Chronic Lyme Disease: Psychogenic Fantasy or Somatic Infection?

Sigal and Hassett published an article about Lyme disease in the *EHP Supplements* (Sigal and Hassett 2002), suggesting that chronic Lyme disease is "psychogenic." I do not think that Sigal and Hassett, nonpsychiatrists, are qualified to speak about psychiatric matters. I, however, actually have had the disease, which they characterize as "medically unexplained," for over 25 years and have 15 years of experience as a patient advocate and educator. I beg to differ.

Many reports in the peer-reviewed medical literature substantiate the notion of persistent infection. *Borrelia burgdorferi*, the causative organism, has been cultured after "adequate" antibiotic therapy from the brain, eye, heart, spleen, spinal fluid, skin, lymph nodes, joints, and synovial fluid (Cimmino et al. 1989; Cimperman et al. 1996; Haupl et al. 1993; Liegner et al. 1992; Oksi et al. 1996; Patmas 1994; Peter et al. 1993; Pfister et al. 1991; Preac Mursic et al. 1993; Reimers et al. 1993; Schmidli et al. 1988). Table 1 provides information on some of the articles supporting persistent infection. A more complete listing of Lyme disease abstracts may be obtained from the Lyme Disease Network (2002).

I and the many other Lyme patients I know are neither "confused" nor "insecure." We did not seek a "societally and morally acceptable explanation" (Sigal and Hassett 2002) for our illness; we sought a scientific and medical explanation. We have been fortunate enough to find informed doctors to treat us with long-term antibiotics and to return to our normal activities. In my experience, patients with Lyme disease who are treated for psychogenic illnesses alone do not fare well.

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Table 1. Available information of Lyme disease.

Reference	Summary
Battafarano et al. 1993	"A patient had chronic septic Lyme arthritis of the knee for 7 years, despite multiple antibiotic trials and multiple arthroscopic and open synovectomies. Spirochetes were documented in synovium and synovial fluid."
Cimmino et al. 1989	"G-penicillin treatment was ineffective.… *Borrelia*-like spirochetes were identified histologically in the spleen."
Georgilis et al. 1992	"Fibroblasts protected *B. burgdorferi* for at least 14 days of exposure to ceftriaxone." Other cell types also protected *B. burgdorferi*, contributing to its long-term survival.
Haupl et al. 1993	The patient had relapsing Lyme borreliosis with choroiditis, arthritis, carditis, and tendinitis. Repeated antibiotic treatment was necessary to stop the progression of disease but did not completely eliminate *B. burgdorferi* from all sites of infection. Viable *B. burgdorferi* was cultured from a ligament sample obtained surgically.
Liegner et al. 1992	Paired CSF and serum tests for antibodies to *B. burgdorferi* and PCR for *B. burgdorferi*-specific oligonucleotides in CSF were negative. Eleven months later, after treatment with cefotaxime and minocycline, a T-cell stimulation test with *B. burgdorferi* antigens was strongly positive. A year later, paired serum and CSF samples were strongly positive for antibodies to *B. burgdorferi* and CSF was culture positive.
Liegner et al. 1993	Patient was ELISA-negative after treatment, but blood was PCR-positive and *B. burgdorferi*-compatible structure was visualized in skin biopsy. Further treatment resolved erythema migrans.
Montgomery et al. 1993	"The macrophage is a known reservoir for a number of infectious agents, and is therefore a likely candidate site for persistence of *Borrelia burgdorferi* …. Although the large majority of spirochetes within a given cell were dead, we saw occasional live ones … and can reculture [them]."
Oksi et al. 1999	One patient had been treated for 47 weeks, including 7 weeks of intravenous ceftriaxone; primary diagnosis was confirmed by positive biopsy and the relapse 44 weeks after treatment confirmed by a positive plasma PCR. One patient had relapse 130 weeks after 16 weeks of treatment. The patient was seropositive initially but seronegative at relapse. Relapse was confirmed by positive PCR, and there was no history of reinfection.
Preac Mursic et al. 1996	Persistence of *B. burgdorferi s.l.* and clinical recurrences occur in patients despite antibiotic treatment. Culture confirmed relapses after 12–14 days of treatment courses in five patients.
Straubinger et al. 1997	"Treatment with high doses of amoxicillin or doxycycline for 30 days diminished but failed to eliminate persistent infection." Antibody titers fell, but 6 months after antibiotic treatment was discontinued, "antibody levels began to rise again, presumably in response to proliferation of the surviving pool of spirochetes."

