EXHIBIT 1

DECLARATION OF DONNA K. ARNETT, PH.D., M.S.P.H.

My name is Donna K. Arnett. I am over twenty-one years of age, am of sound mind, have never been convicted of a felony, and am otherwise competent to make this Declaration. I have personal knowledge of all factual statements contained herein and all such factual statements are true and correct as outlined herein in this declaration-report.

A. Qualifications and Expertise

I am a cardiovascular epidemiologist with over twenty years of experience in the design, conduct, and analysis of epidemiologic studies. Since 1994, I have worked in leading academic research institutions for cardiovascular epidemiology and I have taught postgraduate (masters and doctoral levels) courses in theory, design, and analysis of epidemiologic studies.

I received a B.S.N. degree in 1981 in nursing (magna cum laude) and an M.S.P.H. degree in 1987 in epidemiology and biostatistics from the University of South Florida. In 1992, I received a Ph.D. in epidemiology from the University of North Carolina and was elected into Delta Omega, the honor society for public health. Prior to my graduate training, I worked as a critical care nurse for 5 years and was CCRN certified (i.e., critical care registered nurse) and as a research coordinator for pharmaceutical clinical trials at the University of South Florida for three years. My doctoral research was focused in the area of cardiovascular epidemiology. In 1992, I was awarded my first peer-reviewed grant, a post-doctoral fellowship, and worked two years completing the fellowship at the University of North Carolina at Chapel Hill. From October, 1994 through August, 2004, I rose from a tenure-earning assistant professor to a full professor with tenure as well as holding an endowed chair in epidemiology at the University of Minnesota. During that time, I directed large, complex, multi-center epidemiologic studies funded from the National Institutes of Health, as well as a National Institute of Health T32 Training Grant and cardiovascular genetic epidemiology. At the time I left the University of Minnesota, I was ranked in the top 5% of all National Institute of Health researchers. Since 2004, I have served as chairman and a tenured professor of epidemiology at the University of Alabama at Birmingham, where I have maintained a strong research program. I currently am Principal Investigator for five National Institute of Health projects.

As further evidence of my qualifications and expertise in the field of epidemiology, I am an elected fellow of the American Epidemiologic Society, and serve as editor for the highest ranked journal in epidemiology, namely the *American Journal of Epidemiology*. In addition to serving on numerous research peer-review committees for multiple organizations, including but not limited to, the Veterans Affairs, the American Heart Association and the National Institutes of Health, I was named to the prestigious post of chair for the NIH Cardiovascular and Sleep Epidemiology study section for 2006-2008. I am also a candidate for the Chairman of the Department of Epidemiology at the Johns Hopkins University, the top ranked School of Public Health in the country. My interview is scheduled February 23-24, 2009.

B. Responses to Particular AstraZeneca Assertions

I have reviewed the brief of AstraZeneca that criticizes the veracity of the epidemiologic methodology employed as well as the timing and content of my opinions, and I herein offer a response to these criticisms.

1. AstraZeneca claims that I fail to extensively review the literature and use of flawed methodology.

In addition to several allegations that I failed to employ scientific methodology in forming my opinions in this case, AstraZeneca states that I failed to conduct a complete review of the literature because I "ran out of time," and by my own admission, was too rushed to conduct a methodologically sound analysis. These assertions are incongruent with my expert report, testimony, and experience as an educator of epidemiologic methods and a researcher who employs sound epidemiological principles in her studies. Prior to my deposition in October 2008, I had spent in excess of 80 hours reviewing the New Drug Application (NDA) for Seroquel submitted by AstraZeneca to the FDA in July, 1996, clinical study summaries posted on the AstraZeneca website, various internal documents created by AstraZeneca, clinical study reports, and published observational studies. Since October, I have spent approximately 80 additional hours reviewing additional materials including AstraZeneca's June 2008 Metabolic Parameters submission to the FDA, clinical study reports, declarations by AstraZeneca's experts, and other related materials.

