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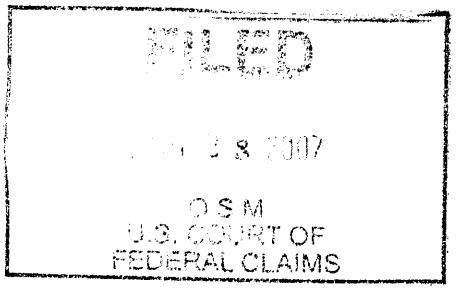

VIA UPS DELIVERY

Clerk
United States Court of Federal Claims
717 Madison Place, NW
Washington, D.C. 20005

Re: In Re: Claims for Vaccine Injuries Resulting in Autism Spectrum Disorder, or a Similar Neurodevelopmental Disorder v. Secretary of Health And Human Services
Autism Master File
Our File No. 054500 - Omnibus Autism Proceeding

Dear Clerk of Court:

Enclosed for filing please find the original and two copies of the following:

1. Sander Greenland, M.A., M.S, Dr.P.H's expert report, bibliography and curriculum vitae,

2. Richard C. Deth, PhD's expert report, bibliography and curriculum vitae.

These submissions are two of the expert reports that the PSC will rely on to prove general causation in the "thimerosal-only" test cases in the Omnibus Autism Proceeding to be heard by the Special Masters in May 2008. Additional reports and supporting documents will be filed separately. Thank you.

Very truly yours,

Lynne Shea

Lynne A. Shea
Paralegal to Thomas B. Powers

Enclosures

cc: George Hastings, U.S. Court of Federal Claims
Denise Vowell, U.S. Court of Federal Claims
Patricia Campbell-Smith, U.S. Court of Federal Claims
Joseph T. Lowe, U.S. Court of Federal Claims (via email only)
John Fabry, Esq., Williams Bailey Law Firm, LLP
Vincent J. Matanoski, Esq., U.S. Department of Justice

REPORT OF SANDER GREENLAND, M.A., M.S., Dr.P.H.

Regarding epidemiologic evidence on the possible link between mercury-containing vaccines and regressive autism

1. Introduction

I herewith provide my evaluation of the current epidemiologic evidence on the possible link between mercury-containing vaccines (MCV) and regressive autism. The conclusion I have reached can be summarized as follows: While the epidemiologic literature to date has not detected an association of MCV with autism in general or autistic-spectrum disorders, it has not ruled out the possibility that MCV is associated with a pre-specified type of autism, the regressive form. The inability of the literature to reject this possibility is largely due to the limited number of autism cases of the regressive type, and the failure of published controlled epidemiologic studies to date to separate regressive autism from other types.

2. Qualifications

My qualifications for this evaluation are extensive, as follows:

a) I have written and published numerous peer-reviewed articles on the summarization, interpretation, and limitations of scientific literature (see my curriculum vitae).

b) The textbook I have co-authored with Kenneth J. Rothman, *Modern Epidemiology* (2nd ed. 1998; Lippincott, Philadelphia), has been selected as the advanced epidemiologic text in numerous schools of public health and medicine. *Modern Epidemiology* also has been authoritatively cited in innumerable peer-reviewed journal articles and reviews including the Federal Judiciary Center's *Reference Manual on Scientific Evidence*.

c) I have been teaching epidemiology and statistics at the UCLA School of Public Health since 1979. I currently am Professor of Epidemiology, UCLA School of Public Health, and Professor of Statistics, UCLA College of Letters and Science.

d) I also have been invited to give and have given over 180 presentations and workshops at such institutions as Oxford University, Stanford University School of Medicine, Harvard School of Public Health, Yale University School of Public Health, Johns Hopkins School of Public Health, Columbia University School of Public Health, Case Western School of Medicine, Emory School of Public Health, Tulane School of Public Health, the National Cancer Institute, the Food and Drug Administration, the Centers for Disease Control, the Royal Statistical Society, and the Universities of Washington, Texas, North Carolina, Minnesota, Michigan, and California, as well as universities, research institutes, and conferences in England, Scotland, Germany, Denmark, Sweden, Finland, Norway, The Netherlands, France, Switzerland, Spain, Italy, Japan, Australia, and New Zealand.

e) I received a Doctorate in Public Health (Dr.P.H. 1978) from the University of California, Los Angeles, where I majored in Epidemiology, minored in Mathematics, and received a Regents Fellowship in Epidemiology. I also received a Master's degree in Mathematics from the University of California, Berkeley (1973), where I received highest honors and a Regents Fellowship in Mathematics.

