Weighing the Universe

Marc Davis

How do you measure the mass of the Universe? You can't use a balance to weigh it, but detailed analysis of hundreds of thousands of galaxies provides an alternative answer.

Cosmology has historically been considered a branch of philosophy rather than physics because of the dearth of data. But there has been dramatic progress in the past few years, and it is now entering an era of large-scale studies and precise measurements. The latest dispatch from this new frontier is the interim report by Peacock and collaborators on page 169 of this issue[1]. By systematically studying not hundreds or thousands of galaxies, but hundreds of thousands of them, they have produced a comprehensive map of the local Universe, which allows them to measure some of the fundamental parameters that define the structure of the entire cosmos.

For more than 70 years, astronomers have known that the Universe is expanding and that the velocity with which a galaxy appears to move away from us is approximately proportional to its distance. This smooth 'Hubble expansion' is direct evidence that the Universe began at a finite time in the past. By studying the spectrum of light from a particular galaxy we can accurately measure its velocity along the line of sight by the magnitude of the resulting Doppler effect, or redshift as it is known, thereby providing a measure of its distance. During the past 20 years, with the advent of sufficiently sensitive detectors and computer-controlled instruments, detailed mapping of the heavens in three dimensions has become possible. Figure 1 shows a three-dimensional slice of the nearby cosmos[2]. This detailed map shows the galaxy distribution to be highly inhomogeneous, with large voids, long filamentary structures stretching in excess of 100 million light years (10^{21} km), and dense clusters of galaxies where the filaments cross.

Peacock *et al.*[1] now present a new map of nearby galaxies up to three billion light years away (see Fig. 1 on page 170). These are the first precise measurements from the 2dF collaboration — a group of British and Australian astronomers using a specially designed instrument at the Anglo–Australian telescope in Australia to study 250,000 galaxies in detail. The resulting map has similar texture to that seen in Fig. 1 here but covers a much larger volume. There is a dramatic contrast between the highly structured clustering of galaxies revealed by these maps and the uniform Universe inferred from measurements of the radiation in the cosmic microwave background[3,4] (CMB). The CMB is a relic from the hot, dense and uniform phase of the initial Big Bang. So how did the striking lumps and bumps that we see as galaxies or clusters of galaxies today develop from such a homogeneous early Universe?

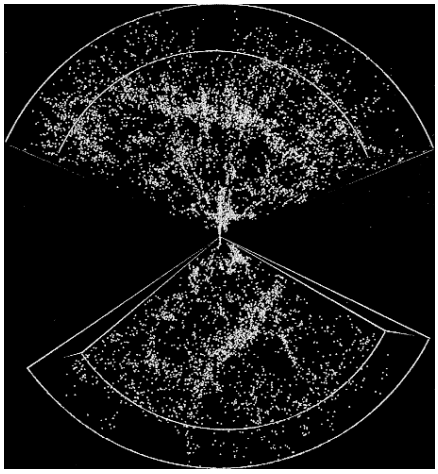

Figure 1 **A slice of the local Universe, in which each galaxy is plotted as a point[2]. The Earth is located at the centre of the plot, and the outer boundary is 400 million light years away (corresponding to a redshift of 0.04). The galaxy distribution appears to be highly inhomogeneous, with large voids and long filamentary structures stretching across 100 million light years. Scientists from the 2dF survey[1] have now produced a much larger version of this map (out to 3 billion light years), which allows them to estimate the mass density of the Universe from the pattern of clustering of some 140,000 galaxies.**

Theorists have long suspected that structures are formed by a process known as 'gravitational instability'. In this picture, the expansion of slightly denser regions in an otherwise smooth, expanding Universe is slowed by gravity more rapidly than the surroundings, increasing the contrast in mass density between high and low density regions. But finding direct evidence in support of this reasonable conjecture has been difficult. The timescale for the growth of galaxy structures (clustering) is the age of the Universe itself, far too long for direct observation. But because the strength of clustering is expected to increase with time, the galaxies must deviate from the smooth Hubble expansion. These deviations away from uniform Hubble flow are known as 'peculiar velocities', and they come in two forms. The first instance occurs within the compressed centres of dense clusters of galaxies. Here, the general outward push of the Universe has long since been overwhelmed by the local tug of newtonian gravity, and these galaxies have high peculiar velocities driven by the local gravitational potential.

