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TEFLON AND AN INCREASED CANCER RISK REMAINS UNPROVEN IN HUMANS

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INTRODUCTION

DuPont manufactures a non-stick coating for pans brand-named Teflon. Teflon-sealed pans make cooking easier – eggs won't stick to the frying pan and cookies won't stick to the cookie sheet. Yet researchers have long worried about negative effects of Teflon and the chemical compounds used to make it, perfluorooctanoic acid (PFOA): is PFOA a carcinogen? Instead of waiting until the Teflon coating sloughs off in black flakes, should households get rid of their non-stick pans now and return to using PAM and Crisco in mass quantities?

PFOA is not exclusively used in Teflon; the chemical is also useful in the production of fast-food paper containers and stain-resistant fabric coatings. The chemical is unique because of how long it lasts in the human body, a fact which perpetuates fears of high cancer risks. DuPont has been resistant to these claims, and maintains that while PFOA is present during manufacturing, its levels are negligible by the time it reaches the kitchen.

Environmentalists, scientists, toxicologists, and consumer advocacy groups, among others, cite numerous short-term animal studies that suggest PFOA increases liver toxicity and risks of liver, pancreatic, and thymus cancers. Human studies of the chemical's effects are more rare – they are mostly limited to studies of workers exposed to high levels of PFOA in their factory work environment. Interpreting this data can be difficult, however: the workers are exposed to a number of toxic chemicals and they are exposed to higher concentrations than the user of a Teflon-coated pan would ever be. And the results from animal studies cannot be simplistically transferred to humans – the animals are exposed to the chemical in ways humans would never be, and most animal tests have been short-term, whereas humans could be exposed to PFOAs across their entire lifespan.

If Teflon can be shown to increase cancer risks, the Environmental Protection Agency (EPA) could take steps to regulate or ban the substance, as advisory panels have already recommended.¹ This would not only cause many Americans to make screening appointments with their physician (and lawyer), but would fundamentally alter the way we cook and the way we in which we regard advances in cookware.

The purpose of this study is to review literature on the epidemiologic relationship between perfluorooctanoic acid (PFOA) and an increased risk of cancer.

1 The Environmental Working Group. "PFCs and PFOA: A Family of Chemicals that Pollute the Planet." 2006. <http://www.ewg.org/reports/pfcworld/index.php>

REVIEW OF LITERATURE

The question of whether PFOA increases the risk of cancer in humans is difficult to scientists, but self-evident in the opinion of consumer-advocacy groups since many non-epidemiological and non-scientific studies have clearly linked PFOA to cancer.

First, studies of laboratory animals have long demonstrated a causal relationship between PFOA exposure and the development of certain cancers. Indeed, PFOA belongs to the same family as PFOS (perfluorooctane sulfonates), the active ingredient in Scotchgard, which was banned by the EPA in 2000. Both PFOA and PFOS have similar chemical and toxic properties: they do not break down in the environment, and they cause cancers in laboratory rats and kill newborn rats at doses that do not affect mothers.²

Second, in the 1980s DuPont itself monitored the pregnancies of seven female employees at its Teflon plant in Parkersburg, West Virginia. The results of the study were not published in a peer-reviewed, scientific journal, but only made public during the course of litigation. According to DuPont, of the seven women, two gave birth to babies with birth defects: eye/tear duct defect and nostril/eye defect.³ To many, this is all the evidence they need to start using their cast iron skillets once again.

Analytic epidemiologic studies, however, are more ambiguous. A 1993 study of cancer mortality in employees working at a PFOA-producing factory found a small correlation between employment in PFOA production and prostate cancer (Gilliland and Mandel, 1993).⁴ The study was a retrospective cohort design that looked at 3,537 people (2,788 males and 749 females) employed between 1947 and 1983 at an unnamed PFOA-producing plant. The study was conducted carefully and with a large sample size, but of all the workers studied (exposed and unexposed) only six died of prostate cancer. Of those, four deaths were exposed workers. This led Gilliland and Mandel to conclude that 10 years of employment at such a plant is associated with a 3.3-fold increase (95% CI, 1.02 to 10.6) in prostate cancer mortality compared with men who were not employed in PFOA production. The researchers' interpretation of this finding was that if prostate cancer mortality truly is related to PFOA, it is because the chemical alters the reproductive hormones of male workers, increasing their prostate cancer mortality rates.

