of sperm than female oocytes. This presents a differential dynamic for the survival of cells expressing maternal X genes compared with those expressing the error-prone paternal X genes. Because many more neurons are produced in the brain than survive, neuronal competition may overcome such DNA errors on the paternal X chromosome by inducing programmed cell death (apoptosis), resulting in a skewed population of surviving cells expressing the maternal X.

Gregg and colleagues' data also associate some 347 genes on autosomal (non-sex) chromosomes in the adult brain with a gender-specific parental allele bias. They show that, compared with male mice, female mice express three times the number of genes with gender-specific imprinting in the preoptic region — an area that mediates maternal care. Intriguingly, most of these genes were of paternal origin in the preoptic areas of both male and female brains. The authors interpret this finding as a mechanism by which parents influence gene expression differently in the brains of their sons and daughters. As the preoptic area is a testosterone-dependent, sexually dimorphic region — with more cells in sons than in daughters — and because it is important for male sexual behaviour and maternal care, these results are open to engaging discussion.

A monumental part of the study by Gregg *et al.* was the generation of a colour-coded 'heat map' of the maternal or paternal genes expressed according to brain region. The authors find that out of 118 brain regions, 26 regions account for more than two-thirds of the imprinted genes expressed in the brain, with half of the regions showing maternal expression and the other half paternal expression. There seems to be a mismatch between this map and their other observations indicating that mostly paternal genes are expressed in the preoptic area (67%) and prefrontal cortex (71%) of the adult brain[4]. Moreover, the increased expression of paternal genes found in the adult brain is also the opposite of the favoured expression of maternal genes in the developing brain. Undoubtedly, brain expression of imprinted genes is more complex than previously thought. But one point is clear: parental influence over gene expression is both spatially and temporally regulated in this organ and may differ between developing and adult brains.

The fate of imprinted genes is sealed by their epigenetic modification in the germ line, to ensure that all the resulting somatic and germ cells retain fidelity for the expression of either the maternal or the paternal imprint[5]. Beyond genomic imprinting, the take-home message from these papers[3,4] extends to the complexity of epigenetic mechanisms that are used in both the developing and adult brain. Several genomic regions in Gregg and colleagues' data set do not match the life-long imprints reported in other studies[6]. Nevertheless, these regions are epigenetically regulated, because the authors observed significant differences in the relative expression of maternal and paternal alleles in hybrid crosses between two strains. Such cross-strain effects were especially notable in the developing brain, with some 774 genes showing a paternal-expression bias and 943 genes showing a maternal bias.

In part, these observations on imprinted gene expression may represent within-species differences in imprinting that emerge in only one of the reciprocal hybrid crosses. However, in the developing brain — for the analysis of which the authors pooled the embryos — these biases may represent genes in which the epigenetic imprinting of control regions may be affected by their different chromosomal position[7]. It is also noteworthy that brain development is not a precise event, and developmental errors result in apoptosis, which reduces the potential brain size by roughly two-thirds. A large proportion of imprinted genes mediate apoptosis, and this in itself may complicate comparisons of adult and developing brains.

Gregg and colleagues' work implicates some ten times more imprinted chromosomal loci than previously appreciated. The extent to which these loci represent imprints sculpted by germline epigenetic modification such as methylation, as well as their tissue variability and background-strain differences, needs more clarification. These studies nevertheless firmly embed the genetic understanding of brain development and evolution in the ever-expanding field of epigenetics. ■

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PLANT PATHOLOGY

Sudden larch death

Clive Brasier and Joan Webber

An aggressive and unpredictable fungal pathogen is devastating larch plantations in Britain. Its remarkably broad host range, and the possibility of further geographical spread, give heightened cause for concern.

For more than a decade, a stream of invasive tree pathogens has been arriving in Europe and North America[1,2]. Among the more damaging and unpredictable arrivals is *Phytophthora ramorum*. For some years, this oomycete fungus has caused 'sudden oak death' in the western United States. In like manner, it now poses a serious threat to Japanese larch, and possibly other tree species, in Europe.

Damaging invasions of tree pathogens are not new, as earlier pandemics of Dutch elm disease and chestnut blight have shown. But these events seem to be accelerating owing to the combined effects of increasing globalization of trade in plants and a flaw in international plant biosecurity protocols. Simply put, for trade purposes, organisms not yet described cannot be legislated against, yet many recent arrivals are completely new to science, presumably introduced from under-explored ecosystems[2]. Indeed some 90% of fungi are probably unknown, and therefore undescribed[3]. In Europe alone, aggressive invasive pathogens are currently spreading on pine, oak, alder, horse chestnut, ash and cypress, and many European nurseries are infested with exotic fungal pathogens, especially species of *Phytophthora* ('plant destroyer')[2].

A new species of unknown origin, *P. ramorum* has since 1995 afflicted evergreen oak and tanoak along 1,500 kilometres of forests in coastal California and Oregon[4,5] (Fig. 1). It is believed to have spread initially from a rhododendron nursery in the San Francisco Bay area and is now estimated to have killed several million trees. It exhibits the classic hallmarks of an invasive plant pathogen: genetic uniformity consistent with a genetic 'bottleneck'[5], coupled with spread from multiple secondary foci of infection resulting from human movement of infected plants[4,5]. *Phytophthora ramorum* is also the first major 'aerial' — as opposed to root-infecting — forest *Phytophthora* species to be identified, attacking mainly foliage and stems, as does the potato blight, *Phytophthora infestans*.

