type of teratogenic effect ever even suggested (although as stated above, later proven to be incorrect) to have been caused by pre-natal exposure to PFOA. Since 1981 there have been additional full-scale teratology studies on PFOA, none of which produced any birth defects (structural or functional abnormalities) in the offspring of test animals.[8] Thus, in 1981 there was no data that would reliably ascribe the child's defects to PFOA, or strongly implicate PFOA as the cause, and no study run in the interim has created any such implication.

As noted previously, the 1981 one-page document also refers to a child born over two years previously as having unconfirmed "eye and tear duct" defects. Assuming that the defects did exist (and we have not been able to obtain any additional information on the unconfirmed defects), again the information contained in the 1981 document does not provide evidence that any such defects could have been reliably ascribed to PFOA exposure, either then or now. The mother's blood samples were taken more than two years after the pregnancy. The document indicates that the employee had worked in the fluoropolymer area for only one month before her pregnancy. Thus, here again, the data in the 1981 document -- listing blood levels more than two years after the pregnancy -- do not provide any reliable information about the presence, levels, or absence of PFOA in the employee's blood during her pregnancy. As such, from the information in this document, it cannot be reasonably concluded -- even assuming PFOA exposure during pregnancy -- that PFOA is "strongly implicated" as the cause of the unconfirmed defects.

The 1981 one-page document also indicates that one of the women, who gave birth a few weeks after the initial blood tests, permitted DuPont to test for PFOA concentration in the blood of the umbilical cord. PFOA was found to be present at a concentration level lower than that found in the mother's blood. In the course of investigating the basis for the information contained in this one-page document, DuPont recently found that the umbilical cord blood of the child of the fourth employee on the list was tested as well. The level reported was 0.43 ppm, again a lower level than that reported in the blood of the employee.

Nothing about this detection of the presence of PFOA in the umbilical cord blood at lower levels than in the mother's blood is unexpected or would reasonably support a conclusion of substantial risk. Indeed, teratology studies (such as were being run in 1981 on PFOA) are run on the assumption that the chemical in question will cross the placenta and will be present in the umbilical cord and come in contact with the developing fetus. The levels that DuPont detected in the umbilical cords simply confirm that there was no unexpected accumulation of PFOA at levels above those in the mother's blood. As explained above, and as supported by the CDC, presence alone does not indicate substantial risk of harm. Both children who had PFOA in their umbilical cords were born normal.

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[8] Gortner, EG. (1981) Oral teratology study of T-2998CoC in rats. Safety Evaluation Laboratory and Riker Laboratories, Inc. Experiment Number: 0681TR0110, December 1981; Gortner, EG. (1982) Oral teratology study of T-3141CoC in rabbits. Safety Evaluation Laboratory and Riker Laboratories, Inc. Experiment number: 0681TB0398, February 1982; Staples, RE; Burgess, BA; Kerns, WD. (1984) The embryo-fetal toxicity and teratogenic potential of ammonium perfluorooctanoate (APFO) in the rat. Fundam. Appl. Toxicol. 4:429-440 (two studies -- inhalation and oral dose administration).