PLANETARY SCIENCE

Jupiter's magnetic field revealed

The magnetic field of Jupiter has been found to be different from all other known planetary magnetic fields. This result could have major implications for our understanding of the interiors of giant planets. SEE LETTER P.76

CHRIS JONES

NASA's Juno spacecraft is currently mapping Jupiter's magnetic field in unprecedented detail. Because the field originates in the planet's interior, it can provide insights into what is going on beneath the spectacular swirling clouds in the planet's surface layers. On page 76, Moore *et al.*[1] analyse data from Juno and find that Jupiter's magnetic field is substantially different in the planet's northern and southern hemispheres. The authors consider what might be happening in the planet's interior to account for this asymmetry.

Juno reached Jupiter on 4 July 2016, and it has been gathering data that are transforming our understanding of the planet's deep interior. Previously, we had only a broad-brush overview of Jupiter's magnetic field[2]. Juno has brought the picture into much sharper focus, allowing a revised model of the field to be constructed[3]. These advances were possible owing to the close approach that Juno makes to Jupiter — the spacecraft flies only about 4,000 kilometres above Jupiter's surface as it dives into the planet's gravitational field once every 53 days[4].

Jupiter has the strongest planetary magnetic field in the Solar System. Ironically, this field is the biggest threat to the Juno mission. High-energy particles from the Sun are trapped in the field, producing a hazard that is dangerous to the electronics on which the mission depends. Fortunately, Juno was designed with protection against this and has survived so far.

The magnetic field of Jupiter is maintained by electric currents that flow in the planet's interior. Jupiter is made up mainly of hydrogen and helium, so it is quite surprising that it can conduct electricity at all. However, the extremely high pressure and density in the planet enable hydrogen to enter a state known as metallic hydrogen[5]. Metallic hydrogen has an electrical conductivity similar to that of metals, allowing electric currents to flow.

Giant planets take billions of years to cool down after they are formed. Consequently, there is as much heat coming out of Jupiter's interior as is received by the planet from the Sun. This heat is carried by convection currents, which stir the interior and produce the swirling clouds and storms — such as the Great Red Spot — that are so beautifully captured by Juno's cameras. The convection-driven flows of fluid in the interior are slower than the surface winds, but they are strong enough to generate Jupiter's magnetic field by a process called dynamo action[6,7].

Earth's magnetic field is also produced by convection-driven flows in the planet's interior, but it is the planet's liquid-iron core that allows electric currents to flow. The fields of both Jupiter and Earth are mainly dipolar — the radial component of the field is mostly positive in the northern hemisphere and mostly negative in the southern hemisphere, as if the planet contained a bar magnet (Fig. 1a). Moore and colleagues report that the non-dipolar part of Jupiter's field is confined almost entirely to the northern hemisphere (Fig. 1b). This is in stark contrast to Earth's field, for which the non-dipolar part is evenly distributed between the two hemispheres.

Moore *et al.* suggest several possible explanations for the morphology of Jupiter's magnetic field. One explanation concerns Jupiter's core, the nature of which is still a mystery. Some models of the planet assume a compact core with a mass about five times that of Earth[8]. But a much larger, dilute core is also feasible[9], and could affect field generation.

Another explanation is that there are one or more stable layers of fluid deep inside Jupiter. Saturn is thought to have a stable layer in its interior, which could account for why its magnetic field is almost completely symmetrical about the planet's rotation axis[10] — vastly different from the fields of Jupiter and Earth. In Jupiter, these stable layers might be regions in which the composition of the fluid changes, partitioning the planet's interior into zones. If the transition regions contained a helium concentration gradient, they could be bottom heavy, altering the fluid flow inside the planet and therefore the magnetic field.

