Scientific Findings Related to Origins of Autism

Exposures of infants to developmental toxins in doses greatly exceeding established safe levels: During the last half century or so, toxins that have become common in the environment have also come to be widely present in human milk.[1] Breast milk in contemporary developed countries now includes three neuro-developmental toxins at levels substantially exceeding governmentally-established thresholds for safety**:**

(a) dioxins (including their chemical relatives, **PCBs), in breast milk in concentrations exceeding the EPA's Reference Dose** (estimated reasonably safe dose, or RfD) **by scores to hundreds of times;**[2]

(b) PBDEs, in breast milk in concentrations normally three times but up to 40 times the EPA's RfD;[3] **and**

(c) mercury, typically in breast milk at four times the maximum allowed by U.S. law in bottled water, but often higher.[4]

All three of the above are present in infant formula in concentrations less than 2% as high, and usually less than 1% as high, as their concentrations in human milk**.**[5]

(For a capsule explanation of why there should be such a difference between toxins in human milk and those in infant formula or cows' milk, note the following: Two leading experts on toxins involved in child development (P. Grandjean and P.J. Landrigan) have stated that "Persistent lipophilic substances (which include dioxins, PCBs, PBDEs and methymercury[5a]) accumulate in maternal adipose tissue and are passed on to the infant via breast milk, resulting in infant exposure that exceeds the mother's own exposure by 100-fold on the basis of bodyweight."[5b] Cows and soybean fields are not exposed to urban and indoor air pollution, diets including (mercury-containing) seafood, and the top-of-the-food-chain bio-accumulations of toxins that are ingested by humans with typical Western diets.)

The above exposures take place while the infant brain is going through a period of very rapid development. (see this chart, from 6) According to EPA researchers, an organ is generally at its greatest vulnerability to environmental toxicants if exposure to the toxins occurs during development of that organ.[7] A 2013 study in the National Library of Medicine reported on converging findings from many studies supporting "*cerebellar* dysfunction…as a contributor to the autism phenotype."[8] Note (in the chart) the predominant development of the cerebellum taking place during the first year after birth.

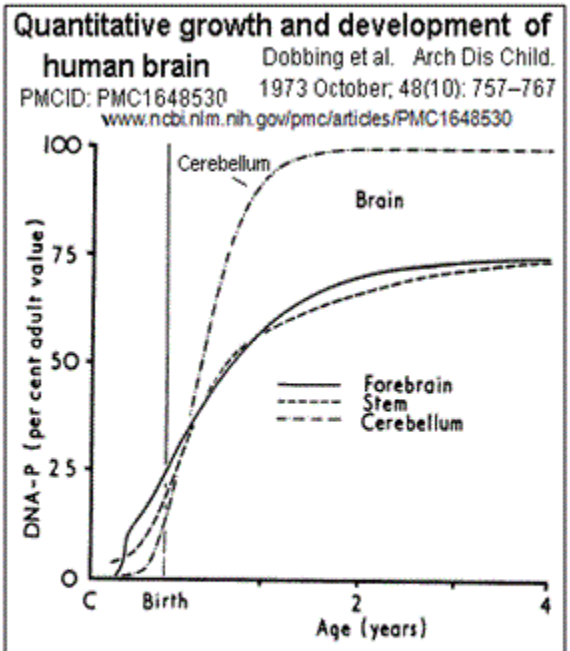

FIG. 11.—*Comparative values for total DNA-P, equivalent to total numbers of cells (see text), in 3 brain regions. Values shown in Fig. 5 for forebrain, cerebellum, and stem have been calculated as a percentage of adult value, and smooth lines drawn through the points.*

According to research supported by NIH grants, one of the forms of mercury that is widely present in human milk (due to its increasing presence in fish and seafood), methylmercury, is one of the "environmental agents with the property of killing neurons as they are born."[8a] A 2006 study determined that, of the three sources of infant mercury exposure, ingestion (breast milk), inhalation, and dermal exposure, the largest contribution was from breast milk, providing 96 to 99.6% of the total exposure.[9] Two authoritative studies have found mercury levels in infants breastfed for six months or a year to be two or three times as high as in bottle-fed infants, and a third study found compatible data.[10]

A characteristic of mercury that is especially relevant to autism is its known *latency of effect* following exposure**:** typically no symptoms appear until months or years after a harmful exposure to mercury.[10a] Latency of neurological effect that can extend into multiple years may be unique to mercury.[11] It is not hard to see a relationship between mercury's latency and the patterns of regression and late emergence in ASD.