f) I have received honors and professional certifications as a Fellow of the American Statistical Association and as a Fellow and a Chartered Statistician of the Royal Statistical Society. I am a member of the Biometric Society and the Society for Epidemiologic Research, I have served as an elected member of the Executive

Committee for the latter society, which is the largest society of epidemiologists in the world today, and as Chair of the Epidemiology Section of the American Statistical Association, which is the largest statistical society in the world today.

g) I have served as a consultant in epidemiology and statistics for numerous government agencies and private corporations, including the National Academy of Sciences, the National Institute of Environmental Health Sciences (NIEHS), the Environmental Protection Agency (EPA), the Food and Drug Administration (FDA), the Centers for Disease Control (CDC), the California State Attorney General's Office (regarding risk assessment), the California State Department of Health, the World Health Organization (WHO), The March of Dimes, General Electric, Southern California Edison, Amgen, and Dow Corning.

h) I am the author of nearly 300 peer-reviewed articles, over 60 published letters, over 130 published abstracts, and over 30 other publications (including book chapters and encyclopedia entries).

i) I have served as an investigator on over 30 grants and contracts from numerous research agencies, including the Rockefeller Foundation, National Cancer Institute, National Institute of Child Health and Human Development, National Institute of Mental Health, National Institute of Environmental Health Science, National Institute of Allergy and Infectious Diseases, National Institute on Drug Abuse, National Center for Health Services Research, National Highway and Traffic Safety Administration, Public Health Institute, Electric Power Research Institute, and the State of California. I have also carried out research for private industry including the Battelle Corporation, Southern California Edison, and General Electric.

j) I have served three terms as an Associate Editor of the *American Journal of Epidemiology* (1984-98) and the journal *Epidemiology* (1989-2006), and now serve as an Associate Editor of *Statistics in Medicine* (1985-present), the *European Journal of Epidemiology* (2003-present). I also serve as a regular referee for American Journal of Epidemiology, Epidemiology, International Journal of Epidemiology, and Statistics in Medicine, serve as an occasional referee for the American Journal of Public Health, the American Statistician, Annals of Epidemiology, Biometrics, Biometrika, Communications in Statistics, Computational Statistics and Data Analysis, Controlled Clinical Trials, International Statistical Review, Journal of the American Medical Association, Journal of the American Statistical Association, Journal of Clinical Epidemiology, The New England Journal of Medicine, and the Scandinavian Journal of Work, Environment, and Health, and have served as a textbook reviewer for Oxford University Press, John Wiley & Sons, and other academic publishing houses.

3. Dilution of Associations Specific to Regressive Autism

Many disease categories (including autism) are composites of distinct types or forms. If an exposure (such as MCV) is associated with a disease category, an exposure will likely exhibit different associations with the different disease types within the broad category. For example, "cancer" is a very broad category. Within that category, smoking is strongly associated with respiratory-system cancers but shows little or no association with most other cancers such as skin cancer.

An association is said to exhibit high specificity if the exposure exhibits associations only with a certain narrow range of disease types within the broad category. Although lack of specificity does not necessarily argue against causality (Rothman and Greenland, 1998, Chapter 2), it has been argued that specificity can be supportive of causal hypotheses (Hill, 1965; Weiss, 2002).

Specificity is a crucial consideration if an exposure has little or no association with the majority of types in a disease category, but some association with one or a few of those types (i.e., if an association is highly specific). In that situation, a study that fails to isolate those few types is likely to miss the association. In other words, if a highly specific association is present, failure to separate the types can severely attenuate or dilute the association of the exposure with the disease category, to the point that it becomes undetectable (indistinguishable from random error). This dilution is a type of systematic error, or bias, in evaluating the potential for the exposure to cause disease.

A simple example may clarify this point. If a vaccine is not associated with any type of disorder in the category, we should expect to see the same risk when comparing vaccinated to the unvaccinated. Suppose, however, that in reality the vaccine is associated with a two-fold increase in the risk of a type of disorder in the category, but not associated with any other type. Suppose also that, without the vaccine, the associated type represents only one-tenth (10%) of the disease category, and that the total number of cases in the category would be 100. Then, without the vaccine, the number of cases with the associated type would be 100/10 = 10. With the vaccine, however, the number of

cases with the associated type would double, to 20, an excess of 10 cases over the original 10 with the associated type. This excess produced by the vaccine would result in a total of 100+10 = 110 cases over the full category, which is only a 10% increase in the risk of any type in the category. Thus, the risk ratio for getting any type in the category would be only 110/100 = 1.1. Such a small risk ratio cannot be reliably distinguished from 1 by ordinary epidemiologic studies.