As stated in my expert report (p. 3) and my deposition (p. 160, lines 1-4, p. 255 lines 9-14), my general causation opinions were formed mostly from the placebo-controlled randomized studies conducted as part of the NDA. In fact, at least half of my report was devoted to data derived from the NDA. This approach is in congruence with sound epidemiologic methodology. On page 6 of my report I indicate:

> "Randomized, double-masked, placebo-controlled clinical trials are the optimal design for testing a hypothesized association between an exposure (or treatment) and disease because such studies offer the best control for confounding (i.e., variables that are associated with the disease and associated with the exposure) and provide for the optimal test for temporality (i.e., exposure precedes disease). Placebo controlled studies are the gold standard for evaluating the risks and benefits of a new treatment."

The assertion that the experimental studies, such as the placebo-controlled randomized studies, are the most optimal design to test causal hypothesis is widely held among epidemiologists. Because of randomization of subjects and controlled administration of the agent under study, experiments are considered more useful than observational studies to demonstrate cause-effect relationships. Woodward, M., Epidemiology, Study Design and Data Analysis, 2nd Ed 2004. Chapman and Hall/CRC Texts in Statistical Science Series. 2004, p. 337). The U.S. Preventive Services Task Force (see www.ahrq.gov/clinic/uspstmeth.htm) has established a ranking for the evidence about effectiveness of treatment, and has deemed evidence from the randomized controlled trial as the best level of evidence, Level 1. According to sound epidemiological

principles, I relied most heavily on Level 1 evidence. However, I supplemented the Level 1 evidence with evidence Level II-B studies regarding Seroquel and diabetes which support the findings from the randomized controlled clinical trials. This does not in any way indicate that I formed my opinions before I completed review of the literature. In fact, it suggests that I used appropriate epidemiologic rigor into writing my opinions.

As stated in my deposition, the method I used to derive the list of observational epidemiologic studies was a PubMed search for studies that contained the words Seroquel (quetiapine) and diabetes and the title or abstract. PubMed is the primary source used by epidemiologists for literature searches, and the one I routinely use in my research. All published cohort or case-control published manuscripts were included in my report. The two articles mentioned by AstraZeneca in its motion that were published before Seroquel was approved for marketing in the United States (Colditz 1995 and Chan 1994) were the product of a different search I conducted for studies on weight gain and diabetes.

I also applied the Bradford Hill criteria in assessing the scientific evidence regarding Seroquel and diabetes. As recognized in the Reference Manual of Scientific Evidence, this is a valid methodology for assessing causality and explores recognized considerations. The Bradford Hill considerations, in determining general causation and whether exposure is the cause of the disease, consider: experimental method; temporal relationship; replication or consistency of results; strength of the association; dose-response relationship; biological plausibility; specificity; analogy and consistency or coherence with other relevant knowledge. With respect to the experimental method, the randomized controlled clinical trial is the optimal design for assessing cause-effect relations because, with a large sample size, the experiment removes confounding as an explanation for the observed association. In the assessment of the metabolic consequences of Seroquel, in my report and my deposition, I indicated that I relied most heavily on evidence from the placebo-controlled randomized studies. The randomized clinical trial also provides the best evidence with respect to the temporal association between Seroquel exposure and metabolic abnormalities: the trials in the NDA, and/or trials 126 and 127 demonstrated that initiation of Seroquel treatment was associated with weight gain, (Report, p. 9), and increased triglycerides, fasting glucose, and HOMA. (Report, pp. 9, 11). Findings, such as weight gain, were consistent across the randomized studies conducted in a variety of clinical populations. From my report, the strength of the association varied across the metabolic indices evaluated: for clinically significant weight gain, but were large: the largest relative risk observed was 4.77, (Report, p. 5), for a glucose value > 200 mg/dl was 4.87, (Report, p. 10), and for diabetes was 2.02. (Report, p. 10). These findings are consistent with strong effects and were found in the strongest of study designs, the randomized controlled trial. Dose-response was also noted with respect to weight gain in Study 13 of the randomized controlled trials provided in the New Drug Application. (Report, p. 5). Several biologically plausible mechanisms were provided in my report that account for the increased metabolic risk with Seroquel. (Report, p. 3). With regard to specificity, this guideline suggests that the exposure has one effect on a population. In the case of drug exposures which have multiple effects that span efficacy to adverse effects, the utility of specificity in assessing causation is difficult, Sir Bradford Hill alludes that specificity is evaluated by the risk of one outcome being more common with the exposure than other outcomes. Since I have focused on the metabolic consequences of Seroquel, I do not employ specificity in my causal assessment. In contrast, I do rely on the guideline of coherence, which

