Networks

More than the sum of its parts

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Claims of scale-free network behaviour need to be approached with caution, say Michael P. H. Stumpf and colleagues.

Many networks in nature and in technology grow by the addition of links to nodes that are already highly connected. Popular websites, for example, are more likely to attract links as new pages are added to the World Wide Web. This 'preferential attachment' process leads to networks that are 'scale-free': there is no preferred scale to the average connectivity of nodes, which instead have a probability distribution of connections described by a power law.

Many networks have been suggested to have this structure, including transport systems, food webs and the networks formed by interactions between proteins or genes. But rarely is such a scale-free network topology identified by looking at the entire network; often, for example, protein networks are constructed from just 10–20% of the total protein complement of an organism.

Stumpf *et al.* point out that, for scale-free networks, such incomplete sampling can be misleading, because subsets of such networks are not themselves scale-free. This contrasts with the properties of other classes of network, such as random graphs, where subsets do reflect the structure of the whole. **Philip Ball**

Systems biology

Many paths, one destination

Phys. Rev. Lett. (in the press)

How does a single type of progenitor cell form the diverse array of cells that make up an organism? Sui Huang *et al.* have approached this question by tracking the route taken by cultured cells differentiating from one cell type into another.

When HL60 cells (a human cell line) are treated with either the solvent dimethyl sulphoxide or the hormone all-*trans* retinoic acid they are stimulated to become neutrophils (a kind of white blood cell). Huang *et al.* simultaneously monitored the expression of 12,600 genes in cells undergoing these two treatments. Some 3,841 genes showed significant changes in expression and these were used as the axes on which the cells' journey from HL60 cells to neutrophils were plotted.

The expression profiles of the neutrophils produced by either stimulant were the same. But this shared end point was reached by very different routes. Despite similar beginnings, the expression paths plotted for each population quickly diverged, ultimately reconverging on their common goal from diametrically opposite 'directions'. Thus, cell differentiation seems to be less the observance of a carefully prescribed path, and more like a ball that can take multiple paths as it rolls over a hill from one valley, or stable cell type, to the next. **Christopher Surridge**

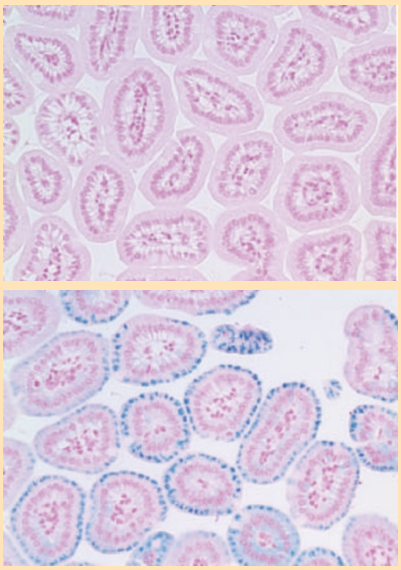
Biochemistry

No escape for iron

Cell Metab. **1,** 191–200 (2005)

A single protein — ferroportin — has been identified as the major exporter of iron from cells, and so is implicated in the accumulation of iron in people suffering from the hereditary disease haemochromatosis. If left untreated, iron overload can cause serious organ damage and is sometimes fatal. The liver and kidneys cannot eliminate iron from the body, as they do other metals, and so cells throughout the body must be able to store and release it when appropriate.

Adriana Donovan and colleagues investigated the role of ferroportin using genetically designed mice with the protein switched off. The animals died early in fetal development because iron was not transferred from mother to embryo. Using a genetic trick, Donovan *et al.* managed to produce live-born mice lacking ferroportin; these animals had large amounts of iron in their intestinal cells (the blue stain on the bottom image) compared with intestinal cells from normal mice (top).



Inactivating ferroportin only in the intestine caused mice to develop anaemia. This result is notable because the intestine normally absorbs iron for transport to the bone marrow, where haemoglobin is made; the intestinal cells could clearly absorb iron, but not release it. The authors say that these mouse models provide the first *in vivo* evidence that the iron export function of ferroportin is essential for mammalian iron homeostasis and survival. **Roxanne Khamsi**

Condensed-matter physics

Chromium condensate

Preprint at http://arXiv.org/abs/cond-mat/0503044

When gases of certain atoms are cooled to extremely low temperatures, they can form a Bose–Einstein condensate, in which all the atoms share the same quantum state. These condensates have unusual properties, such as superfluidity (flowing without friction) and superconductivity (conducting electricity without resistance). Axel Griesmaier *et al.* have now created a condensate using chromium atoms.

Bose–Einstein condensates are usually prepared from alkali metals such as rubidium and potassium. These atoms each have a single unpaired electron, giving them very weak magnetic properties. Griesmaier and colleagues' condensate consists of more than 50,000 chromium atoms at a temperature of about 700 nanokelvin. Each atom carries six unpaired electrons, making their magnetic dipole–dipole interactions 36 times as strong as those of the alkali metals.

This phenomenon should allow researchers to probe the magnetic properties of the condensate, Griesmaier *et al.* suggest. They also expect to be able to adjust both the short- and long-range interactions of the condensate using magnetic fields. And they say that improved lithography processes could result from creating a 'laser' of identical chromium atoms, which could be used to position single atoms exactly on a surface. **Mark Peplow**

Protein dynamics

Folding free of charge

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Many proteins have surfaces peppered with electrically charged groups. It is tempting to imagine that, by offering favourable interactions with water, these help to stabilize the folded state and to promote solubility. Oppositely charged groups might also provide a kind of glue by attracting each other, whereas a protein covered with like charges might be destabilized by electrostatic repulsions.

Katherine L. Gudiksen *et al.* challenge all of these notions. They show that converting 18 of the 27 lysine-NH_3^+ groups on the surface of the enzyme bovine carbonic anhydrase to neutral -$NHCOCH_3$ groups makes virtually no difference to the refolding rate or catalytic activity of the protein following denaturation.This is despite the fact that electrostatic repulsion between the NH_3^+ groups is likely to be far smaller in the denatured, extended polypeptide chain.

So why are the charged groups there at all? Might they prevent aggregation? What do they do to the energetics of hydration? **Philip Ball**