GEOPHYSICS

Earth's Dynamo Powered by Heat

The Earth's magnetic field is generated in its liquid iron outer core at a depth of approximately 3,000 kilometers. The liquid iron is set in motion due to the flow of heat from the core into the rocky mantle, similar to water in a heated pot. Such movements of electrically conductive iron give rise to the "dynamo effect": they induce electrical currents whose magnetic fields are measured on the Earth's surface. Ulrich Christensen at the Max Planck Institute for Solar System Research (formerly Aeronomy) in Katlenburg-Lindau and Andreas Tilgner from the Institute of Geophysics at the University of Göttingen have used computer simulations and laboratory experiments to show that the power needed to sustain the geodynamo is no more than the energy generated by several hundred large power stations – significantly less than was previously assumed (NATURE, May 13, 2004).

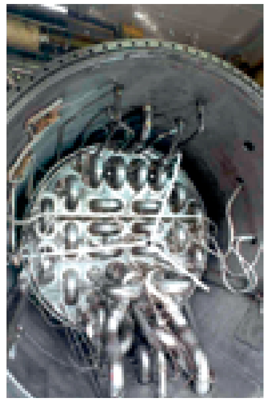

PHOTO: KARLSRUHE NUCLEAR RESEARCH CENTER

Practical experiment: In the Karlsruhe dynamo experiment, liquid sodium is pumped through a system of tubes to generate an artificial geodynamo.

The energy driving the geodynamo has its origin in the heat that has been stored in the Earth's core since its birth. This heat is dissipated so slowly that the Earth's inner core, formed as the liquid iron around it freezes out, may already be more than three billion years old. This figure ties in with evidence showing that rocks of a similar age are also magnetized.

The Earth has a diameter of 12,740 kilometers and harbors a number of natural phenomena, including its magnetic field, with which compass needles align themselves. Current theories suggest that a dynamo mechanism in the liquid iron core is responsible for this magnetism: flows of electrically conductive material through an existent weak magnetic field generate electrical currents by induction, and these, in turn, produce further magnetic fields. Such reciprocal interactions strengthen the Earth's magnetic field to the point where its effects become measurable.

But just how much energy is actually needed to drive such a geodynamo? In an attempt to answer this question, scientists have combined computer models of the geodynamo with results from laboratory experiments. Their computer simulations can successfully explain the strength, dynamic change and large-scale structure of the magnetic field observed on the Earth's surface. However, to calculate the energy requirements of the dynamo, small structural features of the magnetic field in the Earth's core need to be ascertained – but these features are still not accessible for measurement. Whether the computer models correctly convey the information is, as yet, uncertain, since the simulations can only model large-scale fluid movements. The small eddies that can be expected in the turbulent flows at the Earth's core are suppressed by assuming a greater viscosity of the fluid in order to keep such models practicable.

In an attempt to explain the effect of such small eddies, the geoscientists utilized a model known as the "Karlsruhe dynamo experiment." In this experimental model, liquid sodium flows through a system of channels that form a meter-sized cylinder. If the pump rate is high enough, the dynamo springs into life and generates a magnetic field about a hundred times as strong as the Earth's. In contrast to computer simulations, the flow is turbulent; in other words, it includes the eddy component. The power needed to drive such experimental dynamos corresponds closely to values derived from the theoretical models. This shows that eddies have little effect on the dissipation of electrical power. The computer models can therefore be employed to estimate how much energy the geodynamo requires. According to the new models, the power of the geodynamo amounts to approximately 200,000 to 500,000 megawatts – equivalent to the power of several hundred large power stations. Compared with earlier estimates, this figure is relatively moderate. From this, the scientists have concluded that there is no need for an added source of heat in the Earth's core, and that the dynamo is instead driven by the slow loss of the heat that has been stored in the core since the Earth was formed. As the core cools down, liquid iron freezes out and the solid inner core of the Earth grows.

According to previous assumptions, such cooling would occur very rapidly, which would mean that the Earth's solid inner core could have formed only in the last billion years – in other words, in the last quarter of Earth's history. But this contradicts findings from ancient rocks, whose magnetism indicates that the Earth's magnetic field has existed for a much longer period. However, new calculations indicate that cooling occurs very slowly, so the inner core must be more than three billion years old – making it not much younger than Earth itself.

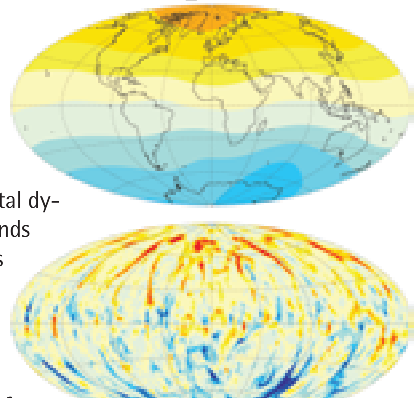

PHOTO: MPI FOR SOLAR SYSTEM RESEARCH

Simulation: The Earth's magnetic field in the computer. Blue represents a "magnetic flux" outward, and red, an inward flux. On the surface of the Earth's core (below), the magnetic field is at a much smaller scale and is more complex than at the Earth's surface (above).