An apparent effect (autism) with no known cause, and unexplained variations in beginnings and prevalence of autism:

-- A typical fourth child's risk of autism is half as high as that of a firstborn. The odds of being diagnosed with autism decrease from first to later children.[11a]

-- In a 2013 study dealing with eye contact of infants, the researchers found that the infants who were later diagnosed with ASD started out with normal eye contact as of a month or so after birth**;** then eye contact declined, and it continued steadily downward. This was seen to be a "derailment" of initially satisfactory development.[12]

-- The above-mentioned sudden downturns should be seen along with the unexplained patterns of "regression" and/or "late emergence" of symptoms of ASD after earlier normal development.

Could there be a link between
(1) an increasing childhood disorder that often arises in the form of mysterious downturns, and which has *no known cause*,
and
(2) increasing exposures to developmental toxins (including one with latent effects) **greatly exceeding established safe levels**, but **with *no recognized effects*?**

Proposed explanations for surprising outcomes without known causes:

-- **Decreasing risk of autism with later birth order.** Infants later in birth order are less likely to be breastfed,[14a] are breastfed for shorter periods on average,[14b] and the milk they receive has levels of developmental toxins that have been reduced as a result of excretion to earlier-born infants during

previous breastfeeding.[14c]

-- **Postnatal derailment of development of eye contact, or regression taking place later, following initial apparently-normal development.** As mentioned, two studies have found mercury levels in infants breastfed for six months or a year to be two or three times as high as in bottle-fed infants**,** and a third study found very large increases in mercury exposure via breastfeeding;[10] and mercury has been found to accumulate in the brain to levels seven times the levels in blood.[16] According to the U.S. Agency for Toxic Substances and Disease Registry, "the major effects (of methylmercury) that are seen across the studies include motor disturbances, such as *ataxia* and tremors, as well as signs of *sensory dysfunction, such as impaired vision.*"[(15)] According to an authoritative definition, "Ataxia is a lack of muscle coordination which may affect *speech, eye movements*,.. and other voluntary movements."[(15a)] In addition to its compatibility with causation of eye dysfunction, remember mercury's known latency of effect.[(10a)] This should be seen in relation to the derailment of initially normal development that has been found to take place in infants months after birth (and therefore months after initiation of breastfeeding). And *mercury's latency extending into multiple years is also consistent with the frequent regressive or late-emerging appearance of ASD symptoms,* appearing years after a surge of exposure that typically starts right after birth.

Autism-related effects of relatively commonly-occurring concentrations of these toxins:

(a) A 2011 study found that 4-year-olds with higher levels of PBDEs had a 160% increased risk of poor social competence.[21a] Also note the following indication of the effect of breastfeeding in determining those higher levels of PBDEs**:** the average total concentration of PBDEs in 4-year-old children was found in a study to be still nearly three times as high in breastfed children as in formula-fed children.[22]

(b) At least five published studies have found high levels of mercury in those diagnosed with autism.[23] The studies finding associations of autism with mercury levels less than twice the normal range should be seen together with the findings in multiple studies of doubling or tripling of infant mercury levels resulting from breastfeeding, taking place during the infant's period of rapid brain growth.[10]

