EXHIBIT 8

IN THE UNITED STATES DISTRICT COURT
NORTHERN DISTRICT OF ILLINOIS
EASTERN DIVISION

ENTERTAINMENT SOFTWARE ASSOCIATION, VIDEO SOFTWARE DEALERS ASSOCIATION, and ILLINOIS RETAIL MERCHANTS ASSOCIATION,	
Plaintiffs,	
vs.	NO. 05C 4265
ROD BLAGOJEVICH, in his official capacity as Governor of the State of Illinois; LISA MADIGAN, in her official capacity as Attorney General of the State of Illinois; and RICHARD A. DEVINE, in his official capacity as State's Attorney of Cook County,	
Defendants.	

DECLARATION OF HOWARD C. NUSBAUM

Pursuant to 28 U.S.C. § 1746, I, Howard C. Nusbaum under penalty of perjury state as follows:

1. I received my B.A. with a major in Psychology from Brandeis University in Waltham, Massachusetts, in 1976, and my Ph.D. in cognitive psychology from the State University of New York at Buffalo in 1981. I was an NIH Postdoctoral Fellow in Speech, Hearing, and Sensory Communication in the Department of Psychology from 1981 to 1984 and an Assistant Research Scientist in the Speech Research Laboratory in the Department of Psychology at Indiana University from 1984 until 1986.

2. I joined the faculty in the Department of Psychology at the University of Chicago in 1986 as an Assistant Professor in the Committee on Cognition and Communication. In 1989, I was promoted to Associate Professor with tenure in the Department of Psychology and became a member of the Committee on Biopsychology. I was promoted to Full Professor in the Department of Psychology in 2001.

3. I became the Chair of the Department of Psychology in 1997 and I continue to serve in that capacity in my third term. I am currently on the editorial board of the journal *Brain and Language*, a journal that focuses on understanding brain mechanisms of language use and I serve as a reviewer for a wide range of journals including but not limited to the *Journal of Cognitive Neuroscience*, *Cerebral Cortex*, *Cognitive, Affective and Behavioral Neuroscience*, *Psychological Science*, and *NeuroImage*. I am a member of the Committee on Computational Neuroscience at the University of Chicago, which grants Ph.D.s in neuroscience, and the Center for Integrative Neuroscience and Neuroengineering. I also serve on the Advisory Board for the Brain Research Imaging Center at the University of Chicago, and I am Co-Director of the University of Chicago Center for Cognitive and Social Neuroscience.

4. My research is in the area of cognitive psychology and cognitive and social neuroscience. This research examines the psychological and neural mechanisms that are important in learning, categorization, and attention and working memory (characterized sometimes as "executive function"). This work has included a study of the role of sleep in learning perceptual skills published in *Nature* (Fenn, Nusbaum & Margoliash, 2003), the role of attention in perceptual learning (e.g., Francis & Nusbaum, 2002), the role of working memory in communication (e.g., Goldin-Meadow, Nusbaum, Kelly, & Wagner, 2001), as well as experiments using functional Magnetic Resonance Imaging (fMRI) on the role of attention in understanding different speakers (Wong, Nusbaum, & Small, 2004), and the role of the motor system in face-to-face communication (Skipper, Nusbaum, & Small, 2005).

5. I started carrying out fMRI research in 1998 and have published three papers concerning the use of fMRI in understanding behavior and psychology. Although there has been a dramatic increase in the amount of fMRI research published in recent years, interpreting the results of fMRI studies can be extremely difficult. Measures of neural activity, such as fMRI provides, are only correlations with behavior and cannot be taken on face value as evidence of causality unless alternative explanations are ruled out. Moreover, behavior and brain activity do not relate in a simple, direct, and unique way. One of my papers, published in the *Proceedings of the National Academy of Science* (Cacioppo & Nusbaum, 2003) addressed problems in interpreting fMRI data regarding the brain mechanisms involved in making risky decisions under uncertainty. A second paper (Small & Nusbaum, 2004) addressed the problems of using fMRI to understand complex behavior that is sensitive to context. The third paper (Cacioppo, Berntson, Lorig, Norris, Rickett, & Nusbaum, 2003) provided guidance to social psychologists interested in using neurophysiological measures such as fMRI to understand complex social and emotional behavior. A copy of my CV is attached as Ex. A.

Purpose

6. I have been asked by counsel for Plaintiffs in this case to respond to the opinions of the expert declarations and reports submitted by the Governor, including in particular the conclusions expressed in the expert report and Declaration of Dr. William Kronenberger.

7. I have reviewed the Illinois Act, which regulates the sale of "violent" and "sexually explicit" video games. I have also been asked to give my opinion on the General Assembly's "finding" that "minors who play violent video games are more likely to: (1) Exhibit violent, asocial, or aggressive behavior[;] (2) Experience feelings of aggression[;] (3) Experience a reduction of activity in the frontal lobes of the brain which is [sic] responsible for controlling behavior."

Background on Effects of Video Game Exposure

8. Dr. Kronenberger's Declaration and opinions about the effects of exposure to media with violent content appear to take as a given that playing video games with violent content leads to aggressive behavior, thoughts, and feelings. Kronenberger Decl. ¶¶ 9-12. This assumption relies to a great extent on Dr. Anderson's research on aggression and the effects of media and video game exposure. In other words, Dr. Kronenberger assumes a causal relationship between exposure to violent media and aggressive behavior, thoughts, and feelings, and then sets out to show the brain activation underlying this causal relationship. However, the research on which Dr. Kronenberger relies has substantial problems that mitigate any possible strong conclusions regarding the relationship between playing video games with violent content and aggressive behavior, thoughts and feelings.

Video Game Exposure, Executive Functioning, and Neurophysiology

Dr. Kronenberger's Background Assumptions About Brain Functioning.

9. Dr. Kronenberger's Declaration argues that video game exposure to violent content leads to aggressive thoughts and behavior because video game exposure has an adverse impact on neural mechanisms related to self-control. Kronenberger Decl. ¶¶ 32-33; 38-39; 42-44. There is no evidence to support this argument. The research presented by Dr. Kronenberger addressing "executive functioning" cannot be used to this conclusion for several reasons.

10. First, there is no clear evidence in Kronenberger et al. (2005), Kalnin et al. (2005), or Mathews et al. (2005)[1] that exposure to violent media has a reliable

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[1] My citations to Wang et al. (2002, Kronenberger et al. (2002a), Kronenberger et al. (2005), Kalnin et al. (2005), and Mathews et al. (2005) refer to the primary studies relied upon by Dr. Kronenberger in rendering his opinions, and cited. Dr. Kronenberger was a co-author on each of these studies.

adverse effect on self-control or, for that matter, the Stroop task (a neurocognitive test used by Kronenberger and his co-authors).

