Monsanto has already made clear its favoured explanation for the contamination: sabotage. "There are folks who don't like biotechnology and who would use this as an opportunity to create problems," the company's chief technology officer Robert Fraley told reporters last month. Activists opposed to genetically modified crops are best known for destroying the plants rather than sowing them, but Fraley argues that those who illegally enter fields to demolish crops could also break into experimental plots to collect seed.

That hypothesis has little support among plant scientists contacted by *Nature*. "I suppose it's possible, but it's a very small possibility," says Norman Ellstrand, a plant biologist at the University of California, Riverside. He says that any saboteur would have been taking a gamble that the GM wheat would be found. It only came to light in Oregon because the farmer had sprayed a non-GM wheat field with glyphosate (the herbicide to which the GM wheat is resistant) in preparation for a new crop, noticed a few remaining wheat plants and notified others of the discovery.

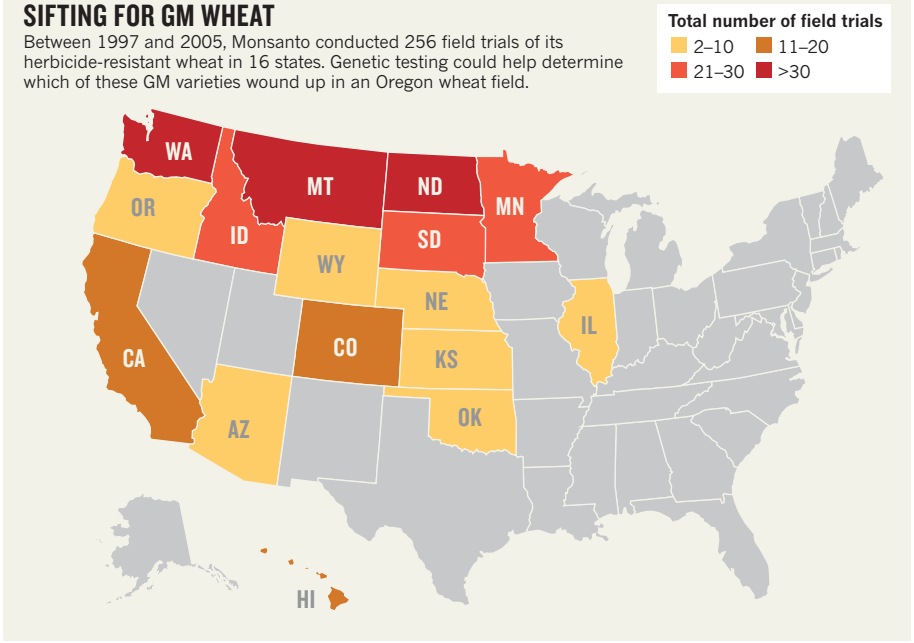
Fraley argues that the distribution of the contaminant plants suggests that a human hand cast them there. They were found in localized patches in only one of two wheat fields that had been planted with the same non-GM seed. But Robert Zemetra, a wheat breeder at Oregon State University (OSU) in Corvallis, says there could be other explanations for such a distribution. If, for example, the contaminant was a spring wheat plant in a winter wheat field, the transgenic wheat would flower and drop most of its seeds before the rest of the crop was harvested. Those seeds would fall straight down, generating a clump of herbicide-resistant offspring.

No explanation is completely satisfying, acknowledges Rene Van Acker, a weed scientist at the University of Guelph in Ontario, Canada. Van Acker and Zemetra carried out separate field trials of the wheat over a decade ago, and both say that Monsanto kept a close watch over the experiments. "We had to account for pretty much every seed in and every seed out, down to the gram," recalls Van Acker. Zemetra not-so-fondly remembers hours spent disassembling harvesters and cleaning each part with an air-compressor, removing seeds with tweezers when necessary.