Abbreviations: CSF, cerebrospinal fluid; PCR, polymerase chain reaction.

Chronic Lyme Disease: It's Not All in Our Heads

Those of us with chronic Lyme disease are not at all confused, as suggested by Sigal and Hassett (2002). We know from years of experience that we have real, specific symptoms that are usually painful and disabling and include severe headaches, crippling arthritis, and heart palpitations, which lead to serious heart disease. Many of us know that our symptoms are kept in check while we are on antibiotics, but they painfully reappear when the antibiotics are withdrawn. Just because the medical community cannot detect a specific causative bacterium and managed health care companies want to maximize profits doesn't mean that those of us afflicted with this terrible condition are delusional and not truly benefiting from antibiotic treatment. We are not all crazy; we are sick and we should not be required to prove it to get medical care.

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Chronic Lyme Disease: Sigal and Hassett's Response

Nowhere in our article (Sigal and Hassett 2002) do we minimize or devalue the pain or suffering of patients with chronic Lyme disease nor do we state that such patients are "crazy" or "delusional." Further, we do not dismiss the possibility that some such patients actually have infection that persists despite adequate prior antibiotic treatment. Nonetheless, we take this opportunity to again issue a caution against making the diagnosis of chronic Lyme disease in the absence of objective clinical and microbiologic evidence of infection.

Without demonstrable physical findings and laboratory proof of the persistence of *Borrelia burgdorferi*, the diagnosis should remain in significant doubt. Certainly, a few, often poorly documented case reports claiming persistence (many in Europe, where the organism, the vector, and the ambient human immunogenetic types are different) cannot be a basis for the widespread diagnosis of chronic infection in certain regions of the United States and in certain practices.

We hope our article (Sigal and Hassett 2002) will help educate physicians and assist them in being more sensitive to their patients and to more correctly identify the true underlying cause of their patients' symptoms in a compassionate manner. However, many patients are misdiagnosed with chronic Lyme disease, a diagnosis often made to provide an explanation for a bewildering array of complaints within an acceptable framework for both patient and physician. Instead, these patients deserve the truth. Improvement and cure require provision of a correct scientific and medical explanation and properly directed therapies. For many of the patients we see, chronic antibiotics, continuous reassurance, and yet another antibiotic regimen when the current one fails have led to frustration, anger, depression, and chronic suffering. Further, the inappropriate use of broad spectrum antibiotics, often for long periods, is not without personal (e.g., *Clostridium dificile* infection, allergies, and other adverse reactions) and societal (e.g., contribution to the development of resistant strains such as methicillin- and vancomycin-resistant *Staphylococcus*) risks.

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Cancer in Beluga from the St. Lawrence Estuary

Martineau et al. (2002) reported that St. Lawrence beluga (SLB) have high cancer rates. Unfortunately, errors in their interpretation of the data have led them to overstate the importance of cancer and its links to environmental sources.

Martineau et al. (2002) compared mortality patterns between the beach-cast, naturally dead animals in the protected, predation-free SLB population with those from a hunted Alaskan beluga population, which were also subject to natural predation (Burns and Seaman 1985). This analysis is incorrect because it compares the raw frequency distribution of ages at death of SLB with an age structure reconstructed by fitting a model to the Alaskan age and reproductive data from the harvest, under the assumption of constant natural mortality (Burns and Seaman 1985). A correct analysis (Burns and Seaman 1985) of the SLB data shows that mortality rises continuously from about 6 years of age. There is no peak in the 20s, and no difference in standing population at 21–25 years of age between Alaskan beluga [8%: Table 7 in (Burns and Seaman 1985)] and SLB [8.1%: stranding data 1982–1998 (Martineau et al. 2002)].