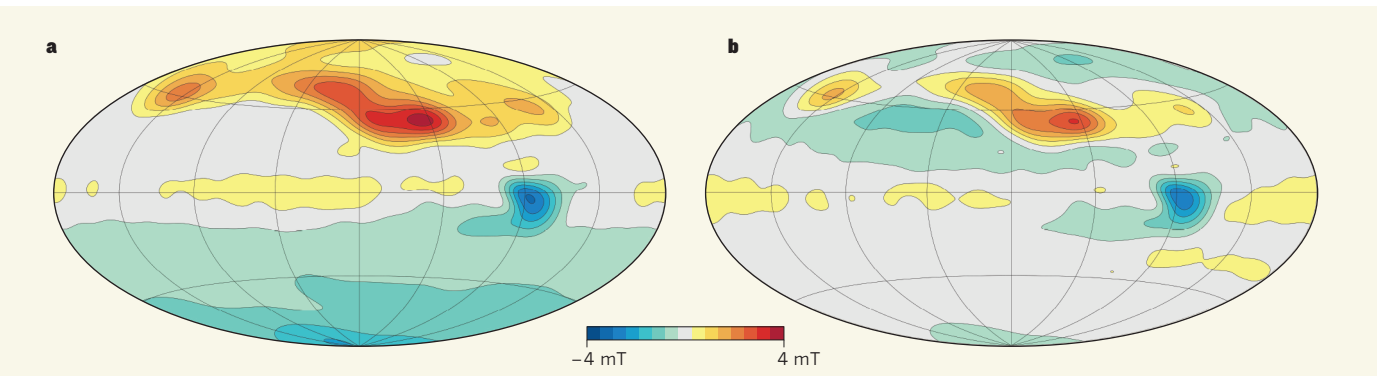

Figure 1 | Maps of Jupiter's magnetic field. **a**, In the northern hemisphere of Jupiter, the radial component of the planet's magnetic field points mainly in the positive (outwards) direction (yellow–red shades). Conversely, in the southern hemisphere, the radial component points predominantly in the negative (inwards) direction (green–blue shades). Such a configuration is known as a dipole. The colour scale depicts the strength of the radial magnetic field in units of millitesla. **b**, Moore *et al.*[1] report that the non-dipolar part of Jupiter's radial magnetic field is almost entirely concentrated in the northern hemisphere — unlike all other known planetary magnetic fields. The maps in **a** and **b** illustrate the magnetic field at a distance of 90% of Jupiter's radius from the planet's centre, under the assumption that substantial electric currents in the planet all reside at distances closer to the centre. (Adapted from Fig. 1e and Fig. 3a of ref. 1.)

To investigate how planetary magnetic fields are generated, it is now possible to solve the fundamental equations that govern the fluid flows and the magnetic fields inside planets. The basic principles of dynamo action were laid down a century ago[11], but solving the fluid–dynamo equations proved difficult. Computers have been able to handle the calculations required to model Earth's dynamo only since 1995 (ref. 12). Nevertheless, much progress has been made, and computational models of dynamos can now capture many of the characteristics of Earth's magnetic field[13].

In the past five years, these models have been adapted to deal with the large variations in density between the interior and atmosphere of Jupiter[6,7], and can now be compared with the field inferred by Moore and colleagues. However, dynamo models depend on the internal structure of the planet, which in turn depends on the planet's thermodynamic properties, electrical-conductivity profile and composition. Although these issues have been extensively explored, some uncertainty remains. Models of fields that are dipolar but broadly symmetric about the equator have been developed[6], as have models of fields that are asymmetric but not dipolar[14]. The challenge is therefore to formulate models of fields that are both asymmetric and dipolar.

Moore and colleagues' suggested explanations for Jupiter's field morphology can now be tested by dynamo modellers to discover whether the explanations are indeed compatible with Juno's observations. Exciting times lie ahead for the study of the interiors of giant planets, as modellers digest the information coming from Juno and begin to work out a clearer picture of the inside of Jupiter. ■

Chris Jones *is in the Department of Applied Mathematics, University of Leeds, Leeds LS2 9JT, UK.*
e-mail: cajones@maths.leeds.ac.uk

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IMMUNOLOGY

An immune response with a sweet tooth

A previously unknown pathway that enables mammalian cells to recognize infection and trigger an immune response requires a kinase enzyme in the host cell to bind a sugar molecule produced by infecting bacteria. SEE LETTER P.122

JOHN-DEMIAN SAUER

Bacterial infections are a major cause of disease and death worldwide. The innate branch of the mammalian immune system, which recognizes and reacts to general characteristics of pathogenic organisms, has a key protective role. On page 122, Zhou *et al.*[1] describe a mechanism by which the innate immune system is activated in response to bacterial sugar molecules. This finding broadens our understanding of the types of molecule that can be recognized as hallmarks of bacterial infection and the host proteins that can recognize such molecules.