(c) A major 2013 study (Roberts et al.), analyzing data from all over the U.S., found associations between autism incidence and variations in atmospheric pollution of various kinds widely present across the U.S., and the pollutant found to have the very highest correlation with autism was *diesel emissions*.[24] Note that diesel emissions contain various chemicals *including the above-mentioned toxins that infants ingest* (via breast milk) *in doses greatly exceeding established safe levels***:** dioxins, PCBs, PBDEs[(24a)] and mercury. Mercury sediment has been associated with proximity to high traffic locations, and all types of vehicles tested in a study were found to contribute to atmospheric mercury levels**;** but mercury in heavy-duty *diesel* emissions was found to be 5 to 28 times as high per mile as the mercury in light-duty gasoline vehicle emissions.[(24b)] Data from the EPA shows that diesel emissions are a leading source and (as of the most recent EPA data) a rapidly growing source of dioxin releases in the U.S.[(24d)]

The above leads to some summary points that might be related to each other**:**

1) Dioxins, PBDEs and mercury are all neurodevelopmental toxins contained in diesel emissions, which are inhaled by many mothers and infants. (see just above) Remember that, due to attraction of lipophilic substances to maternal adipose tissue, breastfed infants receive toxic *"exposure that exceeds the mother's own exposure by 100-fold."* (see Introduction)

2) The levels of those toxins in human milk are undisputedly in concentrations that typically far exceed established safe levels. (see introduction).

Is there a possible relationship between the above and the finding in a large, authoritative study that diesel emissions correlate well with autism incidence?[24] Or could there be a connection with the findings of studies in California of similar correlations between autism rates and residence near freeways?

Possibly related to the above**:** A highly-published scientist and Fellow of the American College of Nutrition (R. J. Shamberger) found, on the basis of data from all 50 states and 51 U.S. counties, that "exclusive breast-feeding shows a direct epidemiological relationship to autism," and also, *"the longer the duration of exclusive breast-feeding, the greater the correlation with autism."*[26] Another U.S. study and a U.K. study arrived at similar findings.[27]

……

The Roberts study (mentioned earlier) that found correlation between autism and approximate-time-of-birth exposure to diesel emissions was sometimes interpreted to mean that harm results from fetal exposure to toxins during gestation**;** but close reading of the study reveals nothing indicating that the harm was necessarily *prenatal*. The effects were at least as likely to have been *post*natal in origin, as indicated by (a) the typical100-fold postnatal increase in exposure to toxins such as are contained in diesel emissions, quoted above, (b) the fact that the bulk of the brain's growth takes place after birth, as indicated in the diagram above, and (c) the expert opinion that an organ is generally at its greatest vulnerability to environmental toxicants if exposure to the toxins occurs *during* development of that organ.[7] A much more complete explanation of why developmental harm is quite likely to result from postnatal exposures can be found at www.autism-research.net/postnatal-effects.htm.

The Shamberger study mentioned above found a dose-response relationship, which is important in establishing causation. A dose-response relationship also exists in the case of declining autism risk with later birth order that was described earlier, given the decline in exposure to breastfeeding toxins with later birth order.

So we have the unexpected close geographic correlations of autism with breastfeeding, *plus* the unexplained outcomes in which autism rates have been found to be surprisingly high or low (earlier- or later-born children)**;** and we have seen a key mechanism of social development (eye contact) proceed to decline after initial carefully-assessed *postnatal normalcy*. The above all lead to important questions**:**

Q #1: Given the following:

a) the presence of three different serious neuro-developmental toxins in typical breast milk, each usually far exceeding recognized safe levels and levels in infant formula;

b) these toxic exposures taking place at an extremely vulnerable time for development of the infant brain, and

c) the toxins all being present in the particular type of pollution that was found in a major 2013 study to correlate best with autism prevalence,

-- is there any reason why those exposures could not be a reasonable explanation for all of the unexpected outcomes mentioned?

Q #2: Are there any developmental toxins known to widely reach infants in doses well in excess of a recognized safe level (e.g., EPA's RfD), **aside from toxins that are ingested by means of breast milk? If so, which ones are those?**

Note that nobody seems to be able to answer the above questions, including the three U.S. physician's associations that promote breastfeeding and the entire science team at the organization, Autism Speaks.

. . . . . .

