Abstractions

FIRST AUTHOR

The hot gases and dust that erupt from a volcano form a plume with a vertical column topped by a horizontal cloud, or umbrella. The umbrella is known to change shape over time from a circle to a wavy, lobular form, but no one knew why. Postdoc Pinaki Chakraborty of the University of Illinois at Urbana-Champaign and his colleagues set out to learn what caused the change. Using satellite images of recent eruptions and an obscure 1811 paper that describes a volcanic plume erupting from the sea, the team concludes that the volcanic plume actually spins about its axis, a phenomenon they have dubbed a volcanic mesocyclone. This rotation model explains the umbrella's changed shape and unravels other mysteries, including why lightning and waterspouts or dust devils usually accompany volcanic eruptions (see page 497). Chakraborty tells *Nature* more.

What is a volcanic mesocyclone?

It's a vortex of air, hot gases and dust that rises and rotates, just like in a violent thunderstorm that spawns a tornado. Everyone had assumed that a volcanic plume doesn't spin. But we discovered that as the hot gases and dust erupt, they engulf atmospheric winds and create this narrowing tube of wind, gases and dust that rotates the column and umbrella.

Why was the 1811 paper significant?

It tied everything together. A sea captain sailing in 1811 in the Azores, west of Portugal, says that the rising plume was rotating — the first and only direct mention of rotation I have ever seen in the literature. He also reports seeing lightning and waterspouts along with the rotation. This was a crucial piece of the mystery for us, and helped us draw an analogy with tornadic thunderstorms.

What did you learn from images of the 2008 eruption of Chile's Mount Chaiten?

They seem to show the whole plume covered in lightning. We found that charged particles in the plume are spat out of the centre of rotation and line up around the edge of the mesocyclone, creating a lightning sheath. Also, the rising plume forms an updraught that begins to spin and may create tornadoes — seen as waterspouts at sea or dust devils on land.

How did you confirm your ideas on rotation?

Using satellite records of the June 1991 Mount Pinatubo eruption in the Philippines, we analysed the umbrella's edge at different times to define its expansion, its rotation and its movement as one element. The umbrella's shape changes because the centrifugal force of its rotation makes the umbrella's edge unstable, and the circle breaks down into a lobular shape. ■

MAKING THE PAPER

Dennis Dougherty

A tiny receptor variation explains nicotine's power over the brain.

Nicotine is one of the most addictive drugs known, in part because it targets the brain's reward system. The compound hijacks brain-cell receptors for the neurotransmitter acetylcholine, which is involved in learning and encoding memories. If nicotine activated the acetylcholine receptors in muscles as efficiently as it does those in the brain, then smoking a cigarette would cause severe, perhaps fatal, muscle contractions. The fact that it doesn't presented scientists with a biochemical conundrum: why does nicotine bind differently to seemingly identical acetylcholine receptors?

Dennis Dougherty, a chemist at the California Institute of Technology in Pasadena, has dissected this paradox over the past two decades. In the late 1980s, he designed a series of artificial chemical receptors to mimic biological binding sites. One turned out to be an excellent binding site for acetylcholine. Dougherty found that an attraction formed between the positively charged acetylcholine and an electron-rich, negatively charged aromatic amino acid in the receptor. He dubbed the effect a cation–π interaction, and wondered whether natural receptors might also use cation–π interactions to bind acetylcholine. He struck up an experimental collaboration with a colleague down the hall — neurobiologist Henry Lester.

Together, they studied the family of nicotinic acetylcholine receptors. Dougherty focused on how complex molecules bind to these receptors; one method he used involved substituting unnatural amino acids at specific sites in a wide range of cellular receptors to test how this affected their function. At the same time, Lester set up a research programme to study the molecular biology of nicotine addiction. "This collaboration between a chemist and a biologist allowed us to do together what neither of us could have done apart," says Dougherty.

In 1998, the duo showed that acetylcholine makes a cation–π interaction when it binds to the muscle acetylcholine receptor. However, nicotine, despite its positive charge, did not make this cation–π interaction in muscle cells.

The next step, looking at how nicotine binds in the brain, presented technical hurdles: for example, the fact that the brain receptor didn't express well in vertebrate cells. Lester's group identified a mutation of the brain receptor that could be used to boost its expression without altering its pharmacological properties. And Dougherty's group found a way to precisely control the ratio of the receptor's five different building blocks, which was needed to effectively incorporate unnatural amino acids.

On page 534, Dougherty, Lester and their colleagues show that a single amino-acid difference between the brain and muscle receptors explains the binding discrepancy. This alters the shape of the brain receptor's binding site so that the crucial π system — a key aromatic residue — is more exposed, allowing nicotine to form a cation–π interaction.

This work shows that nicotine addiction is a biological fluke. "This receptor didn't evolve to bind nicotine. It's simply a coincidence that nicotine activates it," says Dougherty. "If it also activated the receptor in muscles, humans would probably die instantly from smoking."

Dougherty says this work marks the high point of a long and gratifying career arc. He hopes the discovery will help others to find new ways to help people stop smoking, perhaps by developing chemical competitors of nicotine for the brain acetylcholine receptor. Moving forwards, he plans to study other receptors to help him document underlying chemical principles that govern drug–receptor interactions. ■

FROM THE BLOGOSPHERE

Two blog posts this week tell tales — one personal, one global — about how the blogosphere continues to mould scientific publishing. Timo Hannay, publishing director of Nature.com, summarizes all the ways in which pointing-and-clicking removes barriers in scholarly communication. In his Nascent post 'Walls come tumbling down', he describes how the lines between journals and databases are blurring, how publishers can better serve their markets using online research, and how publishing companies are morphing into a mix of broadcasting and technology outlets (http://tinyurl.com/cjpg4e).

Meanwhile, The Great Beyond describes how one scientist recently stood a bit too close to a tumbling wall in online science communication. *Nature* reporter Heidi Ledford recounts how a neuroanatomist found himself in the middle of a tempest when he criticized a conflict of interest in one paper in a letter posted to a rival journal's website (http://tinyurl.com/ctquru). The story, with some very ugly temper-flaring from editors of the publication in question, blew wide open on the *Wall Street Journal* Health Blog. ■

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