

A MEMORANDUM TO MEDIA AND INTERESTED PARTIES REGARDING WATER AND AIR POLLUTION

Mutagenicity and carcinogenicity of airborne organic compounds from fossil fuel emissions and how they relate to recent reports of unexplained pollution in the Washington, D.C. area drinking water sources causing male bass to bear eggs: more support for alternative fuels

(Cancer-causing and gene-altering effects of vehicle exhaust polluting drinking water supplies and how they relate to reports of male fish bearing eggs in the D.C. area water sources: time for alternative fuels?)

Andrew J., Welebir, Ph.D.
October 30, 2006

(Updated 7/19/07, Pollutants in Water and Air – Breast Cancer, Birth Defects, and Autism; Is Drinking Water Safe?)

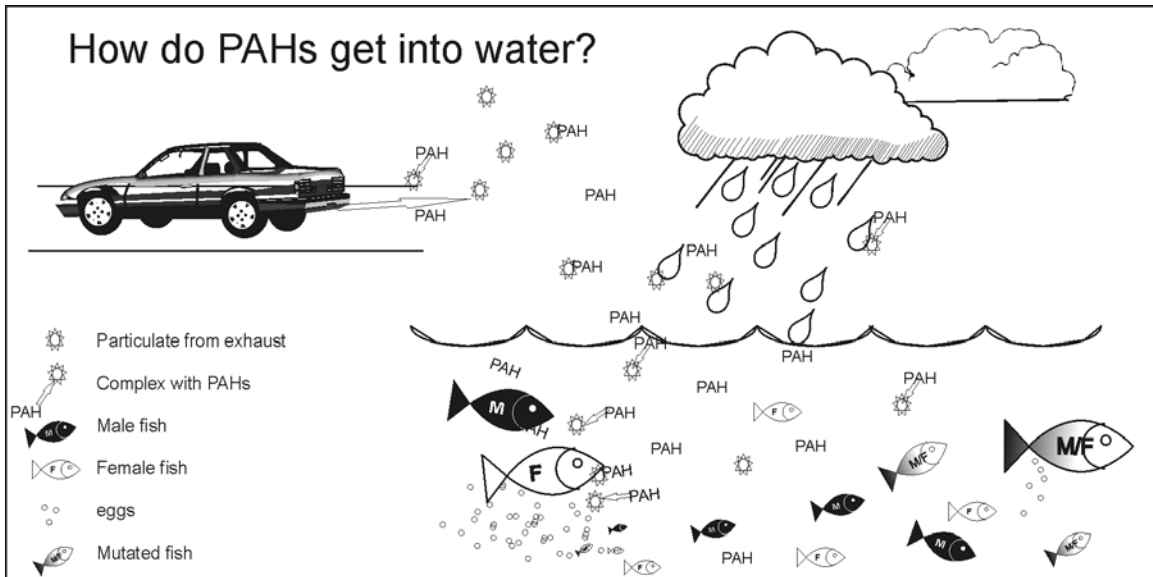
Abstract: This review is presented in two sections. One is an introduction for the lay person, and the other is a technical discussion. It addresses the pollutants in air from vehicle emissions and the effects of rain washing these into the ground water, rivers, and reservoirs and ultimately the area's drinking water. The effects of these pollutants, known as PAHs, and how they pertain to the unexplained effect on local male fish, described in *The Washington Post* reports mentioned in this brief review. Other dangers of these compounds are summarized, and these strongly support a transition to safer, cleaner-burning alternative fuels such as ethanol and bio-diesel. **Updated references at the end of this review.**

Introduction.

There are recent reports in *The Washington Post* (ref. 1) of male bass in the D.C. area waterways, such as the Potomac River, having unexplained “intersex” traits over the past few weeks – they have female eggs. While

these female traits have been attributed to pollution, no solid conclusions have been drawn about this cross-sexual phenomenon. No mention of pollutants from air washing into drinking water supplies was mentioned.

This review summarizes a more probable reason, based on scientific evidence published in past years, which are related to pollution in waterways from atmospheric contaminants which are produced from burning petroleum products. The major cancer-causing and gene-altering compounds from vehicle exhaust (and other processes) are called “PAHs”, or polycyclic aromatic hydrocarbons. These toxic by-products of combustion not only enter our lungs in the air, but fall, or are carried by rain, into our waterways and concentrate there over time, affecting our drinking water supplies.



