depletion trends can be explained by differences between the two types of star in either age or the abundances of heavy elements such as iron. This discovery provides both a valuable method for screening possible planet-hosting systems and a potential window on the planet-formation process.

The authors[1] do not provide direct evidence for the underlying cause of the differences they have discovered. Stellar rotation is an attractive explanation, however. Rotation can induce both turbulence and global circulation currents that can mix lithium into the interior regions, where it can be destroyed. Stars are born with a range of rotation rates, leading naturally to a range of depletion histories. But testing this theory directly has proved difficult. Low-mass stars such as the Sun have magnetized winds that cause the stars to spin down as they age, and these winds cause rapid rotators to spin down preferentially relative to slow ones[5,6]. At late ages, evidence of the initial rotation is erased, leaving only potential fossil evidence, such as the record of lithium depletion. Highly precise rotation-period measurements in coeval stars, such as those in star clusters, may eventually permit this hypothesis to be tested directly.

The total angular-momentum content of a star, which describes its rotational state, is determined by interactions between the protostar and its accretion disk. The formation of planets in such disks could therefore plausibly modify the rotation of the host star. Protostars emerge from their cocoons of gas rotating at a modest rate, despite the fact that the parent gas clouds must have collapsed and spun up by enormous factors during the star-formation process. The most commonly accepted reason for the star's relatively modest initial rotation involves the transport of angular momentum, via a magnetic field, within a circumstellar accretion disk, and between the disk and the parent star[7]. Slow rotation can be explained if angular momentum is extracted from the incoming gas before it is incorporated into the star. Once a star is mostly assembled, it contracts and would ordinarily spin up. However, in the presence of a coupled accretion disk, the star can be made to co-rotate with the inner edge of the disk, preventing it from spinning up until it can become decoupled from the disk.

The observed wide range in stellar rotation rates would be explained by variations in the initial gas-accretion rate and the lifetime of the disks. The formation of giant planets can induce gaps in the accretion disk, disrupting the magnetic star–disk connection and thus plausibly permitting the star to spin up. The next stage in understanding Israelian and colleagues' results[1] is to examine rotation and to look for planets in young stars, which retain a memory of their natal rotation. Although technically challenging because of the associated high variability of young stars, such studies will be necessary to establish a physical explanation for the intriguing link between the lithium abundances described by Israelian *et al.* and the planet-formation process. ■

Marc Pinsonneault is in the Astronomy Department, Ohio State University, Columbus, Ohio 43210, USA.
e-mail: pinsono@astronomy.ohio-state.edu

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LANGUAGE EVOLUTION

The importance of being human

Martin H. Dominguez and Pasko Rakic

The *FOXP2* gene is implicated in the development of human speech and language. A comparison of the human and chimpanzee FOXP2 proteins highlights the differences in function in the two species.

Some genes find instant favour in scientific culture and, like popular celebrities, remain under the close watch of their devoted following. *FOXP2* has many of the qualities of an in-vogue gene — relevance to human disease, evolutionary significance and a prominent role in brain development. The gene was first found to be mutated in a family with language dysfunction[1], hence its implication in the development and evolution of human speech and language, and its reputation as the 'language gene'. Examination of the human *FOXP2* gene and protein sequences[2] reveals accelerated evolution of its amino-acid sequence (specifically, two new amino-acid substitutions) since humans' common ancestor with chimpanzees, suggesting that the gene has a prominent role in the acquisition of language in humans. But like the leading character in Oscar Wilde's play *The Importance of Being Ernest*, *FOXP2* leads a double life — on the one hand, it has a starring role as the language gene, but on the other hand it has roles that are less glamorous, although still worthy of scientific attention. On page 213 of this issue, Konopka *et al.*[3] advance our knowledge of *FOXP2* by publicizing some of its private doings — the underpinnings of its gene-regulatory networks, especially those conferring functions that are unique to humans.

