ORIGINAL

IN THE UNITED STATES COURT OF FEDERAL CLAIMS

OFFICE OF SPECIAL MASTERS

IN RE: CLAIMS FOR VACCINE INJURIES RESULTING IN AUTISM SPECTRUM DISORDER, OR A SIMILAR NEURODEVELOPMENTAL DISORDER, Various Petitioners, v. SECRETARY OF HEALTH AND HUMAN SERVICES, Respondent.	**PSC SUBMISSION RE EXPERT DESIGNATIONS AND EXPERT REPORTS** AUTISM MASTER FILE Special Master George Hastings

In his Order of August 11, 2005, Special Master Hastings directed the Petitioners' Steering Committee to submit two sets of materials in the Autism Master File of the Autism Omnibus Proceeding. First, the PSC was directed to file a statement from petitioners' expert, offering that expert's opinion as to why it is necessary to wait until at least late 2006 for the PSC to file substantive expert reports on general causation in the Omnibus Proceeding. Second, the PSC was directed to disclose a list of experts to be relied on in developing and presenting petitioners' case for general causation.

Enclosed with this filing are true and correct copies of two expert reports responsive to the Special Master's Order.[1] The first (Exhibit A) is from Sander Greenland, detailing why it is reasonable, prudent and necessary to wait until more evidence is available (including ongoing studies identified in petitioners' earlier filings) before requiring expert reports from the PSC.

[1] The PSC has pending a request for an additional two weeks; that is, until February 13, 2006, to submit petitioners' expert disclosure. Respondent consented to this PSC request.

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The second is from Dr. Mark Geier (Exhibit B), explaining why critical research relying on the Vaccine Safety Datalink will not be finished and available until late 2006 at the earliest.

For reasons described by plaintiff's experts, and for all of the reasons described by petitioners in their brief of June 14, 2005, and the exhibits attached to that filing, the PSC respectfully requests an ongoing extension of time in which to file expert reports in the Autism Omnibus Proceeding, at least until late 2006. The extension of time is within the discretion of the Special Master pursuant to 42 USC §300aa-12(g) and 42 USC §300aa-21(b), and is necessary given the proffered testimony of petitioners' experts submitted today.

Petitioners again urge the Special Master not to let these legal proceedings get ahead of the science. Rather, petitioners urge the Special Master to recognize that a significant body of relevant, peer-reviewed, independent, published science, unavailable today, will likely will be available to petitioners, respondent, and the court within the next twelve months. For that reason, petitioners' expert reports should not become due until late 2006 at the earliest.

DATED this 30th day of January, 2006

WILLIAMS LOVE O'LEARY CRAINE & POWERS P.C.

By: _____

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Report to Special Master Hastings regarding the propriety of delaying consideration of general causation issues in the Omnibus Autism Proceeding

Sander Greenland, M.A., M.S., Dr.P.H., C.Stat.
Professor of Epidemiology, UCLA School of Public Health
Professor of Statistics, UCLA College of Letters and Science

1. Introduction

I herewith provide for consideration my scientific evaluation regarding the propriety of delaying consideration of general causation issues in the Omnibus Autism Proceeding. The views I submit concern aspects of the scientific topic of causal inference, including consideration of evidence regarding causation, and related scientific aspects of policy formulation, as opposed to legal matters.

2. Qualifications

My qualifications for this aspect of 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-Raven, 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 most recent (2000) edition of the Federal Judiciary Center's *Reference Manual on Scientific Evidence*.

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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 160 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 I am 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 over 270 peer-reviewed articles, 60 published letters, over 120 published abstracts, and over 30 other publications (inlcuding book chapters and encyclopedia entries).

i) I have served three terms as an Associate Editor of the *American Journal of Epidemiology* (1984-98), and now serve as an Associate Editor of *Statistics in Medicine* (1985-present), the *European Journal of Epidemiology* (2003-present), and the journal *Epidemiology* (1989-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. On The Need for the Further Evidence Regarding Thimerosal and Autism: General Scientific Considerations

In considering divergent views on a matter of general causation, it is important to recognize several general principles of scientific inference in general and of causal inference (the process of evaluating causal hypotheses) in particular.

It is now widely accepted by leading scientific methodologists (including both scientists and those engaged in studies of science, including philosophers of science) that as *matter of science*, scientific hypotheses are never established with complete certainty. Nonetheless, knowledge is gained by precise controlled experimentation (wherein the investigator manipulates the conditions to which the units are subjected – where the units may be particles, animals, humans, etc.) designed to seek refuting evidence, or find how or where the hypothesis breaks down. If little or no such evidence is found after many decades of cleverly designed experimentation, some hypotheses become so well established that they come to be regarded as "facts," which is to say, regarded as true enough to proceed *as if* one were certain, at least to within the level of precision of the experiments.

