal influenza infection prior to closure of neural tube seems to have a high correlation with autism and schizophrenia – verified via large Scandinavian pre-natal care data sets. If you really think about it though, it probably isn't just influenza; it is more likely hyperthermia (fever) related to and possibly linked to some form of infectious/post-infectious insult.

- **Hyperthermia?** An episode of hyperthermia is not uncommon during pregnancy. The consequences depend on the extent of temperature elevation, its duration, and the stage of development when it occurs. Mild exposures during the pre-implantation period and more severe exposures during embryonic and fetal development can result in prenatal death and abortion. Hyperthermia also causes a wide range of structural and functional defects. The central nervous system (CNS) is most at risk probably because it cannot compensate for the loss of prospective neurons by additional divisions by the surviving neuroblasts and it remains at risk at stages throughout pre- and postnatal life. In experimental animals the most common defects are of the neural tube, microphthalmia, cataract, and micrencephaly, with those without physical defects but with associated functional and behavioral problems. Nearly all these defects have been found in human epidemiological studies following (known) maternal fever or hyperthermia during pregnancy.

- **Valproic Acid and Thalidomide?** It is interesting too that the same outcomes can be seen in pregnant moms who take valproic acid (an antiepileptic, anti-depressant and pretty decent pain medication) or thalidomide (no longer available) during early pregnancy, and even throughout gestation. The usual physical deformities can be seen, but there is also a strong correlation with autism and other psychological issues that don't manifest until later – or present as a regression.

SO?

Autism spectrum disorders affect behaviors that emerge at ages when typically developing children become increasingly social and communicative, but many lines of evidence suggest that the underlying alterations in the brain occur long before the period when symptoms become obvious. Studies of the behavior of children in the first year of life demonstrate that symptoms are often detectable in the first 6 months. The environmental factors known to increase the risk of autism have critical periods of action during embryogenesis. Minor malformations that occur frequently in people with autism are known to arise in the same stages of development. Anomalies reported from histological studies of the brain are consistent with an early alteration of development. Congenital syndromes with high rates of autism include somatic that originate early in the first trimester. In addition, it is possible to duplicate a number of anatomic and behavioral features characteristic of human cases by exposing rat embryos to a teratogenic of valproic acid or thalidomide at the time of neural tube closure.

It is still true that the underlying brain injury that leads to autism has been difficult to identify. The diagnostic criteria of the disease are not readily associated with any brain region or system, nor are they mimicked by vascular accidents, tumors, or degenerative neurological diseases occurring in adults. There is a cool paper from the 1990s that provides some evidence that the disease originates by an injury at the time of closure of the neural tube. The human data suggest that the initiating lesion includes the motor cranial nerve nuclei. To test this hypothesis, the investigators first examined motor nuclei in the brainstem of a human autistic case. The autopsy brain exhibited near-complete absence of the facial nucleus and superior olive along with shortening of the brainstem between the trapezoid body and the inferior olive (very high-priced real estate). A similar deficit has been reported in Hoxa-1 gene knockout mice in which pattern formation of the hindbrain is disrupted during neurulation.

Alternatively, exposure to antimitotic agents just after neural tube closure could produce the observed pattern of deficits. Thus, the lesions observed in the autopsy case appear to match those predicted by the thalidomide cases in both time of origin and central nervous system (CNS) location. To produce similar brain lesions experimentally, the team exposed rat embryos to valproic acid, a second teratogen linked to autism. Animals received 350 mg/kg of valproic acid (VPA) on day 11.5 (the day of neural tube closure), day 12, or day 12.5 gestation. Each treatment significantly reduced the number of motor neurons counted in matched sections of the

earliest-forming motor nuclei (V, XII), and progressively later exposures affected the VIth and IIIrd cranial nerve nuclei. All treatments spared the facial nucleus, which forms still later (super interesting). Counts from the mesencephalic nucleus of trigeminal, the dorsal motor nucleus of the vagus, and the locus ceruleus were not affected by exposure to VPA, even though these nuclei form during the period when exposure occurred. Despite its effects on the motor nuclei, valproic acid exposure did not alter the further development of the brain in any obvious way. Treated animals were robust and had no external malformations. The autopsy data and experimental data from rats confirm that CNS injuries occurring during (or just after neural tube closure) can lead to a selective loss of neurons derived from the basal plate of the rhombencephalon. The results add two new lines of evidence that place the initiating injury for autism around the time of neural tube closure.

Whether it is an exogenous agent (ie, medication) given to mom during this critical period or an infection/fever, it seems pretty clear that the damage is done during the period when the neural tube is forming. Not post-natal. There also seems to be a role for VIP, although unclear if that is collateral or an independent process.

There are very few scientists studying this, but the best one is at Caltech. Go Beavers.

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