Other considerations: If breastfeeding is found to be a pathway of harmful effects on infant development, something can be done to rapidly reduce infants' exposures to it. Reduction of pollutants in the environment and in the bodies of mothers is something that can only be worked towards, seeking reductions that would require decades to achieve, if they could ever be achieved**;** but thousands more children would be impaired for life in the meantime, or continuously into the future. On the other hand, most parents could promptly discontinue the type of feeding that is channeling concentrations of developmental toxins to their infants at the most vulnerable stage of the infants' lives.

Of all the disorders that are said by the U.S. Surgeon General to be reduced by breastfeeding, all but one has actually substantially *increased* in the U.S. since breastfeeding greatly increased, after 1971. In the case of that one disorder (alleged to be reduced by breastfeeding) that did not increase, it also did not decline following major increases in breastfeeding.[28] Various studies have found desirable effects to be *associated* with breastfeeding (always with known confounders present), but over 50 scientific studies have found breastfeeding to be associated with *worse* health outcomes.[29] Among those was the study that was apparently the only study on effects of breastfeeding on childhood obesity that was randomized, which is the recognized best way to avoid effects of the confounders that often cause false conclusions in typical studies.

For the U.S. generation born in the mid-20th century, breastfeeding was unusual.[30] That generation did not

have the unexplained childhood epidemics and major increases (diabetes, asthma, allergies, obesity, ADHD and autism) that have become prevalent since then. In the decades since breastfeeding greatly increased after 1971, there have been not only *major increases but also lows and mid-levels of multiple childhood disorders that have correlated closely with preceding increases, lows and mid-levels of breastfeeding rates*.[28]

Various U.S. doctors' associations (American Academy of Pediatrics, American Academy of Family Physicians, American Congress of Obstetricians and Gynecologists) and WHO promote breastfeeding, but they appear to be unable to answer appropriate questions about the basis for their recommendations. Three or more letters to each of those organizations from the director of Pollution Action, asking how they have determined that the known toxins in breast milk are not having harmful effects, have never been responded to, as of a year later.

For additional relevant information, see www.breastfeeding-toxins.info and www.breastfeeding-and-autism.net,

Responses to this are requested, to be sent to:

Donald P. Meulenberg, Director, Pollution Action, 27 McWhirt Loop, Ste. 111, Fredericksburg, VA 22406 or to dm@pollutionaction.org

*For information about **Pollution Action**, see www.pollutionaction.org

(A copy of this statement, **with *working links to most of the references***, in PDF format, is at www.autism-origins.info/A.pdf)

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2) Re: EPA's RfD for dioxin: At www.epa.gov/iris/supdocs/dioxinv1sup.pdf in section 4.3.5, at end of that section, "...the resulting RfD in standard units is 7 × 10−10 mg/kg-day." (that is, O.7 pg of TEQ/kg-d)

Re: breastfed infants' exposures to dioxins, in U.S. and internationally**:**

- Infant Exposure to Dioxin-like Compounds in Breast Milk Lorber (Senior Scientist at EPA) et al., VOL. 110 No. 6 June 2002, Environmental Health Perspectives http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=54708#Download

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- J Grigg, Environmental toxins; their impact on children's health, Arch Dis Child 2004;89:244-250 doi:10.1136/adc.2002.022202 at http://adc.bmj.com/content/89/3/244.full

3) Re: PBDEs ingested by breastfed infants:

-Table 5-4 of EPA (2010) An exposure assessment of polybrominated diphenyl ethers. National Center for Environmental Assessment, Washington, DC; EPA/600/R-08/086F. http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=210404, Schechter study in first page of table. Also Section 5.6.2, near end of section, of above.