FOXP2 is present in vocal and non-vocal animals, is important for muscle coordination in vocalization, but is also associated with many other motor functions in all vertebrate species examined. The most obvious consequence of loss of function of FOXP2 in humans[1] and rodents[4] is impairment of motor skills and coordination. Problems in motor sequencing actions or procedural learning (the acquisition of fine motor skills), including those related to the mouth and face, thus can manifest as disorders of speech and language[5]. The involvement of *FOXP2* in learning corroborates nicely with quantitative changes in synaptic plasticity (changes in the strength of a synapse between neurons) that are seen in the relevant mouse neural circuits when the gene is mutated[4] or replaced with the human version[6].

Impairment of motor-learning skills can have profound effects in humans, in whom a loss-of-function *FOXP2* mutation[1] causes disturbances in language comprehension, grammar and syntax. Such uniquely human aspects of language processing and delivery may be the result of selective elaboration of particular anatomical regions, or the acquisition of novel functions by those regions during human evolution[7]. Indeed, *FOXP2* exhibits region-specific expression in the developing human cerebral cortex, where its expression is highest in cortical regions, such as the orbital prefrontal and perisylvian association areas, thought to be responsible for higher cognitive functions and language[8]. *FOXP2* is also strongly expressed in the human striatum[9], a site in the brain that is involved in cognition and motor coordination, and changes in the branching pattern of dendrites on neurons at this site are observed[6] in mice genetically engineered to express the human version of *FOXP2*.

Complex traits such as language undoubtedly require an equally complex nexus of evolved pathways. To further understand what FOXP2 does on a molecular level, two articles[8,10] have revealed some of its probable targets, but neither study compared the regulatory effects of human and ancestral FOXP2. This is precisely what Konopka and colleagues[3] have done, using whole-genome arrays to detect differences in gene expression in human neuronal cell lines expressing either human FOXP2 ($FOXP2^{human}$) or the ancestral protein, $FOXP2^{chimp}$. The authors find that a substantial number of FOXP2 target genes are differentially regulated by $FOXP2^{human}$ and $FOXP2^{chimp}$. Many of these genes met the criteria for positive selection during human evolution (although the authors had no way of assessing their statistical significance).

This places their findings in harmony with previous results[9–11] that show FOXP2-related genes as evolutionary arbiters. Because the authors examine human-specific gene regulation by FOXP2, their work[3] may provide our first window on the co-evolution of regulatory networks that are important for human-specific features such as language, which probably require a number of genetic changes working in concert. So far, a variant of one FOXP2 target gene, *CNTNAP2*, has been associated[11] with heritable language impairment in children. Additional work is essential to elucidate the relationships between *FOXP2* and the many other genes involved in these pathways.

Konopka *et al.*[3] performed mass spectrometry comparisons of $FOXP2^{chimp}$ and $FOXP2^{human}$ immunoprecipitates, but did not find major differences in the composition of associated proteins, which might have been expected if they interact with different transactivating factors. Furthermore, the region of the FOXP2 protein at which the two human-specific amino-acid changes occurred has an unknown function, leaving open the possibility that different DNA-binding characteristics might account for the observed disparities in gene expression. An alternative explanation for differential target-gene regulation by $FOXP2^{human}$ and $FOXP2^{chimp}$ may be the higher level of the human protein in the tested cells compared with the ancestral protein, perhaps owing to greater protein stability — a larger pool of free FOXP2 might permit a relaxed DNA-binding specificity. However, because $FOXP2^{chimp}$ causes greater changes in gene expression than $FOXP2^{human}$, such an alternative mechanism is less likely. Instead, the DNA that surrounds and includes the FOXP2-binding sites in the regulatory regions of target genes may provide a genomic context for their recognition by $FOXP2^{human}$.

To explore this possibility, the authors[3] chose eight differentially expressed genes, and found that, for six of them, their short promoter regions exhibited differential activity that paralleled the quantitative expression data. A careful analysis of those promoter DNA sequences may reveal common elements that result in disparate activities with $FOXP2^{human}$ and $FOXP2^{chimp}$. Co-evolution may likewise be important in the recognition sites, as similar sequences across the human genome may bear evidence of human-lineage-specific alteration.