The second type of deviation has been seen by Peacock *et al.*[1]. They have convincingly shown that peculiar velocities can also be detected for larger-scale structures that are still expanding. Whereas the peculiar motions within clusters of galaxies are incoherent and random, the clustering in long filamentary chains and other dispersed structures causes opposing sides of the structure to move coherently towards each other, partially cancelling the Hubble expansion. So galaxies on the near side of a collapsing structure are falling away from us, increasing in velocity compared with the rate of Hubble expansion, whereas objects on the far side fall towards us, reducing their velocity. We can detect the Doppler shift of this velocity only along our line of sight, so if we map galaxies using redshift to indicate distance, structures that have not yet collapsed completely will appear to be compressed slightly along our line of sight. Such regions should show up against the background of randomly oriented structures.

Peacock *et al.* report the first convincing detection of this effect in their analysis of the statistical properties of the clustering of galaxies along and perpendicular to the line of sight (see Fig. 2 on page 171). On small scales, peculiar velocities within galaxy groups cause a striking elongation in redshift space (sometimes dubbed 'fingers of god'), which can be used to measure the masses of galaxy clusters. On large scales there is a conspicuous flattening of the galaxy distribution, with considerably higher significance than in previous reports[5,6]. The strength of this flattening measures the mass density associated with the large-scale structure: more mass generates stronger gravity and thus larger accelerations and velocities. Detailed analysis suggests that the amount of mass associated with the clustering is approximately 30% of the cosmic 'critical density', the value at which the mass of the Universe is just sufficient to eventually stop the Hubble expansion because of the backwards pull of gravity. The estimated density is consistent with a variety

© 2001 Macmillan Magazines Ltd

of other methods, suggesting that cosmologists are finally converging on a reliable estimate of the mean mass density of the Universe. All indications point towards an infinite Universe that will expand forever.

Although cosmologists may be close to an accurate measure of the mass of the Universe, and studies currently underway[7,8] will lead to even more precise values in the next ten years, there is still no compelling explanation for why the Universe contains such a complex mix of particles. Ordinary matter contributes about 3% to the critical mass density, whereas heavy neutrinos contribute at least 0.3% to the critical density. The mysterious 'dark matter' — presumably an as-yet undiscovered elementary particle — is thought to comprise the bulk of the mass density in the large-scale structure that Peacock and collaborators are measuring.

Last year, two CMB experiments[9,10] provided convincing evidence that the Universe is 'flat', meaning that the density of the Universe exactly equals the critical density. The year before that, astronomers found evidence that the expansion of the Universe is actually accelerating, as suggested by teams studying distant supernovae[11–13]. Both of these observations can be explained if the Universe today is dominated by smoothly distributed 'dark energy', which constitutes the other 70% of the critical density and is thought to cause 'cosmic repulsion' on large scales. This dark energy might be Einstein's cosmological constant, or it might be the manifestation of an active quantum field of mysterious origin[14].

As additional pieces of the puzzle fall into place, our picture of Big Bang cosmology has become ever more bizarre. A unifying principle is clearly needed to explain the many disparate components of the Universe we have so far discovered. The quest for such unification is likely to keep us busy for decades to come. ■

Marc Davis is in the Department of Astronomy and Physics, University of California, Berkeley, California 94720, USA.
e-mail: marc@deep.berkeley.edu

1. Peacock, J. A. *et al. Nature* **410,** 169–173 (2001).
2. da Costa, L. *et al. Astophys. J.* **424,** L1–L4 (1994).
3. Penzias, A. A. & Wilson, R. *Astrophys. J.* **142,** 419–421 (1965).
4. Bennett, C. L. *et al. Astrophys. J.* **464,** L1–L4 (1996).
5. Cole, S., Fisher, K. & Weinberg, D. *Mon. Not. R. Astron. Soc.* **275,** 515–526 (1995).
6. Taylor, A. N. *et al. Mon. Not. R. Astron. Soc.* (in the press).
7. Sloan Digital Sky Survey, http://www.sdss.org/sdss.html
8. Microwave Anistropy Probe, http://map.gsfc.nasa.gov/
9. de Bernardis, P. *et al. Nature* **404,** 955–959 (2000).
10. Hanany, S. *et al. Astrophys. J.* **545,** L5–L9 (2000).
11. Schmidt, B. *et al. Astrophys. J.* **507,** 46–63 (1998).
12. Goobar, A. *et al. Physica Scripta* **85,** 47–58 (2000).
13. Perlmutter, S. *et al. Nature* **391,** 51–54 (1998).
14. Wang, L. *et al. Astrophys. J.* **530,** 17–35 (2000).