- Costa et al.,Developmental Neurotoxicity Of Polybrominated Diphenyl Ether (PBDE) Flame Retardants, Neurotoxicology. 2007 November; 28(6): 1047–1067. PMCID: PMC2118052 NIHMSID:

- EPA Technical Fact Sheet on Polybrominitated Diphenyl Eithers (PBDEs) and PBBs, p.3 at http://www.epa.gov/fedfac/pdf/technical_fact_sheet_pbde_pbb.pdf Regarding prevalence of tetraBDEs, see Costa LG, et al., Polybrominated diphenyl ether (PBDE) flame retardants: environmental contamination, human body burden and potential adverse health effects. Acta Biomed. 2008 Dec;79(3):172-83 at http://www.ncbi.nlm.nih.gov/pubmed/19260376.

4) Re: Mercury levels in breast milk:

- U.S. ATSDR document on mercury at www.atsdr.cdc.gov/toxprofiles/tp46-c5.pdf, p. 443

- Code of Federal Regulations, Title 21, Chapter 1, Subchapter B, Part 165, Subpart B, Sec. 165-110 at http://www.accessdata.fda.gov/scripts/cdrh/cfdocs/cfCFR/CFRSearch.cfm?fr=165.110

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Re: PBDEs in formula less than 2% of concentration in breast milk:
-Section 4.7 , 2nd paragraph (citing Schechter et al.) of U.S. EPA (2010) An exposure assessment of polybrominated diphenyl ethers. http:/cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=210404
-Section 5.6.2 of above, 2nd paragraph. The EPA states the figure as "44.1 ng/g lwt" (44.1 ng = 44,100 pg). For comparison purposes, the lipid (fat) weight indicated here needs to be converted to whole weight, which can be done as follows: The EPA here assumes a fat content of 4%. Using that figure, 44,100 pg/g lwt becomes 1760 pg/g wwt.

Re: Mercury in formula less than 1% as high as in human milk:
- Food Additives & Contaminants: Part B: Surveillance Volume 5, Issue 1, 2012 Robert W. Dabeka et al., Survey of total mercury in infant formulae and oral electrolytes sold in Canada DOI: 10.1080/19393210.2012.658087 at www.tandfonline.com/doi/full/10.1080/19393210.2012.658087#tabModule

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5b) Grandjean P, Landrigan PJ. Developmental neurotoxicity of industrial chemicals. Lancet. 2006;368:2167–2178. at http://www.reach-compliance.eu/english/documents/studies/neurotoxity/PGrandjean-PjLandrigan.pdf p. 2

6) Dobbing et al., Quantitative growth and development of human brain, Arch Dis Child, 1973 October: 48(10): 757-767 at http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1648530) 80% figure estimated from Figure 11.

7) Rice et al., Critical Periods of Vulnerability for the Developing Nervous System: Evidence from Humans and Animal Models, EPA National Center for Environmental Assessment, at http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1637807, 1st para.

8) Sec. 4 of Gadad et al., Neuropathology and Animal Models of Autism: Genetic and Environmental Factors, Autism Res Treat. 2013: 731935 PMCID: PMC3787615 at http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3787615

8a) Rodier, "Developing Brain as a Target of Toxicity," Environmental Health Perspectives, at http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1518932/pdf/envhper00365-0077.pdf 1st paragraph below Figure 1

-- see also Sokolowski et al., Methylmercury elicits mitochondrial-dependent apoptosis in developing hippocampus and acts at low exposures, Neurotoxicology 2011 at http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3256128

9) Chien LC, et al., Analysis of the health risk of exposure to breast milk mercury in infants in Taiwan. Chemosphere. 2006 Jun;64(1):79-85. Epub 2006 Jan 25 at http://www.ncbi.nlm.nih.gov/pubmed/16442149 Note that "mercury" and "methylmercury" are often used interchangeably, including by the EPA (see www.epa.gov/hg/effects.htm), since it is typically difficult to differentiate the various species of mercury. It is recognized that a high percentage of the mercury in human bodies is methylmercury. According to the U.S. Geological Survey, methylmercury is "the form of mercury that is most easily bio*accumulated* in organisms;" and it "bio*magnifies* (increases in concentration as it travels up the food chain)." Bear in mind that humans are at the top of the food chain, especially with regard to eating of fish and seafood in the case of methylmercury.

