In 2007, before the ordinance went into effect, 93% of employers in San Francisco who would later become subject to the mandate offered health insurance. The mandate went into effect in 2008. By 2009, the percentage of firms offering insurance had increased to 96% in San Francisco. In counties surrounding San Francisco, the percentage dropped from 94% in 2007 to 90% in 2009.

During the same period, there was little difference in the increase in premiums between San Francisco and surrounding counties, but there were significant differences in who paid for those premiums. In San Francisco, employers increased their contributions to premiums by 17%. At the same time, the cost for employees rose only 9%, from $56 to $61. In surrounding counties, the cost of premiums paid for by employees rose by 50%, from $52 to $78. And if not for the ordinance, Colla concluded, another 10% of workers would be without employer-paid health benefits.

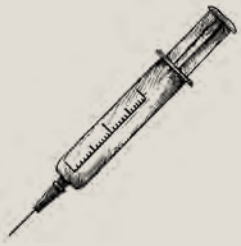
Despite the initial controversy surrounding the policy, including criticism from local business groups and a lawsuit that charged that the mandate was overly burdensome, by 2009 64% of employers supported the mandate, according to the survey.

"I think the main lessons are that it's feasible, that employers support it, and that—most important to us—it actually expanded coverage among workers," Colla says. She adds that these findings might bode well for the implementation of the employer mandate portion of the ACA next year. "I think some of the resistance will die down when employers learn more about it and as it becomes the norm," she says. "I'm hopeful that it will be implemented smoothly. But we'll see."

AMOS ESTY

UNDERSTANDING DIVISION

Writing in the journal *Science*, Geisel researchers from the Department of Biochemistry reported the key role played by the protein INF2 in the division of mitochondria, the organelles that produce energy for cells. The finding may lead to a better understanding of the development of neurodegenerative diseases, such as Huntington's disease and Alzheimer's disease.



HARNESSING A PROTEIN'S POWER

A novel approach to attacking chronic myeloid leukemia (CML) seems to offer the possibility of treating the disease in patients whose cancers are resistant to the standard treatment. In a paper published in the *Proceedings of the National Academy of Sciences*, assistant professor of pharmacology and toxicology Manabu Kurokawa described research he carried out as a postdoc at Duke University. Rather than inhibiting BCR-ABL, the protein that causes the disease, Kurokawa harnessed the protein to force apoptosis in cancer cells. As BCR-ABL is not present in normal cells, this technique could be used to target only cancer cells.

Jon Gilbert Fox

Research led by William Kinlaw was recently put to the test in a clinical trial.

CUTTING CANCER'S FOOD SUPPLY

WILLIAM KINLAW, A GEISEL PROFESSOR OF MEDICINE, has been exploring new ways to prevent cancer cells from making the fat they need to grow and spread.

Working with several other researchers, Kinlaw recently completed a clinical trial that shows how treatment with conjugated linoleic acid (CLA)—a dietary supplement that is sold in health-food stores and used for weight loss—targets key genes involved in fatty acid synthesis, which may significantly reduce the growth of invasive breast cancer tumors. The results of the trial were published in *Breast Cancer Research and Treatment*. According to Kinlaw, this is the first clinical trial to use conjugated linoleic acid as a cancer therapy in patients.

The researchers enrolled 24 women with stage I to stage III breast cancer. All the women took CLA during the 10 to 12 day period between the time they had a biopsy and surgery. The researchers examined biopsy tissue samples (pre-CLA) and surgically removed tissue samples (post-CLA).

The most significant finding was that a gene named *Spot-14*—which cancer cells use to get the fatty acids they need to survive—was suppressed in the patients' tumors after taking CLA for 10 to 12 days. CLA also suppressed Ki-67, a protein found in the nucleus of cancer cells that is commonly used as an indicator of the aggressiveness of a tumor. The reduction of Ki-67 levels seems to indicate that the tumors were becoming less aggressive.

Kinlaw was encouraged by the findings. "I think CLA is probably not going to end up being a drug itself," he says, "but it certainly could be a prototype, and this study is a proof of principle that targeting these pathways in human tumors might be a useful thing to do."

MATTHEW C. WIENCKE