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COLLOID RESEARCH

The Immune System's Rolling Defense

White blood cells, or leukocytes, are the immune system's most important defense tool: they are always there to do battle with germs or to do away with damaged tissue – infiltrating tissue from the blood supply, for example at sites of inflammation or trauma. This assault needs to be both rapid and targeted. An important factor in this process is that they don't just passively swim with the blood flow while on "patrol," but also "roll" along the inner walls of the blood vessels at certain places. More details of the initial steps of this "rolling adhesion" have now been elucidated by Ulrich Schwarz at the Max Planck Institute of Colloids and Interfaces in Potsdam, together with scientists from the Weizmann Institute in Israel (PNAS, Early Edition, April 20, 2004).

In order to carry out their immune defense function, the appropriate type of leukocytes must infiltrate damaged tissue at the right location, from within the bloodstream. They receive the necessary information via substances that are planted on vessel walls close to the affected tissue and that act as emergency signals or guideposts.

This type of "signposting," however, wouldn't be of much use if the leukocytes weren't able to control their movement by rolling down the walls of vessels, stopping in emergencies at exactly the place where they are needed and then migrating out of the vessels. The phenomenon known as "rolling adhesion" solves this problem and involves two sorts of adhesion molecules: selectins and integrins. These are located on the surface of the leukocytes or on the vessel walls and act in conjunction with complementary ligands on the opposing surfaces.

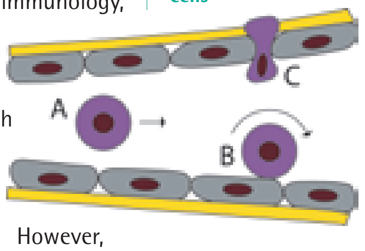
In rolling adhesion, selectin molecules undergo a weak and temporary binding with their ligands. This allows the pressure of the blood flow to push the leukocytes down the walls from one selectin bond to another. If they come across an "emergency" signal, the more strongly binding integrin molecules are activated, braking the rolling adhesion and enabling the leukocytes to penetrate through the vessel walls into the surrounding tissue, and eventually on to the site where they are needed.

Ulrich Schwarz and his colleagues from the Weizmann Institute have been taking a closer look at rolling adhesion. They passed leukocytes down a fluid-filled chamber lined with ligands for selectin molecules, and then followed the movement of the leukocytes with an extremely fast video camera as they rolled down the walls of these "artificial blood vessels." In this way they discovered that, with moderate flow, the contact between selectin molecules and ligands is exceptionally brief. Lasting only four thousandths of a second, they interface the bond between the wall and the leukocytes – "and are, in fact, the shortest molecular binding events yet known to biochemistry," says Schwarz.

Surprisingly, the duration of such bonds seems to increase suddenly when the flow rate is raised above a certain rate – namely by a factor of 14. Initially, this was hard to explain; the duration should rather decrease under the effects of a raised flow pressure. However, further experiments, as well as biophysical modeling and computer simulations, led to a conclusive interpretation of this effect. It seems likely that the raised shear forces cause, not one extremely brief bond, but two selectin bonds to form simultaneously, each renewing their binding at an unusually rapid rate – even faster than the intrinsic bond breaking itself – and stabilizing each other. The effect prolongs the life of selectin-mediated contacts to the seconds range.

Accordingly, rolling adhesion only really becomes a factor when the flow rate reaches a certain threshold – and this for a good, physiologically grounded reason: the leukocytes' adhesive capacity is desirable and useful only within blood vessels. Outside of the vessels it would hinder and delay immune cells on the way to their target.

For now, this new insight into the regulation of rolling adhesion in leukocytes is of particular interest for immunology, as there are diseases caused by adhesion molecule defects, which can, for instance, lead to disturbances in wound healing. However, stem cells also use the phenomenon of rolling adhesion as they migrate via the bloodstream from the bone marrow to their eventual targets. Furthermore, this mechanism enables cancer cells to migrate from a primary focus via the bloodstream and infiltrate certain preferential tissue types, where they lodge and grow into secondary growths (metastases).



GRAPHIC: MPI OF COLLOIDS AND INTERFACES

Circulation of leukocytes: White blood cells are carried through vessels with the bloodstream (A). Due to the presence of adhesive molecules, "rolling adhesion" along the walls of the blood vessels comes into play. This enables the white blood cells to scan the walls of the vessels for stop signals (B). If they encounter a stop signal, stronger adhesion molecules on the white blood cells are activated, bringing the cells to a halt. As a result, the white blood cells leave the bloodstream and enter the surrounding tissue by squeezing through between the cells of the blood vessels (C).

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