SOCIOLOGY

Controversial Study Suggests Seeing Gun Violence Promotes It

A longitudinal study of Chicago adolescents has concluded that even a single exposure to firearm violence doubles the chance that a young person will later engage in violent behavior. The study may once again stoke up the debate over juvenile violence; it has already triggered criticism over the unusual statistical method it employs.

The work is part of the decade-old Project on Human Development in Chicago Neighborhoods, run by Harvard University psychiatrist Felton J. Earls. On page 1323, Earls and two health statisticians describe how they used a relatively new technique called "propensity score stratification" to create, through statistical means, a randomized experiment on propensity toward violence from observational data.

Violence debate. A study of Chicago adolescents indicates that seeing a murder may lead to later gun violence by the observer.

CREDIT: *MIDLAND DAILY NEWS*, RYAN WOOD/AP PHOTO

Over a 5-year period, the researchers conducted three interviews with more than 1000 adolescents initially aged 12 to 15. In the first, they gathered extensive data on variables such as family structure, temperament, IQ, and previous exposure to violence. Halfway through the study, the subjects were asked if, in the prior 12 months, they had been exposed to firearm violence—defined as being shot or shot at or seeing someone else shot or shot at. Then at the end of the period, the 984 subjects remaining were asked if they had engaged in any violence—defined as participation in a fight in which anyone got hurt as well as firearm-related incidents, including carrying a gun.

"If you just compare exposed and unexposed, the exposed were three or four times as likely to be [violence] perpetrators," says lead author Jeffrey B. Bingenheimer, a Ph.D. candidate at the University of Michigan School of Public Health in Ann Arbor.

The authors then went to great lengths to weed out confounding factors. Subjects were ranked according to "propensity" scores: a cumulative tally of 153 risk factors that estimated the probability of exposure to gun violence. They were then divided up according to whether or not they had reported such exposure and whether or not they had subsequently engaged in violent behavior. Those with the same propensity scores but different exposures were compared with each other. In this way, the authors claim, they controlled for a host of individual, family, peer, and neighborhood variables.

Even with this analysis, exposure to gun violence predicted a doubling of the risk for violent behavior—from 9% for unexposed to 18% among the subjects who reported exposure, says Bingenheimer. And it didn't take repeated exposures—"the vast majority" of subjects reported only one, he says. Can a single experience of seeing someone shoot at someone else make an individual more violence-prone? "That doesn't seem improbable to me," says Bingenheimer. "It could be for only a minority, but a very large effect for that minority."

Developmental psychologist Jeanne Brooks-Gunn of Columbia University, one of the scientific directors of the Chicago neighborhoods project, agrees that a single exposure might have a profound effect, even on a hitherto nonviolent individual. "Nobody's done this kind of analysis before," she says, and nobody has focused just on gun violence, which "clearly is a very extreme type of violence."

But a number of other scholars have deep misgivings about both the study findings and the methodology. Psychiatrist Richard Tremblay of the University of Montreal in Canada says the study does not demonstrate that "those who are nonviolent to begin with will become violent." Indeed, the authors didn't address this point directly because a lack of subjects in the lowest-risk category led them to eliminate it from their analysis. ▶

ScienceScope

New Reporting Regs for Globe-Trotting Diseases

The world has a new set of rules for dealing with diseases, such as flu or SARS, that cross borders easily. On Monday, the World Health Assembly, an annual meeting of 192 governments in Geneva, Switzerland, approved regulations making it mandatory for countries to detect and respond to infectious diseases within their borders, notify the World Health Organization (WHO) within 24 hours of any outbreak that could threaten other countries, and collaborate in investigating and controlling such outbreaks.

Similar International Health Regulations have existed for half a century. But even the latest version from 1981 was widely considered outdated; for one, it didn't cover newly emerging infections. The revised treaty, which will formally take effect in 2007, has been debated for more than 10 years. The issue became more urgent in 2003, when China risked a wide spread of SARS by hiding the extent of its outbreak —behavior that would violate the new rules. Although WHO has no sanctions for countries that violate the new regimen, "this gives us much clearer ground rules," says WHO'S Max Hardiman.