But Carol Mallory-Smith, the OSU weed scientist who first tested the Oregon plants three months ago, wouldn't be surprised if one of the field-test seeds had escaped. She has found transgenic crops in stranger places. In 2009, for instance, she found transgenic sugar-beet seedlings in a bag of soil sold to gardeners. "There are so many places in the system where errors can be made," she says. "Once we release these genes into the field, we should just assume that they are going to stay in the environment." ■

SIFTING FOR GM WHEAT

Between 1997 and 2005, Monsanto conducted 256 field trials of its herbicide-resistant wheat in 16 states. Genetic testing could help determine which of these GM varieties wound up in an Oregon wheat field.



SOURCE: USDA, MONSANTO

MEDICINE

NIH gambles on recycled drugs

Early success could bolster congressional support for agency's translational science centre.

BY MEREDITH WADMAN

On Monday, Stephen Strittmatter began recruiting for a clinical trial to test a treatment for Alzheimer's disease. The study is also part of a bigger test for the US National Institutes of Health (NIH) and its fledgling translational research centre.

Strittmatter's team is one of nine that won funding last month from the NIH's National Center for Advancing Translational Sciences (NCATS) in Bethesda, Maryland, to see whether abandoned drugs can be aimed at new targets. Strittmatter, a neurobiologist at Yale University in New Haven, Connecticut, hopes that a failed cancer drug called saracatinib can block an enzyme implicated in Alzheimer's.

The money is not much — US$12.7 million split between the nine awards this year — but the symbolism is potent. NCATS, which launched in December 2011, has promised to find ways around obstacles that stymie the pharmaceutical industry. Legislators have been sceptical about the need for such a programme, but if the drug-resurrection initiative succeeds, it may win over critics in Congress, for whom repurposing drugs has appeal in an age of fiscal austerity.

Agency officials downplay the stakes, arguing that the programme's chief aim is to establish new partnerships between industry and academia. Critics say that its scientific goal may be a tall order. "Others passed [on these drugs] because the scientific odds of success were low," says Scott Gottlieb, a resident fellow at the conservative American Enterprise Institute in Washington DC, and a former deputy commissioner of the Food and Drug Administration. "The odds that NIH has some early wins are equally low."

The projects are tackling conditions including alcoholism, schizophrenia and calcified aortic valves (See 'Old drugs, new tricks'). But none targets an illness of more growing public health concern than Alzheimer's, which is expected to afflict up to 16 million people in the United States by 2050, and will cost the country around $200 billion in 2013. ▶

OLD DRUGS, NEW TRICKS
The US National Institutes of Health has awarded nine grants to find uses for eight drugs abandoned by pharmaceutical firms.

Drug name	Original target(s)	New target(s)
PF-03463275	Schizophrenia	Schizophrenia (new mechanism)
AZD0530 (saracatinib)	Cancer	Alzheimer's disease; lymphangioleiomyomatosis
JNJ-39393406	Schizophrenia	Smoking cessation
ZD4054 (zibotentan)	Cancer; peripheral artery hypertension	Peripheral artery disease
PF-05190457	Type II diabetes	Alcoholism
LY500307	Prostate enlargement; urinary tract symptoms	Schizophrenia
Undisclosed (made by Sanofi)	Undisclosed	Calcific aortic valve stenosis
Undisclosed (made by Sanofi)	Undisclosed	Duchenne muscular dystrophy

Source: National Institutes of Health

Saracatinib inhibits the Src family kinases (SFKs), enzymes that are commonly activated in cancer cells, and was first developed by London-based pharmaceutical company AstraZeneca. But the drug proved only marginally effective against cancer, and the company abandoned it — after spending millions of dollars to develop it through early human trials that proved that it was safe. With that work already done, Strittmatter's group will be able to move the drug quickly into testing in people with early-stage Alzheimer's disease.

The team plans to begin a 24-person safety and dosing trial in August. If the results are good, NCATS will fund the effort for two more years, during which the scientists will launch a double-blind, randomized, placebo-controlled trial with 159 participants. Over a year, the team will measure declines in glucose metabolism — a marker for progression of Alzheimer's disease — in key brain regions, hoping to find that they have slowed.