In summary, Konopka and colleagues' work[3] does what important discoveries usually do: it answers many questions, but raises even more. It confirms evolutionary relationships between FOXP2 proteins in different species revealed through genome sequencing, and uncovers potential mechanisms underlying the elaboration of human-specific traits such as speech. However, it also provides a starting point for future studies of the molecular basis of language and human evolution. ■

Martin H. Dominguez and Pasko Rakic are in the Department of Neurobiology and the Kavli Institute for Neuroscience at Yale University, New Haven, Connecticut 06510, USA.
e-mail: pasko.rakic@yale.edu

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CONDENSED-MATTER PHYSICS

Dirac electrons broken to pieces

Alberto F. Morpurgo

Graphene continues to surprise physicists with its remarkable electronic properties. Experiments now show that electrons in the material can team up to behave as if they are only fragments of themselves.

The fractional quantum Hall effect (FQHE) is a fascinating form of collective electronic behaviour. It arises when electrons in a strong magnetic field — applied at a right angle to the plane in which the electrons flow — act together to behave like particles with a charge that is a fraction of an electron's charge[1]. Its observation requires the use of two-dimensional systems virtually free of disorder. This is why, since its discovery by Daniel Tsui and Horst Störmer in 1982, the effect has been studied in ultrapure semiconductor heterostructures (devices that contain thin layers of one or more semiconductors) grown in an ultrahigh vacuum. In this issue, Du *et al.*[2] (page 192) and Bolotin *et al.*[3] (page 196) show that the FQHE can also be observed in graphene — a one-atom-thick sheet of graphitic carbon, the production of which requires no more sophistication than a common adhesive tape to manually exfoliate graphite in ambient conditions[4].

In the presence of a magnetic field (B), electrons carrying current in a bulk material are subjected to a force (Lorentz force) that deflects them sideways in a direction perpendicular to both the applied field and the current. The deflected electrons accumulate at the edges of the material and generate an electric field that exactly compensates for the Lorentz force. The resulting voltage causes a finite electrical resistance — termed the Hall resistance (R_H) — that, according to classical physics, increases linearly with the strength of the magnetic field, but is otherwise featureless. This unassuming electronic behaviour is what Edwin Hall first reported[5] in 1879.

It was 100 years before Klaus von Klitzing discovered[6] that, in two-dimensional conductors, the Hall effect comes with a twist. The dependence of R_H on B is far from featureless: it exhibits a series of pronounced plateaux at values precisely equal to $R_H = h/ne^2$, where e is the electron charge, h is Planck's constant and n is an integer number — which explains why the phenomenon is dubbed the integer quantum Hall effect. The plateaux originate from the quantization of the motion of individual electrons in a magnetic field, which gives rise to a ladder of energy levels known as Landau levels.

In the centre of the conductor, the Landau levels are separated by 'forbidden' energy gaps in which no electronic quantum state is available. At the edges, however, the Landau levels bend upwards and the gaps close, with each of the Landau levels forming a channel in which electrons can propagate in a single direction. When the Fermi level — the highest energy level occupied by the electrons — is located between two Landau levels in the centre of the system, electronic transport occurs through these 'edge states', and the quantum Hall effect occurs. The integer n indexing the plateaux of the Hall resistance corresponds to the number of edge states occupied by electrons[7], and the quantization of the resistance reflects the fact that the smallest unit of charge that can propagate in such states is exactly the electron charge e.

In the FQHE, n becomes a fraction[1]. This seemingly minor difference has a deep conceptual significance, as first realized by Robert Laughlin[8]. It indicates that the smallest unit of charge in the system is not the electron charge, but a fraction of it. The effect is due to correlations induced by strong interactions between the electrons: it looks as if electrons in the fractional quantum Hall regime spontaneously break into pieces or 'quasiparticles' — a counter-intuitive idea that has proved correct experimentally. But this is only one of the weird aspects of the FQHE, because these fractionally charged quasiparticles are expected to have other unusual properties, such as their odd quantum statistics — that is, the way in which the quantum-mechanical wavefunction of the system changes when two quasiparticles are swapped.

At the end of 2005, the groups of Andre Geim