–MARTIN ENSERINK

Embattled Berkeley Ecologist Wins Tenure

Ignacio Chapela, an ecologist whose views on biotechnology have attracted controversy, has won tenure at the University of California, Berkeley, after appealing an earlier rejection.

Chapela caused a stir with a 2001 report in *Nature* that promoter genes from genetically modified corn had been detected in traditional kinds of corn in Mexico—a finding the journal later disavowed (*Science*, 12 April 2002, p. 236). He also was a persistent critic of a $25 million deal with Novartis in 1998 for exclusive licensing of plant and microbial research.

Chapela claimed that the university denied him tenure in 2003 because of his opposition to the Novartis deal (*Science*, 19 December 2003, p. 2065). Last month, he sued the university, claiming it had also discriminated against him because he was born in Mexico. Berkeley, meanwhile, was reexamining the case as part of an earlier consent agreement, and a nine-member panel voted thumbs-up. "This was a case in which reasonable reviewers could disagree," says spokesperson George Strait. After learning of his victory, Chapela e-mailed supporters that he now fears tenure "may become a [self-imposed] muzzle."

–ERIK STOKSTAD

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Because the remaining subjects already had some violence risk factors, the results don't surprise Tremblay. He compares the work to looking at whether alcoholics are more likely to drink if they are exposed to alcohol. It is already well known, he says, that "if individuals at a high risk of violence are in an environment with violence, they're more likely to be violent."

Economist Steven Durlauf of the University of Wisconsin, Madison, calls the study an "implausible modeling of violence exposure." The authors assume that two individuals with the same propensity rankings are equally likely to encounter violence, he says. But such exposure may not be random; rather, it probably stems from "something that has not been measured"—such as recklessness, says Durlauf. Nobel Prize–winning economist James Heckman of the University of Chicago agrees, calling the study "potentially very misleading." Adds Heckman: "This is why this kind of statistics is not science. This is why you find out orange juice causes lung cancer one week and cures it the next."

But Brooks-Gunn defends the innovative study. The propensity scoring technique "comes the closest we have to any experiment, which is why I think the results are so strong," she says. **–CONSTANCE HOLDEN**

BIOCHEMISTRY

Plant Hormone's Long-Sought Receptor Found

In all of nature, few molecules do more. The plant hormone auxin helps plants grow toward light, grow upward rather than branch out, and grow their roots down. It helps plants flower and bear fruit. Now, more than 70 years after auxin was first discovered, biologists have finally identified its major receptor—a crucial step toward understanding how the hormone works.

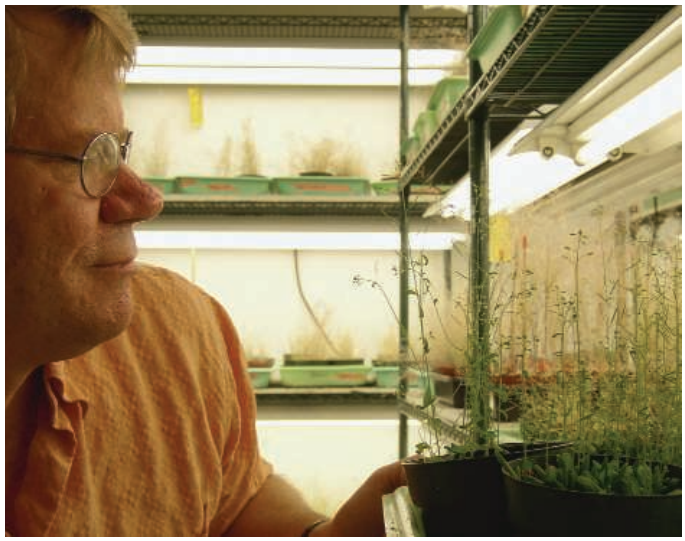
"It's really exciting for auxin biology to know how auxin can be perceived," says plant geneticist Bonnie Bartel of Rice University in Houston, Texas.