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-- Durkin (2008) also referred to another study supporting correlation of increased autism with earlier birth order**:** Glasson et al. Perinatal factors and the development of autism. Arch Gen Psychiatry.

2004;61(6):618–627.

12) Jones et al., Attention to eyes is present but in decline in 2-6-month-old infants later diagnosed with autism: Nature:(2013) DOI:doi:10.1038/nature at http://www.pediatrics.emory.edu/documents/divisions/autism/Jones_Klin_2013.pdf

14a) -- Ryan et al., Program for Women, Infants, and Children Participants, 1978 -2003: Lower Breastfeeding Rates Persist … in journal Pediatrics, at http://pediatrics.aappublications.org/content/117/4/1136.full.pdf+html , Table 2, "Parity" section.
14b) -- CDC chart at www.cdc.gov/breastfeeding/data/NIS_data/2006/socio-demographic.htm

14c) -- PCDDs, PCDFs, and PCBs concentrations in breast milk from two areas in Korea: body burden of mothers and implications for feeding infants, Jiyeon Yang et al. Chemosphere 46 (2002) 419–428); and Infant Exposure to Chemicals in Breast Milk in the United States: Judy S. LaKind, et al., Children's Health Review Environmental Health Perspectives • Volume 109 | Number 1 | January 2001 www.ncbi.nlm.nih.gov/pmc/articles/PMC1242055/pdf/ehp0109-000075.pdf

15) Section 2.2.2.4 of U.S. ATSDR, Public Health Service, Toxicological Profile for Mercury at http://www.atsdr.cdc.gov/toxprofiles/tp46.pdf. For ataxia-producing effect of methylmercury, see also p. 6-21 of U.S. EPA, Mercury Report to Congress, Vol. VII, Dec. 1997, EPA-452/R-97-009 at http://www.epa.gov/ttn/oarpg/t3/reports/volume7.pdf

15a) From a web page of Medical News Today, at http://www.medicalnewstoday.com/articles/162368.php)

16) Burbacher et al., Comparison of Blood and Brain Mercury Levels in Infant Monkeys Exposed to Methylmercury or Vaccines w/ Thimerosal, Environ Health Perspect. PMC1280342 at http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1280342

21a) Gascon M et al., Effects of pre and postnatal exposure to low levels of polybromodiphenyl ethers on neurodevelopment and thyroid hormone levels at 4 years of age. [Environ Int. 2011] . at http://www.ncbi.nlm.nih.gov/pubmed/21237513

22) Section 4.7 , 2nd paragraph (citing Schechter et al.) of U.S. EPA (2010) An exposure assessment of polybrominated diphenyl ethers. National Center for Environmental Assessment; EPA/600/R-08/086F. online at http://www.epa.gov/ncea or directly at http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=210404

23) Geier DA et al., Blood mercury levels in autism spectrum disorder: Is there a threshold level? Acta Neurobiol Exp (Wars). 2010;70(2):177-86, http://www.ncbi.nlm.nih.gov/pubmed/20628441. Also see

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24) Roberts et al., "Perinatal Air Pollutant Exposures and Autism Spectrum Disorder in the Children of Nurses' Health Study II Participants," (Environ Health Perspect; DOI:10.1289/ehp.1206187 online at http://ehp.niehs.nih.gov/1206187):

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Also Wang et al., Emission estimation and congener-specific characterization of polybominated diphenyl ethers from various stationary and mobile sources, Environmental Pollution, Vol. 168, Oct. 2010

(24b) 2004 International Emissions Inventory Conference, Air Toxics Session, Clearwater, Florida, Mercury Emissions from Motor Vehicles, at http://www.epa.gov/ttnchie1/conference/ei13/toxics/baldauf_pres.pdf

Also, Hoyer et al., Mercury Emissions from Motor Vehicles (EPA publication), esp. p 4, at http://www.epa.gov/ttnchie1/conference/ei13/toxics/hoyer.pdf