indicates that a causal conclusion should not fundamentally contradict present substantive knowledge. In fact, the totality of the substantive findings across the multiple domains of metabolic toxicities associated with Seroquel in multiple randomized controlled trials support the criterion of coherence. Finally, with respect to analogy, as Susser has indicated:

> "When one class of causal agents is known to have produced an effect, the standards for evidence that another of that class produces a similar effect can be reduced."

As early as May, 2000, the FDA expressed concerns to AstraZeneca regarding the metabolic consequences of atypical antipsychotics and requested information from AstraZeneca regarding diabetes, indicating that this affect may be a class affect from these agents.

The assertion that I "ran out of time" is patently absurd and was taken completely out of context from my deposition. In fact, it referred to the section where I described the methods used to evaluate the weight data from the AstraZeneca website. In fact, what I referred to in this section was related to the findings regarding weight. What I had observed in the randomized clinical trials from the AstraZeneca website is consistent with what I had found in the NDA, namely, that Seroquel was associated with significant increases in weight:

> Q. Your weight chart here, Table 1, you created this based on the clinical trial summaries, right, that were on the Web site?
> A. Yes.
> Q. Am I right that there were many more than just 11 clinical trial synopses on the AstraZeneca Web site?
> A. Yeah. I took them in sequential order from top to Number 11 until I ran out of time.

(Deposition, pp. 177-178). Additionally, I recently completed service for a Ph.D. dissertation committee where the pharmacogenetics of atypical antipsychotic medications and diabetes was the student's topic. As part of my academic responsibility, I reviewed literature generally related to atypical antipsychotic use and diabetes prior to this litigation

2. AstraZeneca criticizes my review of the NDA.

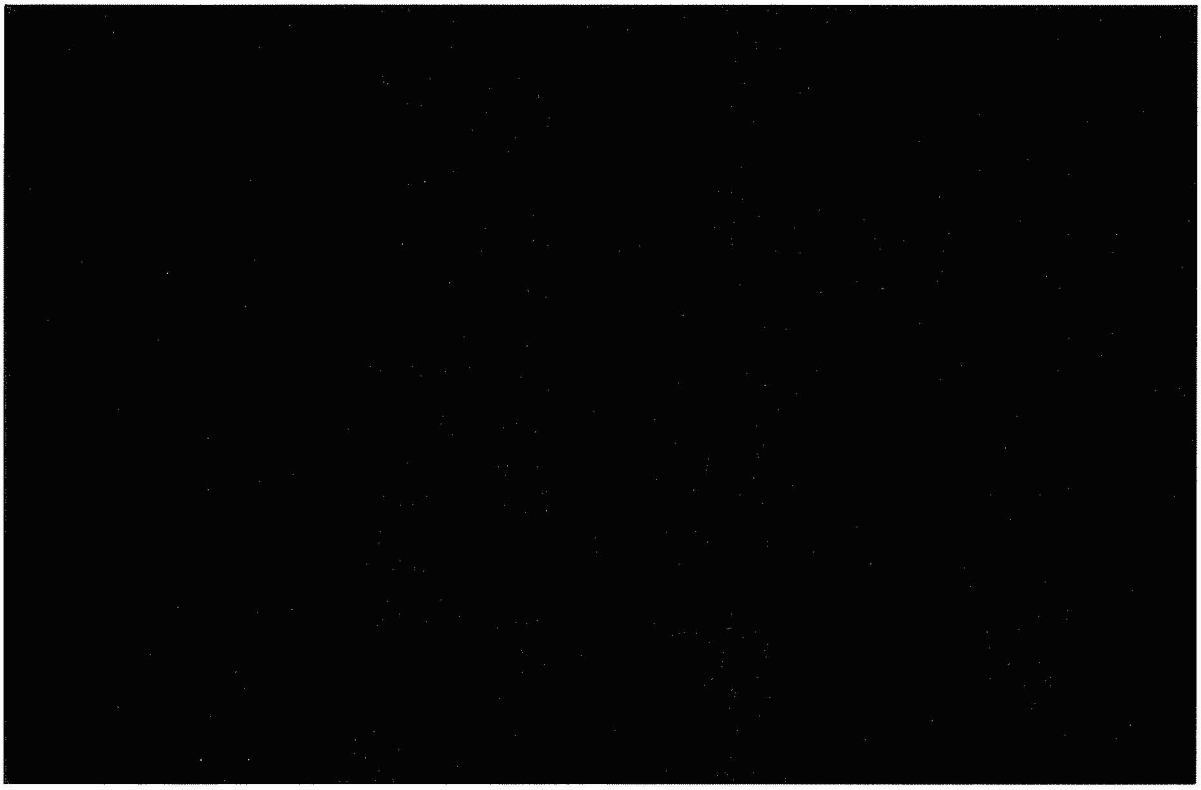
The publications from the earliest AstraZeneca clinical trials that I reviewed did not contain adequate information to evaluate the metabolic risks associated with Seroquel. (REF: Small JG et al, Quetiapine in Patients with Schizophrenia. Arch Gen Psych 1997;54:549-557; Borison RL et al. ICI 204,636, An Atypical Antipsychotic: Efficacy and Safety in a Multicenter, Placebo-Controlled Trial in Patients with Schizophrenia. J Clin Psychopharmacol 1996; 16:158-169). Therefore, I specifically requested the NDA so I could fully evaluate the range of metabolic risk factors measured and the placebo-controlled clinical trials, and the impact of Seroquel on these risk factors.

