In their recent paper in this journal Hewitson and coauthors (2010) reported that exposing infant macaques to the standard US vaccine regimen resulted in increased amygdalae growth, based on neuroimaging results, in the exposed animals compared to control animals. The paper also reported the effects of vaccination on binding of opioids in the amygdala. This claim that vaccination led to increased amygdala volume has attracted considerable attention in the autism media. Unfortunately, there are several serious problems with the methods and analyses used in the study. Further, the results reported by the authors are directly contradicted by their own earlier published findings and the findings of other researchers. These problems render the conclusions drawn by Hewitson and coworkers invalid.

The most obvious problem with the Hewitson and others (2010) study is the number of animals used. A total of 16 animals was enrolled in the study, the plan being to have 12 in the exposed group and four in the unexposed, control, group. However, one animal in the control group was “withdrawn due to a scheduling error” (p. 149). That should have left 12 animals in the exposed and three in the non-exposed group. Even had this been the case, only three animals in the control group would have been inadequate and it would be problematic at best to base any firm conclusions on results from such a small sample. But, it transpires, that, for reasons unstated in the paper, the amygdala volumes of only nine exposed and two unexposed animals were measured. Even in a pilot study, such tiny numbers, especially in the control group, render any results meaningless. The unexplained missing data is a cause for serious concern. While the authors correctly point out that this is a pilot study and only conclude that further research is necessary, the statistical and other limitations of this study do not meet even that low standard.

Another problem with the Hewitson and colleagues study is the use of multiple statistical analyses followed by a selective emphasis on the significant results. Thus multiple variables are examined and significant differences emphasized while negative findings tend to be ignored. Most obvious in this regard is that, overall, there was no difference in amygdala volume between the exposed and control animals. However, these volumes were measured twice, once when the animals were four and once when they were six months old. The authors report a significant interaction with the volumes for the exposed animals increasing and unexposed decreasing over the two month period. Of great interest is the fact that, while Hewitson and her coworkers make much of this interaction, and it was widely discussed as evidence for the harmful effects of vaccination on the internet, it is just the opposite of what Hewitson and coauthors (2008) has reported previously. Specifically, “compared with unexposed animals, exposed animals showed attenuation of amygdala growth”. So, which is it? Reading the results of the current study, especially in light of previous publications, gives an overall impression of a random scatter of data with cherry picking in order to make the argument that there are any meaningful results at all.

Yet another problem with the paper is its serious lack of scholarship. The authors neither mentioned nor discussed a previous paper, the results of which directly contradict their own. Payne with his group (2009)
studied the size of both the hippocampus and the amygdala in rhesus macaques, the same species used by Hewitson and others (2010). Payne and colleagues found that the amygdala (and hippocampus) enlarged from infancy to adulthood. This matches what Hewitson found in their vaccinated animals while their “control” or unvaccinated animals (both of them) showed a shrinkage of the amygdala over time. This combination of results suggests that the differences between the vaccinated and unvaccinated animals in the Hewitson and coworkers study is due to the anomalous shrinkage seen in the tiny control group. It is, of course, difficult to compare across different studies, but it appears that the vaccinated animals in the Hewitson and her group study showed normal amygdala development.

It must be pointed out that the Payne and others (2009) study was published on-line in September, 2009, well before the Hewitson and coauthors (2010) study was submitted for publication, on April 1, 2010. It was accepted June 20, 2010. It is difficult to understand how Hewitson and her coauthors and the peer reviewers of the manuscript were unaware of the Payne and others paper.

Hewitson and colleagues also report decreasing opioid binding in control animals with no change in the exposed animals. They assume these changes, as with the volumetric changes discussed above, are normal “maturational” changes in the control animals, and therefore the lack of change is abnormal in exposed animals, but there is no basis for this assumption.

In summary, the several serious methodological, logical, and scholarly problems with the Hewitson and coauthors (2010) paper make any conclusions about the effect of vaccination on amygdala development totally unwarranted.