Ageing

Yeast longevity gene goes public

David Gems

Changes in gene silencing throughout life might be a general phenomenon underlying ageing and longevity: this mechanism is at work in yeast and, as new work suggests, nematode worms.

Would it make sense to study the weed *Arabidopsis thaliana* to understand human consciousness? How about trying to comprehend human ageing by looking at the budding capacity of a fungus? Research using 'model' experimental organisms often involves gambling on the universality of biological characteristics. Arguably, using budding yeast (*Saccharomyces cerevisiae*) to investigate the genetic determinants of ageing, and consequently longevity, seems almost absurdly optimistic. Ageing yeast do not develop grey hair or poor eyesight, or start complaining about young people today, or have strokes. In fact, it is not even clear that they age at all, and when researchers talk of yeast 'lifespan', what they really mean is the number of times a yeast mother cell can reproduce by producing a bud.

Mutations in several genes can increase the lifespan of yeast. That may be good news for this unicellular fungus, but does it mean anything for multicellular creatures such as ourselves? The answer may be 'yes'. On page 227 of this issue[1], Tissenbaum and Guarente reveal that *sir-2.1*, a relative of a yeast gene that controls lifespan, also controls longevity in an animal — the tiny nematode worm *Caenorhabditis elegans*. After more than a decade of guessing that studies of *S. cerevisiae* might teach us about general mechanisms of ageing, the gamble has paid off.

In *C. elegans*, *S. cerevisiae*[2], the fruitfly *Drosophila melanogaster*[3], and even mice[4,5], there are many genes that, when mutated, increase longevity. In many cases, the proteins encoded by such genes have equivalents in higher animals. For example, the adult lifespan of *C. elegans* can be tripled as a result of reduced activity of a signalling pathway resembling that which responds to insulin or insulin-like growth factor-I (IGF-I) in mammals[2].

But of course what one really wants to know is whether such genes control ageing (and so longevity) in all animals. To put it more broadly, has the biology of ageing been conserved throughout evolution? Clearly, most types of animal grow old and die (although there may be exceptions, such as *Hydra*)[6]. But it does not necessarily follow that ageing involves the same processes in all species. The widespread occurrence of ageing could be attributed to the common evolutionary conditions that give rise to it, in particular the fact that there is little selective pressure in old age on the effects of a gene that have little impact on reproductive success[7]. It is possible that, in different species, the underlying mechanisms of ageing are different.

The gerontologist George Martin has drawn a distinction between 'private' ageing mechanisms, which are unique to one species or animal group, and 'public' ones, such as oxidative damage to DNA, which are likely to be common to all species[8]. To find out whether a given gene, identified as a longevity determinant in one species, affects public or private mechanisms of ageing, one can test whether its equivalent in a very different species has a similar function. This is what Tissenbaum and Guarente[1] set out to do with the *sir-2.1* gene.

The yeast Sir2 protein controls gene silencing, by altering the structure of chromatin — the complex of DNA, RNA and protein that makes up chromosomes. Sir2 silences genes at various DNA sites, such as ribosomal DNA, repeated stretches of sequence that encode ribosomal RNA. Ribosomal DNA is important to yeast ageing: during cell division, circles of ribosomal DNA that are separate from the main chromosomes are generated; these circles reduce yeast lifespan[9]. Experimental overexpression of the *SIR2* gene results in reduced formation of these extrachromosomal DNA circles and extended lifespan[10]. But the accumulation of these circles has not yet been detected in higher organisms. So, like ageing-related accumulation of circles derived from mitochondrial DNA in the fungus *Podospora anserina*[11], the formation of the extrachromosomal ribosomal DNA circles seems likely to represent a 'private' mechanism of fungal ageing.

But *SIR2* is also involved in another ageing mechanism in yeast — a mechanism that is likely to be 'public'. In many animal species (possibly including primates), reduction of caloric intake extends lifespan. In yeast, this requires functional *SIR2* (ref. 12), and is independent of the formation of extrachromosomal ribosomal DNA circles. So it seemed worth testing whether *SIR2* might affect lifespan in other organisms, too. And indeed, Tissenbaum and Guarente[1] show that overexpression of *sir-2.1*, the *C. elegans* counterpart, increases mean lifespan in worms by up to 50%.

The authors then ask whether *sir-2.1* acts through one of the known pathways involved in longevity in *C. elegans* — the insulin/IGF pathway[2]. This pathway includes the *daf-2*, *age-1* and *pdk-1* genes. These genes, perhaps in response to the presence of food[13], inhibit *daf-16*, which encodes a transcription factor.

© 2001 Macmillan Magazines Ltd