"For this to happen in years instead of decades is only possible because of this programme," says Strittmatter. The NIH worked with eight pharmaceutical companies to develop a model agreement for the firms and academic investigators; it specifies how intellectual property will be apportioned and gives the companies first right of refusal to license any discoveries.

Now NCATS must wait for results. Using saracatinib to treat Alzheimer's is "a reasonable idea", says Tony Hunter, director of the Salk Institute Cancer Center in La Jolla, California. The Yale researchers hope that the drug will slow or halt progression of the disease by blocking the activity of Fyn kinase, an SFK that has been shown to induce damage to brain synapses in mouse models of Alzheimer's (J. Chin *et al. J. Neurosci.* **24,** 4692–4697; 2004). But Hunter warns that there may be side effects. SFKs have many functions, so if high doses of saracatinib are needed to inhibit Fyn kinase action in the brain, they could do damage elsewhere.

Strittmatter says that the main side effect of concern to him is reduced white-blood-cell counts, which could increase risk of infection. However, in saracatinib cancer trials, that side effect occurred at higher doses than he intends to use.

NCATS is barred by law from funding trials beyond early phase II, so further money will have to come from another backer, such as AstraZeneca. For that, observers say, Strittmatter will need startlingly good results.

"This is going to be hard for them to do," says John LaMattina, a senior partner at PureTech Ventures, a life-sciences venture-capital company in Boston, Massachusetts, and former president of research and development at the drug-maker Pfizer in New York.

But fans of the NIH programme say that it is not reasonable to expect quick success, given the time-consuming nature of drug development. "The commitment to creative repurposing is wise and timely," says Ann Bonham, chief scientific officer at the Association of American Medical Colleges in Washington DC. "Over time, even with a relatively modest success rate, the potential benefit is worth the investments." ■

ANIMAL DISEASE

Rinderpest research restarts

As moratorium lifts, oversight is put in place to assess studies on eradicated cattle virus.

BY DECLAN BUTLER

Research is set to resume on the rinderpest virus, the cause of a deadly cattle disease that was declared eradicated in 2011 and has been off limits for study ever since. The moratorium — part of efforts to guard against accidental or intentional release of virus that could reintroduce the disease — was lifted on 10 July and replaced by a new international oversight system for such research.

In its heyday, the disease — the only one other than smallpox to be eradicated from nature — killed hundreds of millions of cattle, mainly in Europe, Asia and Africa, often leaving famine in its wake. Under the new oversight system, run by the Food and Agriculture Organization of the United Nations (FAO) in Rome and the Paris-based World Organisation for Animal Health (OIE), the risks and benefits of research proposals will be assessed by a joint advisory committee, and then the FAO and the OIE will decide on approvals. Eligible research must show potential for substantial practical or scientific benefits and be conducted under stringent biosafety and biosecurity conditions.

The first project that has garnered approval will test whether vaccines developed against a closely related virus — *peste des petits ruminants* (PPR), which causes disease in sheep and goats — might also protect cattle against rinderpest. Led by Michael Baron, a rinderpest researcher at the Pirbright Institute in Pirbright, UK, the project, if successful, would eliminate the need to retain stocks of live-attenuated rinderpest vaccine. That would contribute to the goal of reducing the number of labs worldwide holding rinderpest material, thus decreasing the risk of reintroduction.

Some 55 labs in 35 countries still hold some kind of rinderpest virus, according to a 2011 survey published in January 2013 in the journal *Emerging Infectious Diseases*: 37% of them in Asia, 29% in Africa and 26% in Europe (G. Fournié *et al. Emerging Infect. Dis.* http://doi.org/m7w; 2013). The identities of the labs remain confidential. The most dangerous stocks are of live field strains of virus, estimated to be kept in at least 16 labs in 14 countries, and samples of blood and tissues from infected herds,

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For more on rinderpest, see:
go.nature.com/nait5m