(5a) The methyl form of mercury, heavily present in humans due to seafood consumption, is persistent and lipophilic, as per: (Paasivirta, Long-term Effects of Bioaccumulation in Ecosystems, The Handbook of Environmental Chemistry, ,Vol. 2 Part J., Bioaccumulation (ed. by B. Beek) © Springer-Verlag Berlin Heidelberg 2000, at http://www.bio-nica.info/Biblioteca/Paasivirta2000EffectsBioaccumulation.pdf

(24d) An Inventory of Sources and Environmental Releases of Dioxin-Like Compounds in the United States for the Years 1987, 1995, and 2000", EPA/600/P-03/002F, November 2006: Fig. 1-4, and Table 1-17 re rapid growth. 2000 appears to be the most recent year for which the EPA provides national dioxin release data. at http://www.epa.gov/ncea/pdfs/dioxin/2006/dioxin.pdf

(24e) Chien LC, et al., Analysis of the health risk of exposure to breast milk mercury in infants in Taiwan. Chemosphere. 2006 Jun;64(1):79-85. Epub 2006 Jan 25 at http://www.ncbi.nlm.nih.gov/pubmed/16442149

26) Autism rates associated with nutrition and the WIC program. Shamberger R.J., Phd, FACN, King James Medical Laboratory, Cleveland, OH J Am Coll Nutr. 2011 Oct;30(5):348-53. Abstract at www.ncbi.nlm.nih.gov/pubmed/22081621

27) For details, see Appendix 2a at http://www.pollutionaction.org/breastfeeding-and-autism-and-cancer.htm

28) see www.breastfeedingprosandcons.info for many authoritative sources.

29) see www.breastfeeding-studies.info

30) "Breastfeeding, Family Physicians Supporting (Position Paper)," American Academy of Family Physicians at http://www.aafp.org/about/policies/all/breastfeeding-support.html

(A copy of this statement, **with *working links to most of the references***, in PDF format, is at www.autism-origins.info/A.pdf)

Responses to comments from Dr. Alycia Halladay, Autism Speaks senior director of environmental and clinical science**:**

-- "Many important statements (i.e. top of page 2, point b) are missing references."

Acknowledged. Additional references have been provided, or unreferenced statements removed.

-- "Your statement in regards to **reference 26** does not include the analysis with regards to confounding factors nor does it include the conclusions that the authors made regarding their findings."

True. But what I quoted from that study was an accurate statement of the study's findings. The interpretation that I draw from those findings appears to be as valid as that of that study's author, considering the known high levels of developmental toxins in breast milk as I have described (with ample references), levels that are extremely high both in relation to the authoritatively-established safe levels and in relation to levels in infant formula.

-- (re text preceding **Footnote 11a**) Dr. Halladay says, **"**Younger siblings of children diagnosed with autism have a HIGHER risk of autism, not a lower risk (See Ozonoff et al., 2011)"

I do not dispute that, but that does not conflict with my statement to which Dr. Halladay referred **:** "A typical fourth child's risk… The odds of being diagnosed with autism decrease from first to later children." I am talking about children in the *general* population**;** Dr. Halladay refers to children *who have a sibling with autism*, which is a very small subset of the population to which I referred. Correlation of later birth order with reduced autism in the *general* population is well established, as is now indicated at footnote 8.

-- Dr. Halladay's comment that certain footnotes do not lead to peer-reviewed scientific sources:

In the interest of keeping my statement to a length that people would not be inclined to set aside as being too long to read, I referred to non-peer-reviewed web pages *in which the many peer-reviewed references that apply in those cases are properly cited*, along with relevant explanatory material. In trying to accommodate to Dr. Halladay's suggestion without excessive length, I now

(a) have included above many footnotes that had previously been referred to as being in separate web pages, and

(b) hope the readers wanting to check the other sources won't mind going to the websites indicated. Footnote 32 alone refers to a website where *over 50* separate peer-reviewed studies are cited in verification of what is said in this text, and the website referred to at footnote 31 provides *several dozen* such studies and CDC documents to support what is said in the above text.