

tions—or variations in the location of electrons around atoms—might also prove crucial, Haule speculates. In essence, the iron-based materials give more freedom to electrons than cuprates do when it comes to how electrons circle around atoms.

Orbital fluctuations might play important roles in other unconventional superconductors as well, such as ones based on uranium or cobalt, which operate closer to absolute zero, Haule conjectures. Because the iron-based superconductors work at higher temperatures, such fluctuations may be easier to research.

Besides illuminating the theoretical underpinnings of superconductivity, the discovery “makes us ask if there are other high-temperature superconductors we haven’t found yet in unexpected places and if there are even higher temperatures these can work at,” remarks theoretical physicist David Pines of the University of California, Davis, who is also founding director of the Institute for Complex Adaptive Matter. In trying to boost the critical temperature, experiments should focus not only on swapping in other elements but also on layering the compounds. That should im-

prove them just as it does for cuprate superconductors, Haule thinks.

Being based on iron could make these substances more commercially enticing, too. The fragility of cuprates, which as ceramics are quite brittle, has long hampered applications such as superconducting power lines. If iron-based materials are easier to handle and manufacture than cuprates, “they will become very important,” Haule adds.

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NEURODEGENERATION

Your Brain on Diabetes

More signs that insulin ills set off neurodegenerative conditions **BY MELINDA WENNER**

Anyone who is diabetic—or knows a diabetic—recognizes the importance of insulin. The hormone helps cells store sugar and fat for energy; when the body cannot produce enough of it (type 1 diabetes) or responds inadequately to it (type 2 diabetes), a range of circulatory and heart problems develop. But that is not all: recent research suggests that insulin is crucial for the brain, too—insulin abnormalities have been implicated in neurodegenerative diseases, including Alzheimer’s, Parkinson’s and Huntington’s. Among the latest findings is the discovery that a gene linked to insulin processing is located in a chromosomal area linked to Parkinson’s.

Historically, scientists believed that insulin was produced only by the pancreas and had no business in the central nervous system. Then, in the mid-1980s, several research groups spotted the hormone and its receptor in the brain. It appeared that the hormone not only crossed the blood-brain barrier but that it was also produced, at low levels, by the brain itself.

Soon afterward, scientists discovered that insulin plays an important role in learning and memory. People who inject or snort insulin immediately get better at recalling stories and performing other mem-

ory tasks. Learning also raises insulin levels: rats mastering spatial memory tasks have higher brain insulin levels than sedentary rats do.

These observations led neuropathologist Suzanne de la Monte and her colleagues at Brown University to ask whether brain insulin might have a part in Alzheimer’s, which is characterized by severe



SWEET TOOTH, SOUR BRAIN: Recent studies emphasize the connection between neurodegeneration and insulin problems, such as those in diabetes.

memory loss. They compared postmortem insulin and insulin receptor levels in healthy brains and brains of Alzheimer’s patients. Average insulin levels in the neural parts associated with learning and memory were up to four times higher in the healthy brains, which also had up to 10 times as many insulin receptors.

“That made it clear that one could get exactly the same problems as in regular diabetes except confined to the brain,” says de la Monte, who refers to Alzheimer’s as “type 3 diabetes.” Because brain insulin is linked to insulin in the rest of the body via the blood-brain barrier, diabetics are more likely to develop Alzheimer’s, too—nearly twice as likely, according to a 2002 study. They also suffer more memory and learning problems than the general population.

De la Monte and others, including neuroendocrinologist Ignacio Torres Alemán of the Cajal Institute in Madrid, have also found links between Alzheimer’s and low brain levels of insulinlike growth factor 1 (IGF-1) and its receptor—proteins similar in structure to insulin and its receptor (insulin occasionally binds to the IGF-1 receptor, and vice versa). “We have suggested that Alzheimer’s disease originates because of an exacerbated loss of IGF-1 sup-

port to brain cells," Torres Alemán says.

A handful of recent studies have also linked insulin and IGF-1 to Parkinson's and Huntington's. The prevalence of diabetes in patients with Huntington's is seven times higher than average, and at least half of Parkinson's patients have glucose metabolism problems. Robert Smith, an endocrinologist at Brown, recently discovered a protein called GIGYF2 that interacts with insulin and IGF-1 receptors. To better understand GIGYF2's function, Smith mapped the location of its gene in the human genome. Reporting in the April 11 *American Journal of Human Genetics*, he says, "we found that it was smack dab in the PARK11 site," a region of chromosome 2 that is linked to Parkinson's—al-

though he is not certain what the gene's role in Parkinson's may be.

Indeed, one of the biggest remaining questions is how, exactly, defects in insulin and IGF-1 signaling might hurt the brain. "This is a crucial topic—something we're spending a great deal of effort unraveling," de la Monte says. Some scientists believe that insulin is involved in the production of large protein plaques observed in the brains of patients with Alzheimer's and Parkinson's. When Smith added above normal levels of GIGYF2 to neurons in the lab, large GIGYF2 aggregates formed and killed the cells. Other studies have determined that insulin modulates the production and degradation of amyloid beta, the protein that forms

sticky plaques seen in Alzheimer's brains.

Although no one yet knows all the details of what is happening, few scientists in the field doubt that insulin and IGF-1 are crucial players in neurodegenerative disease. Many are working on potential treatments that restore normal insulin function in the hopes of mitigating or even preventing neurodegeneration. For example, compounds that improve insulin response in the brain and body have been shown to lessen cognitive decline in early-stage Alzheimer's patients. "It is so exciting," de la Monte says. "The fact that we have some way to go after it is really cool."

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BANDWIDTH

Fight in White Space

Could future wireless devices destroy HDTV broadcasts? **BY LARRY GREENEMEIER**

Microsoft, Google and several more of the world's largest and most influential technology companies have found a way to provide wireless Internet access that is so fast it makes today's Wi-Fi networks seem as sluggish as dial-up service. The prospect, however, has big media broadcasters up in arms, because this blazing-fast network access may hamper television signals when they go digital next year. In a test conducted last year by the Federal Communications Commission, wireless devices blanked out digital programming on nearby television sets.

At the heart of the dilemma are so-called white spaces, the chunks of unused bandwidth layered between TV channels that are designed to keep broadcast signals from interfering with one another. These spaces will get even bigger on February 17, 2009, the legally mandated day for TV broadcasts to go completely digital, freeing up more of the airwaves. (Digital signals

take up less airwave space than their analog counterparts.)

Tech companies see huge opportunities in these radio-frequency buffer zones. The slices could allow computers, cell phones and other wireless devices to transfer gigabits of data per second (compared with Wi-Fi's megabit-per-second speeds), thereby supporting mesh networks, broadband

access in remote areas and wireless hot-spots. "You may want to call it Wi-Fi 2.0 or Wi-Fi on steroids," said Rick Whitt, Google's Washington, D.C., telecom and media lawyer, during a recent press conference held to promote the effort. In March, Google filed a petition with the FCC stating its support for white-space-sensing technology—such as that proposed by rival Microsoft. Google's interest in wireless technology stems from the company's desire to promote its open-source Android operating system and software for mobile devices, which Google hopes will be available by this fall.

But broadcasters do not want to invest in a digital infrastructure only to have cell phone and Internet traffic infringe on their channels, essentially making digital TV no more reliable than the analog sets that depended on tinfoil-wrapped rabbit-ear antennas. So before Google and the others can exploit white spaces, they must get permis-



FREQUENCY FIGHT: New wireless services using the buffer-zone frequencies among television channels could interfere with digital broadcasts.