Two new studies cast further doubt on the theory that a mercury-based preservative in vaccines causes autism. Called thimerosal, the preservative has already been phased out in many industrialized countries but is still used in the developing world. The new findings “provide additional, extremely reassuring data,” says William Schaffner of Vanderbilt University School of Medicine in Nashville, Tennessee.

Thimerosal first attracted attention in the United States in 1999, when the Food and Drug Administration realized that toddlers, who are typically injected with several vaccines simultaneously, might be receiving higher doses of mercury than allowed by one federal standard. As a precaution, vaccine-makers began to phase out thimerosal that year.

Not long after, parent advocate groups from Safe Minds proposed that mercury, from the preservative and other sources, might be a factor in the rising incidence of autism, which often appears at about the same time that 2-year-olds get a round of shots. Many scientists were skeptical, given the minute amount of mercury and the different symptoms of mercury poisoning and autism. But in 2001 an Institute of Medicine panel concluded that there wasn’t enough evidence to rule out (or accept) the link.

Now the first big epidemiological studies weigh in. One comes from Denmark, which eliminated thimerosal from childhood vaccines in 1992. A team led by Kreesten Madsen of the Danish Epidemiology Science Centre in Aarhus reasoned that if thimerosal were a major cause of autism, incidence of new cases should drop once it was removed. In the September issue of the journal Pediatrics, they report that, instead of declining, the incidence continued to skyrocket after 1992. Like many epidemiologists, Madsen says the rising incidence could be a result of increased awareness and broader definitions of the disease. In any case, Madsen says, because incidence didn’t even slacken, thimerosal is not a major cause of autism.

But Mark Blaxill of Safe Minds argues that the study is “distorted and misleading.” He notes that in 1995, the Danish health registry began tracking a new category of patient, called autism outpatients. This and other factors, he says, are artifacts that confound the interpretation. Madsen responds that an unpublished analysis without outpatients showed the result of dead dopamine neurons. The study in question found surprisingly strong reactions—including two deaths—and “profound dopamine toxicity” in primates given injections of what the researchers thought was MDMA (Science, 27 September 2002, p. 2260). The doses were no higher than the equivalent of what a human would get in one all-night “rave.” They concluded that even brief exposure to MDMA may cause brain damage and raise a person’s risk of developing Parkinson’s disease, which is the result of dead dopamine neurons.

But the team’s subsequent attempts to replicate the results with oral doses, from a different batch of MDMA, failed. So did a repeat of the injection approach. The researchers then became suspicious of their original drug supply. Although the bottle labeled MDMA had been discarded, they discovered that their bottle of “methamphetamine” actually contained MDMA. A check of preserved animal brains from the experiment revealed methamphetamine and not a trace of MDMA.

Ricaurte says he’s not planning to abandon this line of inquiry. MDMA is toxic to dopamine neurons in mice, he says. “In what we’ve done so far, we do not see an effect in monkeys,” but various regimens remain to be tested. From now on, he says, lab members will test selected chemicals to be sure they are what they say they are. —Constance Holden