Martineau et al. (2002) estimated an annual cancer rate (AR) among SLB beluga using

$$\mathrm{AR} = \left(\frac{\mathrm{SLB}_{\text{with cancer}}}{t}\right) \times \left(\frac{100{,}000}{\text{population size}}\right),$$

where t is 17 years. The authors used an aerial survey index estimate of 650 whales, which incorporates a conservative 15% correction factor to account for animals that were below the surface on transect lines and therefore unseen (Kingsley 1999). Our concern is the author's failure to recognize the importance of this correction to their conclusions. Kingsley (1999) applied a factor of 2.09, developed specifically for SLB (Kinglsey and Gauthier. In Press), to the average of five surveys using similar methods between 1988 and 1997, and hence estimated a mean fully corrected population size of 1,100 ± 113 (mean ± SD) whales. Using this estimate of population size reduces the cancer rate from 163 to 96 (95% confidence interval, 88–105).

Martineau et al. (2002) asserted that the rate of winter stranding is as high as or higher than that during the rest of the year because of harsh weather conditions, but that these carcasses are not recovered and adjust their calculations accordingly. These adjustments may be inappropriate for two reasons. First, the beluga is an Arctic species adapted to ice and cold water, and is unlikely to be stressed by winter conditions in the St. Lawrence; mortality rates could in fact be lower during this period. Second, animals that die in winter could freeze into the ice and be beach-cast and thus recovered after breakup. A more appropriate estimated minimum with cancer (EMC) can be estimated from the observed stranding rate as a minimum, resulting in an EMC of 47, or as one end of a range of 47–63. The adjusted estimated annual rate (AAR) of cancer for a complete year,

$$\mathrm{AAR} = \left(\frac{\mathrm{EMC}}{t}\right) \times \left(\frac{100{,}000\ \mathrm{SLB}}{1{,}100}\right),$$

would then be 251 instead of the value of 570 presented by Martineau (2002). The AAR of cancer in SLB is then no longer the highest of the seven species they tabulated.

Martineau et al. (2002) argued that epidemiologically the SLB data resemble

human data more than that from domestic animals because population size is known; however, they then cited incorrect population estimates for the SLB. They also stated that rates among pets are overestimated because pets are protected from other causes of death. Cancer rates in pets are not overestimated; they are high because death is inevitable, and mortality from predation, trauma, and malnutrition is nearly nonexistent—just as is the case for the SLB.

The absence of malnutrition as a cause of death and the generally good body condition of recovered carcasses from this unhunted, predator-free, wild population is unusual. The small number of juvenile carcasses recovered, prima facie indicates a bias toward older adults, whereas the absence of emaciated carcasses may indicate a bias against animals in poor condition that are likely to sink. The beach-cast sample of carcasses is clearly incomplete and must be assumed to be biased. The direction and size of this bias is unknown, but it may increase the apparent significance of cancer.

Martineau et al. (2002) suggested that bottom invertebrates from the sediments of the Saguenay Fjord are important in the diet, and a significant source of contamination with polycyclic aromatic hydrocarbons (PAHs), leading to high cancer rates. However, the Saguenay River is not used extensively by all SLB, particularly west of the Baie Ste. Marguerite area, for which PAH data are available. Furthermore, the only existing diet data come not from the Saguenay, but from collections made elsewhere in the St. Lawrence over 60 years ago (Vladykov 1946).

The SLB population is unique in its accessibility and in its geographic isolation from Arctic conspecifics. The incidence of cancer among a sample of beach-cast animals is interesting, but failure to make the proper links between a sample with unknown bias and the population means that the importance of cancer in this population and links to environmental conditions are overstated.

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Cancer in Beluga: Response

In their letter, Hammill et al. propose an analysis of mortality patterns for the St. Lawrence beluga (SLB) population without submitting data or methods. They state, however, that there is no difference in standing populations at 21–25 years of age between Alaskan beluga and SLB, supporting our conclusion that the high cancer rate in this age group cannot be explained by its overrepresentation in the SLB population (Martineau et al. 2002a). We have also shown in an earlier paper (Béland et al. 1988) and in an updated analysis (Béland et al. Unpublished data) that SLB do not die at a more advanced age than Arctic beluga. Hammill et al. suggest that the SLB population, the denominator used in estimating cancer rate, is larger than the value we used and thus the resulting cancer rate would be lower than that we obtained (Martineau et al. 2002b). We used 650, the population estimate that was both undisputed and published in a refereed scientific journal at that time (and still is) (Lesage and Kingsley 1998). Hammill et al. use a higher, recently developed correction factor of 2.09 to estimate the mean population size at 1,100. Importantly, Kingsley, a coauthor of the letter, previously stated that the mortality rate we used elsewhere, 1.4%, was unrealistically low (Kingsley 2002)—with which we concurred (Martineau 2002). Using a higher, more realistic annual 6% death rate from a population of 960, Kingsley (2002) estimated the SLB mortality rate at 58 deaths/year and derived an adjusted estimated annual rate (AAR) of 1,208, more than twice the AAR we estimated. (Using a population estimate of 1,100, he would have obtained an even higher AAR of 1,433.)

