Cosmology

Density and destiny

David Branch

Will the Universe perpetually expand, or eventually collapse? Is space flat, or curved? On page 51 of this issue, Perlmutter *et al.*[1] report observations of the explosion of a star so distant that its demise preceded the birth of our planet — observations that begin to answer these grand questions, and promise a final answer soon.

If we inhabit a nice, simple Universe, the destiny of the Universe is determined by its density. The present expansion is being decelerated by gravity, at a rate governed only by the average density of matter, Ω_M. If $\Omega_M < 1$, space is negatively curved (like a saddle), and will expand for ever. $\Omega_M > 1$ means positive curvature (like a sphere) and eventual collapse. $\Omega_M = 1$ is supreme simplicity: space is flat and expansion slows asymptotically towards zero.

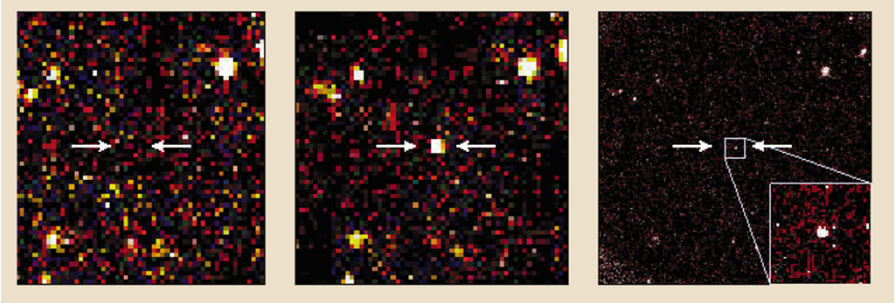
But empty space may have an energy density of its own, measured by the cosmological constant[2], Ω_Λ. Introducing this complicates matters, but there are reasons for doing so. Until recently, the main one was the 'cosmic age problem' — the expansion time of the Universe appears to be shorter than the ages of the oldest stars. The bigger Ω_M, the faster galaxies were separating in the past and the greater the problem (Fig. 1). But a positive Ω_Λ acts like a long-range repulsion which drives acceleration and increases our estimates of the expansion time. The age problem has eased in the past few years, however, as estimates of the Hubble constant (the present expansion rate) and the ages of the oldest stars have come down.

A more roundabout reason for introducing the cosmological constant is the theory of 'inflation'. According to the cosmic inflation hypothesis, the nascent Universe underwent such a phenomenal stretching that it should be nearly flat. But most observers, when they add the luminous matter to the dark matter inferred from galaxy motions, find $\Omega_M \sim 0.2$. This can be reconciled with inflation if $\Omega_M + \Omega_\Lambda = 1$, but in that case Ω_Λ will rise towards one in the future as Ω_M plummets towards zero, and runaway acceleration will lead to another episode of inflation billions of years from now. Why should we just happen to be here at a time when Ω_M and Ω_Λ are about the same size?

When doing the Ω_M sum, however, it is hard to be sure that all matter is being counted; and Ω_Λ is even more elusive. A good way to measure both would be to observe the past history of the expansion, by looking deep into space, deep into the past. This boils down to plotting a Hubble diagram (apparent brightness against redshift) for a sample of 'standard candles' — things that have nearly the same true brightness, so relative brightness indicates relative distance. This is where the brightest kind of supernova comes in.

Type Ia supernovae[3] (SNe Ia) are thought to be such standard candles. They start as white dwarf stars accreting matter from binary companion stars; eventually they approach a critical mass at which nuclear carbon burning begins. But under the electron-degenerate conditions in a white dwarf, this burning happens very rapidly — and the star explodes. These explosions all have about the same brightness because the objects involved are nearly identical: all made of carbon and oxygen, and all of the critical mass.