11. Second, there is a fundamental flaw in the logic of all the brain imaging studies that are discussed by Dr. Kronenberger (including Kalnin et al., 2005, Mathews et al., 2005; Wang et al., 2005). The logic of these studies assumes that a particular pattern of brain activity is a unique and specific predictor of violent or aggressive behavior. While a pattern of brain activity might be associated with a pattern of aggressive behavior, that same pattern might be associated with many other patterns of behavior. As a result, a pattern of brain activity does not cause or uniquely predict a single pattern of behavior.

12. Dr. Kronenbeger's Declaration suggests that his research on the effects of violent media generally can be applied to ascertain the effect of exposure to video games specifically. The rationale for this appears to be a correlation reported in his research, Kronenberger Decl. ¶ 11, that exposure to violence in video games is correlated with exposure to violence in other media. The implication is that because exposure to one is associated with exposure to both, total exposure can be treated as a proxy for the specific causal effects of exposure to video games. However, to the extent that there is concern specifically with the effect of video game exposure, this assumption represents a serious confound that makes valid scientific inferences impossible. Since there is no research that establishes that video game exposure has exactly the same effects as exposure to violence in television or movies, they cannot be treated as equivalent in their effects, even if some children would tend to be exposed to all such media. Measures such as the Media Exposure Measure (e.g., as used in Kronenberger, Mathews, Dunn, Wang, Wood, Giauque, Larsen, Rembusch, Lowe, & Li, 2005) aggregate both television and video game exposure. As a result, any research based on such measures cannot provide evidence regarding the specific effect of video game exposure.

13. Dr. Kronenberger states at ¶ 13 of his Declaration that there is a unique relationship between psychological functions and brain areas. This is certainly not a settled matter of accepted and established science. Dr. Kroneberger's assertion appears incorrect in general and there are many specific counter-examples over the history of fMRI research. One classic example of the problems underlying Dr. Kronenberger's assertion comes from fMRI research arguing that our psychological expertise in face perception (as demonstrated in various behavioral studies, e.g., Yin, 1969) is mediated by a single brain region called the "fusiform face area" (Kanwisher, McDermott, & Chun, 1997). The claim of this research is that this fusiform face area of the brain responds uniquely to information about faces and provides no information about other visual patterns (claim one) and that this fusiform face area is the part of the brain responsible for perception of faces and no other area of the brain has the necessary information (claim two). But in a more sophisticated analysis, researchers using fMRI demonstrated (Haxby, Gonbini, Furey, Ishai, Schouten, & Pietini, 2001) that the fusiform area of the brain conveys sufficient information to distinguish among

objects besides faces, such as houses, cats, and chairs (contrary to claim one) and that many other parts of the brain outside the fusiform area have sufficient information to classify faces (contrary to claim two). Any particular part of the brain may be and typically is (as demonstrated by scientific research) associated with a wide range of psychological functions.

Interpretations of fMRI Activity

14. Dr. Kronenberger also states that activation of certain regions in the frontal lobes occurs during impulse control, self-regulation, choice, attention, and concentration. Kronenberger Decl. ¶ 16. It is not correct to assume that the same regions of the frontal lobes carry out all these functions. For example, while the DLPFC (dorsolateral prefrontal cortex) is active during tasks that involve visual attention (e.g., looking for your car in the parking lot), it is not specifically or uniquely implicated in impulse control or self-regulation (contrary to statements about the DLPFC made by Dr. Kronenberger, e.g., ¶ 13, or in Mathews et al., 2005; *see* Davidson et al., 2000). The frontal lobes have a lot of neurons and cortical areas and represent a broad range of functions that also include motor control and planning, and encoding of information into memory (e.g., what you had for breakfast this morning). Also, many of the functions attributed by Dr. Kronenberger to the frontal lobes, e.g., attention, involve a broad network of brain areas that connect the frontal lobe (e.g., DLPFC) to superior parietal cortex to the thalamus. Although different parts of the frontal lobes may be involved in different psychological functions, few, if any, carry out only a single function and few, if any represent the entire brain involvement in that function.

15. Dr. Kronenberger asserts that reduced activation of certain brain areas is associated with a wide range of problems, including difficulties in attention, self-monitoring, and impulse control, among others. Kronenberger Decl. ¶ 17. This statement is presented so that it implies a causal relationship, which cannot be inferred (e.g., see Uttal, 2001), between the reduced activity in these brain areas and these behavioral or psychological problems.

16. First, reduced activity in specific brain regions is not a clear, unique and specific "marker" of psychological or behavioral problems. Activity in "these regions" can decrease as a result of expertise in attention (e.g., Poldrack, Sabb, Foerde, Tom, Asarnow, Bookheimer, & Knowlton, 2005) as well as deficits in attention.

17. Second, reduced activity in some regions may be accompanied by increased activity in other regions, reflecting a change in the distribution of brain activity, but not necessarily a deficit of any kind. In a comparison of younger and older adults who perform comparably on certain tasks involving memory, the younger adults show less activation than the older adults in some of the brain areas (Reuter-Lorenz, 2002) to which Dr. Kronenberger refers, *see* Kronenberger Decl. ¶ 17. The younger adults are typically thought of generally as better at these tasks, and they show a change in the distribution of brain activity that is similar to

that noted by Mathews et al. (2005) as reflecting the effects of violent media exposure. In the case of younger adults, the reduction has nothing to do with violent media, but rather shows increased brain *efficiency*. Thus, changes in brain activity level downward or upward cannot be interpreted in a single, simple way. Moreover, changing the relative distribution of brain activity cannot be interpreted in any simple way.

18. Third, Dr. Kronenberger's discussion of the functions of attention, self-monitoring, impulse control, etc. does not reflect the true representation of these functions. They are not synonyms for each other and are not even necessarily functionally related. For example, there is not one kind of attention: We can maintain sustained attention, we can shift attention, we can focus attention, we can filter out irrelevant information. Each of these has different psychological processes and may be associated with different brain areas (cf. Posner, 2003) that includes some parts of prefrontal cortex, but also superior parietal cortex and sensory cortices. Problems in attention, self-monitoring, and impulse control cannot be diagnosed from reductions in brain activity in the frontal lobes of the brain.

19. Dr. Kronenberger suggests that reduced activity in the frontal lobes coupled with increased (the term that he uses – "hyperarousal" – implies some kind of absolute clinical standard of fMRI measurement that does not exist to my knowledge) activity in the amygdala and temporal regions "…is responsible for chronic, explosive, and/or severe aggressive behavior." Kronenberger Decl. ¶ 18. Just as decreases in activity in a brain region does not have a unique interpretation (e.g., "functional deficit"), increases in brain activity in particular regions do not have a unique interpretation. As noted previously, patterns of fMRI data simply correlate with patterns of behavior and causality cannot be inferred from this association of measurements.

