heim. Additionally, injecting cycloheximide blocks motorneuron death due to the removal of a developing limb bud that produces a muscle-derived growth factor necessary for motorneuron survival (*Science*, 13 May, p. 919).

The notion that growth factors prevent the production of killer proteins is not restricted to the nervous system, however. Essentially the same phenomenon appears to occur in insect muscle cells and in rat prostate epithelial cells.

After the *Manduca* moth emerges from its pupal case at the end of metamorphosis, the muscle cells needed for that process die within 36 hours. "These are giant muscle fibers," says Lawrence Schwartz of the University of Massachusetts in Amherst. "The triggers for muscle cell death are hormonal and new genes for killer proteins are expressed when the hormones are withdrawn." James Truman of the University of Washington in Seattle, and Brian Kay of the University of North Carolina in Chapel Hill collaborated in earlier phases of the research and Schwartz is now trying to clone the genes that code for what he describes as "endogenous cell death products."

In their studies of epithelial cell death in the rat prostate gland, Debra Wolgemuth, Zahra Zakeri, and Ralph Buttyan of Columbia University in New York and Richard Lockshin of St. John's University in Jamaica, New York, reported earlier this year that a "reactive cascade" of gene activity is associated with cell death (*Molecular Endocrinology*, volume 2, page 650). The epithelial cells require testosterone, the male sex hormone produced in the testes, as a growth factor and will die within 5 days after a rat is castrated.

The Columbia researchers analyzed specific RNAs at 1-day intervals after castration and noted a significant increase in a particular temporal sequence of RNAs for the cellular oncogenes c-*fos* and c-*myc* and a heat shock protein. Interestingly, the same genes are active in many cultured cell types during proliferation and differentiation. The researchers do not yet know if the increased mRNAs that occur as prostate cells are dying represents increased gene expression or the stabilization of existing mRNAs. But they note that c-*fos* gene expression is linked to the degradation of phosphatidyl inositol, a membrane lipid, and increases in intracellular calcium ions.

Whether similar genetic mechanisms account for nerve cell death following the withdrawal of nerve growth factor is still unknown. Johnson, Oppenheim, and Schwartz, however, are beginning to explore that question.

■ **DEBORAH M. BARNES**

New Active Faults in L.A.

A University of Southern California seismologist has confirmed that two potentially destructive but deeply buried faults run beneath downtown Los Angeles, Beverly Hills, and Dodger Stadium. Egill Hauksson had been working on tracing buried active faults in the bedrock beneath the sediments of the Los Angeles basin when the magnitude 5.9 Whittier Narrows shock struck 20 kilometers east of downtown Los Angeles (*Science*, 8 January, 1988). No fault rupture broke the surface. Analyses of the rupture's seismic waves showed that the fault responsible is 11 to 16 kilometers below the surface. That was all the proof anyone needed to confirm suspicions of there being more to the earthquake hazard than meets the eye.

Hauksson recently completed his analysis of 200 far smaller earthquakes in the Los Angeles basin. About half of those were more or less vertical ruptures clustered about the three major strike-slip faults of the basin. These are vertical faults whose opposing faces slip by each other in a northwest-southeast direction, just as the San Andreas does. But about one-third of the small quakes were of the thrust type, in which a crustal block is shoved over a second block along a steeply inclined fault. These events clustered along two trends, including the Elysian Park fault, a buried thrust fault whose existence had been inferred from geological studies (see map).

The buried thrust faults seem to be capable of generating earthquakes, but to estimate the hazard, seismologists must know how much seismic energy is being stored on the faults and how it will be released. For limits on the energy being stored, Hauksson takes two estimates of the rate at which the motions of the Pacific and North American plates are compressing the basin in the direction of the thrusting. Thom Davis, a Los Angeles consultant, has estimated from studies of deformed sediments filling the basin that the rate of compression is 10 millimeters per year.

On the other hand, geodesists using very long baseline interferometry can only estimate that compression is progressing at 3 ± 6 millimeters per year. Thus, the strain requiring release could be zero, which is unlikely given the seismic evidence and the mountains shoved up around the basin, or it could be in a league with that building on the nearby San Andreas. Hauksson assumes enough energy has been stored to produce a magnitude 7.5 earthquake, whose energy release is equivalent to that of 30 events of magnitude 6.5. To this uncertainty must be added the imprecision inherent in identifying the size of the fault segments that will fail in individual earthquakes, whose magnitude will depend on segment length.

These faults have produced only one damaging earthquake during the 200 years of the historical record, which may be the quiet before the storm. Although emergency response planning has included the possibility of buried faults, the adequacy of current building codes must be reconsidered. ■ **RICHARD A. KERR**