Our record includes emaciated individuals, a large number of young animals [22.4% of strandings were ≤ 6 years old (Béland et al. 1988)], and very few beach-cast animals that died over the winter. We assumed that the number of stranding occurring during winter is at least the same as that reported during the rest of the year (Martineau et al. 2002b). It is difficult to conceive that the ice cover and rough weather conditions which prevail in winter would lower mortality rates in animals that need to surface regularly for breathing, especially considering that SLB are often affected by pneumonia (Martineau et al. 1994). In addition, "carcasses with terminal diseases are often found after several days of rough weather" (Martineau et al. 2002b). Predation-induced mortality would muddle the issue, but predation seems not to be an important cause of mortality, as stated by Hammill et al.

Because the populations of pets used to determine cancer rates were exclusively animals examined in veterinary hospitals (Martineau et al 2002b; Priester and Mantel 1971; Priester and McKay 1980), many of these animals, if not most, were sick; thus, cancer rates are more frequent in these animals than in the general population of domestic animals—the same way that cancer patients are proportionally more numerous in a hospital than they are in the general human population (Priester and McKay 1980; Priester and Mantel 1971). In addition, most pet animals are sheltered and receive veterinary curative and preventive care along with abundant well-balanced food. Because cancer develops more frequently in older animals and humans, these factors, by prolonging life span, increase cancer rates relatively to free-ranging animals (Martineau et al 2002b; Fowler 1987).

The Saguenay River has been qualified as "extensively used" by SLB (Michaud 1993). SLB are grouped in three types of herds: adults and juveniles, only adults, and mixed herds. Among seven areas qualified as "extensively used" by SLB, the Saguenay River is one of only two areas frequented a substantial amount of time by the three types of herds. In addition, 5% of the population, on average, is found in the Saguenay River in the summer (Michaud 1993; Environnement Canada 1995). SLB are present in the Saguenay River, occasionally up to 100 km upstream, in the segment most contaminated by polycyclic aromatic hydrocarbons (PAHs) (Martel et al. 1986, 1987; Michaud 1993). Sainte-Marguerite Bay, located 24 km upstream the Saguenay River, is frequented daily by groups of SLB remaining in the bay up to 16 hr, probably for feeding (Michaud 1993). Benthic invertebrates are part of the diet of SLB, and benthic invertebrates are present in the Saguenay River (Lemieux 1996; Vladykov 1946). There is no reason to believe that SLB actively avoid these prey in the Saguenay River.

Mussels at the mouth of the Saguenay River accumulated PAHs produced by the local aluminum smelters, and clean mussels

transplanted in the Saguenay River accumulated benzo[*a*]pyrene concentrations that were 200 times higher than pretransplantation concentrations, demonstrating that PAHs are also being exported into the St Lawrence Estuary (Cossa et al. 1983; Picard-Bérubé and Cossa 1983).

In conclusion, irrespective of uncertainties regarding population size, the percentage of mortality in SLB due to cancer is very high relative to other free-ranging mammals and to free-ranging cetaceans [for instance 2 of 90 dolphins (Cowan DF. Personal communication)] examined in similar conditions, and is higher than in pets and in humans [Figure 4 in Martineau et al 2002b] (Cowan et al. 1986; Howard et al. 1983; Kirkwood et al. 1997; Kuiken et al. 1993; Stroud and Roffe 1979). The contamination of the Saguenay River and immediate St. Lawrence estuary area by PAHs released massively by the local aluminum smelters over half a century and the exposure of belugas to these compounds make PAHs the most likely etiology for certain types of cancer in SLB as we stated in our article (Martineau et al. 2002b).

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Correction

In the editorial in the January issue of *EHP* [111:A14–A15 (2003)], David Michaels was described as the first recipient of the American Public Health Association's David P. Rall Award for Advocacy in Public Health; he was actually the second recipient of the award. Eula Bingham received the first David P. Rall Award in 2000.