20. Moreover, I am relatively certain that I could design a task that in normal participants would produce the pattern of brain activity described by Dr. Kronenberger without any risk of aggressive behavior. Presenting listeners with recorded speech with emotional content can increase, over some baseline resting level, the fMRI measured response in the amygdala and temporal lobes. If one group of subjects (memory group) had to hold certain words in memory from sentence to sentence, and another group (passive group) only had to listen and understand, the memory group would show more prefrontal cortical activity (e.g., in the amygdala and temporal lobes) and, relatively speaking, the passive listening group would show less activity in prefrontal cortex than the memory group. But this would certainly not cause them to have aggressive thoughts or behaviors.

21. To my knowledge, the specific patterns of brain activity as described by Dr. Kronenberger are not in general or accepted use as diagnostic procedures in clinical psychology. Moreover, it is important to note that the amygdala can show patterns of activation in situations without any threat or negative emotion

whatsoever. For example, in a listening task with different sounds in both ears, the amygdala is activated (Pollmann et al., 2004) and in making choices under statistical uncertainty the amygdala is activated (Fukui et al., 2005).

22. Dr. Kronenberger states that fMRI provides images of the location and amount of brain activity based on blood flow. He calls these "actual depictions of activity" in the brain. Kronenberger Decl. ¶ 21. It is important to note that fMRI does not actually depict neural activity in the brain. Real neural activity in the brain (as measured by post-synaptic potentials or firing rates) increases metabolic activity (which is measured by "positron emission tomography" (PET) neuroimaging). Increased metabolic activity increases the "blood oxygen-level dependent" (BOLD) response in the brain, which Dr. Kronenberger refers to as blood flow. fMRI measures the BOLD response and therefore does not directly measure brain activity, but rather it measures a change in the oxygenation of blood due to the metabolic activity that is a consequence of brain activity. Moreover, sometimes in fMRI studies what appears to be a sign of neural activity is instead simply a major supply of blood or a consequence of poor analysis.

23. It is also very important to understand that the analysis of fMRI data depends on the models chosen by the researcher. The data that are presented in tables or in colored displays imposed on gray scale images of the brain are highly abstracted statistical models of the fMRI measurements and not the actual brain or BOLD measurements themselves. Thus, the results that are reported in any particular fMRI study depend entirely on the analytic assumptions of the underlying model chosen by the researcher. The impression that these images from fMRI are direct depictions of neural activity in the brain is incorrect. For example, group data (e.g., as shown in Kalnin et al., 2005; or Mathews et al., 2005) can indicate activity in one area of the brain, even when no single participant actually shows activity in that area (e.g., due to spatial averaging of activity across subjects).

24. Neurocognitive tests are used primarily by clinical neuropsychologists and neurologists to make diagnoses of organic behavioral dysfunctions (without making any accurate claims about the specific brain location of the dysfunction). However, these are not scientific research tools closely calibrated to reflect, in their results, an accurate picture of brain activity, as implied by Dr. Kronenberger in his Declaration. Kronenberger Decl. ¶ 22. While it is true, in fact tautological, to say that behavior reflects brain activity, and vice versa, it is not true that brain dysfunction such as damage from stroke can be accurately (say to the measurement accuracy of fMRI on the order of millimeters or even centimeters) located in the brain from neurocognitive tests.

Dr. Kronenberger's Claims About Activation Of Regions Dealing With "Emotion"

25. In support of the conclusions set forth in his Declaration, Dr. Kronenberger refers to a study by Murray (2001) of brain activity in response to "filmed violence." Kronenberger Decl. ¶ 23. Dr. Murray also describes his study in his Declaration.

26. Dr. Murray's study, which is *not* about video game experience or exposure, measured brain activity for children viewing boxing scenes from a *Rocky* movie compared to a movie of animals at play or about children's literacy. Dr. Kronenberger cites Dr. Murray's study demonstrating activation in areas (such as the amygdala and hippocampus) associated with "threat-arousal appraisal" and posttraumatic stress. Kronenberger Decl. ¶¶ 23, 24-25. But these areas have been associated with a number of brain functions that have nothing to do with aggression or threat.

27. For example, researchers have observed that similar activation in the amygdala is consistent with judging unpleasant words (Maddock, Garrett, & Buonocore, 2003), negative facial expressions (Phillips, Young, et al., 1997), or body expressions (Hadjikhni & de Gelder, 2003), decisions with uncertainty (Fukui et al., 2005), and when pairs of different sounds are presented to both ears (Pollmann et al., 2004) – when no violence or threat or stress or emotional experience for the viewer is involved. This amygdala activity may reflect understanding of an emotional scene in the *Rocky* movie, just as the activity in the hippocampus (another part of the "frontal lobes" referred to by Drs. Kronenberger and Murray) may reflect recognition of faces or places seen earlier in the movie (Dezel, Habib, et al., 2003), and the posterior cingulate may be active due to remembering things the actors had said previously in the movie (Fujii et al., 2002). The frontal lobe regions referred to by Drs. Kronenberger and Murray as being active while watching *Rocky* include areas that are also used to understand another person's physical actions (cf. Farrer & Frith, 2002). Thus, this collection of brain areas may also reflect a higher level of engagement in trying to follow a movie with a plot, actors talking, and physical action (some of which is emotional or has emotional consequences), rather than anything specific to the effects of violence.

28. Dr. Kronenberger also cites a theoretical review by Davidson, Putnam, & Larson (2000) for the proposition that a pattern of reduced brain activity in the frontal cortex and increased activity in the amygdala in "emotionally provocative tasks" is found in people with greater amounts of aggressive behavior. Kronenberger Decl. ¶ 24. As noted above, *supra* ¶ 20, it may be relatively easy to produce this same pattern in normal subjects simply by presenting the right kind of stimuli and tasks without any change in aggressiveness at all. It is quite striking however, that this same pattern of reduced frontal activity and increased amygdala activity also is found in another population not mentioned by Dr. Kronenberger – people with clinical depression (Davidson, Pizzagalli, Nitschke, and Putnam (2002)). Clinical depression also produces the same kind of brain pattern, but clinically depressed patients are the least likely clinical population to display aggressive behavior.

