ASTROPHYSICS

Different stellar demise

Norbert Langer

A decades-old theory of stellar evolution — that the most massive stars end their life in a peculiar type of explosion termed a pair-instability supernova — finally seems to have been confirmed by observations.

Whereas the final evolutionary stage of a low-mass star such as the Sun is that of a simple white dwarf, the life of a massive star ends in a spectacular explosion called a supernova (SN). This theoretical view of stellar demise has been verified many times, most prominently through observations of SN 1987A, a supernova that occurred in a satellite galaxy of the Milky Way, the Large Magellanic Cloud. Neutrinos detected from SN 1987A confirmed the idea that such supernovae are triggered by the gravitational collapse of the iron core of their progenitor star[1]. But theory predicts that stars more massive than 140 solar masses could find another way to blow up — through the thermonuclear explosion of oxygen. On page 624 of this issue, Gal-Yam *et al.*[2] report that SN 2007bi provides the first evidence of such an explosion. Their study opens up the way to understanding the fate — and the mere existence — of the most massive stars, which may have dominated the early evolution of the Universe.

Stars born with a mass in the range 10–140 solar masses form a massive iron core in the final stage of their life[3]. At this point, nuclear fusion or fission ceases in the core, which collapses, owing to its own gravity, into a neutron star. Copious production of neutrinos is then thought to push the star's outer envelope away and produce a supernova. Besides uncertain neutrino interaction and transport processes, the physics of these iron-core-collapse supernovae involves poorly constrained physical processes such as turbulence, pulsations, general-relativity mechanisms, and perhaps rotation and magnetic fields[4]. It remains a challenge for supernova modellers to successfully reproduce iron-core-collapse supernovae on the computer. By contrast, models for the explosions of the most massive stars — with masses larger than about 140 solar masses — are robust.

For such very massive stars, when the core temperature at advanced evolutionary stages — namely before the ignition of oxygen — exceeds about 10^9 kelvin, photons produced in the core are sufficiently energetic to create electron–positron pairs[5]. This pair production reduces the star's radiation pressure, which would otherwise keep it in hydrostatic equilibrium, and the star collapses, igniting oxygen explosively in an event termed a pair-instability supernova (PISN; Fig. 1). Unless the mass of the core is overwhelmingly large (above 130 solar masses[6,7]), the energy released in the explosion disrupts the whole star without leaving a stellar remnant[5,6,8–10]. Although, as in the case of iron-core collapse, the core of the star has to turn a collapse into an explosion, its density and binding energy is much smaller in the PISN case, and so an explosion is much easier to obtain.

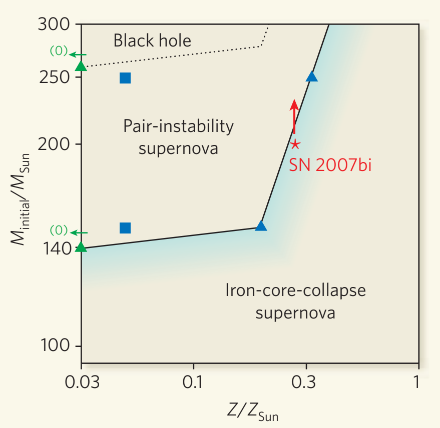

Figure 1 | Fate of the most massive stars. The ultimate fate of a massive star depends on the metallicity (the abundance of elements other than hydrogen and helium; Z) and mass with which it is born (M_{initial}); plotted values are relative to those of the Sun. The solid line represents the boundary between an iron-core-collapse supernova (triggered by the collapse of a star's iron core) and a pair-instability supernova (driven by instability caused by the production of electron–positron pairs in a star's core before oxygen fusion takes place). The blue shaded area denotes a transition region in which stars first become 'pair unstable', but eventually undergo iron-core collapse (pulsational pair-instability supernovae)[5,6]. The dotted line marks the point above which pair-unstable stars are thought to form black holes instead of exploding[5]. Lines are schematic and their exact location is uncertain. Triangles and squares denote values obtained by different theoretical studies: blue triangles and squares for finite metallicity[16]; green triangles for zero metallicity[5]. Gal-Yam and colleagues[2] estimate that the progenitor mass of supernova SN 2007bi (red asterisk) is about 200 solar masses or more.