The effects of PAHs on wildlife can range from almost harmless all the way to possible regional extinction in the extreme. A more detailed explanation follows in the Technical Discussion following this introduction.

PAHs come from a variety of industrial sources, but in non-industrial areas, the primary source is from burning gasoline and diesel fuel, and tobacco smoke. Fuel emissions are the greater source for atmospheric contamination. These compounds, mostly bound to particulates in the exhaust, have been implicated in health problems, ranging from asthma to lung cancer, and even skin and breast cancer.

Another effect on the environment is the ability of these pollutants to affect reproduction since PAHs and their metabolites can mimic and interfere with certain hormones. In aquatic life reproductive system processes, for example, these compounds can act as “endocrine disruptors” – resulting in male fish showing female traits.

Among the 100 or so PAHs in the environment from combustion processes, the more deadly ones include benzo[α]pyrene, or “BaP. This compound has been studied for years as a cancer-causing agent (see Technical Discussion).

Drinking water contains varying concentrations of PAHs, and EPA has set “acceptable limits” for humans. The highest acceptable amounts of BaP, for example, is BaP at 0.2 ng/m³ in air and 0.2 μ g/L in water (or one 10-billionth of a grams per about a quart of water, or one 10-millionth of a gram in air breathed for about 5 minutes). Exposure for a long period varies, as does the source.

The amounts of these pollutants concentrate in water, depending mostly on frequency of rainfall, and include water flow into larger bodies of water. Concentrations of mutagens (gene-altering compounds) have been studied widely, including those in Washington, D.C., Baltimore, and Chesapeake Bay area waters (ref. 36), and these have even been found to be linked to leukemia.

Investigating PAHs in tobacco smoke and those from vehicle emissions show the same dangerous PAHs in both. While cigarettes have warnings as to lung cancer risks, birth defects, etc., the same compounds – more widely spread in the atmosphere from exhaust – are not equated with those warnings even though both combinations of pollutants are virtually the same. No warnings are at the pump.

It is very likely that emissions from petroleum products have a role in the observed changes in male bass in the drinking water supply. This seems to lend strong support that alternative fuels, such as *ethanol* and *biodiesel* will be a necessity in the future if we want to maintain our health.

Technical Discussion

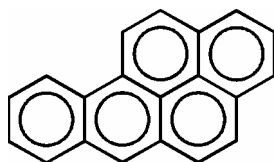
Recent attention has been given to the finding of male bass in the Washington, D.C. area waterways having the ability to produce eggs as reported in three articles in

The Washington Post (1). This was attributed to pollution, particularly from animal waste. However, this phenomenon, in fact, can most accurately be explained through mutagenic pollution in the water supply resources, and possibly *endocrine disruptors*. EPA has recently been questioned by members of Congress on this matter and why it was not investigated when this first became known in 1996 (1).

It is well-documented in the literature that certain airborne combustion products contain trace amounts of carcinogenic and mutagenic *polycyclic aromatic hydrocarbons*, PAHs (2,3). Standards have been established for the allowable concentrations of these in air by EPA and other government agencies, but these are in conflict to some extent (10,11,14,24,25).

These PAHs consist of at least a hundred compounds (5), ranging from virtually innocuous to extremely hazardous as classified under the Clean Air Act (4). The primary concern are the combustion products from fossil fuels, notably emission exhaust from internal combustion engines, such as those in cars, buses, trucks, aircraft, and also tobacco smoke, etc. (2,3,4,8,10,11,18,25).

Among the most toxic, carcinogenic, and mutagenic compounds in the environment covered in the literature is benzo[α]pyrene (BaP), which has been shown to cause cancer in animals for decades. BaP is also implicated in cancer in humans, such as lung, mouth, stomach, esophagus, intestinal, and skin (5,9,11,13,15,19,23,24,25) and breast (32).



benzo[α]pyrene

BaP exists in most ambient air, however the major source of contamination in rural and urban areas is from vehicle exhaust (2,3,4). Human studies abroad show this pollutant is carcinogenic in humans (3,9,15,19,24), however these severe toxic effects are only recognized in the U.S. by EPA as “probably carcinogenic [and mutagenic] in humans” (2). Other countries have concluded that sole reliance on U.S. EPA rules is problematic (14), including countries in Europe (14,24,25).