Phytophthora ramorum probably arrived in Europe in the 1990s, but was only formally named as a new species in 2001 (ref. 6). Despite coming under European Community emergency control measures in 2002, it spread rapidly and widely thereafter, mainly on rhododendron within the ornamental nursery trade. In 2003, it was found to be infecting woodland trees in southwest England and the Netherlands. But, until now, tree infections have been comparatively rare and limited mainly to native beech and non-native oak growing close to affected rhododendron. Interestingly, the European and North American populations of *P. ramorum* are genetically and behaviourally

distinct, indicating separate introduction events into each continent[5].

Many aerial *Phytophthora* pathogens, including *P. infestans*, have a narrow host range. By contrast, *P. ramorum* exhibits a remarkably broad range, at least in its new invasive behaviour[4,5]. Outside nurseries, it has infected more than 40 species across 12 families of tree and non-tree hosts in California, and another 40 species in Europe, although only a minority of tree species have proved highly susceptible. The mechanisms underlying this wide host potential are not known, but their importance is highlighted by another development.

In September 2009, widespread dieback and mortality of mature and juvenile Japanese larch, *Larix kaempferi*, were observed in plantations across southwest England and shown to be associated with infection by *P. ramorum*[7,8]. Symptoms include needle necrosis, branch dieback, stem lesions and heavy resin bleeding. This is the first serious damage caused by *P. ramorum* to conifers and plantation trees. When growing adjacent to larch, other species such as beech, chestnut, birch, rhododendron and Douglas fir are being infected, indicating heavy infection pressure consistent with observations of exceptionally high *P. ramorum* spore counts on artificially inoculated larch needles[7] and frequent infection of larch needle litter.

Between May and July 2010, further outbreaks were detected by aerial and ground surveys in south Wales[8] and southern England (Fig. 1). An estimated 1,900 hectares of larch plantations (about 0.5 million trees) now show symptoms of *P. ramorum* infection: in other words, the fungus is spreading on a landscape scale in Britain, as in the United States. The British Forestry Commission and the private sector are felling all affected trees to minimize further spread, both to forests and to susceptible heathland vegetation.

Larch is not only an important timber tree in western Britain, but is also the only deciduous conifer, providing value for landscape, biodiversity and recreational purposes. This development could therefore have a significant impact on local economies and Britain's strategic reserve of timber. Moreover, *P. ramorum* and the other invasive pathogens now entering Europe could narrow the choice of plantation species for reafforestation, especially in Britain[9], and undermine proposals to use tree planting as a means of mitigating climate change through carbon sequestration[9].

The jump of *P. ramorum* from rhododendron to larch illustrates its behavioural unpredictability. Another issue of concern is its longer-term ecological and evolutionary potential. Introduced pathogens inevitably encounter new hosts (some with little intrinsic resistance) and other novel biotic and abiotic influences. These represent selection forces that, individually or collectively, will result in adaptation. Some recently invasive tree pathogens have also exchanged genes with resident species, resulting in a genetically modified pathogen or even an entirely new pathogen[10]. One example is *Ophiostoma novo-ulmi*, which is responsible for the pandemic of Dutch elm disease and which has acquired 'useful' genes from a related species during its migration[11]. Another is *Phytophthora alni*, which is attacking alder across Europe and is a species hybrid that may have arisen in a European nursery[10]. Each invasive pathogen therefore represents an uncontrolled, open-ended experiment in evolution[2].

It remains to be seen whether *P. ramorum* has already begun to adapt to larch and what other hosts it will attack in the future. It has yet to be found outside Europe and North America, but may represent a threat to trees in climatically suitable habitats in South America, the Himalayas, Asia and Australasia, including *L. kaempferi* in its native Japan. Given current international trade practices and biosecurity protocols, and the considerable epidemic momentum that *P. ramorum* now has in the United States and Britain, prospects for preventing its further spread do not look good. ■

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Figure 1 | Consequences of infection with *Phytophthora ramorum*. Left, dead and dying tanoak in California, July 2010. Right, similarly afflicted larch in southwest England, May 2010.

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HIGH-TEMPERATURE SUPERCONDUCTIVITY

The benefit of fractal dirt

Jan Zaanen

Measurements of X-ray diffraction on small patches of a copper oxide superconductor reveal that oxygen crystal defects form fractal structures that seem to promote high-temperature superconductivity.

In the area of electronic materials, 'oxides' is a buzzword[1] referring to chemically complex solids containing oxygen and transition metals. In these materials, electrons can team up at low temperatures to display collective behaviour that is much richer than that found in conventional metals and semiconductors. The realization of this behaviour started with the discovery of superconductivity at a high temperature in copper oxides in 1986. These 'high-T_c cuprates' (T_c is the temperature below which the material superconducts) are still king of the hill in the oxide landscape, because the myriad of mysterious 'quantum matter' phenomena observed in these systems is among the most intriguing puzzles in modern physics[2].

Conventional semiconductors took off when materials scientists had learned how to grow crystals that are nearly perfect. For the oxides, this is a formidable challenge. Besides the need to control the many elements in the unit cell of the material's crystal lattice, the chemistry is further complicated by oxygen's reputation as the vagabond among the elements. It tends to roam around as an 'oxygen interstitial' in the skeleton formed by