affect learning and memory? Studies involving tissue-specific and developmental-stage-specific loss of *Dlk1* function, as well as behavioural tests, should provide further evidence to support the current study.

More broadly, genomic imprinting serves as an important interface between environment and genes in mature tissues. Determining how *Dlk1* and other imprinted genes operate in concert with signalling cues, as well as discovering the factors that initiate and maintain differential methylation at the genomic region housing *Dlk1*, will lead to a better understanding of adult neurogenesis and brain development. Such knowledge will also inform emerging hypotheses about the role of trans-generational effects (or of their absence in particular processes), in which DNA and histone modifications in the ancestral genetic material affect the phenotypes of subsequent generations[11]. ■

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EARTHQUAKES

The lessons of Tohoku-Oki

An exceptional data set documents surface deformation before, during and after the earthquake that struck northeastern Japan in March 2011. But models for assessing seismic and tsunami hazard remain inadequate. SEE LETTER P.373

JEAN-PHILIPPE AVOUAC

Earthquake science has entered a new era with the development of space-based technologies to measure surface deformation at the boundaries of tectonic plates and large faults. Japan has been at the forefront in implementing these technologies, in particular with the deployment some 15 years ago of a network of continuously recording Global Positioning System (GPS) stations known as GeoNet. Papers analysing the data associated with the devastating Tohoku-Oki earthquake of 11 March 2011 are now appearing. One of these, by Ozawa *et al.*[1], is published on page 373* of this issue.

With a moment magnitude (M_w) of 9.0, the Tohoku-Oki earthquake ranks among the largest ever recorded. The data collected at the GeoNet stations[1] indicate that it resulted from the sudden slip of a remarkably compact area (400 kilometres long by 200 kilometres wide) of the plate interface where the Pacific plate slides beneath the Okhotsk plate, on which northern Japan lies. The rupture area (Fig. 1) lies off the coastline of Honshu, Japan's biggest island, and extends east nearly all the way to the Japan trench — hence the particularly devastating tsunami produced by the earthquake.

Other new papers[2,3] provide further information. A combination[2] of GPS measurements and underwater acoustic sounding shows that the sea bottom in the epicentral area moved seaward by as much as 24 metres and was uplifted by about 3 metres. Therefore, slip along the plate interface at depth must have exceeded the 27-metre peak slip inferred from the GeoNet data[1]; it may have been even more than 50 metres, as suggested from the joint modelling[3] of the GeoNet data and sea-bottom pressure records of the tsunami waves. For comparison, this is about twice the peak slip determined for the giant earthquakes in Sumatra in 2004 (M_w 9.4) and in Chile in 2010 (M_w 9.0) — and larger than that estimated for the biggest earthquake ever recorded[4], the M_w-9.5 event of 1960, which ruptured more than 1,000 km of the plate boundary off the coast of southern Chile.

Over the 15 years preceding the 2011 event, the GeoNet data[5] had revealed the slow accumulation of strain across Honshu, with the Pacific plate squeezing and dragging down the eastern edge of Honshu. We know, however, that the coast of Honshu is being uplifted in the long term, so a significant fraction of that 'interseismic' strain — strain accumulating between earthquakes — must be compensated by sudden episodes of uplift. The current model holds that interseismic strain on the upper plate is purely elastic, and is 'recovered' during seismic rupture of the plate interface, so that in the long run the upper plate does not deform[6]. This assumption provides a rationale to relate slip on the plate interface to interseismic strain on the upper plate. Where the plate interface is creeping, strain on the upper plate is negligible; but where the plate interface is locked, the upper plate is compressed and dragged down, building up elastic strain until it is released when the locked patches slip.

Several earlier studies adopted this assumption[5,7,8] and found that the measured strain

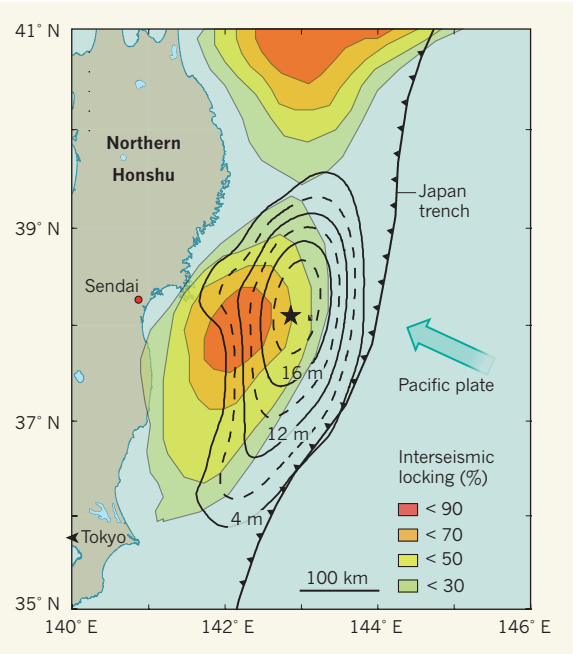

Figure 1 | Location of the Tohoku-Oki earthquake. The earthquake, with its epicentre marked by a star, ruptured the plate interface along which the Pacific plate slides beneath northern Honshu at a rate of 8 centimetres per year. Ozawa and colleagues' analysis[1] shows that the rupture area and distribution of slip (represented by the black contour lines) roughly coincide with a patch of the plate interface that had remained locked over the preceding decades[8] (coloured area east of Sendai). The earthquake source was extremely compact and produced very large slip at relatively shallow depth (less than 20 km depth), hence the devastating tsunami. The other well-locked patch in the north coincides with rupture areas of large historical earthquakes (in particular, the M_w-8.5 Sanriku earthquake of 1896 and the M_w-8.2 Tokachi-Oki earthquake of 1968).