I was provided with a hard drive that contained the entire NDA as it was apparently produced by AstraZeneca. The NDA was incredibly voluminous. I have since been told that it consists of

approximately 340,000 pages. The NDA documents were not indexed through a key word search or table of contents, nor were they provided by AstraZeneca in an easily searchable format. The files were aggregated within a single "file" descriptor in such a way that files were essentially hidden from view. For example, a "file" would be labeled within the NDA disk, but within that file, multiple pages that contained other files that were not listed on the contents of that particular file. Contrary to AstraZeneca's assertion that I merely skimmed the NDA, I spent a number of hours painstakingly searching the NDA for the relevant safety information and finally discovered that a wide range of metabolic risk factor data, such as dyslipidemia, glucose, and thyroid hormone levels, were collected in the randomized controlled trials included in the NDA. These data were not readily available in the published literature. The aggregate of these Level 1 evidence data indicate a metabolic toxicity from Seroquel.

Following the review of the NDA, I also evaluated randomized controlled trial study summaries posted on the AstraZeneca website. These reports, which are summarized in my expert report (p. 8), demonstrate consistent weight gain findings in comparison to those reported in the NDA Integrated Safety Report. Given the consistent, clinically relevant weight gain (as defined by Astra Zeneca as a greater than 7% change in body weight in response to Seroquel treatment), I did not pursue lower levels of scientific evidence from observational epidemiologic studies with respect to weight or other metabolic toxicities, with the exception of type II diabetes. This approach is consistent with sound epidemiological principles.

3. [redacted]



4. AstraZeneca expresses concern that I did not review clinical study reports.

Because of the method in which the NDA was provided, it was nearly impossible to identify the individual clinical study reports. This is not a concern with respect to the formation of my opinions. The integrated safety report provided by AstraZeneca to the FDA summarized all Phase I, II and III study safety measurements that were included in the New Drug Application. Therefore, unless AstraZeneca withheld information from the clinical reports for this Integrated Safety Report, all safety information collected in phase I, II and II studies were reviewed and evaluated in the formation of my opinions.