Continuing this example, note that, among the vaccinated, the percentage in the disease category of the type would be 20/110 = 18%. In a population in which half the children received the vaccine in question, the percentage in the disease category of the type would be (10+20)/(100+110) = 14%. This percentage is similar to reported percentages of regressive autism among all autism cases in nonselective case series (e.g., Lainhart et al., 2002; Rogers, 2004).

Some studies consider more broad categories than all autism, such as "autism-spectrum disorder" (ASD) or even more broadly, "developmental disorders." Regressive autism would constitute an even smaller percentage of these categories, and so an association of MCV with one of these categories would be diluted even more than in the above example, to a value beyond detection by epidemiologic studies.

Broad categories of regressive autism may include cases with early developmental abnormalities (Rogers, 2004; Fombonne, 2007, para. 34). Within such broad categories of regressive autism, there can be cases of autism for which early autistic symptoms are

absent (Werner and Dawson, 2005). I will label such cases as "clearly regressive autism." Clearly regressive cases would be a minority of regressive cases and thus a small minority of all cases of autism. Thus, an exposure associated only with clearly regressive autism would show an association with general autism that was even smaller than that shown in the preceding illustration, again to a value beyond detection by epidemiologic studies. An exposure associated specifically with clearly regressive autism would show a diluted association even in an analysis limited to or heavily weighted with regressive cases, as might arise in study based on a passive reporting system (Woo et al., 2007).

4. Early Abnormalities and Genetic Factors in Regressive Autism

Fombonne (2007, paragraphs 37-39) has argued that most children with regressive autism display subtle developmental abnormalities before the disease is diagnosed, and that (as with other forms of autism), regressive autism likely has a genetic basis. These hypotheses do not however rule out environmental or medical triggers of regressive autism, and in fact are quite compatible with the existence of such triggers (Lainhart et al., 2002).

The argument that most children with regressive autism have pre-existing abnormalities actually implies that clearly regressive autism is very uncommon. Again, if the association of MCV with autism is highly specific to clearly regressive autism and the latter is as rare as some authors suggest (e.g., Fombonne, 2007, para. 37), the association would be subject to a dilution bias even larger than that illustrated above. Furthermore,

the rarity of clearly regressive autism has no logical or factual bearing on whether the disorder has environmental triggers.

As for genetic factors, it is well known that many genetically based diseases require environmental stimulus to become manifest. A classic example is phenylketonuria (PKU), a genetic disorder. This condition will cause of mental retardation in childhood, but only when phenylalanine (an ordinarily harmless amino acid) is present in the diet. Despite its genetic basis, PKU-based retardation can be prevented by avoidance of the trigger, dietary phenylalanine. Thus, in general, the existence of a genetic origin for a disease does not rule out or even argue against the presence of equally necessary environmental triggers; to assert otherwise is a common but simple logical mistake (Rothman and Greenland, 1998, Chapter 2).

5. Controlled Epidemiologic Studies: Necessity and Limitations

By analogy with established examples of environmental triggers, it has been argued that MCV may trigger regressive autism in a susceptible subgroup of children (Blaxill et al., 2004), although this view has been disputed (Fombonne, 2007). A reliable (valid and precise) epidemiologic evaluation of this hypothesis would require a series of controlled studies that specifically examined the association between MCV and subsequent occurrence of regressive autism.

A controlled epidemiologic study is one that compares two groups that would be expected to exhibit differences if and only if MCV is associated with autism. Most such

studies are either cohort studies or case-control studies. Cohort studies compare autism incidence among vaccinated children (the index group) to incidence among unvaccinated children (the control group). Case-control studies compare vaccination frequency among autistic children (the index group) to the frequency among normal children (the control group). In each type of study, the control group provides a reference point for comparison to the index group.

Controlled studies are essential for the formation of sound inferences regarding the presence or absence of an association. Nonetheless, all epidemiologic studies, including controlled ones, suffer various limitations inherent in the process of collecting data on human subjects. These problems lead to biases (systematic errors) in the results of the studies, and must be considered in any reliable evaluation of the studies (Rothman and Greenland, 1998, Chapters 8 and 19).

In the topic of vaccines and autism, it is important to distinguish *ecologic studies* from controlled studies. By definition, ecologic studies lack data linking the exposure (MCV) to the disease (autism) in individuals, and thus cannot compare vaccinated with unvaccinated children or autism cases with noncases (Morgenstern, 1998). In other words, such studies do not compare index and control groups. As a substitute, ecologic studies examine trends in the disease rate across times or places with different exposure prevalence.