Indeed, the peak brightnesses of normal SNe Ia are so similar that treating them as standard candles gives relative distances to within 15 per cent. Correlations between peak brightness and distance-independent observables such as colour or the rate of fading can be used to standardize the candles further, and get distances accurate to 10 per cent. The absolute level of the SN Ia peak brightness is needed for measuring the Hubble constant[4], but not for Ω_M and Ω_Λ. The challenge there is to discover and accurately measure the apparent brightnesses of many



SAUL PERLMUTTER

Figure 1 **The furthest supernova. The first two images, from the Cerro Tololo Interamerican Observatory 4-metre telescope, show a galaxy halfway across the Universe transformed by the apppearance of a type-1a supernova (resolved more clearly in a follow-up observation using the Hubble Space Telescope). Being of well-known brightness, these supernovae help to determine the deceleration, and so the eventual fate, of the Universe.**

100 YEARS AGO

"Do the crystalline gneisses represent portions of the original earth's crust?" is the question asked, and answered in the affirmative, by Mr. J. Lomas, in his recent presidential address to the Liverpool Geological Society ... Excluding gneisses of later igneous or metamorphic origin, there remain the great series of fundamental gneisses, world-wide in distribution and uniform in general character, which must have had some world-wide cause of origin. As a possible cause of their foliation, Mr. Lomas suggests tidal action in the incompletely-consolidated crust. Prof. G. H. Darwin has shown that huge tidal wrinkles must have been raised by the moon when near the earth, forming ridges and troughs which ran north and south near the Equator, and curved to the eastward as they approached the Poles. The strike of the gneisses of Britain and Scandinavia corresponds to the direction of these tidal wrinkles in those latitudes, and there is evidence that the Palæozoic strata were deposited in troughs parallel to the gneissic ridges.

From *Nature* 30 December 1897.

50 YEARS AGO

In a previous communication, a preliminary account was given of observations and experiments on the relative edibility of the flesh of birds. During the past two years, this work has been extended to an investigation of the relative edibility of birds' eggs. ... Each sample was tested in the form of a scramble, prepared over steam, a numerical score being awarded on a scale ranging from 10.0 (excellent flavour) to 2.0 (inedible). Eggs of eighty-one species have now been examined. In the following list these are arranged in order of palatability:

Domestic fowl	8.8
Coot (*Fulica a. atra* Linn.)	8.3
Moorhen (*Gallinula c. chloropus* (Linn.))	8.3
...	
Great tit (*Parus major newtoni* Prazak)	3.5
Blue tit (*Parus coeruleus obscurus* Prazak)	3.3
Wren (*Troglodytes t. troglodytes* (Linn.))	2.0

An extensive series of experiments has also been carried out to test the preferences of small egg-eating mammals such as the ferret, rat and hedgehog.

From *Nature* 3 January 1948.

very remote (high-redshift) SNe Ia.

Perlmutter's team has met this challenge by developing a search strategy that virtually guarantees the discovery of batches of high-redshift ($z > 0.3$) SNe Ia during an observing run at a large ground-based telescope[5]. The guarantee means that coordinated observations by other telescopes can be arranged in advance.

In this issue, Perlmutter *et al.*[1] present observations of SN1997ap, at the very high redshift $z = 0.83$. (The Universe has expanded since then by a factor of 1.83.) The brightness against time of SN1997ap was measured from the ground and from the Hubble Space Telescope, and the Keck telescope contributed a good spectrum to prove that it was a normal type Ia. The data do not support $\Omega_M = 1$. A rival pack of high-redshift-supernova hunters[6] has also tentatively concluded that Ω_M is low. So if space is flat, there must be a cosmological constant.

The prospects for more precisely measuring Ω_M and Ω_Λ are bright. Both groups already have data on dozens of remote SNe Ia, including a number near $z = 1$; these will provide the leverage needed to determine Ω_M and Ω_Λ separately[7]. Astronomers will be wondering whether these analyses could be underestimating Ω_M. The inferred value would come out higher if remote SNe Ia were observed to be brighter than they are, relative to low-redshift supernovae. Could the high-redshift SNe Ia of the younger Universe be intrinsically dimmer than the nearer ones? (Not likely, if they have normal spectra.) Could there be wavelength-neutral intergalactic obscuration? Could the low-redshift sample be seriously biased by observational selection effects?

