PLANETARY SCIENCE

Hyperion the sponge

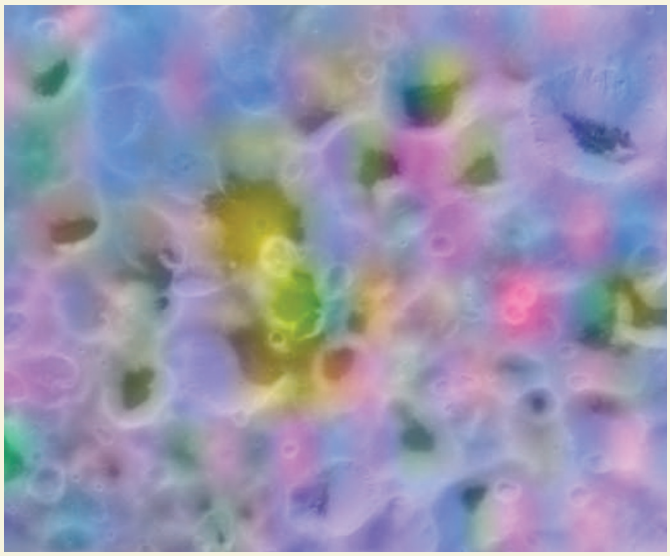
On 1 July 2004, seven years after its launch from Earth, NASA's Cassini orbiter fired its main engine, throttled back its speed and allowed itself to be pulled in by Saturn's gravity. In the three years since then, the spacecraft has provided snapshots of the strange and fascinating worlds that inhabit the Saturn system: the eerily Earth-like Titan, for instance, the rain on whose plains is mainly methane; or Enceladus, from near whose 'tiger-striped' southern pole an icy plume spouts into space.

No moon, however, seems quite as odd as the body that hove into Cassini's close view on 25 September 2005. Irregularly shaped and chaotically rotating, Hyperion, the eighth-largest saturnian moon, looks for all the world like a bathroom sponge.

Two papers in this issue take a detailed look at this odd character. In the first of these, Thomas *et al.* (P. C. Thomas *et al. Nature* **448,** 50–53; 2007) conclude that Hyperion's sponge-like appearance (Fig. 1 on page 51) comes from an unusually high density of well-preserved craters between 2 and 10 kilometres across. According to the authors' theory, these discrete craters survived, rather than having been eroded away or filled in by material ejected in the impacts that formed them, because Hyperion's interior is indeed porous. A meteorite hitting a significantly porous body will compress its surface rather than excavate it, and produce much less ejected material.

The authors arrive at this explanation by first calculating Hyperion's mean radius — no mean feat, as its irregular shape and random spinning make imaging it in one shot impossible. They were also able to compute the moon's mass through a dynamical model requiring that Hyperion maintain a stable orbit in the complex gravitational environment of the Saturn system. This mass was only just over half that expected for a body of Hyperion's 135-km calculated mean radius, if it were made of water ice — and considerably less if higher-density materials are present. In other words, there is far less within Hyperion than had been expected.

In the second of the papers, Cruikshank *et al.* (D. P. Cruikshank *et al. Nature* **448,** 54–56; 2007) delve more deeply into Hyperion's make-up. Using data from ultraviolet and infrared spectrometers onboard



Cassini, they divide the moon's surface into distinct areas of high reflectivity, seemingly dominated by water ice, and low reflectivity, concentrated at the craters' bases. These latter areas have a considerably diminished water-ice signature, but a prominent absorption band best explained by the presence of solid carbon dioxide in complex with a further, unknown material.

Hyperion's spectra, like those of other saturnian satellites such as Phoebe and Iapetus, also provide evidence for irregularly scattered, nitrogen-rich, organic molecules. The image shown here of three surveys overlaid on the moon's surface topography (blue, H_2O; red, CO_2; green, carbon nitrides, CN) gives an idea of the complexity of the picture. Cyan indicates areas with strong H_2O and CN signals, but little CO_2; yellow areas are dominated by CO_2 and CN, with little H_2O; and magenta equates to CO_2 and H_2O, but little organic material.