Ecologic studies are subject to a number of biases not present in controlled studies, which can easily generate false associations or conceal real associations. For example, by definition at least one of the groups compared in ecologic studies will be a mixture of exposed and unexposed individuals, and hence its difference from the compared groups will be attenuated. Thus, such studies are generally not considered adequate substitutes for controlled studies, and are especially unable to reliably distinguish small associations from no association (Morgenstern, 1998; Greenland, 2004).

In the present context, the primary ecologic studies have been time-trend studies of general autism. Such studies would be unable to detect a specific association of MCV with regressive autism, because a diluted association would be submerged by the large background trends reported (Madsen et al., 2003; Stehr-Green et al., 2003).

6. Statistical Significance and Confidence Limits

Evaluation of a study result in terms of whether it was statistically "significant" or "nonsignificant" addresses only the possibility that random errors alone could have produced the finding. It fails to address systematic errors, such as the dilution problem that arises from grouping different disease types (discussed above).

In addition, classification of studies on the basis of their statistical significance is seriously inadequate even for evaluation of random error because it does not address the possibility that chance obscured (rather than created) an association. This possibility is addressed by the confidence limits from the study (Rothman and Greenland, 1998, Ch.

12). These limits show the range of values for a risk ratio that could have easily given rise to the observed results by chance alone. For example, suppose a study reports a risk ratio of 1.00 (no association observed), but with 95% confidence limits of 0.50 and 2.00. These limits indicate that the observed risk ratio is not significantly different (at the 0.05 level) from risk ratios as small as 0.50 or as large as 2.00; in other words, chance alone could have easily produced the observed risk ratio of 1.00 even if the true risk ratio were 0.50 or 2.00.

The pair of confidence limits taken together define a *confidence interval* for the risk ratio, and the pair is often written in parentheses. Thus the confidence interval in the above hypothetical example is (0.50, 2.00). The ratio of the upper and lower confidence limits (2.00/0.50 = 4 in this example) is often taken as a measure of the width of the interval, with smaller values indicating less random error (greater precision) in the study result.

The confidence interval says nothing about systematic errors in the study, however, which can be much larger than random errors. If one additionally accounts for systematic errors such as the dilution described earlier, the resulting interval estimate will be wider, usually much wider (Greenland, 2005). Therefore, the confidence intervals from a study should be taken as showing the absolute *minimum* range of risk ratios compatible with the data.

7. Controlled Epidemiologic Studies of Mercury-Containing Vaccines and Neurodevelopmental Disorders

As of this writing (August 2007) I am aware of no peer-reviewed controlled epidemiologic study of MCV and regressive autism *per se*; all studies identified failed to separate regressive autism from other types of autism. They are thus unlikely to have detected an association of MCV with regressive autism, especially clearly regressive autism, for the reason explained earlier: Dilution due to failure to separate autism types in the analysis. This fact, along with other problems of the studies, make the published studies almost uninformative regarding the hypothesis that MCV can trigger regressive autism specifically.

To further explain this point, I will briefly comment on the published controlled epidemiologic studies that have been accorded reasonable credibility by most reviewers and have been generally judged to be negative (providing no support for a link of MCV to autism), as in the review by Parker et al. (2004). I will also comment on three ecologic studies covered by the latter reviewers, but will specifically exclude from consideration the ostensibly positive studies by Geier and Geier, which have been criticized by these and other reviewers (e.g., Fombonne, 2007) as too flawed for use in scientific evaluations. For brevity, I will not here address validity issues of the studies other than the potential dilution bias mentioned earlier. Addressing these issues would further increase the uncertainty regarding the association of MCV with regressive autism.

Hviid et al., Journal of the American Medical Association, 2003. This cohort study was based on records from Danish registries. It reported a risk ratio for any MCV versus no MCV of 0.85 with 95% limits of 0.60 and 1.20, and a risk ratio for the highest dose category (3 doses of MCV) of 0.96 with 95% limits of 0.63 and 1.47. Thus the results are

compatible with a substantial association of MCV with regressive autism. As the authors note, the children vaccinated in this study followed Danish vaccination schedules, which result in roughly half the total mercury exposure from MCV in early childhood than do the American vaccination schedules (Ball et al., 2001; Hviid et al., 2003). The study should thus be expected to exhibit an even weaker association with autism than would an American study, if MCV is indeed associated with regressive autism. This weakness would be in addition to the dilution problem discussed above.

Andrews et al., Pediatrics, 2004. This British study examined various developmental disorders in relation to estimated mercury exposure from MCV in cohorts assembled from an official database. The risk ratio for autism associated with total MCV was 0.99 with 95% confidence limits of 0.88 and 1.12. In their discussion, the authors note that the children vaccinated in this study followed British vaccination schedules, which (like the Danish schedules) result in less total mercury exposure in early childhood than the American vaccination schedules. Thus, as with the Danish studies, the British studies should be expected to exhibit a weaker association with autism than would an American study, if MCV is indeed associated with regressive autism.

