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## **Polyethylene Terephthalate May Yield Endocrine Disruptors**

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**Abbreviations:**

<i>bis</i> (2-ethylhexyl) phthalate	DEHP
<i>bis</i> (hydroxyethyl) terephthalate	BHET
Degree of polymerization	DP
Diethyl phthalate	DEP
Dimethyl phthalate	DMP
17 $\alpha$ ethinyl estradiol	EE2
Polyethylene terephthalate	PET

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**Abstract**

Background: Recent reports suggest that endocrine disruptors may leach into the contents of bottles made from polyethylene terephthalate (PET). PET is the main ingredient in most clear plastic containers used for beverages and condiments worldwide, and has previously been generally assumed not to be a source of endocrine disruptors.

Objective: I begin by considering evidence that bottles made from PET may leach various phthalates which have been putatively identified as endocrine disruptors. I also consider evidence that leaching of antimony from PET containers may lead to endocrine-disrupting effects.

Discussion: The contents of the PET bottle, and the temperature at which it is stored, both appear to influence the rate and magnitude of leaching. Endocrine disruptors other than phthalates, specifically antimony, may also contribute to the endocrine-disrupting effect of water from PET containers.

Conclusions: More research is needed in order to clarify the mechanisms whereby beverages and condiments in PET containers may be contaminated by endocrine-disrupting chemicals.

**Introduction**

Polyethylene terephthalate (PET) is the material most commonly used to make the clear plastic bottles in which bottled water is sold. PET bottles are also in widespread use as containers for soda beverages as well as for sports drinks, and condiments such as vinegar and

salad dressing. PET bottles are also commonly used for the packaging of cosmetic products, such as shampoo, particularly when such products are sold in clear plastic bottles.

The potential of plastic packaging to introduce endocrine disruptors into foods and beverages has gone largely unrecognized until quite recently (Muncke 2009). The plastics industry generally asserts that PET bottles are not a source of endocrine disruptors (e.g. American Chemistry Council 2009). In this Commentary, I present evidence that PET bottles may leach endocrine disruptors, and I consider the conditions under which this leaching may occur.

### **Synthesis of PET**

The synthesis of PET begins with the esterification of either terephthalic acid or dimethyl terephthalate with ethylene glycol, yielding *bis*(hydroxyethyl) terephthalate (BHET). The BHET is then polymerized up to about 30 repeat units (Awaja and Pavel 2005). Next, to achieve a degree of polymerization (DP) of about 100 repeat units, polycondensation is then performed at temperatures above 270 °C and pressures above 50 Pa (Ravindranath and Mashelkar 1986). In order to produce bottle-grade PET, the DP must be greater than 150 repeat units, which is typically accomplished via solid state polymerization, a process which requires temperatures above 200 °C, pressures above 100 Pa, and incubation times of at least 15 hours (Al-Ghatta, Cobror, and Severini 1997).

It is becoming increasingly common for manufacturers to market co-polymers for purposes previously filled by homopolymer PET. Co-polymer blends, such as polybutylene terephthalate/polyethylene terephthalate, have certain advantages over homopolymer PET with regard to mechanical properties and resistance to degradation (Grossetête et al. 2000; Guerrica-

Echevarría and Eguiazábal 2009). In the United States, a clear plastic bottle may be made with co-polymers and still be legally marketed as PET, according to applicable federal regulations e.g. 21 CFR §177.1630.

## Phthalates

The term “phthalates” refers to the diesters of 1,2-benzenedicarboxylic acid, better known as phthalic acid. A growing literature links many of the phthalates with a variety of adverse outcomes, including increased adiposity and insulin resistance (Grün and Blumberg 2009), decreased anogenital distance in male infants (Swan et al. 2005), decreased levels of sex hormones (Pan et al. 2006), and other consequences for the human reproductive system, both for females and males (reviewed by Hauser and Calafat 2005). Infants and children may be especially vulnerable to the toxic effects of phthalates (Sathyanarayana 2008). Indeed, legislatures and government agencies in Australia, Canada, the European Union, and the United States have already acted to restrict or prohibit the use of phthalates in consumer products (Canadian Department of Health 2009; USA Consumer Product Safety Commission 2008).

The plastics industry has been keen to emphasize the distinction between PET and phthalates. In a letter to *Environmental Health Perspectives*, a spokesperson for the American Plastics Council wrote:

*“Plastic beverage bottles sold in the United States are made from a type of plastic known as polyethylene terephthalate (PET). Although polyethylene terephthalate (the plastic) and phthalate (the additive) may have similar names, the substances are chemically dissimilar. PET is not considered an orthophthalate, nor does PET require the use of phthalates or other softening additives.”* (Enneking 2006)

Indeed, phthalates are not used as substrates or precursors in the manufacture of PET. However, as we will see, several reports suggest that phthalates may leach from PET bottles into the contents of the bottle.

