

is doubtful whether analyses of overall cranial shape have the diagnostic power to distinguish between closely related taxa, as is indeed demonstrated by some of the analyses presented in the report. Species are defined by specific morphological features, not by overall cranial shape. Lordkipanidze and colleagues' list of individual features could have been informative in this respect, but it is not analysed systematically, nor is a distinction made between traits that are derived (absent in the last common ancestor of a group) or primitive (already present in the last common ancestor) — a distinction that is essential to establishing phylogenetic relationships. Moreover, the features are categorized in a way that sometimes obscures, rather than highlights, important differences. For example, two crania attributed to *H. rudolfensis*⁹ clearly differ from all other early *Homo* specimens in the degree of facial projection around the mouth. This distinction is not revealed in the authors' table of features because of the arbitrary way the associated angle is categorized. Finally, the authors make no reference to the available non-cranial fossil evidence, even though biomechanical analyses of specimens attributed to *H. habilis* and *H. erectus* indicate marked differences in locomotive behaviour¹¹.

The new cranium's small brain size, projecting face and large cheek teeth are primitive for *H. erectus* (in the conventional use of this species name), but the specimen also shows derived morphological features that are typically found in this species, but not in specimens attributed to *H. habilis* or *H. rudolfensis*. These include its thick and protruding brow ridges, the distinct shape of the occipital bone (Fig. 1) and the arrangement of the temporal bone in basal view. This pattern of combined primitive and derived morphology is seen in other Dmanisi specimens as well, but in the new cranium the primitive aspect is particularly prominent. As such, this morphology seems to correspond to what one would expect not too long after the *H. erectus* lineage diverged from a more generalized form of early *Homo*. It would also be compatible with the centrifugal model of speciation¹², in which central populations in Africa are more derived, and peripherally distributed ones in western Asia and southern Africa (such as *Homo* at the Swartkrans site) retain primitive features.

The discovery of the new Dmanisi cranium will greatly help with the evaluation of the fossil record of early *Homo* in eastern Africa, which is temporally and geographically more

diverse, and generally less well-preserved. This should contribute to a better understanding of where and when the *H. erectus* lineage first emerged, and how it relates to other taxa of early *Homo*. ■

Fred Spoor is in the Department of Human Evolution, Max Planck Institute for Evolutionary Anthropology, Leipzig 04103, Germany, and University College London, UK. e-mail: f.spoor@eva.mpg.de

1. Lordkipanidze, D. *et al.* *Nature* **449**, 305–310 (2007).
2. Rightmire, G. P. & Lordkipanidze, D. in *The First Humans* (eds Grine, F. E., Fleagle, J. G. & Leakey, R. E.) 39–48 (Springer, 2009).
3. Gabunia, L., de Lumley, M. A., Vekua, A., Lordkipanidze, D. & de Lumley, H. *C.R. Palevol* **1**, 243–253 (2002).
4. Skinner, M. M., Gordon, A. D. & Collard, N. J. *J. Hum. Evol.* **51**, 36–49 (2006).
5. Rightmire, G. P., Van Arsdale, A. P. & Lordkipanidze, D. *J. Hum. Evol.* **54**, 904–908 (2008).
6. Lordkipanidze, D. *et al.* *Science* **342**, 326–331 (2013).
7. Wood, B. *Nature* **355**, 783–790 (1992).
8. Spoor, F. *et al.* *Nature* **448**, 688–691 (2007).
9. Leakey, M. G. *et al.* *Nature* **488**, 201–204 (2012).
10. Suwa, G. *et al.* *Anthropol. Sci.* **115**, 133 (2007).
11. Ruff, C. *Am. J. Phys. Anthropol.* **138**, 90–100 (2009).
12. Groves, C. P. *A Theory of Human and Primate Evolution* (Clarendon, 1989).

ASTROPHYSICS

Recipe for regularity

A detailed astrophysical model has been laid out that not only reproduces the far-infrared–radio correlation for galaxies that are actively forming stars, but also predicts how the correlation is modified at high redshift.

ELLEN ZWEIBEL

Galaxies, particularly those that, like our own Milky Way, actively form stars, are complex systems in which a vast array of physical processes operate simultaneously (Fig. 1). Patterns of regularity in galaxy behaviour are often interpreted, therefore, as evidence for global self-organization, and thus for the workings of the system at a fundamental level. One such pattern is a remarkably tight correlation between the rate of star formation and that of synchrotron radiation from cosmic-ray electrons gyrating in the galactic magnetic field. This correlation has now been reinterpreted by Schleicher and Beck, through the lens of modern ideas about magnetic-field amplification in galaxies, in a paper¹ published in *Astronomy & Astrophysics*. The authors provide predictions about the evolution of the correlation, and the physical quantities underlying it, over cosmic time.