What these discoveries tell us of Hyperion's origin and the wider history of the Saturn system is as yet unclear. But as the pictures from Cassini continue to roll in, the varied worlds they depict do not cease to surprise.

Richard Webb

perspective speculate on what kinds of glycolipid other than αGalCer might promote the best TCR–CD1 interaction. Their structure also shows how the α-linked sugar lies down to allow a tight CD1–TCR interface, whereas an upright β-linked sugar might block the approach of the TCR to CD1d.

The TCR footprint on CD1 is perhaps the most pleasing aspect of this study, because it provides a simple explanation for the observation that nearly all NK T cells require the same kind of TCR α-chain to function[6]. Imagine that the α- and β-chains of TCRs are legs. MHC-reactive TCRs generally adopt a confident, two-footed stance, with both chains contacting their peptide antigen near the centre of the MHC platform. With some variation[13], this mode of binding holds true for 15 TCR–MHC structures[14] and was predicted to apply also to CD1. But the NK TCR is a ballerina in a pirouette: it leans sideways and has spun clockwise, contacting its antigen only with the α-chain, while using the β-chain to graze CD1 at its edge (Fig. 1). Whether other TCRs dance on one foot is not yet settled, as T cells responding to CD1a, CD1b and CD1c do not seem to share the same type of α-chain for antigen recognition[5].

Another notable feature is the displaced location of the TCR β-chain — it is pushed to an extreme lateral position so that it hangs over the edge of CD1d. This lateral approach is reminiscent of a TCR found on γδ T cells[15]. Perhaps these two unusual footprints will serve as counterpoints to help explain how most MHC proteins guide TCRs to their centres[14].

During the long wait for the first TCR–antigen–CD1 crystal, specialists guessed that the TCR would align near the centre of CD1, as occurs with the MHC. This reasonable — but apparently mistaken — prediction did not arise from lack of consistency in MHC footprints, but rather from the faulty assumption that the behaviour of the MHC could be used to predict that of CD1. Such thinking has a chequered past, and dates back to the description of CD1 proteins as MHC-like molecules. This oft-used comparison provides a good introduction to CD1, but is superficial and even misleading. Modern evidence shows that these systems differ in their levels of polymorphism, mutability of antigens, loading mechanisms, trafficking pathways and other features. CD1 is more than just the MHC for lipids, and its footprint provides another reminder that we should think about CD1 biology in its own terms. ■

D. Branch Moody is in the Department of Medicine, Division of Rheumatology, Immunology and Allergy, Brigham and Women's Hospital, 1 Jimmy Fund Way, Boston, Massachusetts 02115, USA.
e-mail: bmoody@rics.bwh.harvard.edu

1. Calabi, F. & Milstein, C. *Nature* **323,** 540–543 (1986).
2. Porcelli, S., Morita, C. T. & Brenner, M. B. *Nature* **360,** 593–597 (1992).
3. Beckman, E. M. *et al. Nature* **372,** 691–694 (1994).
4. Borg, N. A. *et al. Nature* **448,** 44–49 (2007).
5. Grant, E. P. *et al. J. Exp. Med.* **189,** 195–205 (1999).
6. Cui, J. *et al. Science* **278,** 1623–1626 (1997).
7. Moody, D. B., Zajonc, D. M. & Wilson, I. A. *Nature Rev. Immunol.* **5,** 387–399 (2005).
8. Kawano, T. *et al. Science* **278,** 1626–1629 (1997).
9. Mattner, J. *et al. Nature* **434,** 525–529 (2005).
10. Kinjo, Y. *et al. Nature Immunol.* **7,** 978–986 (2006).
11. Gadola, S. D. *et al. J. Exp. Med.* **203,** 699–710 (2006).
12. Kjer-Nielsen, L. *et al. J. Exp. Med.* **203,** 661–673 (2006).
13. Hahn, M., Nicholson, M. J., Pyrdol, J. & Wucherpfennig, K. W. *Nature Immunol.* **6,** 490–496 (2005).
14. Rudolph, M. G., Stanfield, R. L. & Wilson, I. A. *Annu. Rev. Immunol.* **24,** 419–466 (2006).
15. Adams, E. J., Chien, Y. H. & Garcia, K. C. *Science* **308,** 227–231 (2005).