This model of science works well in experimental sciences such as physics, chemistry, and many branches of life sciences. It reaches severe limits in health and social sciences, however, where for many topics (including thimerosal and autism)

experiments cannot be done on the units of interest – people – due to ethical or logistical reasons. In that case the best direct evidence one can obtain is called observational, by which is meant evidence obtained by passive observation, without experimental intervention by the investigator. The problems are compounded by the fact that the available observational data may not have been collected within experimental standards of accuracy.

All other things being equal, observational evidence is widely regarded as inferior to experimental evidence. Three major reasons are often given for this inferiority:

1) Confounding: Those who receive treatment may differ from those who do not in ways that affect disease.

2) Selection bias: Forces may affect entry into the data base of the study in ways that generate spurious relations or mask true relations of treatment to disease.

3) Measurement error: Inaccuracies or mistakes in measurement of treatment or outcome (disease) can again generate spurious or mask true relations.

(see e.g., Rothman and Greenland, 1998, Ch. 8). While these problems can occur in experiments with protocol violations, they are the norm in observational studies. Consequently, for an enormous number of hypotheses regarding health outcomes, evidence must remain limited, and nothing like the status of fact is warranted.

Nonetheless, actions are often taken (and sometimes must be taken) *as if* a hypothesis were correct or incorrect, on the basis of current, uncertain, and incomplete evidence. At other times, however, the evidence is taken as so incomplete that to behave

as if the hypothesis is correct or incorrect is seen as courting disaster, or at least is premature. Because human data that record both thimerosal and autism is observational, the question at issue in the present decision is whether the current evidence justifies an *as if* approach (regardless of whether it is a decision to behave "as if correct" or "as if incorrect" that thimerosal causes autism).

There are many examples from recent history that parallel the present question and provide some valuable guidance. A famous one is the administration of unopposed estrogens to menopausal women, which is now well accepted as the cause of an epidemic of endometrial cancer (cancer of the body of the uterus) in the 1970s. As recounted in many sources (e.g., Rothman and Greenland, 1998, p.139), at the time the first studies making the connection appeared, there was considerable objection on the grounds that the observed relation was merely the result of increased detection of endometrial cancer among estrogen users (in the above general scheme, the objections claimed that the system was failing to adequately measure cancer occurrence among the untreated, and that failure had led to a spurious association of treatment with disease).

Despite these objections, it was viewed by many physicians as prudent to proceed *as if* unopposed estrogens were as dangerous as they appeared and to cease prescribing them, or at least carefully monitor women who were using them. The reason for this decision is two-fold: (1) the benefit of use was strictly in relief of symptoms, so stopping the treatment seemed to demand little cost other than a woman's discomfort, whereas the cost of continued was risk of serious illness; (2) the size of the association observed was enormous: a 10-fold or more increase in endometrial cancer rates among women taking unopposed estrogens. To explain an association so large, the effects of the proposed

alternative explanatory factor (here, enhanced detection) must be as powerful as the observed association of treatment with the disease. It was not believed credible that detection effects could be so enormous, and indeed when data on those effects became available they revealed incomplete detection incapable of explaining more than a small fraction of the observed association (because surveys found that only a small minority of cancers among the untreated were being overlooked).

The relevant point here is that, despite uncertainty about the true source of the observations linking unopposed estrogen to endometrial cancer, nonetheless alternative hypotheses appeared much less able to explain dramatic observations than did the causal hypothesis at issue; and the cost of stopping treatment did not appear serious. It thus appeared wise to proceed *as if* the hypothesis that unopposed estrogen caused endometrial cancer were true. (In fact the same sort of argument was among those used to justify the Surgeon General's first warning against smoking in 1964.)

Now consider an opposite example, recounted at length in the June 2004 issue of the *International Journal of Epidemiology*, in which early action favoring wider use of a treatment eventually came to appear premature and incorrect. The controversy concerned the use of hormone replacement therapy (HRT), of which unopposed estrogen treatment was an early special form. In 1990, renowned Harvard investigators summarized a literature more extensive than that now available for thimerosal and autism, and concluded that HRT (which was again largely prescribed for relief of menopausal symptoms) reduced risk of heart disease among treated women. Again the human data linking HRT to heart disease was solely observational, yet some physicians began prescribing HRT for prevention of heart disease. Two key differences from the cancer

example, however, were (1) the cost-benefit equation was less clear: the action--expanding treatment-- might prevent heart disease, but carried serious risks of its own, possibly including cancer; (2) the observed association was not strong (less then twice the risk of disease observed among the untreated compared to the HRT treated). Thus, some health researchers objected that (unlike in the cancer example) the alternative explanations (1-3) could just as easily account for the observations as could the hypothesis that HRT prevented heart disease; and, in view of possible negative consequences of the proposed action (of expanding treatment), it was unwise to proceed *as if* HRT indeed prevented heart disease. Eventually, results from human experiments with HRT became available that suggested HRT in reality offered no protection, and possibly even increased the risk of heart disease in women.

