Cancer in Belugas from the St. Lawrence Estuary

In their paper published in the March 2002 issue of *EHP*, Martineau et al. (*1*) reported the results of 100 necropsies of St. Lawrence Estuary (SLE) belugas found dead, drifting, or stranded along the shoreline during 1983–1999. They gathered a remarkable set of pathology information on these dead animals and are to be commended for their work.

In their report, Martineau et al. (*1*) used epidemiologic techniques to compare cancer rates among the belugas with those of other animal species and humans. Because their work involved a cohort of dead animals found on shoreline, comparison with cohorts of live or hunted animals or with human statistics is indeed challenging. These comparisons raise the questions of whether the information collected is representative of the source population and whether the referent groups used for the comparisons are valid.

In Figure 2 of their paper, Martineau et al. (*1*) compared the age structure of SLE belugas found dead with that of northwest Alaska (NA) belugas obtained through subsistence hunting. They concluded,

> Thus, the age structure of SLE beluga dying of natural causes appears clearly different from that of NA beluga, principally because most SLE belugas die at an earlier age.

We do not concur with this interpretation. The age distribution of the NA belugas that died of hunting and the age distribution of belugas found stranded on the SLE shoreline are two very different things. To conclude that SLE belugas died at an earlier age, one would need to know the age structure of the two populations—the NA belugas and the SLE belugas—and the death rate by age categories in each of them. The authors did not have this information and therefore could not state that the "SLE belugas die at an earlier age."

Martineau et al. (*1*) estimated that the annual cancer mortality rate of the SLE belugas was 570 per 100,000, a crude rate. It would have been more informative to present age-specific cancer rates. The crude rate may simply be reflective of the age distribution of the beluga population. If a large proportion of the population is in the age groups with elevated cancer rates, the overall cancer rate will be high. Comparing these crude rates to those of other animal species or humans without taking the age distribution of the population into consideration is bound to lead to erroneous interpretations.

In their Figure 4, Martineau et al. (*1*) compared the annual cancer rate in the SLE belugas with that of humans and domestic animals. They concluded that

> … The AAR [adjusted annual rate] in SLE beluga is much higher than that observed in cattle, horse, and sheep examined in veterinary hospitals, higher than the rate observed in dogs and cats examined in veterinary hospitals, and higher than the rate in humans.

Here again, we do not concur with this interpretation. On one hand, the comparisons are made without considering the age distribution of these groups, and on the other hand, the source of information, the profile of life and death of these groups, and the way the information was collected for each comparison groups (sheep, horses, cats, dogs, cattle, and humans) are totally foreign to the life profile of the SLE belugas and the way the information about them was obtained. These were simply not comparable data; thus the authors' conclusion is not sustainable.

Martineau et al. (*1*) referred to our studies on bladder and lung cancers among aluminum workers and the association that we reported of these cancers with exposure to polycyclic aromatic hydrocarbons (PAHs) in the workplace (*2,3*) as an argument in support of their hypothesis that cancer in belugas was caused by PAHs in the sediments of the river. We are not sure of the relevance of our study findings to their hypothesis because the exposure that led to increased lung and bladder cancers in workers involved the inhalation of high concentrations of airborne PAHs over several years.

Martineau et al. (*1*) referred to a higher incidence of stomach, digestive tract, and breast cancer in members of the Saguenay population not working in the aluminum industry, and inferred that this may be related to drinking water that they claim is contaminated with PAHs. We have reservations on these views. First, the referred statistic may be inflated because it does not exclude workers. Second, the hypothesis of a relationship between high incidence of cancers of these sites and drinking water seems highly improbable in light of the confirmation of the Ministry of Environment of Quebec that the norm of PAHs (benzo[*a*]pyrene) (10 ng/L) is present in untreated water as well as in water from the filtration plants (*4*). Third, to date, no association has been observed between PAH and stomach cancer among aluminum workers, even if they drank the local water and worked in the aluminium industry environment.