The theoretical prediction of PISNs was never in doubt. In fact, the most recent model predictions for PISNs seem to be beautifully confirmed by Gal-Yam and colleagues' analysis[2] of SN 2007bi. In particular, the large derived amount of the radioactive isotope ^{56}Ni, the very high total mass and kinetic energy, and the slow expansion velocity and brightness evolution of SN 2007bi fit well the models of PISNs, and are incompatible with the classic iron-core-collapse supernovae.

So is SN 2007bi a textbook example of a PISN? Maybe, but it leaves us with one puzzle. First, one might wonder why it took so long (decades) to discover a PISN, which can obviously be very bright. The fact that none has been detected before did not discourage theorists because it was known that stars in our Galaxy, however massive they are at birth, would lose so much mass during their evolution, owing to radiation-driven stellar winds, that they would end up undergoing iron-core collapse[11,12]. To observe PISNs, one would have to withdraw to the early Universe, in which the abundance of metals (in astronomy, elements other than hydrogen and helium) was low — preferably even to the cosmic epoch that witnessed the formation of the very first stars, which lacked any metals. Indeed, most PISN models, including those used by Gal-Yam and colleagues[2], were constructed for metal-free stars. It was only recently that PISNs were predicted — on the basis of the most recent stellar-wind theories and provided that the metal content of the host galaxy is sufficiently low — to occur also in the local, present-day Universe (Fig. 1). The criterion of low-metal content is met by local dwarf galaxies such as the Small Magellanic Cloud. In view of this, it is reassuring that SN 2007bi was found in a metal-poor dwarf galaxy[13].

The puzzle about SN 2007bi is that no hydrogen was detected in its spectrum[2]. This means, on the one hand, that the progenitor star must have undergone substantial mass loss, but, on the other hand, that it must have avoided the drastic mass loss that massive hydrogen-free stars (called Wolf–Rayet stars) are known to suffer[14,15]. Such drastic loss would have swiftly reduced the mass of the progenitor star to below that required for a PISN. So we are left with an unlikely, finely tuned timing that led the star to explode shortly after the last of its hydrogen stores were lost to the star's wind, or — perhaps more likely — a situation in which some amount of hydrogen was still present in the supernova progenitor but was insufficient to produce a clear signal in the supernova spectrum.

Whatever the case, SN 2007bi pushes the door wide open for studies of the early, metal-poor Universe. The consequences of PISNs for the chemical evolution of metal-poor galaxies can be enormous, because a single PISN can release more metals than a whole generation of iron-core-collapse supernovae[16]. Gal-Yam and colleagues' study[2] indicates that SN 2007bi produced about 22 solar masses of silicon and more than three solar masses of radioactive nickel. This and future PISNs will also remain the only empirical way to quantify the stellar-wind mass loss in the most massive stars. Knowledge of the mere existence of stars sufficiently massive to undergo a PISN may have a considerable impact on our understanding of star formation. And, perhaps most importantly and exciting of all, finding PISNs in the local

Universe argues most convincingly that they existed — or even dominated — in the early Universe, in which stars are thought to have been more massive than they are today, and where stellar winds are predicted to have been weak. ■

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NEUROSCIENCE

Unbearable lightness of touch

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Following inflammation or nerve injury, stimuli that are normally perceived as innocuous can evoke persistent pain. A population of neurons that contributes to this syndrome has now been identified.

For many people with persistent pain syndromes, even the touch of something as innocuous as a shirt on their skin can be agonizing. Determining the underlying cause of this debilitating pain is essential for developing effective therapies. However, the precise identity of the neural circuits involved has so far remained elusive. In this issue (page 651), Seal *et al.*[1] pinpoint a previously enigmatic population of small sensory neurons that respond to light touch, and show that they have a crucial role in the painful sensitivity to touch or pressure that follows injury or inflammation.

The sensations of pain and touch, like other bodily sensations such as temperature and itch, begin with the activation of distinct subsets of primary sensory neurons by physical stimuli affecting the body. Once activated, these neurons excite circuits in the dorsal horn of the spinal cord. It is there that the sensory input is processed and then relayed to centres in the brain, where it is perceived and interpreted.