Gilliland and Mandel collaborated again in 1996 to conduct and publish the results of a cross-sectional study of workers at DuPont's Cottage Grove, Minnesota plant (Gilliland and Mandel, 1996).⁵ This time, the two looked at liver cancer in 115 occupationally-exposed workers, and – after controlling for the confounder of employment length – found no significant associations between PFOA and clinical hepatic toxicity.

A two-part study, in which Gilliland was also involved, was published in 1998 and sought to determine if PFOA exposure led to (1) increased serum estradiol levels, which is shown to be linked with cancer in laboratory animals, and (2) increased mortality rates from liver, pancreatic, and testicular cancers (Olsen, *et al.* 1998).⁶ Two cross-sectional studies were done of 111 workers in 1993 and 80 workers in 1995. Olsen, *et al.* measured the levels of serum PFOA in the workers, and found a 10% increase in mean estradiol levels in workers with the highest levels of serum PFOA. However, serum levels can be confounded by body mass index (BMI) and few subjects were found at the

highest and lowest levels of serum, so the researchers were forced to conclude that there was not a significant association between PFOA-exposure and risky increases in serum estradiol levels.

For the cancer portion of the study, researchers were also unable to establish a link clear enough to impress the EPA. There was no significantly increased cause-specific standardized mortality ratio for either male or female employees at the PFOA-producing plants. While there were four employee deaths from prostate cancer, compared with only 1.97 expected (95% confidence interval [CI], 0.55-4.59), only one of those employees had worked directly in the PFOA production buildings. Researchers could only conclude a link was “biologically plausible.”⁷

The fifth study in this review was another cross-sectional study of individuals who worked in two DuPont plants – one domestic, one overseas. The study was conducted in 2000 and published in 2003 as part of the company’s fluorochemical medical surveillance program (Olsen, *et al.* 2003).⁸ Researchers did not compare actual cancer rates of exposed and unexposed employees, but instead took a cross-section of employees and analyzed the presence of serum PFOA concentrations in the blood of the workers. Because PFOA is known to cause cancer in laboratory animals at high concentrations, and Olsen, *et al.* sought to determine if those concentrations were present in the humans directly exposed to PFOA at the factory site. The sample size was 255 employees (206 male, 49 female) at the plant in Antwerp, Belgium and 263 employees (215 male, 48 female) at the plant in Decatur, Alabama. Age, BMI, current alcohol consumption (drinks per day) and cigarette use (cigarettes smoked per day), years worked, and type of job (PFOA production versus nonproduction), were potential confounding factors that were considered in the analyses. Researchers adjusted for these factors and found that the mean serum PFOA concentrations for Decatur employees were 1.32 parts per million (men) and 1.78 ppm (women). Mean concentrations were approximately 50% lower among Antwerp workers. Because both figures are substantially lower than the amounts in laboratory animals that lead to cancer, the researchers concluded that there is no connection between PFOA and an increased risk of cancer.

SUMMARY AND CONCLUSIONS

After reviewing the above five studies, one can only say that a link between PFOA exposure and an increased risk of cancer is unproven. One study found a small correlation between employment in PFOA production and prostate cancer (Gilliland and Mandel, 1993), a second found no significant associations between PFOA exposure and liver cancer, specifically (Gilliland and Mandel, 1996), a third found no association between PFOA exposure and risky increases in serum estradiol levels (Olsen, *et al.* 1998), a fourth found only a “biologically plausible” link between PFOA exposure and cancer rates (Olsen, *et al.* 1998), and a fifth study concluded that there was no connection between PFOA and an increased risk of cancer (Olsen, *et al.* 2003).

Yet neither DuPont nor consumers can be overconfident in these findings. PFOA, the active ingredient in Teflon, is proven to be a carcinogen in laboratory rats, and

though epidemiologists have not yet been able to prove that this translates to a similar risk for humans, it is because studies are difficult to conduct (they must be very long-term), and types of controlled studies that would be conclusive would also be unethical and indefensible. Many of the studies on people who were occupationally-exposed to PFOA were conducted by the same sets of researchers, and all were sponsored in part by 3M, a chemical supplier to DuPont. The studies have also been small in scope, involving so few people that it would have only taken one or two more cancer cases to change the link from “no increase” to “statistically significant increase.”

Additional studies are needed – analytic, long-term studies conducted on people with a more common and relevant exposure (eating food cooked on Teflon-coated cookware) that are objective and not tied to the interests of 3M or DuPont. The studies available have only been conducted on people with a decade or more of occupational exposure to PFOA, but because PFOA does not break down in the environment or the human body, exposure to the chemical over the course of a human’s lifetime must be measured, not simply 10 years of exposure (as most of the studies did).⁹

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