In this Commentary, I first review evidence from various bioassays that PET may yield endocrine disruptors. I then consider evidence that phthalates leach from PET bottles, followed by evidence that antimony leaches from PET bottles

### **Bioassays**

Wagner and Oehlmann (2009) employed two bioassays to investigate the estrogenicity of water within PET bottles. First, they used a yeast estrogen screen, employing a strain transfected with the human estrogen receptor  $\alpha$ . They evaluated 20 brands of mineral water, nine of which are available both in glass and in PET bottles. Three of nine brands sampled in glass demonstrated significant estrogenic activity in this bioassay, compared with seven of nine brands of water from PET bottles. However, one cannot say with certainty that the estrogenic substance or substances necessarily leached from the bottles; the contaminant may have been introduced into the water prior to bottling.

The second bioassay by Wagner and Oehlmann addresses this concern. In the second bioassay, these investigators emptied the bottles of their contents, then filled the empty PET bottles and glass bottles with a defined culture medium (pH  $8.0 \pm 0.5$ ) and incubated New Zealand mudsnails, *Potamopyrgus antipodarum*, for 56 days. Production of embryos was significantly enhanced among snails incubated in the PET bottles compared with snails incubated in glass bottles, across all brands ( $p < 0.001$ ). Production of embryos incubated in PET bottles of brand D was roughly double the production of embryos incubated in glass bottles of brand D, for

example; curiously, on the yeast estrogen screen, this same brand showed no difference in estrogenic activity between PET-bottled water glass-bottled water. This finding suggests that the *in vivo* snail bioassay might be more sensitive than the *in vitro* yeast estrogen screen.

Regarding the snail bioassay, Wagner and Oehlmann concluded that “it is obvious that the observed effects can only be attributed to xenoestrogen leaching from these plastic bottles. Moreover, the compounds released by the PET material were potent [enough] to trigger estrogenic effects *in vivo* similar to those of E2 [17 $\alpha$  ethinyl estradiol] at a concentration of 25 ng/l” (p. 284). The maximum estrogen activity which they detected in any brand of water was equivalent to 75 ng/l of ethinyl estradiol.

Pinto and Reali (2009) also employed a yeast bioassay to investigate estrogenic activity in samples of water obtained from PET bottles. Like Wagner and Oehlmann, they found large variations between brands of water obtained from PET bottles: in ethinyl estradiol equivalents, their results ranged from a low of 0.9 ng/l to a high of 23.1 ng/l. Pinto and Reali explained this variation by noting that “not all PET materials are of the same chemical quality. Quality may vary depending on the raw material as well as the technology used in bottle production . . .” (p. 230). Pinto and Reali, using waters bottled in Italy, obtained results which were substantially lower in estrogenic activity than those obtained by Wagner and Oehlmann, who purchased their bottled waters in Germany. It is possible that the German PET had a greater propensity to leach endocrine disruptors than the Italian PET: it is possible that the yeast bioassay employed by Pinto and Reali may be less sensitive than the yeast bioassay employed by Wagner and Oehlmann.

In a third report, investigators at the University of Missouri tested the effect of an unspecified brand of PET-bottled water on the proliferation of breast cancer cells. They found

that the PET-bottled water triggered a 78% increase in the growth of the breast cancer cells compared to the control water: 1,200 breast cancer cells multiplied to 32,000 in 4 days when incubated in PET-bottled water, versus 18,000 for the control sample (Naidenko et al. 2008). This report must be interpreted with caution, as it is available only as a posting on the Web and has not yet been published in any peer-reviewed journal. Furthermore, the results from the University of Missouri bioassay, like those from Pinto and Reali, do not prove unequivocally that the presumptive endocrine disruptors leached from the PET bottle wall; they might conceivably have been present in the water prior to bottling.

### **Presence of Phthalates in PET-Bottled Water, Soda, and Food Simulants**

Montuori et al. (2008) tested 71 commercial brands of bottled water, all of which were available both in glass bottles and in PET bottles. Across all brands, they found that the concentration of all phthalates combined was “more than 12 times higher in PET than in glass bottled water” (p. 515). In most cases, the concentration of phthalates in water from glass bottles was below the limits of detection. The most abundant phthalates which they found in PET-bottled water dibutyl phthalate, diisobutyl phthalate, and diethyl phthalate (DEP). The 50<sup>th</sup> percentile for the sum of all phthalates in all PET bottles tested by Montuori et al. was 1.32 µg/l. Montuori et al. assert that the phthalates they detected in the PET-bottled water must have leached from the PET bottle wall (p. 515); however, their data do not compel this conclusion, since they did not measure the concentration of phthalates as a function of time. It is conceivable that the water in the PET bottles was contaminated with phthalates prior to bottling.

