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fatigue syndrome, fibromyalgia, multiple sclerosis, and motor neuron disease contributes to the misdiagnosis and inadequate treatment of this spirochetal illness.^{2,17-19}

As numerous specialists are consulted, the patient may feel unheard and trivialised, and become overwrought in dealing with multiple diagnoses, each aligned with a physician's specialty yet not contributing to improved health. The suggestion that unresolved emotional issues are causing the patient's symptoms can be overwhelming for the patient and lead to questions of factitious or psychoneurotic illness. Cognitive impairment^{2,9,20} and chronic pain from neuropathy can activate depressive illness.^{2,9} Neuropsychiatric manifestations of Lyme borreliosis in school-age children are often misdiagnosed as learning, behavioural, or attention deficit disorders.^{9,20}

Lyme disease is a complex and extremely serious illness that affects

patients and the entire medical community. I hope my comments will broaden the perspective on Lyme borreliosis presented in Hengge and colleagues' review.

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Human monkeypox

Daniel Di Giulio and Paul Eckburg's review¹ of human monkeypox in Africa is informative. Monkeypox is an important, emerging zoonosis and we applaud the authors' support for a vigorous research agenda. However, we wish to clarify some of Di Giulio and Eckburg's statements about the outbreak that occurred in the USA in 2003.

Rabbits were not found to be enzootic hosts of the monkeypox virus during the 2003 outbreak. DiGiulio and Eckburg cite the first in a series of five reports on the monkeypox outbreak published in the *Morbidity and Mortality Weekly Report (MMWR)* as evidence that a rabbit became infected with the monkeypox virus after having been exposed to an infected prairie dog at a veterinary clinic.² They place emphasis on the virus being transmitted from one New World species to another and the increasing potential for establishment of an enzootic reservoir. These authors further assert that the infected rabbit transmitted

the virus to a human being. This *MMWR* article described the association of an ill rabbit and a possible human monkeypox case; however, laboratory testing of rabbit necropsy specimens did not find monkeypox virus infection (Centers for Disease Control and Prevention, unpublished data). 1 month later the update published in the *MMWR*³ correctly reported that the cases of monkeypox in human beings were associated with exposure to prairie dogs.

The case counts presented in Di Giulio and Eckburg's review was also derived from case totals released in an interim report.⁴ These numbers were compiled before completion of laboratory testing, follow-up interviews, and in some cases, clinical evaluations of people suspected of having had monkeypox. Updated case counts were published 1 week after those cited by Di Giulio and Eckburg.³

These authors' account of the introduction of monkeypox virus into

the USA via a shipment of exotic rodents from Africa is not complete. The six species of African rodent referred to in the review were part of the suspect importation from Africa, but several other African non-rodent species, including palm civets, genets, and cusimanses, were present as well. The permissiveness and reservoir potential of these species for monkeypox virus is not known, but their presence in the shipment may prove noteworthy. Additional epidemiological and laboratory studies will address this matter.

Since the latest public-health update on the US outbreak of monkeypox,³ new laboratory test results have been developed and data collection and analysis continue. A comprehensive publication describing the epidemiology of this outbreak is forthcoming.

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Indiana, and Wisconsin, 2003. *MMWR Morb Mortal Wkly Rep* 2003; **52**: 537–40.

- 3 Anon. Update: multistate outbreak of monkeypox—Illinois, Indiana, Kansas, Missouri, Ohio, and Wisconsin, 2003. *MMWR Morb Mortal Wkly Rep* 2003; **52**: 642–46.
- 4 Anon. Update: multistate outbreak of monkeypox—Illinois, Indiana, Kansas, Missouri, Ohio, and Wisconsin, 2003. *MMWR Morb Mortal Wkly Rep* 2003; **52**: 616–18.

Authors' reply

We appreciate the comments of Mary G Reynolds and colleagues regarding the 2003 US monkeypox outbreak. Our review¹ states that a rabbit “was implicated as the source of primary infection in one US case”. This statement is based on an issue of the *Morbidity and Mortality Weekly Report* (MMWR) that reported, “One patient had contact with a rabbit (family Leporidae) that became ill after exposure to an ill prairie dog at a veterinary clinic”.² Reynolds et al cite unpublished data to inform us that later testing of necropsy specimens from this rabbit showed no evidence of infection with the monkeypox virus. We welcome this clarification since the four subsequent MMWR updates on the US outbreak do not document this.^{3–6} We do not agree with Reynolds and colleagues’ suggestion that a particular MMWR update⁶ sufficiently clarified this issue for readers. That report indicated that prairie dog exposures were associated with the 35 confirmed cases of monkeypox in human beings; it did not indicate that prairie dog exposures were associated with all of the 71 reported

human cases that comprised the outbreak.⁶ It simply states that “the majority” of all cases were exposed to prairie dogs,⁶ thereby leaving open the possibility that the earlier report implicating the rabbit² was still accurate.

We recognise that in infectious disease outbreaks initial case counts are commonly revised because of updated case definitions and ongoing investigations. We have addressed the revised counts noted by Reynolds et al in a published letter.⁷

We agree with Reynolds and colleagues that the full breadth of potential animal reservoirs for the monkeypox virus are poorly characterised and that the presence of non-rodent species in the contaminated African shipment may prove noteworthy. We look forward to the publication of additional findings from their ongoing laboratory and epidemiologic research into this outbreak.

Finally, we would like to acknowledge the Armed Forces Institute of Pathology website (<http://www.afip.org/Departments/infectious/mp/index.html>), from which we identified figures 1 and 2 in our review.¹

html), from which we identified figures 1 and 2 in our review.¹

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- 7 Di Giulio DB, Eckburg PB. Human monkeypox. *Lancet Infect Dis* 2004; **4**: 199.

Injecting reason

Your editorial on the medically supervised safe-injection facility in Vancouver, Canada,¹ correctly noted that intravenous drug use accounts for about one third of all AIDS and one half of hepatitis C cases in the USA. The figures for women are much higher. The US Centers for Disease Control and Prevention estimate that 57% of AIDS cases among American women are linked to injection drug use or sex with partners who inject drugs (<http://www.cdc.gov/hiv/pubs/facts/idu.htm>). This easily preventable public-health

crisis is a direct result of zero-tolerance laws that restrict access to clean syringes.

The good news is that Canada has already adopted many of the harm-reduction interventions first pioneered in Europe. The bad news is that Canada’s southern neighbour continues to use its superpower status to export a dangerous moral crusade around the globe. I am confident that the prospective cohort study conducted by Evan Wood and colleagues in Vancouver will confirm what public-health advocates in North

America have been saying for years. Canada cannot afford to emulate the harm-maximisation approach of the USA.

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