

Deformed Nuclei Spit Out Protons

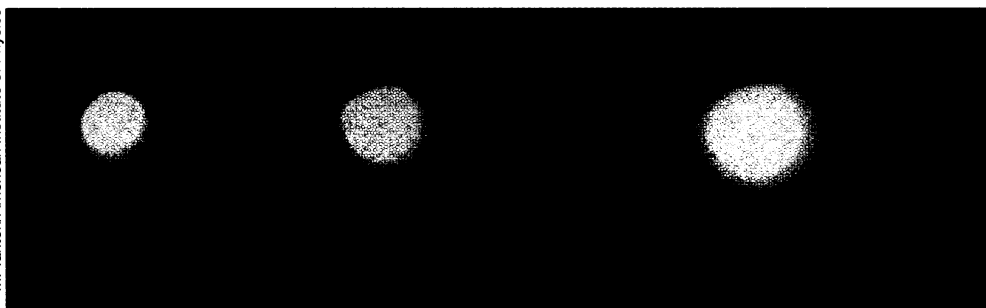
A rare type of radioactive decay can signal the shape of an atom's nucleus.

By determining the rate at which the unstable nuclei of two elements emit protons, researchers have for the first time obtained experimental evidence that these particular nuclei look more like flattened globes than true spheres.

"We haven't actually measured the deformation, but our results show these nuclei to be highly deformed," says Cary N. Davids of Argonne (Ill.) National Laboratory. Davids and an international team of researchers describe their findings in the March 2 *PHYSICAL REVIEW LETTERS*.

"It's a new example of proton radioactivity and, more than that, an excellent example of the ability to deduce something about the shape of a nucleus from the properties of the decay," says Richard F. Casten of Yale University.

Atomic nuclei are made up of protons and neutrons. Normally, neutrons help



prevent the electrostatic repulsion between the positively charged protons from splitting the nucleus apart.

Many nuclei are unstable, however, because they contain too many protons for the number of neutrons present. Such proton-rich nuclei sometimes become more stable by spontaneously ejecting a proton.

In proton radioactivity, a proton deep inside the nucleus penetrates an outer

surface shell of protons. To get through the barrier, it takes advantage of a quantum effect known as tunneling. During the last few years, Davids and his colleagues have been smashing stable nuclei together to create unstable, proton-rich nuclei and looking for evidence of proton radioactivity among these short-lived isotopes. Nearly all of their proton decay results were consistent with tunneling out of a spherical nucleus. Recent improvements in detector sensitivity have enabled the researchers to detect proton radioactivity among certain unstable isotopes of elements containing between 55 and 69 protons. Theorists had predicted that many of these nuclei would be nonspherical. Davids and his team recently created the isotopes holmium-141 (67 protons and 74 neutrons) and europium-131 (63 protons and 68 neutrons). In both cases, the measured half-lives and energies associated with proton decay indicated that the protons must have tunneled through a barrier that could not be uniform in all directions. A spherical model of the nucleus failed to fit the data. "Such proton-emitting nuclei are fantastic laboratories for testing our understanding of quantum tunneling through a deformed barrier," comments Witold Nazarewicz of Oak Ridge (Tenn.) National Laboratory. Davids and his team aim to detect proton radioactivity in unstable isotopes of other elements that contain an odd number of protons, from which a single proton is most likely to escape. Such results, Casten says, will help physicists refine their models of the forces that shape a nucleus.

—I. Peterson

Exploring a genetic link to smoking

Dopamine, a chemical essential to brain function, has a reputation for being the life of the party. Dopamine's day job is to act as a neurotransmitter, passing messages between nerve cells. However, when a person ingests substances that induce pleasure—such as nicotine, alcohol, cocaine, opiates, or even food—extra dopamine is released, enhancing the effect.

Now, researchers at the University of Texas in Houston studying nicotine suggest that an individual's genetic makeup may influence the impact of this dopamine reward. Their findings could help explain why quitting smoking is easy for some people and difficult for others.

Dopamine exerts its effects through receptor molecules in the brain. One gene encoding such receptors has components called A and B, each of which comes in two forms. These components, which are inherited, always occur in pairs. The A variation appears as A1A1, A1A2, or A2A2, and a similar pattern holds for the B variation.

The Texas researchers took blood samples from 283 people, 157 of whom had recently been diagnosed with lung cancer, to determine which genes they possess. Overall, 37 percent of the participants smoked.

The dopamine receptor genes appear to be linked to smoking habits. People with A1 or B1 in their genetic makeup started smoking a year earlier, on aver-

age, than those with A2A2 or B2B2, the researchers report in the March 4 *JOURNAL OF THE NATIONAL CANCER INSTITUTE*.

Smokers with an A1 or B1 version of the gene also reported few serious attempts to quit, says coauthor Margaret R. Spitz. Those showing A1 averaged four attempts and those with B1 averaged six attempts, compared to eight for their A2-only and B2-only counterparts.

Of 76 people in the study who had a B1 gene, less than 3 percent had never smoked. Among 193 people with the B2B2 pairing, 13 percent had never smoked.

Earlier research showed that roughly 10 percent of the U.S. population has a pairing that includes either A1 or B1. "This raises an interesting question: Are these people so genetically affected that they have a great problem quitting smoking?" asks Ernest P. Noble of the University of California, Los Angeles Medical Center.

While that question remains unanswered, the study supports the earlier hypothesis that people with A1 or B1 have fewer dopamine receptors, says Spitz. Such people experience a deficiency in the natural dopamine effect, making the reward from nicotine or other drugs more pronounced, Noble argues. He adds that this dopamine receptor gene "is not a smoking gene per se but a pleasure gene."
—N. Seppa