*This article and the paper under discussion[1] were published online on 15 June 2011.

across Honshu required a large, locked patch off the coast of Sendai (Fig. 1). The rupture area of the Tohoku-Oki earthquake coincides quite well with that area[3]. A noteworthy discrepancy, however, is that the rupture reached closer to the Japan trench, where interseismic models suggested there was little locking. The particularly extensive, shallow slip seen in the Tohoku-Oki event could be due either to high preseismic stress left over from previous ruptures of the plate interface that failed to reach the trench, or, as seismological investigations suggest[9], to specific properties of the plate interface. In any case, the observed slip requires the shallow plate interface to have remained locked, at least partially, in the period before the earthquake.

The published interseismic models[5,7,8] indicated little locking at shallow depth, essentially as a result of built-in methodological assumptions: the shallow portion of the plate interface is actually not well constrained if only onshore data are used[1]. These models may have been misleadingly interpreted as discounting the possibility of extensive shallow slip, which in fact occurred. Therefore, in the absence of direct constraints from sea-bottom geodesy, it may be preferable for models to assume maximum locking of the shallow zone of the plate interface. In fact, the interseismic data do not exclude a locked region off the coast of Sendai extending all the way to the shallow plate-boundary zone at the trench. Such an assumption raises questions for assessing the frequency of Tohoku-Oki-like earthquakes.

The estimated slip along the plate boundary off the coast of northern Honshu — due to earthquakes over the past few centuries — falls well short of balancing the slip deficit that should have accumulated over that period owing to interseismic locking. So it might seem that a large earthquake there was overdue. Indeed, according to the published interseismic models, interseismic strain builds up really fast on that boundary: it should take only a few centuries to accumulate enough strain to generate an M_w-9.0 earthquake. Such large events should recur even more often if locking of the shallow portion of the plate interface is assumed in the modelling of interseismic strain.

By contrast, on the basis of historical and palaeo-tsunami records[10], large earthquakes would be predicted to return only once every 1,000 years, or even less frequently. The way out of this conundrum is not clear. There is no evidence for particularly frequent episodes of large aseismic slip in that area, and postseismic afterslip, although significant[1], is much too small to balance the slip budget. So either the slip deficit accumulating in the interseismic period is overestimated (which might happen if, for example, a fraction of interseismic strain is not recoverable), or it is incorrect to assume that geodetic rates measured over a decade or so are representative of strain build-up over periods of centuries to millennia.

Another paradoxical and possibly related observation is that the Tohoku-Oki earthquake induced more than 1 metre of systematic coastal subsidence, whereas uplift would have been expected to balance the subsidence rate of 5 millimetres per year during the interseismic period. The long-term coastal uplift requires deformation events that are large and frequent enough to compensate for that subsidence. This might call for a review of both the assumption that interseismic deformation of the upper plate is purely elastic, and the corollary that interseismic elastic strain is relaxed only by earthquakes that occur along the plate interface.

Finally, the geodetic data acquired both before[5,7,8] and after[1] the Tohoku-Oki earthquake suggest that the plate interface south of the rupture area is mostly creeping aseismically. There is thus no indication of a major zone of strain build-up on that portion of the plate boundary that might threaten Tokyo. But it is clear that although geodetic networks are invaluable instruments for observing strain accumulation and seismic release at plate boundaries and major faults, we don't yet have an adequate theory to use these data for earthquake and tsunami hazard assessment. ■

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MICROBIOLOGY

Molecular syringes scratch the surface

New data suggest that the most recently discovered class of bacterial 'molecular syringes' inject proteins only across the outer membrane of target cells during interbacterial competition. SEE ARTICLE P.343

PEGGY COTTER

Many bacteria use specialized secretion systems to inject proteins or DNA into cells of eukaryotic organisms (such as animals and plants) or into other bacteria. Little is known about the mechanism of secretion in the most recently discovered class of these molecular syringes, the type VI secretion system (T6SS)[1]. On page 343 of this issue, Mougous and colleagues (Russell *et al.*[2]) show that the T6SS of the bacterium *Pseudomonas aeruginosa* delivers two proteins into target bacteria. These proteins degrade peptidoglycan, a highly cross-linked lattice that lies just below the outer membrane of Gram-negative bacteria in a region called the periplasm, causing lysis of the target cell. These findings strongly suggest that the T6SS 'needles' puncture only one membrane (the bacterial outer membrane in this case), providing substantial insight into this system's mechanism of action.

T6SSs were discovered on the basis of their contribution to symbiosis and virulence in bacterial interactions with eukaryotes. Until recently, the only proteins that had been shown to enter host cells through T6SSs were the VgrG proteins of the bacterium *Vibrio cholerae*, which seem to form the membrane-puncturing tip of the T6SS needle[3].

Last year, Mougous and co-workers[4] identified three candidate T6SS-dependent 'effector' proteins in *P. aeruginosa*. One of these proteins, called Tse2, was toxic to both mammalian and bacterial cells if the cells were engineered to produce it intracellularly. The co-production of an immunity protein called Tsi2 prevented this toxicity. Surprisingly, however, *P. aeruginosa* itself could not intoxicate mammalian cells if co-cultured with them, but it could outcompete other Gram-negative bacteria in a manner dependent on both cell–cell contact and Tse2.

Subsequently, Pukatzki and colleagues[5] showed that *V. cholerae* could also outcompete other Gram-negative bacteria, and that it did so using the same T6SS that it uses to inject proteins into amoeba and mammalian macrophages. These results suggested that at least some T6SSs can deliver proteins into both mammalian cells and Gram-negative bacteria.

That T6SSs can inject proteins into both