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► **COVER:** 'Gateways' by Tom Wilson, inspired by the perspective on p312.



DAVID O'CONNELL



SUSAN JONES



SHEILAGH MOLLOY



SHANNON AMOILS

During initial HIV-1 infection in humans, the viral entry site is attacked by different HIV-1 subtypes, including the CXCR4-utilizing HIV-1 (X4 HIV-1) and CCR5-utilizing HIV-1 (R5 HIV-1). But during transmission, an as-yet-unidentified selective mechanism filters out the X4 subtype, restricting initial HIV infection to the R5 subtype. The identity of this elusive 'gatekeeper' is much sought after, as an understanding of this physiological antiviral barrier could hold the key to effective preventive therapies.

On page 312, Leonid Margolis and Robin Shattock propose that, instead of a single gatekeeping mechanism, X4 entry is restricted by the cumulative effect of a series of 'leaky' gatekeepers that provide a selective advantage for R5 transmission. The authors argue that, rather than focusing on a single restriction mechanism, researchers should define the contributions of each potential gatekeeper, and that similarly, effective microbicides and vaccines should target multiple stages of mucosal transmission.

On page 307, Siouxsie Wiles and colleagues highlight the inherent limitations of using animal models to study infectious diseases. Focusing on mouse models of bacterial pathogens, they show that a failure to recognize fundamental differences between models and natural disease can produce misleading results. In particular, the exclusion of natural transmission from models of infection can profoundly influence pathogenic phenotypes.

Last, on page 249, Laurent Keller and Michael G. Surette take a fresh look at the phenomenon of quorum sensing. They examine bacterial interactions in the context of ecological and evolutionary frameworks that are commonly applied to studies of higher social animals. These insights reveal that bacterial communications are not merely cooperative in nature, but also have a role in competition within and between species.

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Nature Publishing Group, 75 Varick Street,
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nature@naturejpn.com
Chiyoda Building 5F, 2-37-1 Ichigayatamachi,
Shinjuku-ku, Tokyo 162-0843, Japan
Tel: +81 3 3267 8751;
Fax: +81 3 3267 8746
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Harpal Singh Gill
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