NSU NOVA SOUTHEASTERN UNIVERSITY
College of Pharmacy

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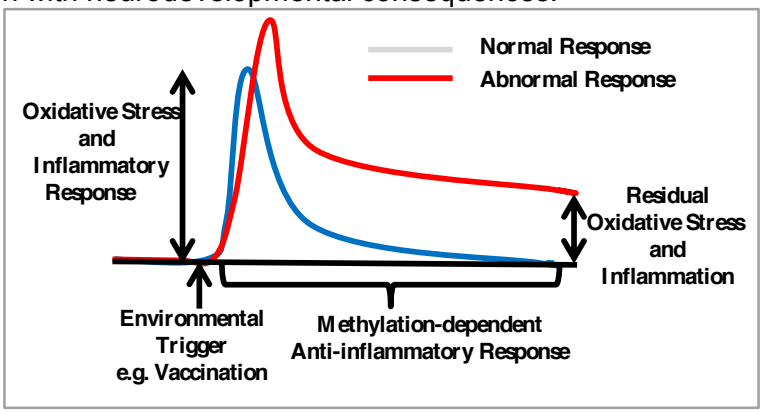
To Whom It May Concern:

As a researcher with expertise in the molecular origins of neurodevelopmental disorders as well as the adverse effects of vaccines, I would like to provide my perspective on the issue of individual genetic vulnerability, as it relates to the current debate over mandatory vaccination.

My personal background includes a B.S. degree in Pharmacy from the State University of New York at Buffalo (1970) and a Ph.D. degree from the University of Miami (1975), followed by 38 years as Professor of Pharmacology at Northeastern University, before taking my current position at Nova Southeastern University (2014). Throughout this time I have conducted laboratory-based research into various aspects of neuroscience and development, and I have authored over 100 peer-reviewed research papers as well as a book entitled "Molecular Origins of Human Attention: The Dopamine-Folate Connection". I have also served as an expert witness in a number of vaccination-related court cases.

For the past 10-15 years my lab has focused on the metabolic and molecular origins of autism. Much of our effort has been directed toward understanding the factors which regulate gene expression during neurodevelopment and their sensitivity to various environmental exposures. This includes the relatively recent recognition of **epigenetic regulation**, which involves turning genes on or off by the reversible addition of carbon atoms (methyl groups) to DNA. This process of **DNA methylation** is fundamental to neural development but is also involved in ongoing brain functions, including the capacity for memory formation. Our work, as well as that of many other scientists, shows that DNA methylation and the epigenetic regulation it provides is highly sensitive to environmental exposures, and, not surprisingly, it is particularly sensitive to **neurodevelopmental toxins**. Underlying this extreme sensitivity is the ability of these toxins to promote oxidative stress and since autistic children have about one-third less antioxidant than normal, they are most likely to develop oxidative stress.

Vaccination provokes inflammation and causes oxidative stress. Indeed, these responses are integral to successful vaccination. As such, vaccination represents an "environmental" challenge, both to antioxidant capacity and to DNA methylation-dependent epigenetic regulation. Individuals with sufficient antioxidant and methylation capacity can withstand this challenge with little or no interruption in epigenetic regulation and can restore their systems to normal after vaccination. However, as illustrated below, individuals with only limited antioxidant capacity or limited methylation capacity are less able to restore normal status after vaccination, placing them at higher risk of sustained oxidative stress and impaired methylation, which can lead to disruption of epigenetic regulation with neurodevelopmental consequences.



Naturally occurring genetic variations play a significant role in determining who will be more likely to have problems with vaccination-induced oxidative and impaired methylation. In a very common example, the gene known as MTHFR (methylenetetrahydrofolate reductase) has several different variants which differ in their activity and people carrying the lower activity forms are at greater risk of impaired methylation. A higher incidence of adverse reactions to vaccination has been linked to MTHFR status[1] and a number of studies have reported an association between autism and MTHFR variants[2-4], as well as other genes affecting antioxidant and methylation capacity[4].

The bottom line is that the risk of adverse responses to vaccination is significantly greater for certain individuals and medical science is beginning to identify genetic factors which place people at greater risk. Personalized medicine based upon our genetic vulnerabilities is becoming a reality and it is foolhardy to compel such vulnerable individuals to place themselves or their children at extraordinary risk by enacting mandatory vaccination legislation. This is especially true when vaccines are given so early in life prior to assessment of genetic risk factors. Moreover, in my view, enacting such laws will place states in the position of assuming liability for the health consequences of vaccinating high risk populations, with significant legal and financial implications.

While vaccination provides a substantial benefit to society, this benefit is not without cost. Until the necessary research into vaccine safety is completed and sources of individual vulnerability are better defined, we need to maintain caution and maintain accommodation for individual exemptions. This is a circumstance where the guidance of *"First do no harm"* makes both scientific sense and common sense.

I hope you find this perspective of value and I would be happy to provide further details as needed.

Sincerely,

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[1]Reif DM *et al.* Genetic basis for adverse events after smallpox vaccination. J Infect Dis. 2008;198(1):16-22.
[2]Park J *et al.* MTHFR 1298A/C is a risk factor for autism spectrum disorder in the Korean population. Psychiatry Res. 2014;215(1):258-9.
[3]Mohammad NS *et al.* Aberrations in folate metabolic pathway and altered susceptibility to autism. Psychiatr Genet. 2009;19(4):171-6.
[4]James SJ *et al.* Metabolic endophenotype and related genotypes are associated with oxidative stress in children with autism. Am J Med Genet B Neuropsychiatr Genet. 2006;141B(8):947-56.