If the answers to these and other such questions prove to be no, it will become clear that expansion is here to stay. We should keep in mind, though, that if the cosmological principle (large-scale homogeneity) isn't valid, we could simply be exploring the nature and future of just one bubble in a cosmic sea of champagne. □

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Kaposi's sarcoma

Coupling herpesvirus to angiogenesis

Chris Boshoff

Since the discovery of Kaposi's sarcoma-associated herpesvirus (KSHV) only three years ago[1], and its detection in every Kaposi's sarcoma biopsy (Fig. 1), there have been many hypotheses to explain how this virus contributes to the abundant vasculature and spindle-cell proliferation that are associated with the disease. Spindle cells are considered to be the 'tumour' cells, and they contain (and secrete) large amounts of a powerful angiogenic agent called vascular endothelial growth factor (VEGF)[2]. Angiogenesis — the sprouting of new blood vessels from pre-existing ones — is essential for tumour progression and, on page 86 of this issue, Bais *et al.*[3] report that the G-protein-coupled receptor (GPCR) encoded by KSHV induces an 'angiogenic switch' in transformed NIH3T3 cells.

Like experimentally mutated cellular GPCRs, KSHV-GPCR is fully active in the absence of chemokine ligands[4]. Bais *et al.*[3] show that KSHV-GPCR can, indeed, act as a viral oncogene to transform NIH3T3 cells, and that this transformation is accompanied by secretion of VEGF. Spindle cells in Kaposi's sarcoma belong to the endothelial lineage, but they often express antigens characteristic of endothelial, macrophage and smooth-muscle cells. This indicates that they either represent pluripotent mesenchymal precursors, or are endothelial cells undergoing aberrant differentiation. KSHV DNA is present in most spindle cells, and also in endothelial cells and monocytes in Kaposi's sarcoma lesions.

The KSHV-GPCR gene is not the only viral oncogene to induce tumour formation and angiogenesis. Cells transformed by v-Ha-*ras* and v-*raf* (the constitutively active retroviral forms of cellular *ras* and *raf*) express increased

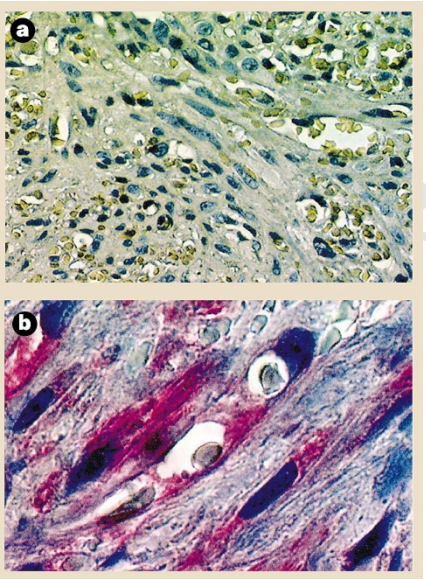

Figure 1 **Kaposi's sarcoma biopsy.** a, **Numerous vascular spaces and extravasated red blood cells are surrounded by spindle ('tumour') cells.** b, **Higher magnification of spindle cells, showing abundant magenta staining for the powerful angiogenic protein, vascular endothelial growth factor. (Reproduced, with permission, from ref. 2.)**

Viral pirates on a cellular sea

KSHV-GPCR is just one of an extraordinary number of genes that are pirated[8,10] from eukaryotic cellular DNA by KSHV. The structural proteins and viral enzymes that are common to most herpesviruses probably originated from an ancient progenitor of contemporary herpesviruses. But the recognizable cellular genes occur only sporadically in some herpesviruses. They are probably more recent acquisitions from the host genome, and they might support viral replication in a specific microenvironment (which, for KSHV, could be the microvasculature).

The captured eukaryotic genes have acquired unique properties (through accelerated evolution within the viral genome) which can give us insight into the biology of their cellular counterparts, and may even be exploited for development of therapies. For example, the D-type cyclin encoded by KSHV binds to its activating cellular partner cyclin-dependent kinase 6 (Cdk6). This association seems to be resistant to proteins that inhibit Cdk6 (ref. 11), meaning that the KSHV–cyclin D complex can drive cellular proliferation, and that this is not blocked by the normal cell-cycle control mechanisms. Similarly, the chemokines that are encoded by KSHV are more promiscuous than their cellular counterparts[12]. Moreover, the viral chemokines induce angiogenesis and might, therefore, also be directly involved in the pathogenesis of Kaposi's sarcoma[12]. **C.B.**