In spite of our reservation concerning the epidemiologic aspects of this paper by Martineau et al. (*1*), it remains an important contribution on the pathological aspects of cancer in wildlife.

The authors are currently involved in the conduct of a mortality study of the aluminum workers from the Saguenay region as a follow-up to their previous studies. The study is financed to G. Gibbs by Alcan Inc.; C. Tremblay and G. Theriault are consultants on the project.

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St. Lawrence Beluga Whales, the River Sweepers?

Many polycyclic aromatic hydrocarbons (PAHs), among which the most extensively studied is benzo[*a*]pyrene (BAP), are carcinogenic in mammals (*1,2*). Because humans are also mammals, these PAHs are reasonably anticipated to be human carcinogens (*3–5*).

Further support for PAH carcinogenicity in humans has been provided by epidemiologic studies such as those carried out in aluminum workers of the Saguenay-Lac Saint-Jean region of Canada (*6–8*). In these studies, the epidemiologic association between exposure to PAHs and lung and urinary bladder cancers has been strong enough to warrant the compensation of affected workers by the Quebec Health and safety Workers Compensation Board (*6–8*).

The types and sites of cancer induced by PAHs depend on the route of exposure (*1,2,9*). Thus, it should be no surprise that PAHs cause lung and urinary bladder cancer in aluminium workers who inhale these compounds and that PAHs may cause cancer of the digestive system in belugas, because these animals ingest fish that feed on plankton and invertebrates living in the contaminated sediments of the Saguenay river (*10*).

Advanced age is related to the high incidence of cancer. Thus, the high rate of cancer in SLE belugas might have been associated with the old age of stranded belugas, but this is not the case. Belugas that died as a result of being hunted in Alaska and belugas found stranded in the SLE were used to infer the age structures of the respective populations (Figure 1), following accepted procedures in field population biology that are appropriate to each case (*11–15*). The records (*13–15*) show that age-specific mortality rates in classes 18 and above are consistently higher in the SLE than in Alaska; thus, "St. Lawrence estuary beluga die at an earlier age," as we stated in our paper (*16*). Further, the age distribution of adult belugas that died of cancer is the same as adult belugas that died from other causes (*16*). The hypothesis that the SLE population includes a large proportion of animals in the age groups with elevated cancer rates is not supported by the data (*13–15*). Within the 18–30-year age group—the age group of SLE belugas with intestinal cancer (*16*)—there are more belugas in the Alaska population (19.8%) than in the SLE population (18.9%) (Figure 1).

The data in our paper relating to pet animals (*17*) were collected from animals seen in veterinary colleges and thus comprise a large proportion of sick animals, where cancer is most likely more prevalent than in the general population of domestic animals.

Smith and Levy (*18*) estimated that, downwind of the smelters, 40,000 tons of PAHs had accumulated in the Saguenay watershed by 1980; 20 tons of the accumulated PAHs are released annually, of which 3% is BAP (*18*). They stated that this represents a serious, chronic hazard to this environment and its inhabitant (*18*). The smelter company estimated that 100–200 tons of PAHs were released annually between 1964 and 1976 through the liquid waste discharged directly into the Saguenay River from the scrubber of one plant [Alcan, personal communication, cited in (*19*)]. Smith and Levy (*18*) more conservatively estimated that 80 tons of PAHs were discharged in the Saguenay before 1976 from that plant.