Prior to my deposition, I had actually reviewed some of the clinical study reports though at the time; however, at that time I was unable to recall which ones. (Deposition, pp. 91-92). Nevertheless, I have reviewed the 4,582 page clinical study report for Study 125 which is described below. With respect to Studies 126 and 127, I reviewed the clinical trial summaries for those studies that AstraZeneca made available on their website and the June 2007 *Clinical Overview: Glucose dysregulation in patients treated with Seroquel*, specifically Tables 5.1.1.3, which contained glucose data from Studies 126 and 127 and was provided to the FDA. AstraZeneca criticizes me for not knowing of certain potential confounders associated with those studies. AstraZeneca's criticism seems unwarranted given that AstraZeneca failed to disclose this information even to the FDA. Nevertheless, I am in the process of reviewing the Study 126 and 127 clinical study reports, which are 6135 and 6434 pages respectively, and will supplement my report should the data contained therein alter the substance of my opinions.

5. AstraZeneca suggests that I appeared to be unaware of Study 125's existence and have not taken Study 125 into account.

As I testified during my deposition, I did review Study 125 though it was not a placebo-controlled randomized clinical trial, but rather, was an open-label study designed to contrast the effects of Seroquel on a glucose metabolism in comparison to two of their active comparators. (Deposition, p. 32). Open-label studies fall into the Level II-1 evidence according to the U.S. Preventive Services Task Force (see www.ahrq.gov/clinic/uspstmeth.htm), a level of evidence that falls below that of the double-blind randomized clinical trial, and one of the reasons I did not include it in my Expert Report. In addition to these limitations, as with other Seroquel randomized, controlled trials, there was a larger drop-out among Seroquel users, 59/168 compared to only 23/169 for olanzapine users and 40/173 for risperidone users, raising the possibility that people who had metabolic side effects could have dropped out more often among the Seroquel users. Nonetheless, even though the primary outcome measure (i.e., the change at 24 weeks of the "area under curve" in a 2 hour oral glucose tolerance test) the secondary results indicate statistically significant increases in both mean fasting blood glucose (3.19 mg/dl) and HbA1c (0.122%), showing that Seroquel impacted regulation of glucose in a fasting state. Fasting C-peptide (a measure of endogenous insulin production and an indicator of insulin resistance) increased. Further, patients taking Seroquel experienced a mean weight gain of 3.65 kg (8 pounds) in 24 weeks. In aggregate, these findings lend Level II-1 evidence in support of the effects of Seroquel on metabolic toxicities, including weight gain and insulin resistance.

6. AstraZeneca claims that I improperly relied on case reports.

As I stated during my deposition, case reports alone cannot establish causality. However, case reports along with evidence from studies, can begin to build a case for causation and they are properly considered as one piece of evidence in epidemiology. Indeed, the Food and Drug Administration's MedWatch program (http://www.fda.gov/medwatch/What.htm) is essentially a series of case reports for detection of safety information about drug products in an effort to detect safety signals. The only case report series included in my expert report was from the MedWatch program.

7. AstraZeneca suggests that I failed to account for confounding factors.

I am aware of the importance of assessing confounding in the design, analysis, and the interpretation of epidemiologic studies. I currently teach the second course in a series of three graduate-level epidemiology methods courses where design, analysis, and interpretation of confounding is the primary learning objectives. For confounding to occur, the confounder must be associated with both with the disease (e.g., diabetes), and the exposure (e.g., Seroquel). If the confounder is associated with only the exposure or only the disease, it cannot confound the exposure-disease association. This is the advantage of randomization as a design strategy to reduce confounding and why I relied most heavily upon randomized, placebo controlled trials in rendering her opinion: through randomization, the association between the exposure (i.e., Seroquel) and the confounder (i.e., a family history of diabetes as an example) is broken. In this particular example, a positive family history of diabetes is made equal in Seroquel users versus other treatments, and confounding does not occur even though a positive family history is associated with diabetes.

AstraZeneca states, "Dr. Arnett fully recognizes the importance of controlling for alternative causal factors." This is a wrong definition of confounding and implies that I considered only one of the two requirements for confounding, namely, that the confounder is associated with the disease. I purposely sought the most "confounding-free" source of data available to render her opinion, namely, the randomized clinical trial. Moreover, it is unclear the degree to which confounding biased the results from the observational epidemiological studies. For confounding to occur, Seroquel use would have to be associated with the confounder, such as a positive family history as an example, in order for the Seroquel – diabetes association to be biased. In the observational studies, that would require that health care providers prescribed Seroquel more often than other antipsychotic drugs based on the presence of the confounder.