29. Furthermore, although Dr. Kronenberger claims that Kalnin et al. (2005) (in an unpublished and unreviewed study) report finding the same pattern of brain activity for adolescents with "aggressive/violent behavior," this does not seem to correspond the data that are reported. *See* Kronenberger Decl. ¶¶ 38, 43. The pattern described by Davidson et al. (2000) for aggressive and violent people depends on a brain region called orbitofrontal (and associated ventromedial frontal) cortex. The Kalnin et al. (2005) study does not describe results for this area of the brain. Indeed, the Mathews et al. (2005) study (which appears to have used the same fMRI methods as the Kalnin et al. study) explicitly states (p. 291-292) that they did not have the technical ability or capability to image brain activity in the orbitofrontal cortex. The Kalnin et al. (2005) study does not explicitly identify (as would be standard in most scientific brain imaging) the brain regions that are investigated. Thus it appears that this research cannot investigate the brain regions that were identified by Davidson, et al. (2000) as relevant for aggressive behavior – but which in any event are also relevant to depression (Davidson et al., 2002).

Methodological Biases In Dr. Kronenberger's Measures of "Aggression" and "Media Exposure"

30. Another problem with Dr. Kronenberger's methodology is how he and his team measured "aggression." Although Dr. Kronenberger describes the participants in each Phase of his research as "adolescents with aggressive/violent behavior", these are in fact described in the studies as adolescents with Disruptive Behavior Disorder (DBD). Kronenberger Decl. ¶ 26. Dr. Kronenberger's description of DBD (¶ 27 and in studies such as Mathews et al., 2005) indicates that subjects can be classified as DBD simply by breaking rules and challenging authority. Although Dr. Kronenberger's research team found that each subject had at least on incident of "aggression" in the 6 months prior to the study, the subject's aggressive behavior (which can include verbal aggression) was based solely on the report of a caregiver. In other words, there is no objective evidence of aggressive behavior in these subjects and it is entirely possible that adolescents who defy the authority of their caregiver may be reported as displaying overt aggression due to reporting bias.

31. As noted previously, *supra* at ¶ 12, the measure of violent media exposure employed by Dr. Kronenberger in his research did not separate out exposure to "violent" video games from "violent" television. It is therefore not possible to draw any conclusions from research using this measure regarding the exposure to video games containing violence. A correlation in exposure among the different types of media cannot mitigate this criticism. Furthermore, it is important to note that, contrary to the guidelines of the best standards of evidence-based medicine, this measure of exposure is based on self-report and parent reports, which are subject to reporting biases and are not substantiated by objective measurements of actual video game exposure. The detailed nature of the reports, Kronenberger

Decl. ¶ 28, cannot substitute for objective measures of observed exposure; retrospective measures, Kronenberger Decl. ¶ 30, are subject to recall error among other biases.

Measuring "Executive Functioning"

32. In his Declaration and expert report, Dr. Kronenberger asserts that his research on the "frontal lobe functioning" (which he sometimes refers to as "executive functioning") of adolescents has shown that those with higher exposure to media violence is associated with impaired "frontal lobe functioning." Kronenberger Decl. ¶¶ 29-33. As an initial matter, Dr. Kronenberger's discussion of "executive functioning" is at best incomplete. The frontal lobes are, relatively speaking, large and complex neural networks. While cortical activity in parts of these regions is associated with decision-making, behavioral control, attention, and other cognitive functions, it is incorrect to treat these regions as homogeneous or undifferentiated either anatomically or with respect to their functions, as Dr. Kronenberger appears to do.

33. For example, the tasks referred to by Dr. Kronenberger as requiring attention and "concentration," Kronenberger Decl. ¶ 31, are not directly relevant to aspects of frontal cortex activity as such activity might relate to behavioral control. Mathews et al., (2005) and Kronenberger et al. (2005a) used variants of a task called the "Stroop task." This is a response selection task in which subjects have to ignore one property of a stimulus (e.g., the word RED) and name (or press a button to classify) the color of the ink in which it is printed (e.g., green). Since reading the word (i.e., RED) and naming the color of the ink (i.e., GREEN) are both responses in terms of color names, and because reading is a highly practiced skill and ink color naming is not, the response based on the printed word interferes with naming the color. Subjects have to filter out one response and focus on the other. Other forms of the Stroop task such as the Counting Stroop (as in Mathews et al., 2005) present a possible response conflict in the number of items (to be named) and the numbers that make up the items. For example, if presented with 3333, the correct response of the number of items (i.e., 4) is in conflict with the name of the items themselves (i.e., 3).

34. It is important to note that "response conflict" in the context of a Stroop task only means that there are two possible responses (e.g., in Color Word Stroop, the word RED and the ink color green) and only one is correct (i.e., the ink color green). It does not imply "conflict" as in conflict between people, and response selection, in this situation has nothing to do with behavioral regulation or control. In addition, Kronenberger et al. (2005) used a vigilance task called the continuous performance task. These tasks do not require impulse control or self-control, as suggested by Dr. Kronenberger.

35. Dr. Kronenberger cites the Wang et al. (2002) study as showing that "adolescents with a history of DBD showed less activation in parts of the frontal lobe to be

potentially responsible for concentration and self-control) compared to Control adolescents." Kronenberger Decl. ¶ 32. The Wang et al. (2002) study is not published, and the presentation slides provided by Dr. Kronenberger do not provide sufficient data to support its conclusions. It does appear, however, that the Wang et al. (2002) study examined brain activity without any kind of "executive function" task. Thus, the changes in frontal lobe activity cannot be interpreted specifically about executive functioning within this task. The comparison of observing a James Bond game (but not playing the game) and a car racing game is more akin to watching a movie than to an actual task that specifically requires explicit decisions and responses.

36. Without any data on the actual behavior of the subjects during the fMRI measurements, it is unclear how the Wang et al. (2002) study's patterns of brain activity in attentional areas should be interpreted. The presentation slides contain tables that purport to show differences between DBD subjects and Control subjects, and between high and low media violence exposure, in fMRI images. The tables report "cluster size" in certain brain regions (the number of activated analysis elements or voxels above a certain statistical threshold), but the lack of presented data makes it difficult to draw any conclusions. It is quite surprising that watching the James Bond video game did not appear to have produced increased amygdala activity, given the assertion of Dr. Kronenberger in his Declaration (e.g., ¶ 18), that high violent media exposure and DBD diagnosis lead to "hyperarousal" of the amygdala in such situations. If this is due to the failure to separately analyze brain responses to the two different types of stimuli (violent and non-violent video games), thereby mitigating any conclusions about arousal due to violence, the same caveat and disqualification should be valid for examining the pattern of frontal lobe activation as well.

37. Group differences of the type used in all the studies cited by Dr. Kronenberger cannot serve as the basis for drawing any causal conclusions about the relationship between media exposure and executive functioning. Two groups such as DBD and Control subjects may differ in a number of ways that they are not matched on, and these differences may be responsible for any measured differences in performance on a test. Moreover, assignment to the groups is not done by the standards of evidence-based medicine, making it impossible to draw any strong conclusions regarding these differences.