Figure 1 | An ultra-luminous infrared galaxy. Galaxy IRAS 19297-0406, shown here in a composite image, is an extreme example, in terms of its star-formation rate, of the type of galaxy described by Schleicher and Beck¹.

These predictions will be testable with the radio telescopes currently under development.

Observations at wavelengths from radio to γ -rays are providing a detailed picture of the structure and evolution of galaxies, from their formation just a few hundred million years after the Big Bang to the present. One of the most tantalizing results to emerge from multi-wavelength studies is that the far-infrared (FIR) and radio luminosities (L_{fir} and L_{rad}) from galaxies over several orders of magnitude in galaxy luminosity and size show a weakly nonlinear correlation: $L_{\text{rad}} \sim L_{\text{fir}}^x$, where the exponent x has a value in the range 1.15–1.3 (refs 2,3).

The radio luminosity is primarily emitted by relativistic cosmic-ray electrons circling galactic magnetic-field lines and is roughly proportional to the product of magnetic-field and cosmic-ray-electron energy densities. The FIR luminosity is emitted by interstellar dust heated by the ultraviolet radiation from massive stars. Because the lifetimes of these stars are short by galaxy-evolution standards (a few million years), the number of these stars in a galaxy is proportional to the rate at which they form. Thus, the FIR–radio correlation suggests that the product of magnetic-field and cosmic-ray-electron energy densities scales with the star-formation rate, with exponent x and a scatter of only about 2 over a wide range of galaxy properties. The sensitivity and resolution of telescopes have now improved to the point

that this correlation has been confirmed in galaxies that have cosmological redshifts of about 2, which we observe at a time when the Universe was only about one-fifth of its present age.

A model illustrates the plausibility of the FIR–radio correlation. Because massive stars end their lives as supernovae, the supernova rate scales with the star-formation rate. There is good evidence that cosmic rays are accelerated by supernovae. Suppose a fraction of the energy of each supernova is converted to cosmic rays. Suppose further that the main energy sink for cosmic rays is synchrotron radiation. Then, the energy density of cosmic-ray electrons is directly proportional to the star-formation rate and inversely proportional to the magnetic-energy density, whereas the synchrotron emissivity is independent of magnetic-energy density and directly proportional to the star-formation rate. So, by assumption, is the FIR emissivity; therefore, the synchrotron and FIR emissivities are correlated.

This model is a simplified version of so-called calorimeter models of the FIR–radio correlation⁴. More general versions, which include mechanisms of electron-energy loss other than synchrotron radiation, such as inverse Compton scattering of electrons by ambient photons or electron escape from the galaxy, do yield synchrotron emissivity that depends on magnetic-energy density. This introduces an element of uncertainty into the models, because galactic magnetic fields are difficult to measure, and the theory of how

they originated and grow is still incomplete⁵.

In their paper, Schleicher and Beck have laid out a more detailed model that reproduces the observed FIR–radio correlation. The model's new ingredients are an estimate of galactic magnetic-field strength based on recent results⁶ from the theory of magnetic-field amplification by galactic turbulence, and an estimate of the level of galactic turbulence which ties it to the star-formation — or supernova — rate. There are good theoretical and empirical bases for both estimates. Although the origin of the large-scale magnetic fields seen in many galaxies is still unclear, the idea that turbulence regulates the amplitude of a small-scale turbulent magnetic field such that the energy densities of the two are proportional is well established⁶. What matters for the FIR–radio correlation is magnetic-energy density, not large-scale field structure. Likewise, it has long been argued, on general energetic grounds, that energy supplied by massive stars and supernovae is a primary driver of turbulence in the interstellar medium. But up to now, these well-founded ideas had not been used quantitatively in a model of the FIR–radio correlation.

