2008 [that includes an annual payment of $6.5 million], and we are living up to that contract," says Amgen spokesperson Jeff Richardson. "We're ending our affiliation with them, but if someone were to pay me until 2008, I would think they were still supporting me."

The institute's director, molecular geneticist Tak Mak, says that he's grateful for Amgen's support but that "we are happy to be again on our own, concentrating on the science." Although he says that the settlement "is not consistent" with Amgen's previous level of support, he acknowledges that there's little he can do about it: "If you're the boss, you can call the shots."

The institute is now part of the new Advanced Medical Discovery Institute within Toronto's University Health Network. UHN research vice president Christopher Paige says the settlement buys the university enough "breathing room" between now and 2008 to raise an estimated $65 million needed to maintain the institute's current level of operations, including a half-dozen or so principal investigators and as many as 90 technicians, students, and support staff. One investigator, Josef Penninger, had previously announced that he was moving his lab to his native Austria.

Both parties agree on one thing: The new setup gives scientists more freedom to pursue their research and disseminate the results. "Having those researchers in a university setting doing proprietary research did not allow them to speak with their colleagues about what they were doing," says Richardson. "Now they have academic freedom, and they really didn't have that before."

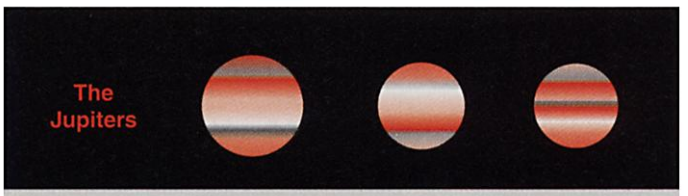
Mak says that he's relieved to be shedding 10 years of corporate ties and that having UHN own the intellectual property rights to any discoveries "allows us to be more free in terms of giving away animals and reagents, without six lawyers signing off." Mak has been criticized in the past by colleagues for being unresponsive to such requests (*Science*, 23 June 1995, p. 1715). **–WAYNE KONDRO**

Wayne Kondro writes from Ottawa.

EXOPLANETS

Jupiter's Brother Joins the Family

Last week American astronomers announced the discovery of a new yet familiar-looking planetary system—not "a sibling of the solar system," but "a first cousin." The distant relation made front-page news, but in the hour before the Americans' televised press conference got under way at NASA headquarters, European astronomers were discreetly spreading the word via e-mail that they had uncovered a nearer relation: an exoplanet that more closely resembles Jupiter in a planetary system far more like our own, "a younger brother" of Jupiter, as one of the discoverers put it. The find marks the true beginning of an expected string of discoveries of planetary systems in which Earth-like planets might be hiding.



	55 Cancri's	HD 190360's	Jupiter
Mininum mass	4.3 x Jupiter's	1.1 x Jupiter's	1.0
Mean orbital distance	6.0 AU	3.7 AU	5.2 AU
Eccentricity	0.16	less than 0.1	0.05

Do the numbers. When the two Jupiter-like planets are compared, the one orbiting HD 190360 announced this week is closer to Jupiter in mass and orbital eccentricity.

Both discoveries came as astronomers searched for telltale stellar wobbling, a sign that the gravity of a massive unseen planet is tugging the parent star back and forth. Before last week, 76 exoplanets had been discovered, but none resembled Jupiter, the solar system's most massive planet. All either were "hot Jupiters" orbiting closer to their stars than Mercury does to the sun or were on wildly elongated orbits.

Last week astronomer Geoffrey Marcy of the University of California, Berkeley, and his colleagues announced their "near analog to our Jupiter" (*Science*, 14 June, p. 1951). It is a body at least 4.3 times the mass of Jupiter. It orbits the star 55 Cancri at a distance of 6.0 times Earth's orbital distance (6.0 astronomical units, or AU). And it is in an orbit "just a little out of round," having an eccentricity of 0.16 (Jupiter's eccentricity is 0.05).

Unfortunately, planet 55 Cancri d hangs out in a distinctly un-Jovian neighborhood. The American team had already found one hot Jupiter orbiting 55 Cancri and announced a second last week. Like all hot Jupiters, these must have formed farther out and drifted inward, driving everything before them into the star and vaporizing any inner, Earth-like planets. "It's not like our solar system," says exoplanet searcher William Cochran of the University of Texas, Austin, "which is what people are looking for."