38. For example, Kronenberger et al. (2005) report that the DBD and Control subjects were matched on IQ. However, there are many cognitive functions that can vary among individuals that do not specifically correlate with IQ. For example, it is possible that there are working memory differences between the groups such that Controls have greater working memory ability. Working memory refers to the ability to hold information in mind while using it for some purpose (e.g., Baddeley, 1986). Or there may be differences in reading ability between the groups, or math ability, or some other cognitive skill or function that has not been assessed. If so, the difference between these groups in performance of a task such

as Stroop may be entirely due to these uncontrolled differences, rather than the claimed difference of aggressive or disruptive behavior.

39. Similarly, two groups that differ in reports of exposure to violent media could differ for a number of reasons beyond this exposure, which could be similar to the DBD vs. Control group difference. For example, DBD kids may not read as much as Control kids, and kids with high exposure to media with violent content may read less than those with low exposure to media with violent content because this content is more exciting (Anderson & Dill, 2000). In the Wang et al. (2002) study comparing a James Bond "violent" video game and a "non-violent" car racing game, control subjects appear to have rated the James Bond game more exciting, interesting, fun, and requiring attention than car racing (although this difference may not be significant and the scales have been manipulated so the difference in the numbers is not readily visually apparent). Therefore, ratings of violent media exposure might be predicted by reading ability and interest, such that poor readers play more video games that are fun, exciting, and interesting (and have violent content) that than good readers who spend more time reading. And poor readers might show more Stroop interference than good readers (cf. Johnson et al., 2003). There is no way to draw any conclusion from this study regarding the causal role of violent media exposure and certainly there is no basis for concluding anything regarding the effects of exposure to video game violence (because the researchers did not differentiate between television and video game violence).

"Phase I" Of Dr. Kronenberger's Research

40. Dr. Kronenberger states that the Mathews et al. (2005) study found differences in brain activation, as shown in fMRI, among the groups when performing "concentration tasks." Kronenberger Decl. ¶ 32. Dr. Kronenberger asserts that the Mathews et al. study showed "reduced activity" in certain areas of the brain that, according to Dr. Kronenberger, "are thought to be associated with concentration, choice, self-regulation, and self-control." Kronenberger Decl. ¶ 32. That conclusion is problematic for a number of reasons.

41. The Mathews et al. study reported that DBD subjects showed activation in the Middle Frontal Gyrus on both sides of the brain, whereas Control subjects showed activation in the Anterior Cingulate, left Middle Frontal and Left Inferior Frontal gyri. These are not areas involved specifically in behavioral control or self-regulation, as asserted by Dr. Kronenberger. Kronenberger Decl. ¶ 32. Such a description would be a more appropriate for the Orbitofrontal or the Ventromedial Frontal cortex (see Davidson et al, 2000). But none of the research done by Dr. Kronenberger and his colleagues looked at those areas.

42. The differences identified in the Mathews et al. study do not support the conclusions drawn by Dr. Kronenberger. The areas covered by the Mathews et al. study are, roughly speaking (with the caveats previously described about making such identifications between brain and behavior), areas of the brain more typically

associated with monitoring responses for errors, holding things in mind temporarily (working memory) and directing attention to something. The difference between the groups in the Mathews et al. study (DBD vs. Control, high media violence exposure vs. low) is similar to the difference between children and young adults in their cortical responses while performing the Stroop task (Adleman et al., 2002): Adults showed Anterior Cingulate activity not seen in the younger children and the younger children showed more Middle Frontal Gyrus activity. Thus, the difference between DBD and Control subjects in the brain activity accompanying the Stroop performance may be more a reflection of maturation than aggressive tendencies. Similarly, young adults and more senior adults have shown different patterns of cortical activity as a result of normal aging. Both groups maintained equal performance on memory tasks, but the younger adults activated dorsolateral prefrontal cortex (DLPFC) on one side (like Control subjects) and the seniors showed activation on both sides (like the DBD subjects) (Reuter-Lorenz, 2002). In both these age comparison studies, cognitive performance in different groups was manifest with different patterns of cortical activity that mirrored aspects of the group differences reported by Mathews et al. (2005) – but without involving any aggression or violence, suggesting that these are the merely the result of cognitive processing differences between groups. Similarly, differences as a result of media exposure within the Control subjects, reported by Mathews et al. (2005), appear only to reflect slight differences in the cortical networks that are active in ways that are similar to the developmental or aging effects.

43. Even accepting Dr. Kronenberger's methodology and underlying assumptions – which, for the reasons already detailed, are seriously flawed – the research he cites is itself internally inconsistent. For example, the pattern of difference in cortical activity reported by Mathews et al. (2005) is very different from the results of the Wang et al. (2002) study. On several measures, the brain activity for the DBD group versus the Control group, and for the high and low media exposure Control subgroups, were exactly the opposite in the two studies. Nevertheless, Dr. Kronenberger claims that the apparently conflicting results of each study support his conclusion that high media violence exposure results in "impaired" executive functioning. Dr. Kronenberger's claim that the results of Phase II research replicated the results of Phase I findings also does not appear to be based on consistent results. *See* Kronenberger Decl. ¶ 43.

44. The argument that Dr. Kronenberger proposes in Paragraph 33 of his Declaration is that exposure to media containing violence results in patterns of cortical activity during a Stroop task that are similar to adolescents with a history of aggression and violence. This pattern of cortical activity is interpreted as reduced functioning in brain areas involved in self-control. However, it is not established that the DBD kids have a clear history of aggression and violence, and it is not established that playing *video games* with violent content has any role in the results that are reported, given that there is no separate measure of game exposure. The data reported by Mathews et al. (2005) in fact show three clusters

of activity in the frontal lobes of both the Control subjects and the DBD subjects. Although the clusters are distributed differently, the difference is not one that relates to "self-control." Controls with high media exposure show one cluster of activation and controls with low media exposure show two clusters, all in the frontal lobe. However, there is no data reported on the size of the clusters or percent signal change in the regions, leaving open the possibility that this represents equivalent amounts of brain activity distributed slightly differently in the two groups. Even if the amounts differed, relative decreases in cortical activity do not necessarily reflect deficits and could reflect relative increases in cortical efficiency due to improved attentional processing in subjects with high media exposure.