In the 26 May issue of *Nature*, two teams, led by Ottoline Leyser of the University of York, U.K., and Mark Estelle of Indiana University, Bloomington, independently report that auxin binds to a protein called TIR1. When auxin attaches, TIR1 helps mark for destruction another protein that represses a set of genes that are known to be activated by auxin's presence; when the cell destroys that protein, the genes turn on.

For decades, biochemists fished around in extracts of growing plants for proteins that bound to auxin (also known as indole-3-acetic acid). Plants lacking one such protein, auxin-binding protein 1 (ABP1), die, demonstrating that it is essential. But ABP1 does not resemble other hormone receptors, and it doesn't seem to turn genes on or off, a property that's needed to explain auxin's myriad effects, Estelle says. So beginning in the mid-1980s, he and his coworkers began anew, identifying lines of a small plant called *Arabidopsis thaliana* (wall cress) that respond abnormally to auxin. They reasoned that the defective genes in these mutant lines might be part of the machinery that enables the plant to respond to auxin.

One such defective gene encoded an F-box protein, a family of proteins found in plants and animals that tag other proteins with a molecule called ubiquitin, which signals the cell to destroy the tagged proteins. That suggested that the plant auxin response involved protein degradation, and that this particular F-box protein, called TIR1, played a key role. By 2001, Estelle and Leyser, a former postdoc of Estelle's who by then ran her own laboratory, had shown that auxin causes a protein complex containing TIR1 to bind to so-called Aux/IAA proteins, which repress certain genes known to be triggered by auxin. Auxin apparently activates genes by marking Aux/IAA proteins for destruction.

To establish precisely how, the two teams first spent several years running down "a lot of blind alleys," Estelle says. It turned out that the pathway was a lot simpler than assumed, Leyser says. They'd expected an auxin receptor to activate genes the way other hormone receptors do: through a signal cascade involving a series of enzymes in which the last one activates gene-regulating protein. Both teams isolated TIR1-containing complexes from plant extracts, thinking they'd have to find and add back other enzymes to allow the complexes to detect auxin and bind Aux/IAA. But nothing else was needed. To prove the point, both teams added radioactively tagged auxin and showed that it bound to purified TIR1 complexes but not to Aux/IAA proteins.



Hormone helper. Two teams, one led by Mark Estelle (*above*), have finally identified a key receptor that enables the hormone auxin to guide plant growth.

CREDIT: DAVID BRICKER, 2005

Stefan Kepinski, a postdoc in Leyser's laboratory, also took the gene encoding TIR1 and injected it into hundreds of frog embryos in order to mass-produce the protein. After purifying TIR1 from the ground-up embryos, Kepinski showed that the auxin caused the protein to bind to a purified piece of an Aux/IAA protein. Nihal Dharmasiri, a postdoc in Estelle's group, did similar experiments with TIR1 protein produced in insect cells and got similar results. Because no other plant proteins were present in either case, the work shows that TIR1 is an auxin receptor, Estelle says.

"We're happy to have a receptor for auxin," says plant biologist Joanne Chory of the Salk Institute for Biological Studies in La Jolla, California. "Auxin has been such an enigma."

What's more, according to results from Estelle's group that will appear in *Developmental Cell*, TIR1 is just one of four related F-box proteins, each of which functions as an auxin receptor; when all four are missing, a plant's development is severely damaged. These results suggest that a family of TIR1-like proteins, working with a family of AUX/IAA proteins, could direct many of the diverse physiological responses to auxin.

The discovery of this auxin receptor may also shed light on additional plant signaling pathways. Plants have roughly 700 F-box proteins, but little is known about them. Researchers suggest that some of them may mediate responses to other hormones, such as jasmonate, which mediates plant defenses, and the gibberellins, which promote germination and stem growth. "It's a whole new type of receptor," Bartel says. That's "the big story."

–DAN FERBER

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