The dorsal horn has a laminated, or layered, structure, with each lamina receiving input from multiple but distinct classes of sensory neurons that extend from the body's periphery and convey information about various sensations (Fig. 1). These neurons release the excitatory neurotransmitter glutamate to activate neurons in the dorsal horn. Glutamate is packaged into synaptic vesicles in neurons by proteins called vesicular glutamate transporters (VGLUTs), of which there are three types. Previously, only the two most common types, VGLUT1 and VGLUT2, had been characterized in sensory neurons. VGLUT1 is expressed in touch-sensing fibres (heavily myelinated Aβ fibres with low activation thresholds) that terminate in lamina III/IV and the inner part of lamina II (IIi) in the dorsal horn[2,3]. By contrast, sensory neurons that innervate the more superficial laminae, I and II, are generally smaller and mainly associated with sensing painful (including strong mechanical) stimuli, temperature and itch. These neurons have thin, either lightly myelinated Aδ fibres or unmyelinated C fibres, most of which express VGLUT2 (ref. 3). (Myelination increases the speed at which electrical impulses pass along a neuron.)

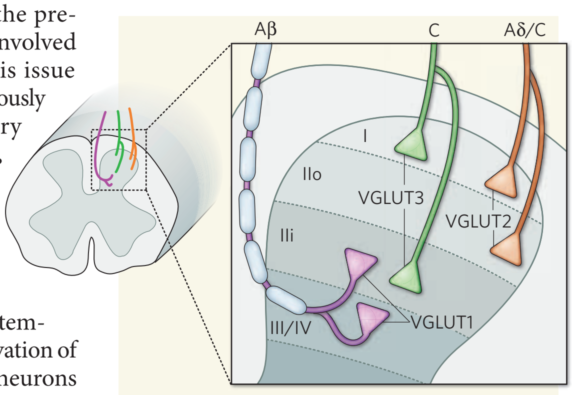

Figure 1 | Schematic view of peripheral sensory neurons terminating in the spinal-cord dorsal horn. Sensory neurons activate dorsal-horn neurons by releasing the neurotransmitter glutamate, which is packaged into synaptic vesicles by vesicular glutamate transporters (VGLUTs). Low-threshold Aβ fibres (pink) enter the dorsal horn medially, terminating in lamina III/IV and in lamina IIi. These heavily myelinated touch-sensitive fibres express VGLUT1. High-threshold, lightly myelinated Aδ fibres and unmyelinated C fibres (orange) that sense pain, temperature and itch enter the dorsal horn more laterally and terminate in laminae I and II. These fibres may express VGLUT2. The novel mechanosensors with C fibres described by Seal *et al.*[1] (green) terminate at the lamina II/III border and in lamina I, and express VGLUT3.

Seal *et al.*[1] show that the little-studied VGLUT3 is expressed in a specific subpopulation of small sensory neurons that have unmyelinated C fibres. These fibres terminate in lamina I and also in lamina IIi, where they overlap with the most dorsal innervation of conventional, touch-sensing Aβ fibres (Fig. 1). It has been known for some time that there is a small subset of sensory neurons in the C-fibre population that terminates primarily in lamina II and that responds to innocuous mechanical stimuli, such as light touch, rather than to noxious stimuli[4]. By performing electrical recordings on a preparation of sensory neurons with their fibres still attached to skin, Seal *et al.* demonstrate that the VGLUT3-expressing sensory neurons are activated by brush and light touch, and so represent these previously ill-defined, low-threshold C-fibre mechanoreceptors.

The authors then assessed the function of low-threshold C-fibre mechanoreceptors by studying mice in which the *vGlut3* gene had been knocked out[5]. The absence of VGLUT3 functionally disconnects this subset of peripheral sensory neurons from their dorsal-horn targets by preventing the normal release of glutamate. Surprisingly, in the absence of injury or inflammation, these knockout mice had small defects in their responses to noxious mechanical stimuli, but responded normally to low-intensity mechanical stimuli and also to hot and cold stimuli. This somewhat paradoxical result suggests that, despite the low-threshold mechanical sensitivity of the VGLUT3-expressing neurons, they can contribute to the detection of noxious mechanical pain.

Seal and colleagues' most striking findings[1], however, came in the next round of tests, in which the authors used three manipulations in mice as models of persistent pain states. These comprised: injection of an irritant substance into the footpad, causing a massive inflammatory response; an incision in the paw that is considered a model of post-surgical pain; and a lesion of the peripheral nerve to model neuropathic pain. In the last case, injury to peripheral nervous tissue causes a persistent pain state, best defined[6] as a pathological response of the pain system to nerve damage. In all of these conditions, animals whose paws are exposed to mechanical and thermal stimulation will usually tolerate a far lower stimulation intensity than normal mice before withdrawing their paws. In all three models, the VGLUT3-knockout mice and normal control mice showed