In the 1990s two studies were carried out by the Quebec Environment Ministry, each using a different norm (*20,21*). The Quebec Environment Ministry classified 16 PAHs as "group 1 PAHs," which comprises PAHs that are possibly and probably carcinogenic and those for which there is sufficient evidence of carcinogenicity. The first study was carried out on surface waters from 1997 to 1999 (*20*) using a norm from the U.S. Environmental Protection Agency (*5*); this norm of 4.4 ng/L (group 1 PAHs), which was designed to protect an individual who drank this water for his/her life time and ate aquatic organisms containing bioaccumulated material from water contaminated at the determined level, was exceeded in this study. For instance, 1 km downstream of the drinking water intake of LaBaie city, "Rivière à Mars" contained up to 19.7 ng/L in 1997 ($n = 2$) and 16.4 ng/L in 1999 of group 1 PAHs. Total PAH levels were much higher—up to 83 ng/L—in Rivière Chicoutimi in 1997 (*20*).

Cancer, the ultimate result of a progressive accumulation of mutations over a long period, requires exposure to mutagens over a significant proportion of the life span of animals and people (*22*). In marked contrast, PAH levels in the surface waters of the Saguenay-Lac Saint-Jean area have been measured only twice over a short and recent period (1997–2000) (*20,21*).

The smelter company claims that, in 2002, PAH emissions have been reduced 88% from the 1983 levels (*23*). This implies that PAH emissions were about 8 times higher in 1983 and that, in 1983 and over the previous decades, PAH levels in the surface waters, downwind of the smelters, were most likely elevated in the same proportion. Supporting this inference, Picard-Bérubé et al. (*24*) reported in 1983 that the initial BAP levels of blue mussels transplanted from a nonpolluted area to various sites along the fjord increased 200 times within 1 month (*24*). Aluminium workers, predominantly male, have been exposed to airborne PAHs (*6–8*). A significant part of the daily water intake of aluminium workers has occurred at work, as opposed to the spouses whose water intake has been largely at home.

Although no attempts have been made to detect PAHs in the tissues of people living in that region, the Quebec worker compensation board compensates aluminium workers affected with urinary bladder cancer based on their exposure to PAHs (*8*). In contrast, such attempts—which have demonstrated PAH contamination—have been made in terrestrial and aquatic wildlife of that region (*25,26*). The results and observations presented in our paper (*16*) warrant undertaking studies that would address this gap.

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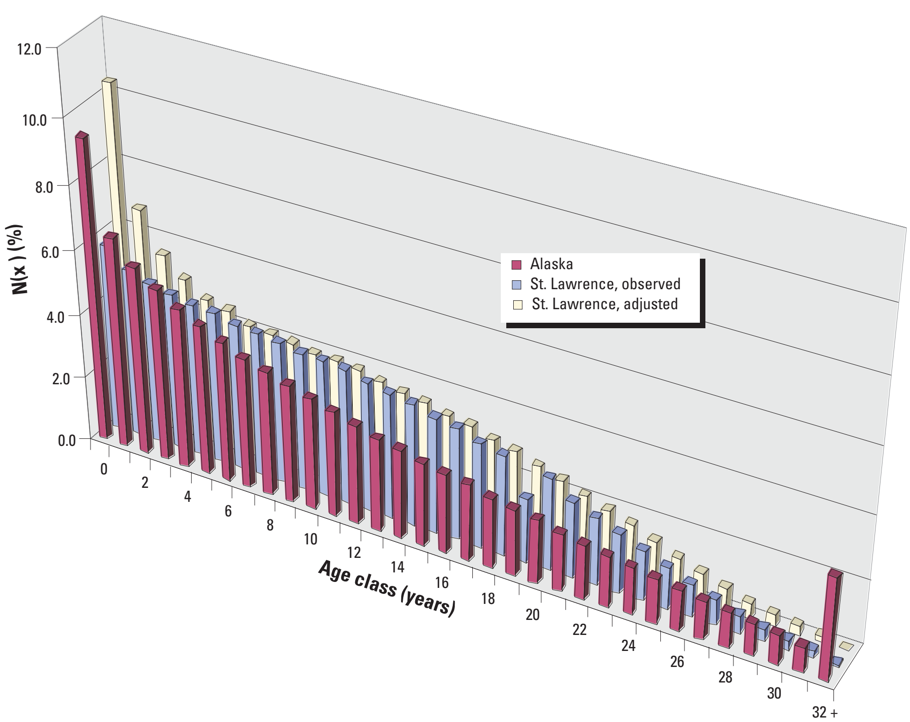

Figure 1. Age structures of beluga populations from Alaska [as in (*13*), with juvenile mortality adjusted] and from the St. Lawrence [with (adjusted) or without (observed) modifying the record to account for underrepresentation of juvenile mortality (*14,15*)]. N(x), proportions of individuals in each age class.