Casajuana and Lacorte (2003) investigated the effect of prolonged incubation on the concentration of various phthalates in water from PET bottles compared with water from glass

bottles. In all their samples, both from glass bottles and from PET bottles, the concentration of phthalates was initially very low, at or below the limits of detection in almost every case, when first sampled. Prolonged incubation had little effect on the concentration of phthalates in glass-bottled water; phthalates in water from glass bottles were still generally undetectable after ten weeks' storage. However, in their samples of water from PET bottles, three out of five brands showed measurable levels of *bis*(2-ethylhexyl) phthalate (DEHP) after ten weeks' incubation, with an average DEHP concentration after 10 weeks of 0.134 µg/l; and all five brands showed measurable levels of DEP after ten weeks' incubation: average DEP concentration after 10 weeks was 0.214 µg/l.

Schmid et al. (2008) sought to determine whether solar water disinfection (SODIS) would promote leaching of phthalates into water in PET bottles. SODIS is a technique used in developing countries to disinfect water by incubating water in PET bottles in direct sunlight. After 17 hours of incubation in direct sunlight, maximum concentrations of di(2-ethylhexyl)adipate (DEHA) and DEHP were 0.046 and 0.71 µg/L, respectively.

Biscardi et al. (2003) went to a bottling plant in order to obtain mineral water prior to bottling. They then filled PET bottles and glass bottles with mineral water, both carbonated and still. All bottles were stored at room temperature. Each subsequent month, for 12 months, samples of water were lyophilized, the powders then shaken with acetone, and the acetone extracts analyzed using GC/MS. Throughout the first eight months, no phthalates were detected in any sample. Beginning at month 9 for PET-bottled still water, and month 10 for PET-bottled carbonated water, the acetone extracts increased from 0.4 to more than 3.0 mg/l. GC/MS analysis of the extracts identified the presence of DEHP.

Farhoodi et al. (2008) studied the interaction of incubation time with storage temperature on the leaching of DEHP from PET bottles. Using a solution of 3% acetic acid as a food simulant, they incubated the solution in PET bottles for up to 120 days, either at 25 °C or at 45 °C. On day 0, at the beginning of the trial, the amount of DEHP in PET bottles was below the limits of detection. On day 25, the amount of DEHP in the solution incubated at 25 °C was 1.2 mg/l, while the amount of DEHP in the solution incubated at 45 °C was 2.1 mg/l. By day 66, the amount of DEHP in the solution incubated at 25 °C had peaked at 1.4 mg/l, while the amount of DEHP in the solution incubated at 45 °C had plateaued at 2.5 mg/l.

Bošnjir et al. (2007) sought to determine how the contents of the PET bottle influenced the concentration of phthalates in the contents. They compared the concentrations of various phthalates in PET-bottled mineral water with PET-bottled soft drinks preserved with phosphoric acid or with sodium benzoate and/or potassium sorbate. They reported large variations in the concentrations of phthalates both across beverages and across manufacturers. For example, they were not able to detect dimethyl phthalate (DMP) in any brand of mineral water after 30 days' incubation, whereas DMP was the most abundant phthalate detected in the soft drinks they tested. Among soft drinks preserved with both sodium benzoate and potassium sorbate, the concentration of DMP in samples incubated for 30 days ranged from 18 to 2,666 µg/L, with a mean of 501 µg/L; by contrast, the concentration of DMP in mineral water was consistently below the limits of detection. They conjecture that the lower pH of the soft drinks might account for this difference (p. 93). However, the concentrations of DEHP (unlike DMP) did not differ between soda beverages and mineral water: they found average levels of DEHP less than 100 µg/L in all their specimens, with no significant difference between soda beverages and mineral water.

Bošnjir et al. asserted that leaching of phthalates from the PET bottle must be the source of the phthalates they measured; they assumed that the concentration of phthalates was below the limits of detection when the mineral water or soft drinks were originally bottled. Their basis for this assumption is the fact that “Raw materials for soft drinks and final products (both soft drinks and mineral water) are under obligatory and continuous public health validity control [in Croatia] which excludes possible contamination with phthalates” (p. 92). However, they provide no measurements to support their assumption that the soft drinks and mineral water they tested were phthalate-free when bottled. Once again, as with Montuori et al., one cannot exclude the possibility that the water or soft drinks may have been contaminated with phthalates prior to bottling.

