enhanced operational management and field coordination of outbreak response. The monitoring of disease spread would be further enhanced by widespread use of practical, tested, field-based digital surveillance instruments that could be used for many diseases. There is enormous potential to mitigate the lost lives and economic burdens of the EVD epidemic by the development, adoption, and monitoring of these instruments.

Future research should incorporate transportation networks as well as EVD case data at higher spatial resolutions (eg, subprefectures, districts, chiefdoms). Furthermore, these findings for EVD velocity should be compared with other diseases in Guinea, Liberia, and Sierra Leone that are transmitted from person-to-person. This would provide further insight into disease transmission patterns in Guinea, Liberia, and Sierra Leone.

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1 US Centers for Disease Control and Prevention. 2014 Ebola outbreak in West Africa. http://www.cdc.gov/vhf/ebola/outbreaks/2014-west-africa/index.html (accessed May 15, 2015).

2 Wesolowski A, Buckee CO, Bengtsson L, Wetter E, Lu X, Tatem AJ. Commentary: containing the ebola outbreak—the potential and challenge of mobile network data. *PLoS Curr* 2014; **6**. DOI:10.1371/currents.outbreaks.0177e7fcf52217b8b634376e2f3efc5e.

3 Bogoch II, Creatore MI, Cetron MS, et al. Assessment of the potential for international dissemination of Ebola virus via commercial air travel during the 2014 west African outbreak. *Lancet* 2015; **385:** 29–35.

Emergent chikungunya virus and arthritis in the Americas

The Correspondence letter by Emilie Javelle and colleagues[1] emphasises new insights into arthritic disease after chikungunya virus infection. We are gratified that new evidence is confirming our argument that chikungunya virus is likely to exacerbate or increase susceptibility to underlying joint diseases.[2] The need to intensify drug treatment for underlying arthritis after chikungunya virus infection[1] provides a clear example of the importance of chikungunya virus infection in these patients. We agree with Javelle and colleagues that it will be important to develop data about possible interactions between chikungunya virus bone pathology and osteoporosis.

Clinical manifestations during chikungunya virus infection are typically self-limiting, although a few patients develop complications, particularly neurological, and also fulminant hepatitis and cardiological manifestations. Despite a typical one in 1000 low fatality ratio associated with chikungunya virus infection,[3] a substantial increase in mortality rate was reported during the 2005–2006 Indian ocean outbreak. The cause of this increase is not clear, although comorbidities were present in many cases. More than 150 deaths have been reported in the outbreak in the Americas, and this should be monitored carefully, particularly in patients with comorbidities, to detect changes in virulence. The first clinical reports of chikungunya virus infections in the USA are beginning to emerge. Miner and colleagues reported clinical and immunological characteristics of ten chikungunya-virus-infected American travellers who had returned from Haiti.[4] This study identified chikungunya virus as a major mimic of seronegative rheumatoid arthritis, thus emphasising the need to consider chikungunya virus in patients presenting with symmetric arthritis, particularly after travel to endemic regions. Acute clinical symptoms including fever, rash, symmetrical joint pain, and swelling, and morning stiffness were observed in most of the patients. Joint pain persisting for at least 6 weeks was reported in eight out of ten patients. The clinical features closely resembled seronegative rheumatoid arthritis based on American College of Rheumatology/European League Against Rheumatism 2010 criteria. Two patients who continued to experience debilitating joint pain at 4 months had clinical evidence of synovitis in the hands and feet. The patients had raised levels of natural killer cells and activated T-killer cells, both of which might contribute to virus clearance. CD8+ T cells can also regulate bone resorption, potentially leading to bone loss.[2] The striking similarity in the clinical features between patients infected with chikungunya virus and patients with rheumatoid arthritis and the elevated cytotoxic T-cell profile suggest that bone loss might be the underlying cause of chronic persistent joint pain experienced by the patients in Miner and colleague's study.

Although the emergence of chikungunya has been overshadowed in the media by Ebola virus, the spread to the Americas is an international public health emergency. The virus has become established in South America. As noted by Alfaro-Toloza and colleagues,[5] many of the affected countries have already-stretched health-care sectors that are being seriously tested by the growing outbreak. On a related note, there is a growing awareness that chikungunya virus might endanger the blood supply in outbreak areas, with a consequent risk of transfusion-associated chikungunya virus infections.[6] Additionally, the presence of endemic mosquito vectors of high transmission efficiency will likely accelerate spread of chikungunya virus around

the Americas. In this regard, *Aedes albopictus*, the Asian tiger mosquito, is highly competent as a chikungunya virus vector.[7] Up to ten North American and four South American *A albopictus* strains have the potential to transmit chikungunya.[8]

The present situation in the Americas is on a knife-edge: will this debilitating virus, which has infected millions of people in the Asian region in recent years, become established in the Americas? The chikungunya virus outbreak is developing on a daily basis and we hope this Correspondence will act to stimulate the thinking of researchers and public health officers.

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1 Javelle E, Gautret P, Simon F. Chikungunya, the emerging migratory rheumatism. *Lancet Infect Dis* 2015; **15:** 509–10.
2 Burt F, Chen W, Mahalingam S. Chikungunya virus and arthritic disease. *Lancet Infect Dis* 2014; **14:** 789–90.
3 Morens DM, Fauci AS. Chikungunya at the door—deja vu all over again? *N Engl J Med* 2014; **371:** 885–87.
4 Miner JJ, Aw Yeang HX, Fox JM, et al. Chikungunya viral arthritis in the United States: a mimic of seronegative rheumatoid arthritis. *Arthritis Rheum* 2015; **67:** 1214–20.
5 Alfaro-Toloza P, Clouet-Huerta DE, Rodríguez-Morales AJ. Chikungunya, the emerging migratory rheumatism. *Lancet Infect Dis* 2015; **15:** 510.
6 Appassakij H, Promwong C, Rujirojindakul P, Wutthanarungsan R, Silpapojakul K. The risk of blood transfusion-associated Chikungunya fever during the 2009 epidemic in Songkhla Province, Thailand. *Transfusion* 2014; **54:** 1945–52.
7 Vega-Rúa A, Zouache K, Girod R, Failloux A, Lourenço-de-Oliveira R. High level of vector competence of *Aedes aegypti* and *Aedes albopictus* from ten American countries as a crucial factor in the spread of Chikungunya virus. *J Virol* 2014; **88:** 6294–306.
8 Mitchell CJ. Vector competence of North and South American strains of *Aedes albopictus* for certain arboviruses: a review. *J Am Mosq Control Assoc* 1991; **7:** 446–51.