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Corrections and Clarifications

In Table 1 of the article ("Serum Dioxin Concentrations and Endometriosis: A Cohort Study in Seveso, Italy") by Eskenazi et al. [*EHP* 110:629–634 (2002)], two values for Zone B (row 2) were reported incorrectly. The value for total study participants (third column) should be 504 instead of 540, and the value for "Uncertain" (fifth column) should be 255 instead of 225.

In "A New Mechanism for DNA Alterations Induced by Alpha Particles Such as Those Emitted by Radon and Radon Progeny," by Lehnert and Goodwin [*EHP* 105(suppl 5):1095–1101 (1997)], Alina Deshpande was acknowledged for her technical assistance. More appropriately, she should have been included as a co-author of the article. The authors regret this oversight.

In "Health Effects Assessment for Environmental Perchlorate Contamination," by Greer et al. [*EHP* 110:927–937 (2002)], the triangle and diamond symbols in the key were transposed. The figure appears here correctly. *EHP* regrets the error.

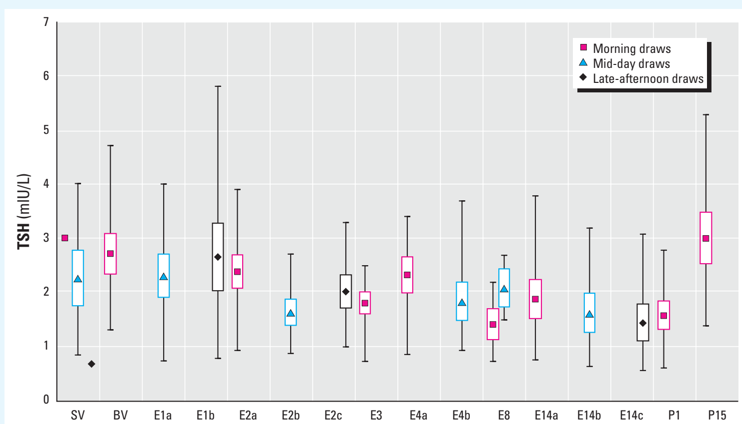

Figure 6. Values of serum TSH in all blood samples from main study subjects in the 0.5-mg/kg-day dose group, categorized according to the 16 scheduled blood-draw events. BV, baseline visit; SV, screening visit. Where more than one blood draw was scheduled on a given day, a, b, and c denote the first, second, and third draw, respectively. In the SV category, the square and diamond correspond to the lone morning and late-afternoon blood draws, respectively, for that event. Boxes indicate SE; whiskers indicate maximum and minimum. The *x*-axis is not to scale regarding the timing of events. For all events except the following, $n = 8$. Morning draws: SV ($n = 1$), E4a ($n = 7$), E8 ($n = 5$); mid-day draws: SV ($n = 5$), E2b ($n = 7$), E8 ($n = 3$); afternoon draws: SV ($n = 1$), E2c ($n = 7$), E14c ($n = 7$).

In the September Focus article, "Economy and Environment: China Seeks a Balance" [*EHP* 110:A516–A522], part of the last paragraph was inadvertently omitted. The final paragraph should have read:

> Ultimately, Western governments and industries would be wise to help China on its path to a cleaner environment. As an enticement, the market opportunities are potentially huge. And with nearly a quarter of the world's population affected, the consequences of success or failure will be truly global.

EHP regrets the error.