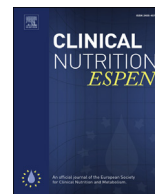




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Original article

Evidence the U.S. autism epidemic initiated by acetaminophen (Tylenol) is aggravated by oral antibiotic amoxicillin/clavulanate (Augmentin) and now exponentially by herbicide glyphosate (Roundup)

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SUMMARY

Because certain hereditary diseases show autistic behavior, and autism often runs in families, researchers seek genes underlying the pathophysiology of autism, thus core behaviors. Other researchers argue environmental factors are decisive, citing compelling evidence of an autism epidemic in the United States beginning about 1980. Recognition that environmental factors influence gene expression led to synthesis of these views – an ‘epigenetic epidemic’ provoked by pervasive environmental agents altering expression of vulnerable genes, inducing characteristic autistic biochemistries in many mothers and infants. Two toxins most implicated in the U.S. autism epidemic are analgesic/antipyretic acetaminophen (Tylenol) and oral antibiotic amoxicillin/clavulanate (Augmentin). Recently herbicide glyphosate (Roundup) was exponentially implicated. What do these toxins have in common? Acetaminophen depletes sulfate and glutathione required to detoxify it. Oral antibiotics kill and glyphosate inhibits intestinal bacteria that synthesize methionine (precursor of sulfate and glutathione, and required to methylate DNA), bacteria that synthesize tryptophan (sole precursor of neuro-inhibitor serotonin), and bacteria that restrain ammonia-generating anaerobes. Sulfate plus glutathione normally sulfate fetal adrenal androgen dehydroepiandrosterone to DHEAS – major precursor of placental/postnatal estrogens. Glyphosate (and heavy metals) also inhibit aromatase that turns androgens to estrogens. Placental/postnatal estrogens dehydrate/mature brain myelin sheaths, mature corpus callosum and left hemisphere preferentially, dilate brain blood vessels, and elevate brain serotonin and oxytocin. Stress-induced weak androgens and estrogen depletion coherently explain white matter asymmetry and disconnection in autism, extreme male brain, low brain blood flow, hyperexcitability, social anxiety, and insufficient maternal oxytocin at birth to limit fetal brain chloride/water and mature GABA.

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The history of American and European agriculture over the last hundred years has been a history of the increasing dominance of industrial capital over farming.... The creation and adoption of genetically modified organisms are the latest steps in this long historical development of capital-intensive industrial agriculture. Roundup Ready herbicide-resistant soybeans have been created by Monsanto so that farmers will be able to use its powerful herbicide, Roundup, while at the same time buying Monsanto seed. The farmers accept the cost of the new variety and its chemical partner because the use of such a powerful general weed killer will reduce the number of herbicide treatments or mechanical tillage passages through the fields, freeing them for the hours in the automobile assembly plant that they need to keep their farms.

Richard Lewontin *It Ain't Necessarily So* [1]

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1. Introduction: is there a “single key mechanism” in autism?

[F]or every disease there is a single key mechanism that dominates all others. If one can find it, and then think one's way around it, one can control the disorder.... In short, I believe that the major diseases of human beings have become approachable biological puzzles, ultimately solvable.

Lewis Thomas *The Medusa and the Snail* [2]

Certain aspects of autistic disorders (ASD) are obvious though little understood. As different as autistic behavior looks from child to child, it is recognizably *autistic*. Martha Herbert noted many behavioral, neurological, and metabolic *autisms* – and that transient autistic behavior has been seen in many different disorders, including metabolic, allergic and epileptic conditions. “What is it about brain biology that may allow many different underlying biological mechanisms to produce a set of behaviors that look so similar?” she asked [3].

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Clearly these diverse mechanisms have *some* pathophysiology in common. Lewis Thomas noted several diseases with multiple environmental causes and multiple pathologies linked to a “single key mechanism”: “This generalization is harder to prove, and arguable – it is more like a strong hunch than a scientific assertion – but I believe that the record thus far tends to support it. The most complicated, multicell, multitissue, and multiorgan diseases I know of are tertiary syphilis, chronic tuberculosis, and pernicious anemia.... Before they came under scientific appraisal each was thought to be what we now call a ‘multifactorial’ disease, far too complex to allow for any single causative mechanism. And yet, when all the necessary facts were in, it was clear that by simply switching off one thing – the spirochete, the tubercle bacillus, or a single vitamin deficiency – the whole array of disordered and seemingly unrelated pathologic mechanisms could be switched off, at once.” [2].

Thomas's view of pathological mechanisms switched on or off is apt as researchers investigate genetic vulnerabilities in autism. Although gene *structure* (DNA) remains stable over a lifetime (except for mutations) gene *expression* is very labile. Genes are readily turned on or off by epigenetic mechanisms – environmental factors like toxins and even stress [4,5]. Three environmental toxins most implicated in the U.S. autism epidemic are (1) analgesic/antipyretic acetaminophen (Tylenol), (2) oral antibiotic amoxicillin/clavulanate (Augmentin), and most recently (3) herbicide glyphosate (Roundup) (Table 1). Do these toxins have a common “key mechanism”?