"Phase II" of Dr. Kronenberger's Research

45. "Phase II" of Dr. Kronenberger's research is subject to the same problems discussed above. *See* Kronenberger Dec. ¶¶ 34-36. This research relied on the same methods establishing group differences (e.g. DBD/Control; high/low media violence exposure). Thus, Phase II still leaves unclear specific exposure to games, as compared to television. Group differences are still not established according to the accepted standards of evidence-based medicine. Thus, this research cannot license any conclusions about the effects of playing games with violent content, nor can it establish any causal effects of exposure to media with violent content. Moreover, the Kalnin et al. (2005) study, like the Wang et al. (2002) study is unpublished and therefore not subjected to the standard scientific review process. There are no real data presented in this presentation, just summary descriptions of the claimed findings and thus there is insufficient technical and scientific information to evaluate the soundness of the work, the results, or the conclusions.

46. The Stroop task used by Kalnin et al. (2005) in their fMRI study is described by Dr. Kronenberger (parag. 37) as "emotionally provocative." This is highly unlikely and is not substantiated by any mood ratings or ratings of the task by the participants. The task involves naming the color of ink of words related to violence (e.g., hit or kill) or words that are not related to violence (e.g., walk or run). In our studies of word-related decisions, such tasks are not typically "emotionally provocative," although the words that have more emotionally loaded meanings (such as kill compared to run) may grab the participant's attention. Kalnin et al. (2005) did not report using other kinds of emotionally loaded words (such as *discouraged*, *gloomy*) as negative words or emotionally positive words (such as *cake*, or *cheer*) as emotionally positive words. Thus, there is no evidence that the DBD adolescents responding more slowly to the "emotional" words in Kalnin's study did so on the associations with aggression or emotion. As a result, the behavioral results cannot be attributed to aggressive thought processing as claimed by Dr. Kronenerger. Kronenberger Decl. ¶ 37.

47. The Kalnin et al. (2005) conference presentation slides show one axial, one sagittal, and one coronal image of a brain for fMRI comparisons. These comparisons show activation greater for one group than another during the Stroop task (e.g., DBD greater than Control). However, there are no tables that specify the spatial coordinates (which is standard for scientific presentation) of the activation clusters, making it difficult to independently ascertain the brain locations showing activation. Dr. Kronenberger makes claims about what brain areas are activated in the Kalnin et al. study, Kronenberger Decl. ¶ 38, 43, but the presentation slides do not support his claims.

48. Dr. Kronenberger claims that the Kalnin et al. study shows "more activation in the parahippocampal gyrus and amygdala" for DBD participants and for subjects with high exposure to media violence compared to those with low exposure. Kronenberger Decl. ¶ 38. But that finding, even if substantiated, does not specifically implicate anything having to do with aggression. The fMRI results are not separated out with respect to brain activity produced by the emotional words compared to the non-emotional words. The group differences reported by Kalnin et al. (2005) might have been found for the words *walk* and *run* as well as the verbs related to aggression. If the reported differences in brain activity are valid, the pattern of fMRI results could reflect differences in emotional responses (not aggressive responses) to performing the Stroop task.

49. In any event, the areas showing greater activity do not correspond uniquely or specifically to brain areas involved in aggression or threat arousal as implied by Dr. Kronenberger. Kronenberger Decl. ¶ 39. While these areas may be active in response to emotional stimuli, what that activity means is completely unknown currently and is certainly not simply a center of threat arousal. Moreover, as stated above, *supra* ¶ 43, although Dr. Kronenberger asserts that the Kalnin et al. (2005) study replicates the Mathews et al. (2005) study, there is no basis for this claim in the results that are reported by Kalnin et al.

Summary

50. In summary, there is no evidence from either the Phase I or Phase II studies described by Dr. Kronenberger (e.g., summarized in ¶ 43 of the his Declaration) that playing video games with violent content has any adverse effects on brain function or executive function. Game playing experience is completely confounded with all media exposure in all the studies described by Dr. Kronenberger, so no conclusions can be drawn from this research that are specific to the effects of game playing.

51. The basic assumption of much of this research is to compare DBD adolescents with Control adolescents as a measure of what an "aggressive brain" looks like. However, uncontrolled group differences, and failure to use the standards of evidence-based medicine make this assumption unwarranted.

52. The assumption that specific brain regions are uniquely causal in behavior (e.g., anterior cingulate for self-control of impulsive behavior) is not consistent with research on neuroscience. Most areas of the brain participate in many different psychological processes. Moreover, correlations between brain activity and behavior does not establish a causal link between them (Uttal, 2001).

53. The DLPFC and ACC identified by Dr. Kronenberger as important for control of behavior are more generally viewed as important for attention than behavioral control, whereas orbitofrontal cortex (OFC) and ventromedial frontal cortex – comprising an area that was not specifically examined by Kronemberger – is more typically thought of as an area for behavioral control. Indeed it is OFC that Davidson et al. (2000) are referring to as possibly important in controlling violence. Moreover, in a Stroop task study of pathological gamblers, a group with real clinical problems in behavioral control, the reduced activity compared to normal subjects was in the ventromedial prefrontal cortex (closely associated with OFC) and not in the DLPFC or ACC (Potenza et al., 2003). In other words, when a real clinical group with behavior control problems is examined using the same kind of task as Dr. Kronenberger's group has used, the difference in performance is in a very different brain area. Thus, real behavior control problems are not manifest by reductions in DLPRC and ACC, contrary to Dr. Kronenberger's claims.

54. As cited by Dr. Rich in his Declaration (¶ 45), extensive experience playing violent video games has substantial benefits to prefrontal cortical functions such as attention and response selection. Green and Bavelier (2003) showed that extensive experience playing games with violent content such as *Grand Theft Auto 3*, *Half-Life*, and *Halo* produce substantial improvements in cognitive function in visual attention tasks that involve selection and control of processing. Moreover, this paper showed that these improvement are specifically obtained as a result of experience with video games with violent content and not for video games without violent content. Green and Bavelier gave non-game playing subjects experience playing either *Medal of Honor: Allied Assault*, which has "violent" content, or Tetris, which has no violent content but engages eye-hand coordination and interest. Subjects trained on the first-person shooter game showed significant improvements in visual information processing and attentional control compared to subjects trained on the non-violent puzzle game. The researchers do not attribute these benefits to the violence but to the way these games engage the player. (The Green and Bavelier (2003) study is one of the few studies that selected subjects based on specific video game experience to examine using performance tests the psychological consequences of this experience, and then tested the conclusion by specifically providing that experience to novices and administering the same tests.) It is extremely important to note that these improvements in behavior would likely be accompanied by reductions in fMRI measures of brain activity in the DLPFC and ACC since these areas are involved in attention (LaBerge, 1995). Indeed, Poldrack et al. (2005) found just such reductions in cortical activity after attentional training.

