By adjusting the intensity and frequency of the laser, Gerritsma and colleagues could vary at will the effective mass of the simulated free Dirac particle and the effective speed of light, which appears in the Dirac equation and constrains the particle's motion. They first observed the Zitterbewegung for an ion with zero average momentum, the internal states of which would be in a superposition (corresponding to the superposition of the positive- and negative-energy states of a free Dirac particle) with equal relative strengths. The frequency of this quasi-periodic motion extends from about 10 kHz to 80 kHz — a range that was accessible in the authors' experiments.

Next, the researchers created another superposition state of positive- and negative-energy states, but one in which these two components moved in opposite directions. They observed that the Zitterbewegung disappears as soon as these parts leave the space they had initially jointly occupied. Furthermore, they showed that a pure negative-energy state results in no Zitterbewegung. These results, obtained by controlling the ion's initial state, confirm that it is indeed the interference between positive- and negative-energy states that gives rise to the Zitterbewegung. When the authors changed the particle's effective mass and kept its momentum constant, both in the non-relativistic limit (large effective mass) and in the highly relativistic case (small effective mass), the Zitterbewegung disappeared, whereas this quivering motion was clearly present in the regime in between.

The measurement of the ion's average position as it evolves in time requires exacting experimental control, because it needs to be carried out with a precision of a few nanometres to be able to resolve the Zitterbewegung. Gerritsma *et al.* achieve this precision by mapping, using a sequence of laser pulses, the ion's motion onto its internal states, which can in turn be measured through the detection of scattered laser light.

Gerritsma and colleagues' experiment[1] not only demonstrates a much-sought-after effect in a real system, but also marks important progress in bringing quantum simulations closer to yielding new insight even in scientific fields that lie beyond the realm of quantum-information science. Trapped ions[5], neutral atoms[6], superfluids[7] or optical fields[8] may be used to further our understanding of relativistic quantum mechanics and astrophysical processes. Furthermore, simulating many-body physical phenomena with neutral atoms may help in deciphering hitherto unsolved problems in condensed-matter physics — for instance, the nature of high-temperature superconductivity[4,9]. Similarly, internal states of a collection of trapped ions can be made to interact as particle spins do, with the interaction strength designed by the experimenter[10]. Thus, trapped ions could be used to investigate phenomena such as quantum magnetism[11,12]. Finally, coupled cavity arrays in the solid state, although still in their infancy in terms of experimental work, offer promise for quantum simulations[13,14].

Although quantum-information research progresses in unforeseen, and sometimes spectacular, steps, building a universal quantum computer — one that would be able to simulate other quantum systems and thus solve problems that are intractable on a classical computer — still poses formidable challenges. Specialized quantum simulations, such as that performed by Gerritsma and colleagues[1], promise to be a versatile and, at the same time, more amenable scientific tool. ■

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VIROLOGY

Bornavirus enters the genome

Cédric Feschotte

A survey of mammalian genomes has unexpectedly unearthed DNA derived from bornaviruses, leading to speculation about the role of these viruses in causing mutations with evolutionary and medical consequences.

Some people might find it disquieting that a hefty 8% of human genetic material originates not from our vertebrate ancestors but from viruses. The assimilation of viral sequences into the host genome is a process referred to as endogenization. It occurs when viral DNA integrates into a chromosome of reproductive germline cells and is subsequently passed from parent to offspring. Until now, retroviruses were the only viruses known to generate such endogenous copies in vertebrates. But on page 84 of this issue, Horie *et al.*[1] report that non-retroviral viruses called bornaviruses have been endogenized repeatedly during mammalian evolution. The finding unveils bornaviruses as a potential cause of mutation and also as an unforeseen source of genomic innovation (Fig. 1).