The examples above and many others point to the following guidelines on the matter of whether action or delay pending further evidence is warranted. First, as do wise physicians and scientists, one needs to consider all possible states of reality and decisions from a cost-benefit perspective, and recognize that any current *as if* choice may eventually be revealed as erroneous. Whether the choice defensible is not then a matter of whether the *as if* choice is right or wrong, but whether it hedges wisely, which is to say allows for the consequences of error.

4. On The Need for the Further Evidence Regarding Thimerosal and Autism: Considerations Specific to the Present Matter

In simplest terms, the hypotheses at issue are that thimerosal has not caused any autism (the null hypothesis) and its opposite, that thimerosal has caused autism (the causal hypothesis). It is not known for certain which of these hypotheses is correct;

logically one and only one of them can be correct, but scientifically the human evidence is observational and cannot establish either with absolute certainty. Thus, one must consider the consequences of any decision under both hypotheses. Further evidence regarding these two competing hypotheses can be employed in deciding whether it is best to proceed as if the first hypothesis is correct or the second is correct. Thus, from a scientific perspective, the key advantage of deciding to wait for further evidence is that it reduces the risk that any *subsequent* decision based on the totality of evidence will turn out to be an incorrect decision.

This reduced risk of erroneous decision is a benefit of waiting. Equivalently, a cost of not waiting is an increased risk that a subsequent decision will turn out to have been incorrect. This tangible cost of not waiting has to be weighed against the cost of waiting. While the cost of waiting in each of the above examples could be measured in health consequences (higher cancer risks for delaying cessation of unopposed estrogens; lower cancer risks for delaying the wider adoption of HRT), no such health cost appears associated with waiting for further evidence regarding thimerosal and autism.

The arguments thus far favor waiting, as is often the case in science-oriented analyses, but are purely qualitative and general. Policy analyses and decisions require at least some quantitative considerations, which here translate into the value of the information that would come available if one waits, relative to the information available thus far. One should recognize that the value of information to come (as opposed to that already available) is necessarily not known with certainty (since that cannot be established until after the studies in question are completed, reviewed, and published). Nonetheless, one can compare the type of studies done so with those to come to obtain a

sense of whether the future information will be merely incremental, or have some qualitative superiority to current information. In this regard the following points seem salient.

When properly analyzed, comparative studies that link treatment and disease status in *individuals* (comparative individual-level studies, typified by what are known as case-control studies and cohort studies) are generally regarded as providing stronger evidence than "ecologic" or group-level (aggregate) data, which by definition associate only rates of treatment with rates of disease over place or time. This view arises because aggregate studies are subject to various artifacts of grouping, and inevitably rely on unvalidated records that may have a high error rate for both treatment and disease reporting (for details see e.g., Greenland, 2004). Individual-level studies escape grouping artifacts because for each member of the study both the treatment status and disease status is determined; hence if one sees that treatment and disease are positively associated in the study, one can be sure that this arises because those treated reported more disease than those untreated (in a cohort study), or those with disease reported more treatment than those without disease (in a case-control study). Furthermore, such studies can incorporate protocols to validate treatment histories and diagnoses; they thus can minimize errors in treatment and (especially) disease classification, which reduces consequent concerns about distortions due to measurement error.

Regardless of which type of study is superior, the two types of data (individual and aggregate) are qualitatively different, and any paucity of one type of data is a source of uncertainty. In their literature search and summary, Smeeth et al. (2004), report only four published peer-reviewed studies involving individual-level data on vaccine and

autism or autism-spectrum disease which they considered conducted well enough for inclusion in their meta-analysis. While the total number of subjects in these studies is large, the number of studies is not. In evaluating a topic, it is important to have a number of different studies by different teams in different settings, to reduce vulnerability to misleading conclusions due to a problem shared by several studies (pervasive bias). For example, by the time the Surgeon General's office issued its first warning about smoking and lung cancer, there were over 20 published comparative individual-level studies. As another example, one similarly controversial to vaccines and autism, there are today at least 15 useable comparative individual-level studies of magnetic-field exposure and childhood leukemia, and that still may be insufficient for secure policy formulation (Greenland and Kheifets, 2006).

It is also important to have supporting theory and relevant laboratory data regarding proposed mechanisms of action. The importance of mechanistic research is illustrated by the above two examples: The Surgeon General regarded the coherence of the entire body of evidence as crucial in reaching a decision that action against cigarette use was needed, and made clear that this action was not predicated on epidemiologic evidence alone. In contrast, a major reason that no official actions regarding residential magnetic fields have been taken is the absence of laboratory evidence supporting an effect or even indicating how an effect of fields at the observed strength could occur.