### **Origin of Phthalates in PET-Bottled Water and Beverages**

Farhoodi et al. were not able to detect DEHP in their samples when first tested; yet after 66 days of incubation at 45°C, the concentration of DEHP in their sample reached 2.5 mg/l, i.e. 2,500 µg/L. Likewise, Biscardi et al. reported similar concentrations of DEHP after nine months’ incubation of water in PET bottles at room temperature. Where did the DEHP come from? i.e. how did DEHP get into the PET bottle wall in quantities sufficient for such an amount to leach into the bottle contents?

One possibility may have to do with the use of recycled PET. In 2008, 27.0% of PET containers sold in the United States were recycled (National Association for PET Container Resources 2009). “New” PET may therefore contain PET which has been recycled from a previous use. PET recycling begins by washing the used PET to remove contaminants; however,

this washing is not effective in removing organic molecules once they have been sorbed into the bottle wall (Safa 1999).

PET from different suppliers may differ in the degree to which it is homopolymer or copolymer; the extent to which the material is “virgin” or recycled PET; and in details of the manufacturing process. As noted above, Bošnjir et al. (2007) detected DMP in concentrations as high as 3,000 µg/L in PET-bottled soda, whereas they were unable to detect DMP at all in PET-bottled mineral water. One possible explanation is that the soda, perhaps due to its lower pH, promoted leaching of DMP from the PET bottle wall. However, it is also possible that the PET used in production of the bottles intended for soda had a different provenance than the PET used in production of the bottles intended for mineral water. Shampoo often contains DMP (e.g. Sathyanarayana 2008). If the bottles used for soda included PET recycled from shampoo bottles, while the PET-bottled mineral water did not, that difference might contribute to the much higher concentrations of DMP in PET-bottled soda.

### **Estrogenicity of Antimony; Leaching of Antimony from PET**

Measuring the concentration of phthalates in soft drinks and 3% acetic acid, respectively, Bošnjir et al. (2007) and Farhoodi et al. (2008) both found phthalates in concentrations above 1,000 µg/L in at least some of their samples. However, among the other reports cited above which measured phthalates in bottled water rather than in soda or acetic acid, only one (Biscardi et al. 2003) identified any phthalates in concentrations above 1,000 µg/L, and that was only after at least nine months’ incubation. Nevertheless, the bioassays described at the beginning of this article incubated their specimens for less than two months, and employed water or a water-based culture medium with a neutral or near-neutral pH, not with soft drinks or acetic acid. This raises

the possibility that a non-phthalate endocrine disruptor or disruptors may have mediated the estrogenic effects documented in the bioassays. Some evidence suggests that antimony may be at least partially responsible for these estrogenic effects.

Choe et al. (2003) reported that antimony chloride has “high estrogenicity” in two bioassays. In an estrogen-receptor-dependent transcriptional expression assay using human breast cancer cells, they found that antimony chloride at a concentration of 1  $\mu\text{M}$  had estrogenic activity which was 61% equivalent to 17 $\beta$ -estradiol at a concentration of 1 nM. In an E-screen assay measuring proliferation of human breast cancer cells, they reported that antimony chloride at a concentration of 1  $\mu\text{M}$  had estrogenic activity which was 49% equivalent to 17 $\beta$ -estradiol at a concentration of 10 nM.

In the United States, the Environmental Protection Agency (EPA) has established an MCL (Maximum Contaminant Level) of 6 ppb for antimony, which is the same limit set by Health Canada; the German Federal Ministry of Environment has set a limit of 5 ppb, while the Japanese drinking water standard requires levels of antimony below 2 ppb (Shotyk and Krachler 2007). However, these cutoffs are generally based on older research on antimony toxicity, related to cardiovascular risks and carcinogenicity; for example, the EPA’s web site on antimony in drinking water makes no mention of antimony’s possible endocrine-disrupting effect (EPA, 2009).

Antimony is widely used as a catalyst in the polycondensation of PET (Pang et al. 2006). PET resin typically contains antimony in concentrations between 100 and 300 mg/kg (Duh 2002). However, PET resin made in Japan is sometimes manufactured using titanium rather than antimony as a catalyst. Nishioka et al. (2002) investigated antimony concentrations in PET bottles manufactured in Japan. They found a bimodal distribution of concentrations, with some

bottles having antimony concentrations between 170 and 220 mg/kg, while in other bottles, antimony concentrations were below the limit of detection (<0.1 mg/kg).

