**Acetaminophen – Please hold off on using for now**

I want to re-address this fever control issue yet again. I know that a parent's first instinct when their child has a fever is to get rid of it as quickly as possible; to make things better. Remember that fever has a beneficial function during illness. It enhances immune function and allows the child to get over the infection more expeditiously. When we drop that fever, the immune system may founder. In the process of trying to "make things better", sometimes we impede the very process that will facilitate that. The use of acetaminophen has taken a growing center stage in some medical circles as we wrestle with the realities of a logarithmic growth in autism. What on earth do the two have to do with each other? In this review article emanating from the reputable halls of Harvard University School of Medicine and Duke University, some early alarm bells have started ringing, begging for caution in use of a drug that we have used for 30 years in almost as caviler a manner as water… Tylenol (paracetamol, acetaminophen). To gain a quick perspective of the link, think of what happens to a child when they overdose on Tylenol. The ED doc will put an NG tube down into the stomach and try grab the drug with charcoal. The next will be the use of a IV nutrient called N-acetylcysteine. What exactly are we doing when giving NAC (N-acetylcysteine)? It is one of the direct precursors of glutathione, our master antioxidant. The doc is trying to dramatically stimulate the production of this molecule. Glutathione is the molecule that gives us our ability to survive the stress of infection, pesticide exposure, emotional stress, heavy metals (to a degree), and oxidative stress originating from the challenges of living in a high oxygen environment (remember we breath 21% oxygen with every breath; there are consequences to that); without it, we die. Acetaminophen uses up glutathione to a tremendous degree and leaves small children vulnerable to oxidative stress in their most vulnerable stage of development. We are slowly beginning to grasp that autism may very well result in part from a dramatic reduction in ability to handle oxidative stress from the environment. In a genetically vulnerable child, acetaminophen simply is the "straw that breaks the camel's back". If a child is walking on an oxidative knife-edge, the use of this drug can give that little push that it takes to make that child fall off the knife-edge into overtly clinical manifestation such as autism. Until we get this figured out, I am urging parents to stop using acetaminophen during pregnancy and in infants under 2 yrs of age. The abstract of this review article underscores this urgency:

"The wide range of factors associate with the induction of autism is invariably linked with either inflammation or oxidative stress, and sometimes both. The use of acetaminophen in babies and young children may be much more strongly associated with autism than its use during pregnancy, perhaps because of well-known deficiencies in the metabolic breakdown of pharmaceuticals during early development. Thus, one explanation for the increased prevalence of autism is that increased exposure to acetaminophen, exacerbated by inflammation and oxidative stress, is neurotoxic in babies and small children. This view mandates extreme urgency in probing the long-term effects of acetaminophen use in babies and the possibility that many cases of infantile autism may actually be induced by acetaminophen exposure shortly after birth."

The second analysis is by the former director of Great Plains Laboratory, William Shaw, PhD. He widens the evidence of causation to other chronic disorder such as asthma, ADHD, and other inflammatory conditions. He has been sounding this alarm for two years!!

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5536672/>

<https://www.greatplainslaboratory.com/articles-1/2015/11/13/evidence-that-increased-acetaminophen-use-in-genetically-vulnerable-children-appears-to-be-a-major-cause-of-the-epidemics-of-autism-attention-deficit-with-hyperactivity-and-asthma>