Fortunately, as has been documented in other filings in the current matter, there are several comparative individual-level studies of thimerosal and autism that are underway or planned, including the Vaccine Safety Datalink (VSD) study and other CDC-funded efforts, as well as laboratory research regarding theorized mechanisms of

action. For several reasons, the proposed expanded VSD study is of particular interest and importance.

Whether justified or not, deep suspicions have arisen regarding the published CDC report (Verstraeten, 2003) from the VSD database, due to a series of events that preceded publication (among them, changes in data, methods and results that occurred between the initial unpublished analysis from early 2000 and the 2003 publication, including exclusion of data from a large HMO; move of the lead author to Glaxo Smith Klein; and refusals to provide access to the data). Further analyses of the data by independent parties would address concerns that the published results had been manipulated to conceal thimerosal effects.

Regardless of their origin, the published VSD study did have many questionable aspects of analysis that may have obscured effects, if any effects were present. Most of these problems were pointed out by Dr. Harland Austin at the Omnibus Hearing in September 2004, and I can corroborate his testimony. Of great concern to me is the failure to pool results across the HMOs. As Dr. Austin stated, pooling is standard practice, and should be done using sound techniques. Instead, the published VSD study used a very small HMO C (Pilgrim) to "validate" findings from the much larger HMO B (Kaiser) and A (Puget Sound), which as Dr. Austin noted, is not good practice because it has extremely low power (is very likely to miss effects, compared to correct pooling methods). The published results from HMOs A and B also employed questionable restrictions on the comparison children, to clinic or emergency-room attendees, which further reduces power. The authors of the published study also excluded comparisons when the case total was less than 50, on the grounds of "power considerations." This is a

fallacious reason for exclusion, because all power considerations after data are collected can be addressed by examining the confidence intervals for the comparison; the latter show what effect sizes cannot be reasonably excluded, based on the analysis.

In addition to having serious analytic problems as just enumerated, the published VSD study did not employ births beyond 1998, and did not fully justify exclusion of data from all the other VSD HMOs. The use of births in subsequent years would further enhance the power to detect effects, if any are present, as would use of data from other VSD HMOs as appropriate. Thus, a new analysis of the VSD database addressing the analytic problems of the published analysis, and incorporating as much data from subsequent years and other HMOs as feasible and justifiable, would both allay concerns about impropriety of the 2003 publication, and provide considerable additional scientific information.

5. Conclusion

In light of the above considerations, I respectfully submit that it would be prudent and advisable to allow experts in this matter to wait for further scientific evidence, as will be provided by forthcoming studies; such evidence will better inform any decisions based on general causation considerations.

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1/30/06

To: Thomas B. Powers
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From: Dr. Mark R. Geier
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Re: Status Update Regarding Vaccine Safety Datalink Studies

Dear Mr. Powers,

As requested, I have prepared some information for the court pertaining to my present attempts to access and publish results of studies based upon examination of the Vaccine Safety Datalink (VSD) database.

My original researcher proposals to analyze the VSD database were submitted to the Centers for Disease Control and Prevention (CDC) back in August 2002. I believed at this time (and consistent with other research programs that I have undertaken) that it would require several months for the CDC to provide me access to the entire VSD database, and that following several months of analyses, a scientific paper would have been able to be submitted for publication regarding analyses from VSD database within one year of my initial request to access data in the VSD database.

My original submitted research proposals submitted in 2002 consisted of requests for extensive data from the VSD database, including information that would have allowed for study of potential association between Thimerosal administration at a wide range of exposure and neurodevelopmental disorder outcomes including autism. After almost a half-year delay on the part of the CDC my proposals were approved by the CDC,

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and I subsequently submitted and received approval of my proposals from several different Institutional Review Boards (IRBs) of several of the Health Maintenance Organization (HMOs) that participate in the VSD database.

Despite having the appropriate approvals from the CDC and the HMO's IRBS to access data in the VSD database since mid-2003, at this point, I still have not been able to adequately collect data sufficient to conduct the analyses described in my proposals. Data collection has been slowed by significant problems with access to the VSD database including: (1) Data has been found to be missing; (2) Data has not been assembled in a usable form; and (3) Data has been found to be possibly destroyed.

At present, attempts are still underway to attempt to collect VSD data, and then complete studies based upon analysis of the data. It appears under the current constraints to access, and until the problems with the data itself are resolved, there is very little probability that I will be able to complete and publish results from analyses of the VSD database by the end of 2006.

Sincerely,

Dr. Mark R. Geier

Dr. Mark R. Geier

EXHIBIT B

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CERTIFICATE OF SERVICE

I hereby certify that on January 30, 2006, I served the foregoing **PSC SUBMISSION RE EXERT DESIGNATIONS AND EXPERT REPORTS** on the following individual(s):

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Electronically and by United Parcel Service, next morning delivery.

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