8. AstraZeneca suggests that I violated one of the fundamental principles of epidemiology by improperly and frequently relying on statistically insignificant data.

In my report, I presented numerous statistically significant findings with respect to weight gain, triglyceride increase, measures of insulin sensitivity, waist circumference, and thyroid abnormalities in relation to Seroquel treatment. Additionally, I presented epidemiologic studies that indicated the Seroquel was associated with statistically significant increased risk of diabetes. Nonetheless, the assertion that statistical significance is an exclusive requisite for evaluation of causation is a frank misrepresentation of accepted epidemiologic methods. In the recent text,

Modern Epidemiology, Third Edition, by Kenneth Rothman et al., page 159 states (ref Rothman, Greenland and Lash. Modern Epidemiology, Third Edition, Lippincott Williams and Wilkins 2008, pages 157-159):

> "Confidence limits and P value functions convey information about size and precision of the estimate simultaneously, keeping these two features of measurement in the foreground. The use of a single P value -- or worse dichotomization at the P value into significant or non-significant -- obscures these features so that the focus of measurement is lost. A study cannot be reassuring about the safety of an exposure or treatment if only a statistical test of the null hypothesis is reported. As we have seen, results that are not significant may be compatible with the substantial effects. Lack of significant alone provides no significance against such effects."

The authors state that the confidence limits around a point estimate must be interpreted with respect to the point estimate, namely that point nearer to the center of the range are more compatible to the data of them than points farther away from the center. As I testified during my deposition, this would mean that the true effect of Seroquel on diabetes would be nearer to the point estimate of 2.02 rather than from the extremes of the 95% confidence limits. (Deposition, pp. 286-287). This value is comparable to a range of point estimate derived from the observational epidemiologic studies (e.g., ranged of 1.15 – 3.02 from my report, page 11).

9. AstraZeneca expresses concern that I did not calculate post-hoc power calculations.

In light of the totality of statistically significant data discussed in my export report and deposition concerning the effects of Seroquel on diabetes risk, I did not find it necessary to calculate statistical power in the setting of the consistent and statistically significant findings previously cited. In my twenty years of work in epidemiology, and having served as Chair of the NIH Study Section on Cardiovascular and Sleep Epidemiology Study Section where I routinely evaluate statistical power, I assert that I have a solid understanding of the factors that contribute to statistical power. In the case of the specific calculation requested by AstraZeneca's counsel, the idea that the study was adequately powered was due to the number of events required for adequate power (i.e., the error or claiming the null hypothesis is true when indeed it is not). If therefore, I did not invest time in calculating the power as it was most assuredly low.

Though I maintain it is unnecessary to calculate the power of the relative risk calculations, I have now done some of these calculations (to the extent they could be done) at the request of plaintiff's counsel and to demonstrate my theory. In aggregate, exactly as I assumed would occur based on my years of experience in analysis as an epidemiologist, the power was never within a range considered appropriate for making inferences (i.e., power of at least 80%) to say that a non-significant finding is real. I would only report that the power was low (i.e., below 80%).

The power for the relative risk for diabetes (2.02, p=.49, 95% CI 0.31-12.04) found on page 10 of my report cannot be calculated using the program that I use, and one would have to be careful regarding any calculation of power because for this particular relative risk, there was only one

event among the placebo-treated subjects. Below is a sampling of the relative risk power I was able to calculate:

Study 13 (Report, p. 5): "In comparing low dose Seroquel (75 or 150 mg) verses placebo, the relative risk of weight gain was 3.54 (p=.06, 95% CI .95-16.1) . . . ." The power of this relative risk calculation equals .42% at an alpha (p value) = 0.05.

Study 15 (Report, p. 6): "Clinically significant weight gain occurred in 50/209 (23.9%) of the Seroquel participants compared to 4/38 (10.5%) of the haloperidol-treated subjects (relative risk=2.27, p=0.066, 95% CI=0.94-7.55)." The power of this relative risk calculation equals 65% at an alpha (p value) = 0.05.