Conclusion

55. The Response to Plaintiff's Motion for Preliminary Injunction states that the General Assembly determined that minors who play video games with violent content are more likely to engage in violence or aggression, are more likely to experience feelings of aggression, and are more likely to experience a reduction in brain activity in the frontal lobes. As noted previously, there is no strong scientific evidence supporting the first two of these assertions that playing video games with violent content causes aggressive behavior and thoughts. And as I have explained above, the research put forth to support the third proposition is seriously flawed. However, even if the third contention were correct, given all the qualifications and concerns already raised previously, it is not clear that it has any significance regarding the first two claims. Reductions in frontal lobe activity, as reported in the research cited by Dr. Kronenberger, are not in areas that have been most closely identified with problems of behavior or aggression control, namely orbitofrontal or ventromedial cortex. Instead, the areas that are demonstrated to show reductions, dorsolateral prefrontal cortex and anterior cingulate, are areas of the brain more closely associated with attention. The assumption that such reductions reflect deficits in brain function is not warranted, given that extensive experience with video games leads to improvements in attentional function and that studies that examine brain activity following improvements in attention function reveal reductions in these areas.

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I declare under penalty of perjury that the foregoing is true and correct.

EXECUTED on October 17, 2005.

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1987-1988	Principal Investigator, *Attention and Vigilance in Speech Perception*. Grant to The University of Chicago from the Air Force Office of Scientific Research, Total direct costs: $133,479.
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2001-2006	Senior Personnel, Steven Small, Principal Investigator. *Functional neuroanatomy of normal and impaired language.* RO1 proposal funded by NIDCD. Direct Costs: $1,664,668.
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Research Interests

Speech Perception and Production, Psycholinguistics, Attention and Working Memory, Perceptual Learning, Auditory Perception, Computational Models, Cognitive and Social Neuroscience, Speech Technology, Applied Cognitive Psychology

Teaching Interests and Capabilities

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Journal Articles

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Nusbaum, H. C. Choosing the right voice response system: All applications are not created equal! Colloquium presented at Telesensory Speech Systems, Palo Alto, September, 1982.

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Nusbaum, H. C., & Morin, T. M. (1988). *Perceptual normalization of talker differences*. Presented at the 1988 meeting of the Psychonomics Society, Chicago, IL, November.

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Nusbaum, H. C. Understanding speech perception from the perspective of cognitive psychology. Invited talk presented at the Workshop on Spoken Language Understanding, Department of Psychology, SUNY/Buffalo, May, 1989.

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Nusbaum, H. C. Hearing speech and understanding language: Implications for a theory of consciousness. Invited talk presented at States of Consciousness, Satellite Symposium of the 14th World Congress of Neurology, New Delhi, October, 1989.

Nusbaum, H. C. Paying attention to the sounds of speech. Invited paper presented at the 62nd meeting of the Midwestern Psychological Association, Chicago, May, 1990.

Nusbaum, H. C., Paying attention to differences among talkers. Invited presentation for the ATR Workshop on Speech Perception and Speech Production, Kyoto, Japan, 1990.

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Nusbaum, H. C. Paying attention to speech. Invited colloquium, Department of Psychology, University of California at San Diego, La Jolla, January, 1991.

Nusbaum, H. C. Why we need a cognitive theory of speech perception. The Dean's Inuagural Lecture, Spring, Social Sciences Division, The University of Chicago, 1991.

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Nusbaum, H. C. Explaining speech perception with cognitive principles. Talk presented to GUV, Ann Arbor, September, 1992.

Nusbaum, H. C. Towards a cognitive view of speech perception. Invited colloquium, Cognitive Science Group, Department of Psychology, University of California, Los Angeles, January, 1993.

Nusbaum, H. C. Understanding speech perception from the perspective of cognitive psychology. Invited colloquium at the Institute for Research in Cognitive Science, University of Pennsylvannia, March, 1993.

Magnuson, J. S., & Nusbaum, H. C. Talker differences and perceptual normalization. Paper presented at the125th meeting of the Acoustical Society of America, Ottawa, May, 1993. *Journal of the Acoustical Society of America, 93*, 2371.

Baldwin, A. K., & Nusbaum, H. C. Changing the deployment of attention to phonetic structure. Paper presented at the125th meeting of the Acoustical Society of America, Ottawa, May, 1993.

Magnuson, J. S., Yamada, R. A., & Nusbaum, H. C. Variability in familiar and novel talkers: Effects on mora perception and talker identification. Paper presented at the September 1994 meeting of the Acoustical Society of Japan Technical Committee on Hearing, Kanazawa, Japan, 1994.

Nusbaum, H. C. & Francis, A. L. The effect of lexical complexity on segmental intelligibility. Paper presented at the 131st meeting of the Acoustical Society of America, Indianapolis, May 1996.

Francis, A. L., & Nusbaum, H. C. (1997). Computational constraints on spoken language understanding. Presented at Computational Psycholinguistics 1997, Berkeley, CA, August.

Francis, A. L., & Nusbaum, H. C. (1998). Perceptual learning of synthetic speech. Paper presented at the 136th meeting of the Acoustical Society of America, Norfolk, VA, October, 1998.

Nusbaum, H. C., & Francis, A. L., (1998). Attentional effects of variability in phonetic context. Paper presented at the 136th meeting of the Acoustical Society of America, Norfolk, VA, October, 1998.

Nusbaum, H. C., & Francis, A. L., (1998). Processing lawful variability in speech perception. Paper presented at the Psychonomics Society, Dallas, TX, November, 1998.

Nusbaum, H. C., Alperin, N., Towle, V. L., Francis, A. L., Barshes, N., Yarger, R., Small, S., & Solodkin, A. (1999). Cortical localization of linguistic expectations. Paper presented at the Psychonomics Society, Los Angeles, November, 1999.

Nusbaum, H. C., & Francis, A. L. The role of learning and adaptive processing in speech perception. Invited talk to be presented at a workshop on The Nature of Speech Perception: The psychophysics of speech perception III, Utrecht, Holland, July, 2000.

Cognitive Mechanisms of Spoken Language Acquisition, invited talk at the Bilingual Brain Conference, GASLA 2000 at MIT, Spring, 2000.

Goldin-Meadow, S., Nusbaum, H., Kelly, S., & Wagner, S. Gesturing helps us remember. Paper delivered as part of the symposium "Gestures in thinking, speaking and communicating: A developmental perspective" at the biannual meeting of the Society for Research in Child Development, Minneapolis MN, April 2001.

Goldin-Meadow, S., Nusbaum, H., Kelly, S., & Wagner, S. Gesturing helps us remember. Paper presented at Orage 2001, Aix-en-Provence, France, June 2001.

Skipper, J., Nusbaum, H. C., Hlustik, P., & Small, S. L. (2001). On the Non-Independence of Language Skipper, J. I., Nusbaum, H., Small, S.L. (2001), Language Processing without Motor Processing is Bilaterally Symmetric, American Academy of Neurology Annual Meeting.