When these PAHs are released into the atmosphere by internal combustion engines, they remain in the air at varying concentrations mostly attached to particulates from the sources. When these compounds fall (or are washed by rain) into water supplies (2), they become of concern which is additional to just air contamination problems (2,3,6). These can cause mutagenic effects in fish (26), and *alter sex steroid receptors*, increasing the vulnerability to *endocrine disruptors*, according to a recent *EPA report* (33). Ultimately, they concentrate in water sources and to an extent, show presence in drinking water (2,3,6).

PAHs have been implicated in detrimental health effects besides cancer, including *reproductive* and development problems (4,7,15) and other problems, yet PAHs and BaP levels in drinking water are not currently reported in local water reports to the general public. EPA and NIOSH have set “acceptable levels” of PAHs similar to BaP at 0.2 ng/m³ in air and 0.2 µg/L in water (4,7,15). EPA further states that BaP is expected to “bioconcentrate in aquatic organisms that cannot metabolize it” (7). Occupational air concentrations have been shown to be as high as 1,200 ng/m³ (14), and water concentrations vary greatly by area, e.g., rural vs. urban, etc. (24). PAH concentrations are markedly *reduced* by addition of various amounts of alcohols to fuel, such as *ethanol* (18).

Water supplies in the Washington, D.C.-Baltimore area, the Chesapeake Bay, and even Los Angeles have been shown to contain PAHs from airborne particles, containing about 15% human cell mutagens (36). Other reactions with PAH content from fuel emissions, such as those with environmental ozone and ·NO₃ radicals from engine emissions, are known to produce other atmospheric mutagens and procarcinogens in research studies partly funded by EPA, Ford Motor Company, and DOE (12,13,18).

Airborne PAH sources (mostly bound to particulates) affect people prior to combining with water. There is no safe level of PAHs known for children exposed to diesel exhaust from school buses (29), and other problems with contaminated air and lung cancer have been summarized (25). *Tobacco smoke* has been compared to emissions, and found to have basically the *same PAH content* (20), however cigarettes have *lung cancer warnings* while EPA still lists PAHs from emissions as “probably cancer-causing in humans” (16,17).

PAH-DNA adducts can account for the gene-altering potential of these compounds (30,31,32). In fact, these adducts are implicated in effects *in utero* and chromosome damage in the fetus in the 1st trimester (27), which have been related to childhood cancers (31). PAH-DNA adducts resulting from vehicle emissions and tobacco smoke have been cited in the literature as leading to breast cancer (32), since PAHs are stored in the fatty material of the breast (23). PAHs attack lymphoblasts and have been implicated in lymphoblastic leukemia (28,36). Furthermore, purification of water for drinking at local facilities can usually remove only up to 80% of PAHs (32).

It is interesting to note that EPA has updated its report on sex steroid endocrine disruptors (33), and issued a paper on EPA agenda for studying same to the year 2010 (34). This apparently did not come up in the Congressional questioning as set forth in *The Washington Post*, October 5, 2006 (1). This report includes endocrine disruptors in mammals, birds, and *fish* following exposure to PAHs and the development of methods for endocrine disruptor challenge to *fish* from populations exposed to PAHs (33). Also, the report states, “PAHs could bring about an increased vulnerability to additional exposures to agonists/antagonists of sex steroid receptors (EDC’s)” (updated March 2, 2006). Other such studies are reported to be in progress (35).

It is also interesting to note that population growth demands a higher rate of drinking water, and more vehicles will occupy the roads traveling at a slower pace. With the limited water-processing facilities available, the increasingly contaminated drinking water resources will have to be processed at a much high rate. The faster water is processed, holding current capacity constant, the lower the amounts of impurities removed due to decreased PAH adsorption rate during the last steps of purification.

Reportedly, EPA states that it has reviewed several thousands of papers on these issues, and about 120,000 comments. However, they were not included in the 2006 report, and the next report will not be issued until 2010 (22).