2. Environmental toxins most implicated in the U.S. autism epidemic

Two new sets of statistics, one released by the U.S. Office of Education and the other by the California Department of Developmental Services (DDS), also provide clear evidence of an autism epidemic. The federal statistics ... show an average 700 percent increase nationwide in the incidence of autism in less than ten years.... Of the autistic children identified by DDS, eight out of 10 were born after 1980. Bernard Rimland 2002 [6].

The total number of people now thought to have autism compared to what it was before and based on the estimate of what part of that is real, we probably have a four-to six-fold increase in autism over the last twenty years or so. Martha Herbert 2012 [7].

Table 1
Environmental toxins most implicated in intensifying U.S. autism epidemic.

Acetaminophen (Tylenol)
- Depletes sulfate and glutathione required to detoxify it. - Kills cerebellar Purkinje neurons. - Use multiplied after 1980 CDC warning re aspirin/Reye's syndrome. - Commonly given for circumcision
Amoxicillin/clavulanate (Augmentin)
- Kills gut bacteria that make methionine, precursor of other sulfur AAs, sulfate, glutathione, metallothionein, DHEAS; needed to methylate DNA/express genes. - Kills gut bacteria that make tryptophan, limiting brain serotonin thus oxytocin. - Enables Clostridia and other anaerobic bacteria to flourish, increasing ammonia. - May also increase ammonia because made with ammonia. - Launched in 1981 as general oral antibiotic. - Recommended by pediatric groups for otitis media in daycare centers.
Glyphosate (Roundup)
- Inhibits gut bacteria that make methionine for sulfur AAs, sulfate, GSH, metallothionein, DHEAS, DNA methylation, gene expression. - Inhibits gut bacteria that make tryptophan, limiting brain serotonin thus oxytocin. - Spares anaerobes notably Clostridia, increasing ammonia. - Inhibits aromatase, limiting estrogens – as do heavy metals notably mercury. - Use multiplied after glyphosate-resistant soybeans introduced in 1996.

In 2002 biochemist Jon Pangborn reported a dramatic increase in autism incidence in the U.S., based on thousands of parents' reports to the Autism Research Institute (ARI/ICBR) since 1967 [8]. Until about 1980, 50–60% of autistic children were autistic at birth, and 40–50% regressed into autism at about 18 months. “Around 1980,” Pangborn reported, “all this began to change. The total frequency of occurrence doubled, doubled again, and by 1995 was approximately 10 times that of 1980. Furthermore, while the onset-at-birth type had increased 3 to 4 times, the onset-at-18-months type had skyrocketed to considerably more than 10 times its 1980 level.” (see Fig. 1. Incidence of autism types). In 2015 the Centers for Disease Control and Prevention (CDC) reported the incidence of autism in the U.S. was now one in 45 children [9] – about 4:1 boys.

Psychologist Fred Previc concluded: “The incidence of autism has risen 10-fold since the early 1980s, with most of this rise not explainable by changing diagnostic criteria. The rise in autism is paradoxical in that autism is considered to be one of the most genetically determined of the major neurodevelopmental disorders and should accordingly either be stable or even declining. Because a variety of epigenetic influences, particularly those occurring during the prenatal period, can override or masquerade as genetic influences, these should be considered as prime contributors” [4].

2.1. Acetaminophen (Tylenol) rapidly replaced aspirin for children after 1980 CDC warning

Navy scientist Stephen Schultz also implicated 1980 as the year our U.S. autism epidemic began – the year the CDC warned the American public that aspirin could cause Reye's syndrome in children [rare but often fatal high ammonia after viral illness] and parents, pediatricians and hospitals rapidly switched to acetaminophen (Tylenol) [10]. An online parent survey by Schultz and colleagues found children given acetaminophen for pain/fever of the measles–mumps–rubella vaccine (MMR) were far more likely to become autistic than children given ibuprofen: “Acetaminophen use after MMR vaccination was associated with an increase of sixfold in the likelihood of AD when considering only children 1–5 years ... fourfold after limiting cases to children with regression in development ... and eightfold when considering only children who had post-vaccination sequelae.” [11].

They noted sulfation by the liver is the primary pathway to detoxify acetaminophen in children under ten. When sulfation is impaired (common in autistic children) acetaminophen oxidizes to a toxic metabolite that requires glutathione (GSH) to detoxify. Acetaminophen thus depletes the liver's sulfate and glutathione, impairing detoxification of other harmful agents. Orlowski and colleagues compellingly debunked the association of aspirin and Reye's syndrome [12,13].

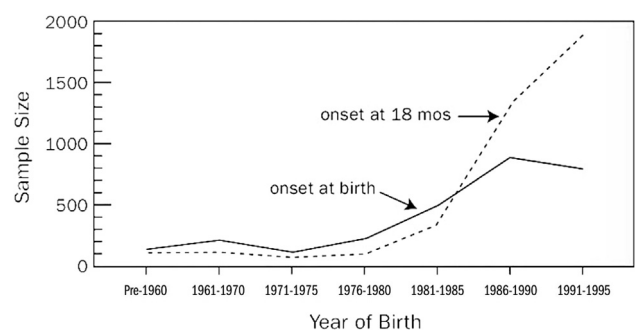


Fig. 1. Incidence of autism types. From: Pangborn JB. Introduction to the diseases of autism and laboratory testing options. In: Pangborn JB, Baker SM. Biomedical Assessment Options for Children with Autism and Related Problems. San Diego, Ca: Autism Research Institute; 2002.

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