Borna disease virus (BDV) owes its name to the town of Borna, Germany, the site of a dreadful virus epidemic that decimated a regiment of cavalry horses in 1885. However, it is only recently that BDV has been characterized genetically: it belongs to the order Mononegavirales, and is a negative-sense RNA virus (in which the single-stranded RNA genome has the opposite sequence to messenger RNA). BDV infects a range of birds and mammals, including humans, and is unique among RNA viruses in that it naturally infects only neurons, establishing a persistent infection in its host's brain. In addition, the entire life cycle of BDV takes place in the nucleus of the infected cells, and does not require chromosomal integration[2]. This intimate association of BDV with the cell nucleus prompted Horie *et al.* to investigate whether bornaviruses may have left behind a record of past infection in the form of endogenous elements.

Horie *et al.* searched the 234 currently available eukaryotic genomes for sequences that are similar to that of BDV, and unearthed a plethora of endogenous Borna-like N (EBLN) elements in diverse mammals. The sequences of these elements resemble the nucleoprotein (*N*) gene of BDV, which encodes a structural protein involved in packaging the viral RNA into a nucleocapsid[2]. The authors show[1] that bornavirus endogenization has occurred in multiple mammalian lineages and at different times, ranging from more than 40 million years ago in anthropoid primates to less than 10 million years ago in squirrels. These molecular fossils add to the growing evidence[3–6] for the long-term coevolution of RNA viruses and their mammalian hosts.

All instances of endogenization described by Horie *et al.*[1] correspond to the *N* gene, and although most EBLN sequences are fragmentary and seem to be non-functional (they have decayed into pseudogenes), surprisingly, two EBLNs in the human genome are annotated as protein-coding genes. They retain long open reading frames (sequences that seem to encode proteins) and are transcribed from

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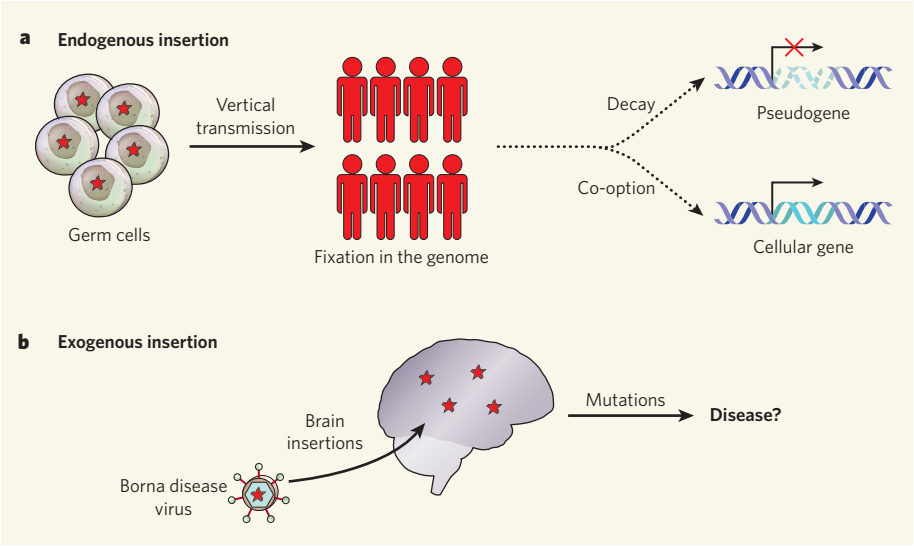

Figure 1 | Bornavirus in the genome, for better or worse. a, Horie *et al.*[1] report that bornavirus gene sequences (red stars) became integrated into the germline of our ancestors, and through vertical transmission (by conventional inheritance) have become 'fixed' in the genome, thereby becoming endogenous viral insertions. A fixed viral insertion can follow one of two evolutionary fates: it can either decay into a pseudogene or be co-opted to form a new gene whose product has a cellular function. **b**, Circulating bornavirus sequences can become integrated into the genome of brain cells (the current target of Borna disease virus) after infection (exogenous insertion). These sequences are not heritable, but might cause mutations that interfere with brain function and may contribute to the development of psychiatric disorders.