The conclusion suggested by this review is that it would be extremely advantageous to pursue fuel alternatives, such as *ethanol*, which is clean burning, and *biodiesel* fuels, which pollute less than other diesel fuels. **Updated References 37 to 45 refer to subsequent *Washington Post* articles and reviews on PAHs causing breast cancer, birth defects, and autism.**

The author: Dr. Welebir analyzed water samples, including drinking water, while working for his M.S. and Ph.D. degrees in the 1970's under grants from the Naval Research Laboratory and the U.S. Army (Ft. Belvoir, VA). His area of expertise is in organic/biochemistry. He worked in cancer drug research and patenting new anti-cancer agents, and he currently works in the organic agricultural products field (over 25 years) for his company, Bio-Gard Agronomics, Inc., P.O. Box 4477, Falls Church, VA 22044. E-mail: Calcium25@aol.com.

References

1. "Male bass across the area found to be bearing Eggs" *The Washington Post*, Sept. 6, 2006, pages A1, A8; "Wildlife is a Major Polluter, Study Says" *The Washington Post*, Sept. 29, 2006, pages A1, A8; "House Panel Prods EPA to Speed Probe of Pollutants, Abnormal Fish" *The Washington Post*, October 5, 2006, page B3.
2. U.S. Dept. of Health and Human Services, *Toxicological Profile for Polycyclic Aromatic Hydrocarbons*, Aug. 1995. URL: www.atsdr.cdc.gov/toxprofiles/tp69.html.
3. Wisconsin Dept. of Health and Family Services, *Polycyclic Aromatic Hydrocarbons*, POH 4606, Dec. 2000.
4. "Polycyclic Aromatic Hydrocarbons", summary, URL: www.wisc.edu/~wang/EJBaldwin/PCR/pcrPAH.htm.
5. Agency for Toxic Substances and Disease Registry, *ToxFAQs for Polycyclic Aromatic Hydrocarbons (PAHs)*, Sept, 1996.
6. "Ground and Drinking Water Contaminants", U.S. Environmental Protection Agency, URL: www.epa.gov/safewater/hfacts.html.
7. "Technical Factsheet on: POLYCYCLIC AROMATIC HYDROCARBONS (PAHs), *ibid.*, URL: www.epa.gov/safewater.dwh/t-soc/pahs.html.
8. Goldman, *Cancer Res.* **61**, 17:6367-6371.
9. "Lung Cancer", from www.oncologychannel.com/lungcancer/causes.shtml.

10. "Diesel Exhaust Particulates", *Report on Carcinogens, Eleventh Ed.* (2000).
11. *PAH Health Review*, International sources on PAHs, URL:
www.ephc.gov.au/pdf/Air_Toxics_Health_Review.pdf.
12. Atkinson, R. and Arey, J., *Environ. Health Perspectives*, **102**, Supp. 4, Oct., 1994.
13. "Mobil Source Emissions – Past, Present, and Future" U.S. Environmental Protection Agency. EPA-OTAQ – Mobile Source Emissions – Definitions.
14. *Environ. Health Perspectives*, Annual Review Issue, **112** 9 June, 2004.
15. Hardy, J., "Polycyclic Aromatic Hydrocarbons", excerpts, URL:
<http://delloyd.50megs.com/hazard/PAH.html>.
16. *Framework Convention Alliance for Tobacco Control*, Geneva, Switzerland, e-mail fca@globalink.org
17. National Center for Chronic Disease and Health, CDC, 2000, URL:
www.cdc.gov/tobacco/sgr/sgr_2000/factsheete/factsheet_labels.htm.
18. Westerholm, R. and Egeback, K-E., *Environ. Health Perspectives*, **102**, Supp. 4, Oct., 1994.
19. Sinha, R., Kulldorff, M., Gunter, M.J., Strickland, P., and Rothman, N. *Cancer Epidemiol. Biomarkers Prev.* **14**(8):2030-2040 (2005).
20. *Energy Independence Now*, "How Do Tobacco and Car Exhaust Compare?" Fact Sheet (714 Bond Ave., Santa Barbara, CA 93103, 805-899-3399). URL:
www.energydependencenow.org.
21. "Air Pollution", Summary from http://en.wikipedia.org/wiki/Air_pollution.
22. EPA Standards Revision – 2006, URL:
www.epa.gov/oar/particlepollution/naagsrev2006.html.
23. URL: www.breastcancer.org/research/diet/080002.html
24. Santodonato, J., P. H. Howard and D. Basu. 1981. Health and ecological assessment of polynuclear aromatic hydrocarbons. *J. Environ. Pathol. Toxicol.* **5**(1):1-364.
25. Rantanen, J., "Community and Occupational Studies of Lung Cancer and Polycyclic Organic Matter, *Environ. Health Perspective*, **47**:325-332 (1983).
26. "Biomarkers of Contaminant Exposure and Effect in Flatfish from Southern California", URL: www.sccwrp.org/pubs/97/ar06.htm.
27. "Airborne Cancerous Agents Can Reach Fetus", URL:
www.webmd.com/content/article/84/98277.htm.
28. Sasaki, J.C., *et al.*, *Mutat.Res.* **393**(1-2):23-35 (1997).
29. "Children's Exposure to Diesel Exhaust on School Buses", Environmental & Human Health, Inc., URL: www.ehhi.org.
30. Perera, F.P., *J. Natl. Cancer Inst.* **92**, No. 8, 602-612 (2000).
31. *Cancer Epidemiology Biomarkers & Prevention*, **14**, 506-511 (2005).
32. "Polycyclic Aromatic Hydrocarbons and Breast Cancer" (July 2001), URL:
<http://envirocancer.cornell.edu/FactSheet/general/4s41.pah.cfm>.
33. EDRI Federal Project Inventory: Development of Gene Probes for Diagnosis of Exposure to Sex Steroid Endocrine Disruptors, EPA Report, updated March 2, 2006. <http://www.epa.gov/endocrine/inventory/EXP-TOTH.html>
34. Endocrine Disruptors Multi Year Plan, December, 2003, contact:
toth.greg@EPA.gov. Full text: <http://www.epa.gov/osp/myf/edc.pdf>