A key advance in our understanding of how the innate immune system functions was the identification of proteins called pattern-recognition receptors (PRRs), which recognize 'non-self' molecules termed pathogen-associated molecular patterns (PAMPs). Beginning with the Toll and Toll-like receptor PRRs[2–4] in the late 1990s, the identification of PRRs and the PAMPs that they recognize has proceeded at a breathtaking pace.

A key function of PRRs is to help drive the expression of secreted proteins called cytokines, which alert the immune system to the presence of infection. The transcription factor NF-κB is a central regulator of cytokine expression. Zhou and colleagues studied human cells grown *in vitro* to try to identify pathways that activate NF-κB in response to infection by the bacterium *Yersinia pseudotuberculosis*. This bacterium has a needle-like, multiprotein structure called a type III secretion system (T3SS), which is required for the direct transfer of bacterial proteins into host cells. T3SSs are evolutionarily conserved in many pathogenic bacteria.

Zhou *et al.* took an unbiased approach and screened a collection of *Y. pseudotuberculosis* genetic mutants to identify bacterial genes that are linked to NF-κB activation in response to infection. This led the authors to focus on the enzyme HldE, which catalyses steps in the biosynthetic pathway that generates lipopolysaccharide (LPS) molecules.

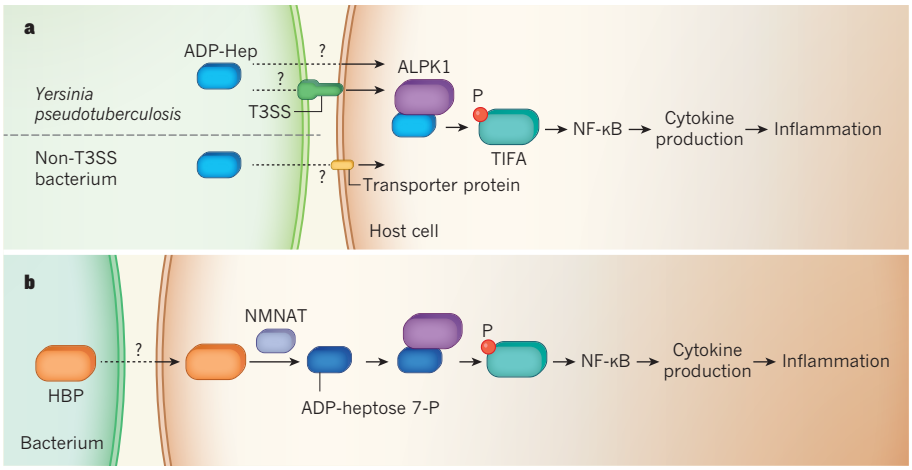

Figure 1 | Bacterial sugars trigger a host immune response. **a**, Zhou *et al.*[1] demonstrate that in bacteria such as *Yersinia pseudotuberculosis*, which has a multiprotein complex called a type III secretion system (T3SS), and in other bacterial species lacking a T3SS, the sugar molecule ADP-β-D-manno-heptose (ADP-Hep) can enter a host cell, by an unknown route (possibly through a transporter protein), and can trigger a signalling pathway that drives inflammation. When ADP-Hep enters the host cell, it binds to ALPK1, which activates the protein TIFA by adding a phosphate group (P) to it. The downstream signalling pathway, not all the steps of which are shown, leads to activation of the protein NF-κB, which drives the expression of cytokine proteins that stimulate an immune response to the infection. **b**, The authors also report that if the bacterially produced sugar D-glycero-β-D-manno-heptose 1,7-bisphosphate (HBP) enters the host cell (by a route that remains to be determined), it can be converted by host enzymes of the NMNAT family into the molecule ADP-heptose 7-P. This binds to ALPK1 and activates the same pathway as that activated by ADP-Hep.