Jick and Kaye, New England Journal of Medicine, 2004. This case-control study drew subjects from the same British data base as used in the Andrews et al. study, and thus suffers from the same weaknesses. The authors reported an odds ratio (estimate of the risk ratio) for the relation of diphtheria-tetanus-pertussis vaccine to autism of 1.6 with 95% confidence limits of 0.7 and 3.3. The authors claimed their findings "provide further support for the view that exposure to mercury in vaccines is not the cause of the rising

incidence of autism." In reality, however, their findings are compatible with risk ratios ranging from 0.7 to 3.3, and thus provide no support for either side of the issue.

Heron et al., Pediatrics, 2004. This British cohort study examined various developmental disorders in relation to estimated mercury exposure from MCV. Due to the small size of the cohort, however, there were too few subjects to examine autism in general, let alone regressive autism: Only about 13,000 births had useable records (Heron et al., 2004, Fig. 1), which would have produced only 10-15 autism cases (based on rates reported by Andrews et al., 2004) and only 2 or 3 clearly regressive autism cases. Consequently, no analyses for autism were reported, and the study could provide almost no information regarding MCV and regressive autism.

Verstraeten et al., Pediatrics, 2003. This American study examined various neurodevelopmental disorders in relation to estimated mercury exposure from MCV in cohorts assembled from records of three health maintenance organizations (HMOs). Due to small numbers of cases in two of the HMOs, however, risk ratios for autism associated with the higher levels of estimated cumulative mercury exposure from MCV were presented for only one of the HMOs (Verstraeten et al., 2004, Table 5). The confidence intervals reported for these comparisons were very wide, ranging from (0.62, 1.46) to (0.55, 3.48). In other words, the results of the study appear compatible with risk ratios for autism in a very broad range, including values well above 1. This observation matches the conclusion reached by the first author of the study, who said "an association between thimerosal and neurological outcomes could neither be confirmed nor refuted, and therefore, more study is required" (Verstraeten, 2004).

Madsen et al., Pediatrics 2003, and Stehr-Green et al., American Journal of Preventive Medicine, 2003. Both of these articles review of ecologic data concerning the relation of trends in MCV and trends in general autism. Although these articles found no association, like other studies they did not separate regressive autism from other types. Both studies do note large trends in general autism diagnoses. These trends are large enough so that a specific association of MCV with regressive autism, if it existed, would have been completely submerged.

Fombonne et al., 2007. This article analyzed ecologic data relating MCV to "pervasive developmental disorder" (PDD), a very broad category which subsumes autism as well as other disorders. It is thus subject to larger dilution bias than the other studies when considering regressive autism. Furthermore, as the authors note, not all children in the "thimerosal exposed" cohorts in the study were exposed to thimerosal, which would result in further dilution bias. Finally, the article failed to present any estimate or confidence interval for the MCV-autism association, so it is not possible to tell from the reported results how large an association could be compatible with the data. From the data reported in the article, there would be about 60 cases of general autism in the study, so only about a dozen cases of regressive autism should be expected in the study. Thus the article is uninformative about a possible association of MCV with regressive autism, and a reanalysis of the study data would not be capable of detecting such an association if it existed.

8. Conclusion

As with most epidemiologic issues, full assessment of the literature examining MCV and autism would require a detailed quantitative analysis of the biases present in the different data sources, using methods such as those described in Greenland (2005) and Greenland and Kheifets (2006). The brief overview given above supports the idea that the association of MCV with autism is small or nonexistent. Nonetheless, if MCV is specifically associated only with a less common type of autism, such as regressive or clearly regressive autism, its association with general autism would likely be very small. Because the currently published evidence cannot rule out a very small association of MCV with autism in general, it cannot rule out an association of MCV with regressive autism, even one large enough to correspond to a risk ratio of 2.

Further progress would require analysis of a pediatric data source capable of distinguishing regressive autism from other types, and providing vaccination information and disease status on each child. In order to provide narrow enough confidence intervals to support a reliable assessment, the data source would have to have large numbers of cases with exposure to American dose schedules of MCV prevalent in the 1990s, as well as large numbers of cases with no MCV exposure over the same time period. It appears that such an analysis has not been published. Hence the question of whether MCV is associated with regressive autism remains unanswered by the current epidemiologic literature.

Sander Greenland Date: 8/13/2007

Sander Greenland

9. References

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