Several investigators have now demonstrated significant levels of antimony in water bottled in PET containers. Shotyk and Krachler (2007) measured antimony concentrations in 132 brands of bottled water purchased in 28 countries. They found a wide variation in antimony concentrations, with dramatic differences in the leaching of antimony over time. In 14 brands of PET-bottled water purchased in Canada, antimony concentrations increased on average 19% during six months' storage at room temperature. By contrast, 48 brands of PET-bottled water purchased in Europe increased on average 90% during six months' storage, under identical storage conditions in the same laboratory. Shotyk and Krachler also reported wide variations in antimony concentrations even among the same brand of PET-bottled water, depending on the location of purchase. For example, one brand of PET-bottled water yielded 1650 ng/l of antimony when first purchased in Hong Kong, increasing to a concentration of 1990 ng/l when tested six months later; while the same brand of bottled water purchased in Europe had a concentration of 725 ng/l when first purchased, increasing to 1510 ng/l six months later.

Westerhoff et al. (2008) found that raising the ambient temperature significantly increases the leaching of antimony into nine brands of PET-bottled water purchased in the United States. At room temperature (22°C) they found no significant change in the concentration of antimony over time: the average antimony concentration from nine brands of PET-bottled waters was  $0.195 \pm 0.116$  ppb at the beginning of the study and  $0.226 \pm 0.160$  ppb after 3 months indoors at 22°C. When the bottles were incubated at 70°C, however, the concentration reached 6 ppb in just 12 days; at 80°C, in just 2.3 days. After seven days at 80°C, the antimony concentration reached 14.4 ppb. Noting that temperatures within a closed-container truck may

easily exceed 60°C in Arizona, where this study was conducted, they concluded that “short duration exposure to elevated temperatures during transit or storage by the seller or consumer could yield antimony concentrations that approach or exceed the 6 ppb MCL” (p. 555). Previous research has demonstrated that the temperature inside a car parked in the sun, with windows closed, can reach 78°C after six hours (Surpure 1982).

Keresztes et al. (2009) studied ten brands of PET-bottled water, all purchased in Hungarian supermarkets. Unlike Westerhoff et al., Keresztes et al. found that the concentration of antimony in PET-bottled water increased monotonically over time at room temperature, although they reported large differences between brands. The antimony concentration in one brand of PET-bottled water increased from an average initial concentration of about 0.1 ng/ml to an average concentration of about 0.9ng/ml after two years’ incubation at room temperature. However, another brand of PET-bottled water showed almost no additional leaching of antimony even when warmed to 60°C for 24 hours (the concentration always remaining below 0.2ng/ml); while the concentration of antimony in a different brand of PET–bottled water increased from 0.2 ng/ml at 22°C to more than 1.5ng/ml after 24 hours’ incubation at 60°C (Keresztes et al. figure 5).

## **Discussion**

The available research suggests that the concentration of phthalates in the contents of PET bottles varies as a function of the contents of the bottle, with phthalates leaching into lower-pH products such as soda and vinegar more readily than into bottled water. Temperature also appears to influence the leaching both of phthalates and of antimony from PET, with greater leaching at higher temperatures.

The effect of temperature may account for some of the variation in the results noted previously. For example, Pinto and Reali 2008 noted that “Cell toxicity was observed for water samples of the same lot of three different brands purchased from the same retailer”; they conjectured that “toxicity might be attributable to the storage conditions of the product” (p. 230). Perhaps that retailer left the bottles exposed to the hot sun, while other retailers did not.

Lower-pH condiments such as table vinegar and salad dressing may warrant particular attention. The findings of Farhoodi et al. (2008) suggest that ingesting several servings of salad dressing which had been stored in a warm warehouse for a month might result in a dose of DEHP on the order of several hundred micrograms, possibly reaching the reference dose limit of 20 µg/kg/day (US Environmental Protection Agency 2006, p. 3).

## **Conclusion**

The evidence suggests that PET bottles may yield endocrine disruptors under conditions of common use, particularly with prolonged storage and elevated temperature. Important questions for future research include: What substances in the water are responsible for the estrogenic effects observed in the bioassays – is it one or more of the phthalates, and/or antimony, and/or as yet unidentified substances? How do variations in the composition and manufacture of PET influence the leaching of these substances into the contents of the bottle? Would special measures – such as a special coating on the inner wall of the bottle (e.g. Pennarun et al. 2004), or transportation under controlled-temperature conditions – minimize the leaching of these substances into the contents? Because of the widespread use of PET plastic worldwide in containers for water, soda beverages, and condiments, the safety of PET under conditions of common use certainly merits further investigation.

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