Short-term, placebo-controlled trials as reported by AstraZeneca in response to the FDA's May 2000 request (Report, p. 9): "In the short-term (i.e., less than 6 weeks duration) placebo-controlled studies, only 230 Seroquel treated subjects and 143 placebo-treated subjects had glucose measurements analyzed, and Seroquel treated subjects had higher values of glucose than their placebo counterparts (3.6 (1.52 SE) vs. -0.26 (1.93), p=.12, respectfully)." The power of this relative risk calculation equals 34% at an alpha (p value) = 0.05, unpaired t-test, assuming variance to be from Seroquel group.

Short-term, placebo-controlled trials as reported by AstraZeneca in response to the FDA's May 2000 request (Report, pp. 9-10): "Additionally, 3.4% of 323 Seroquel treated subjects verses .07% of 143 placebo-treated subjects had a glucose value in excess of 200 mg/dl during the short term trials (relative risk 4.87, 95% confidence interval 0.83-29.30, p=0.116)." The power of this relative risk calculation equals 64% at an alpha (p value) = 0.05.

10. AstraZeneca suggests that I did not examine and cannot offer evidence of a dose-response relationship between Seroquel and diabetes.

This is a misrepresentation of both my expert report and my deposition testimony. As just one example of several offered in my report, for study 13 alone, low dose Seroquel (75 or 150 mg) versus placebo was associated with a 3.54 greater relative risk of clinically significant weight gain and higher doses (300 or 600 mg) was associated with a 4.77 greater relative risk of weight gain (p. 5). Additionally from my deposition, I stated the following:

A. There's a dose response relationship with all of the metabolic parameters that are a part of the diabetic -- Type II diabetes. There's some indication from the observational studies that there is a dose response between diabetes incidence and dose of Seroquel.
Q. Are you testifying to a reasonable degree of scientific certainty that there's a dose response relationship between Seroquel and diabetes?
A. Yes.

(Deposition, pp. 210-211).

11. AstraZeneca suggests that I did not examine and cannot offer evidence of a mechanism of action.

In my report, I describe three different biological mechanisms that support the weight gain and diabetic consequences of Seroquel treatment. (Report, pp. 3-4). As stated during my deposition, because of my work in pharmacogenetics I have to understand how drugs work in the body (page 53, line 19-20). Additionally, I stated that I had evaluated the literature and had an understanding of how Seroquel worked specifically in relation to weight gain and diabetes. (Deposition, p. 55, lines 2-4).

The relation between weight and weight gain and diabetes is an established risk factor for diabetes. As stated in my report, weight gain is also associated with features of the multiple metabolic syndrome and the metabolic syndrome is an important risk factor for diabetes incidence. (Report, p. 4). Beyond weight gain, Seroquel causes metabolic derangements, such as increased waist size (Meyer JM et al., Change in metabolic syndrome parameters with antipsychotic treatment in the CATIE Schizophrenia Trial: prospective data from phase 1, Schizophr Res. 2008;101(1-3):273-86) and hypertriglyceridemia. (Report, p. 11). Data from the Atherosclerosis Risk in Communities Study, having three of the components of the metabolic syndrome correctly identified 81% of the future cases of diabetes in the cohort. (REF: Ballantyne CM et al., Metabolic syndrome risk for cardiovascular disease and diabetes in the ARIC study. *International Journal of Obesity* (2008) 32, S21–S24). Collectively these data point to the importance of Seroquel-induced weight gain and its impact on diabetes risk. Finally, with respect to the correlational analysis between Seroquel weight gain and diabetes I indicated in my deposition that AstraZeneca has not evaluated the data in that way and therefore, I cannot scientifically offer an opinion regarding that correlation. (Deposition, p. 209, lines 9-10).

I hold additional relevant opinions as set forth in my expert report in this matter, which is attached and incorporated by reference. Additional opinions were elaborated in my deposition.

I declare under penalty of perjury that the foregoing is true and correct. Executed this 23rd day of February, 2009.

Donna K. Arnett

Donna K. Arnett, Ph.D., M.S.P.H.

Rebecca Thomas Putzulo

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