On the basis of their simple models of turbulence driving and magnetic-field amplification, Schleicher and Beck derive a weak nonlinearity of the FIR–radio correlation. Galaxies with low star-formation rates have less turbulence, weaker magnetic fields, smaller synchrotron losses and lower radio fluxes; at high star-formation rates the opposite holds. The model also

predicts how the FIR–radio correlation should scale with cosmological redshift. The density of cosmic microwave background photons that permeate the Universe and mean star-formation rates increase with redshift, enhancing the importance of inverse Compton emission relative to synchrotron emission at high redshift (inverse Compton emission is produced by cosmic-ray electrons interacting with photons, so if there are more cosmic-background and starlight photons there is more inverse Compton emission). This alters the correlation at high redshift, a change that should be observable with the Square Kilometre Array (SKA) radio telescope now under construction. Verification of Schleicher and Beck's prediction would be evidence for rapid turbulent amplification of magnetic fields in the early lives of galaxies. ■

Ellen Zweibel is in the Departments of Astronomy and Physics, University of Wisconsin, Madison, Wisconsin 53706, USA. e-mail: zweibel@astro.wisc.edu

- Schleicher, D. R. G. & Beck, R. *Astron. Astrophys.* **556**, A142 (2013).
- de Jong, T., Klein, U., Wielebinski, R. & Wunderlich, E. *Astron. Astrophys.* **147**, L6–L9 (1985).
- Helou, G., Soifer, B. T. & Rowan-Robinson, M. *Astrophys. J. Lett.* **298**, 7–11 (1985).
- Völk, H. J. *Astron. Astrophys.* **218**, 67–70 (1989).
- Kulsrud, R. M. & Zweibel, E. G. *Rep. Prog. Phys.* **71**, 046901 (2008).
- Tobias, S. M., Cattaneo, F. & Boldyrev, S. in *Ten Chapters in Turbulence* (eds Davidson, P. A., Kaneda, Y. & Sreenivasan, K. R.) 351–404 (Cambridge Univ. Press, 2013).

could be synthesizing a signalling molecule that, when secreted, promotes fat uptake by the muscle. Indeed, they find that blood serum collected from normal mice in the dark phase of the day can promote fat uptake by cultured muscle cells, but that serum from mice lacking PPAR δ in the liver cannot.

Extensive analysis narrowed down the factors transmitting the effects of PPAR δ through the blood to a handful of lipid candidates, and Liu *et al.* focused on a phosphatidylcholine dubbed PC(18:0/18:1), demonstrating that treatment with this phospholipid, but not with other closely related phosphatidylcholine species, induces fatty-acid uptake into muscle cells both *in vitro* and *in vivo*. This is a hallmark of PPAR α activation, and, consistently, PC(18:0/18:1)-mediated fatty-acid uptake was diminished in PPAR α -deficient muscle cells and in mice.

Thus, this dance starts at night when liver PPAR δ is activated, increasing PC(18:0/18:1) production. In an exchange of partners, PC(18:0/18:1) crosses from the liver to muscle, where it joins with PPAR α in the next step, promoting fat uptake and fatty-acid oxidation. The cycle is completed as the levels or activities of all three partners fall during the day, setting up the next round.

Now that they have been worked out, these

PHYSIOLOGY

A metabolic minuet

Two related nuclear receptors mediate circadian fat metabolism in two different tissues using a lipid messenger as an intermediary. This signalling pathway might be relevant to the understanding of metabolic disorders. SEE LETTER P.550

DAVID D. MOORE

In the minuet, a popular court dance of the baroque era, couples exchange partners in recurring patterns. This elaborately choreographed exercise comes to mind when reading Liu and colleagues' paper¹ on page 550 of this issue. In this study, the nuclear receptors PPAR α and PPAR δ are two of the three stars in a metabolic minuet that promotes appropriate fat utilization.

PPAR α drives fat use in muscle and liver and is a well-known target of the fibrate class of lipid-lowering drugs. By contrast, PPAR γ is essential for the development of white-fat tissue, mediating fat storage. PPAR δ is more broadly expressed than its two brothers and is more enigmatic, having functions that overlap with both. In muscle it promotes fatty-acid

breakdown and increases muscle endurance^{2,3}. And in the liver, it stimulates fatty-acid synthesis, or lipogenesis, as Liu and co-workers have previously demonstrated⁴. This lipogenic activity is now shown to generate a 'dancing partner' for PPAR α .

The circular pattern for this dance comes from the circadian activity of PPAR δ in the liver (Fig. 1). Mice eat at night, storing excess calories as fat. During the day, Rev-erba and Rev-erbb, two nuclear receptors that also have circadian activity, repress lipogenesis in this organ⁵. Liu *et al.* report that nocturnal expression of at least a subset of key lipogenic enzymes in the liver depends on PPAR δ . They also make the surprising observation that mice lacking PPAR δ in the liver have defective fat uptake in muscle, but only at night. The authors deduce that the night-time liver