Now astronomer Michel Mayor of the Geneva Observatory in Sauverny, Switzerland, and his colleagues believe they have discovered a true Jupiter orbiting the star HD 190360. The new planet, which they formally announced this week at a meeting* in Washington, D.C., has a minimum mass just 1.1 times that of Jupiter and orbits at 3.7 AU. In the solar system, that would put it outside Mars between the asteroid belt and Jupiter. And its eccentricity is less than 0.1, indistinguishable from Jupiter's. Best of all, Mayor's HD 190360 has no hot Jupiters. In Doppler-shift observations, its planetary system looks nearly identical to what alien observers would see if they looked at ours. Astronomers are welcoming both discoveries as the vanguard of a coming Jupiter bonanza. "I think it's great," says astronomer David Trilling of the University of Pennsylvania in Philadelphia. "In the next few years, there will be dozens and dozens more."

–RICHARD A. KERR

* "Scientific Frontiers in Research on Extrasolar Planets," 18 to 21 June, sponsored by NASA and the Carnegie Institution of Washington.

JAPAN

New Program to Aid Smaller Universities

TOKYO—Like most of Japan's smaller universities, Fukui University doesn't have a big research budget. But a new program to help universities build up strengths in specific areas will give scientists there a chance to compete against the scientific heavyweights at Tokyo, Kyoto, and Tohoku universities for precious government funding.

Last week the Ministry of Education launched its 21st Century Centers of Excellence program and invited all universities, public and private, to compete. The $160-million-a-year program will concentrate resources, in contrast to Japan's traditional approach of scattering small grants across the academic research enterprise. Another novel wrinkle is that applications must be submitted by the president of the university, rather than by a research group or individual scientist. The idea is to ensure the institution's commitment to the project. "From now on, universities will have to begin thinking strategically," says Yoshihide Akatsuka of the ministry's University Reform Office.

The ministry hopes to fund 20 or so centers in each of five areas: life sciences,

chemistry and materials sciences, electronics and information sciences, humanities, and the deliberately vague category of interdisciplinary studies. A separate review committee in each area will select centers based on both the track record of its investigators —especially publications and awards—and the likelihood of achieving "epochmaking results." Grants will range from $800,000 to $4 million a year for 5 years.

Akatsuka admits that the amounts might not be large enough to build a new program. But Yukinori Urushizaki, an administrator overseeing research support at Fukui University, says that winning a grant "would be a considerable boost" to the image of the university, located 350 kilometers west of Tokyo. "We are different from the big universities … we have a limited budget for research," Urushizaki adds.

The program's funding levels are "not so attractive to the major universities," acknowledges Keiichi Kodaira, president of the Graduate University for Advanced Studies in Hayama, near Tokyo. But that doesn't mean his institution and other research heavyweights won't compete. Kodaira, a member of an advisory committee that vetted the program, predicts that "many [large universities] will apply simply because if they don't, their [programs] will be regarded as below standard."

The first batch of centers is expected to be selected by the end of September. Ministry officials hope to receive enough new money in the next fiscal year, which begins 1 April, to add five more areas and another 100 centers. The program's long-term status, however, has not yet been determined.

–DENNIS NORMILE

GENE TRANSCRIPTION

Demolition Crew Gets a Hand From Chaperones

The body's cells react rapidly to the ups and downs of the hormones that control their activities. But that presents a puzzle: The biochemical machinery that responds to certain hormones is so large and seemingly cumbersome that researchers have long wondered how it manages to react quickly to changes in hormone concentrations. New results, described on page 2232 by Brian Freeman and Keith Yamamoto of the University of California, San Francisco (UCSF), suggest a solution to this long-standing conundrum—one that might provide a new role in regulating gene expression for the proteins known as chaperones.

Donald DeFranco of the University of Pittsburgh School of Medicine calls the new results "novel and important" and predicts that "this will be an 'impact' paper." There is, however, at least one competing view of which molecules to thank for a cell's rapid-response capabilities.

The hormones for which the conundrum arises include the thyroid hormone thyroxine and the steroids, such as the sex hormones and the glucocorticoids that regulate cell metabolism. When one of these hormones binds to its receptor, the resulting complex moves to the nucleus, where it, together with several other proteins, binds to regulatory sequences on the DNA. This, in turn, activates or inhibits appropriate genes. The mystery has been how the receptor can, while buried deep in this regulatory complex, detect when hormone levels fall off—a feedback needed to tell the regulatory complex to stop overseeing the genes.