35. Research is under way to determine the mechanism of **PAH** ... capstone course titled "Endocrine **Disruptors** in the Environment." ... registrar.duke.edu/bulletins/Nicholas/2001/faculty.pdf
36. Durant, John L., Pederson, Daniel U., *et al.*, *Environ. Sci. Technol.*, **32**(13), 1894-1906 (1998), *ibid.* **33**(24, 4407-4415 (1999), *ibid.*, **39**(24), 9547-9560 (2005).

UPDATED REFERENCES 7/19/07

37. **Cancer-causing PAHs in the D.C. water supply** – the Anacostia River contains 290 time the present EPA limit of PAHs. “Polluted Water Stains D.C. Shining Vision” David A. Fahrenthold, *The Washington Post*, Jan. 9, 2007, pp. A1, A8.
38. “D.C. Area Sees Spike in Rate of Emissions” David A. Fahrenthold, *The Washington Post*, April 29, 2007, pp. A1, A16.
39. **Fish death in Virginia Rivers** – “A Mystery of Fish Mortality” David A. Fahrenthold, *The Washington Post*, June 20, 2007, pp. B1, B2.
40. “New Fears Raised over Safety of D.C. Water” Carol D. Leonnig, *The Washington Post*, pp.A1, A8 (**PAHs weren’t included, other carcinogens were**).

BREAST CANCER

41. “Polycyclic Aromatic Hydrocarbons and Breast Cancer Risk”, *Cornell University Program on Breast Cancer and Environmental Risk Factors in New York State*, Fact Sheet #41, July, 2001 (PDF file available online).
42. “Breast Cancer Risk Linked to Traffic Emissions at Menanche, First Birth” April 30, 2005, www.medicalnewstoday.com/medicalnews.php?newsid=23611.

BIRTH DEFECTS

43. “Urban Air Pollution Linked to Birth Defects for the First Time” UCLA report at: www.herelectricvehicle.com/toxic.html.

AUTISM, CHILDHOOD DEVELOPMENT

44. Harder, Ben, “Air Pollutants Linked to Slow Childhood Mental Development”, *Science News*, April 29, 2006, Included in: www.childproofing.org/kidsnewspage1.htm.
45. “*Prioritization of Toxic Air Contaminants under the Children’s Environmental Health Protection Act*” California Environmental Protection Agency Report, Oct., 2001 (PDF file available online).