Skipper, J., Nusbaum, H. C., Hlustik, P., & Small, S. L. (2001). On the Non-Independence of Language Subcomponents. *Conference on Human Brain Mapping*. Brighton, England.

Nusbaum, H. C. (2001). On the Neural Coding of Speech. Talk presented at the First Neural Coding Workshop, Committee on Computational Neuroscience, September, 2001, The University of Chicago, Chicago, IL.

Nusbaum, H. C., & Small, S. L. (2001) Perceptual learning of spoken language: Cognitive mechanisms and implications for aphasia, Invited talk presented at the Academy of Aphasia. Boulder, CO: Sept, 2001.

Nusbaum, H. C., & Fenn, K. (2001) The role of feedback in perceptual learning of synthetic speech, Talk presented at the 42nd Meeting of the Psychonomic Society, Orlando, Florida, November, 2001.

Nusbaum, H. C. (2002). Expectations and attention in speech perception. Invited talk presented at Speech Perception in Context, the Ninth Biennial Linguistics Symposium, Rice University, Houston, TX March 2002.

Fenn, K., Nusbaum, H. C., Margoliash, D. (2002). The role of sleep in perceptual learning of synthetic speech. Talk presented at the Midwestern Psychological Association, Chicago, IL, May, 2002.

Small, S. L., Uftring, S. J., & Nusbaum, H. C. (2002) Naturalistic language imaging: Hierarchical event analysis. Poster presented at the 9th Annual Meeting of the Cognitive Neuroscience Society, San Francisco, April 2002.

Skipper, J. I., Nusbaum, H. C., & Small, S. L. (2002). Speech perception and the inferior frontal neural system for motor imitation. Poster presented at the 9th Annual Meeting of the Cognitive Neuroscience Society, San Francisco, April 2002.

Fenn, K., Nusbaum, H. C., Margoliash, D. (2002). The effects of sleep on perceptual skill acquisition. Poster presented at the 43rd Meeting of the Psychonomic Society, Kansas City, November 2002.

Francis, A. L., & Nusbaum, H. C. (2002). On the flexibility of phonetic categories. Talk presented at the 143rd Meeting of the Acoustical Society of America, Pittsburgh, PA, June, 2002.

Wong, P. C. M., Nusbaum, H. C., Skipper, J. I., & Small, S. L. (2003). Cortical activation associated with lexical tone acquisition. Poster presented at the 10th Annual Meeting of the Cognitive Neuroscience Society, New York, April 2003.

Atkins, A. S., & Nusbaum, H. C. (2003). Cognitive load sensitivity of the PRP effect. Talk presented at the Midwestern Psychological Association, Chicago, IL, May, 2003.

Fenn, K., Nusbaum, H., & Small, S. (2003). Neural correlates of perceptual learning of synthetic speech. Talk presented at the Midwestern Psychological Association, Chicago, IL, May, 2003.

Skipper, J. I., Nusbaum, H. C., & Small, S. L. (2003). Face to face social interaction and inferior frontal processing. Talk presented at the Midwestern Psychological Association, Chicago, IL, May, 2003.

Skipper, J. I., Nusbaum, H. C., & Small, S. L. (2003). Sentence-level repetition priming as measured by brain activity is modulated by syntactic complexity and task demands. Poster presented at Human Brain Mapping, New York, June 2003.

Small, S. L., Uftring, S., & Nusbaum, H. C. (2003). A hierarchical design for functional imaging of language comprehension. Poster presented at Human Brain Mapping, New York, June 2003.

Shintel, H., Okrent, A., & Nusbaum, H. C. (2003). Spoken Gesture: Analogue Expression of Meaning in Speech, Presented at the 44th Meeting of the Psychonomic Society, Vancouver.

Norris, C. J., Chen, E., Zhu, D., Nusbaum, H. C., Solodkin, A., Small, S., & Cacioppo, J. T. (2003). *Neural Mechanisms Activated by Emotional Pictures.* Poster presented at the annual meeting of the Organization for Human Brain Mapping, New York.

Skipper, J., Nusbaum, H. C., & Small, S. L. (2003). Sentence-level repetition priming as measured by brain activity is modulated by syntactic complexity and task demands. Paper presented at the Organization for Human Brain Mapping, New York, NY.

Fenn, K. F., Nusbaum, H. C. & Small, S. L. (2004). Cortical Mechanisms of perceptual learning of spoken language. Presented at the 11th annual meeting of the Cognitive Neuroscience Society, San Francisco, CA.

Nusbaum, H. C., & Small, S. (2004). Language as a social module. Presented at Social Neuroscience: People thinking about people. Multidisciplinary perspectives on neural substrates and mechanisms. Chicago, May.

Skipper, J. I., Nusbaum, H. C., van Wassenhove, V., Dick, F., & Small, S. L. (2004). Your brain says what it sees: motor mechanisms of audiovisual speech perception. Paper presented at the Organization for Human Brain Mapping, Budapest, Hungry.

Skipper, J. I., van Wassenhove, V., Nusbaum, H. C., & Small, S. L. (2004). Hearing lips and seeing voices in the brain: motor mechanisms of speech perception. Paper presented at the Cognitive Neuroscience Society, San Francisco, CA.

Wagner, S. M., Whealton Suriyakham, L., Nusbaum, H. C., Goldin-Meadow, S., & Small, S. L. (2004). An fMRI Study of Gesture-Speech Perception: Hands Help Brains Reduce Cognitive Load. Presented at 16th Annual APS Convention, May, 2004, Chicago.

Wymbs, N. F., Nusbaum, H. C., & Small, S. L. (2004). The Informed Perceiver: Neural Correlates of Linguistic Expectation and Speech Perception. Cognitive Neuroscience Society, San Francisco.

Fenn, & Nusbaum, H. C. (2004). Change deafness, Presented at the 45rd Meeting of the Psychonomic Society, Minneapolis.

Nusbaum, H. C., Norris, C. T., & Cacioppo, J. T. (2004). Attention in perception of emotional information. Invited paper presented at the 44th Annual Meeting of the Society for Psychophysiological Research, Santa Fe.

Skipper, J. I., Nusbaum, H. C., van Wassenhove, V., Dick, F., & Small, S. L. (2004). Your brain says what it sees: Motor mechanisms of audiovisual speech perception. Paper presented at the Organization for Human Brain Mapping, Budapest, Hungary.

Skipper, J. I., Nusbaum, H. C., van Wassenhove, V., Barber, C., Chen, E. E., & Small, S. L. (2005). The role of ventral premotor and primary motor cortex in audiovisual speech perception. Paper to be presented at the Organization for Human Brain Mapping, Toronto, Canada.