The new results by Freeman and Yamamoto indicate that two so-called chaperone proteins, named p23 and Hsp90, help disassemble regulatory complexes shortly after they form on the DNA. When thus released, the receptor can, as Yamamoto puts it, "quiz the cell: Is the hormone around?" If it is, the regulatory complex can be reformed, but if not, the response can be terminated.

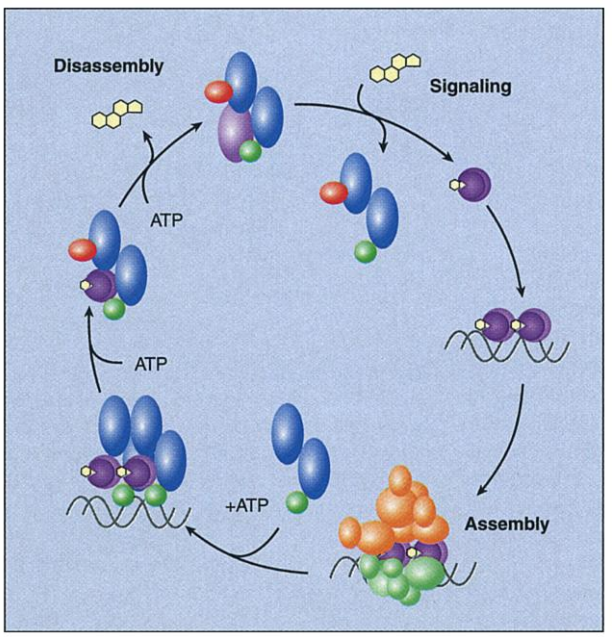
Freeman and Yamamoto were inspired to undertake the current experiments partly by a finding from Gordon Hager's team at the National Cancer Institute in Bethesda, Maryland: Despite their size, steroid hormone regulatory complexes get on and off DNA very quickly (*Science*, 18 February 2000, p. 1262). "Here we are building these immense protein machines, and they are turning over within seconds," Yamamoto says.

Chaperones, which help proteins fold into their proper three-dimensional shapes, were known to facilitate the binding of steroid hormones to their receptors. But earlier hints from Yamamoto, DeFranco, and others that the proteins might also act later to break down the resulting regulatory complexes were met with skepticism. Disassembly of the complexes takes place in the cell nucleus, and chaperones' actions were thought to be limited to the cell cytoplasm.

The current work appears to demonstrate chaperones' role in ending a receptor's effects on transcription. For example, in vitro experiments revealed to Freeman and Yamamoto that p23 both triggers the release of the thyroxine-thyroid hormone receptor complex from the DNA to which it binds and also decreases the gene transcription normally caused by the complex. And in living cells, the investigators showed that p23 binds to glucocorticoid-induced regulatory complexes—which it must do in order to trigger their breakup—and blocks transcription induced by the hormones. Hsp90 had similar, but weaker, effects. What's more, the UCSF researchers found that p23 blocked transcription by two very different regulatory complexes—ones not containing steroid hormone receptors—suggesting a more general role for chaperones in controlling gene activities.

DeFranco says that these experiments provide "by far the clearest and most striking evidence" that chaperones help disassemble regulatory complexes containing hormone receptors. But Hager is not so sure. Some cellular actor is needed to release receptors from the complexes, he says, but his work, including results that appear in the May issue of *Molecular and Cellular Biology*, indicates that the job is performed by the so-called Swi/SNF complex, which alters the way that the DNA and its associated proteins are wound together. Hager notes that both Swi/SNF and the chaperones might have a role, depending on the circumstances. He and Yamamoto are collaborating to see if they can reconcile their differences.

–JEAN MARX



Going in circles. From the upper right, when a steroid receptor (purple, top) binds a hormone (yellow), it kicks off chaperones (blue, green, and red). It then settles on DNA and draws in several other proteins (green and orange) to form a regulatory complex. Disassembly of this complex is triggered by chaperones that the energy compound ATP aids, ultimately returning the receptor to its initial state.

CREDIT: ADAPTED FROM B. C. FREEMAN AND K. R. YAMAMOTO, *TRENDS BIOCHEM. SCI.* 26, 285 (2001)