DNA into mRNAs in the various tissues and cell lines examined[1]. Also, one of the two proteins (LOC340900) has been reported[7] to interact with several well-known cellular proteins. Thus, the discovery of EBLNs uncovers two cases of viral DNA that has apparently been co-opted to form cellular genes (Fig. 1a). Although it is not known whether EBLN-derived proteins are functional in human cells, these proteins may have been usurped by the host, at least initially, to serve an antiviral function. There are precedents for this in mice and sheep[8,9], in which endogenous retroviral capsid proteins offer protection against exogenous retroviral infections.

How are EBLN elements generated? Unlike retroviruses, BDV does not need to integrate into the host DNA to replicate, and therefore the virus genome does not encode the machinery for reverse transcription of its RNA into DNA. Despite this, using the polymerase chain reaction, Horie *et al.*[1] were able to detect BDV DNA in various infected cell lines and in the brain of persistently infected mice. Furthermore, after infecting human cells for 30 days, the authors could isolate chromosomally integrated BDV DNA along with flanking host genomic sequences. These BDV insertions resemble EBLN elements in that they are derived from the *N* gene and exhibit the hallmarks of retroposition (the process by which RNA is integrated into a DNA genome), including a stretch of adenine nucleotides at the 3′ end of the inserted element and a short duplication of the target site.

In mammalian genomes, retroposition is primarily driven by the activity of L1 long interspersed nucleotide elements — pieces of mobile DNA that make copies of themselves and reinsert into the genome[10]. L1 has colonized the genome of mammals for more than 100 million years and continues to replicate actively in several species, including humans. The L1 enzymatic machinery can reverse transcribe its own RNA into DNA, but can also act on non-L1 RNA templates — throughout evolution, this promiscuity has caused the bombardment of mammalian genomes with millions of DNA inserts[10]. The abundance of BDV RNA in the nucleus of persistently infected cells, coupled with some peculiar properties of the *N* gene RNA, might have promoted their fortuitous recognition by the L1 machinery.

The fact that Horie and colleagues[1] could readily detect BDV DNA and chromosomal insertions in human cells suggests that BDV retroposition might occur at an appreciable frequency during BDV infection, creating a source of mutation in infected individuals (Fig. 1b). This yields a tantalizing and testable hypothesis for the alleged, but still controversial, causative association of BDV infection with certain psychiatric disorders, such as schizophrenia and mood disorders[2,11]. This possibility becomes even more intriguing when considering the recent demonstration of L1 hyperactivity in the human brain[12], the primary site of BDV infection. ■

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PALAEONTOLOGY

Muddy tetrapod origins

Philippe Janvier and Gaël Clément

The tracks left by organisms are among the most difficult of fossils to interpret. But just such evidence puts debate about the origins of four-limbed vertebrates (which include ourselves) on a changed footing.

The term 'tetrapodomorph fishes' scarcely rolls off the tongue, but these are fossil animals that have a special place in the evolutionary history of vertebrates. It was through the stepwise transformation of paired fins in this lineage of lobe-finned fishes that paired limbs with digits arose, marking the advent of the four-limbed vertebrates, or tetrapods. This event occurred sometime during the Devonian period, between 416 million and 359 million years (Myr) ago. On page 43 of this issue, Niedźwiedzki *et al.*[1] describe fossil tracks that were clearly made by a four-limbed animal possessing digits (see image on the cover of this issue). But they date to a time well before tetrapods were thought to have existed.

The temporal and taxonomic context for this discovery is outlined in Figure 1. The earliest complete evidence for limbs with distinct digits is provided by the articulated skeletons of the iconic early tetrapods *Ichthyostega* and *Acanthostega*, which date to the Famennian stage (374–359 Myr ago)[2]. But the record extends much further back, to the Frasnian and possibly the late Givetian (385 Myr ago), thanks to the identification of several skeletal 'signatures'