

Immunity and Crises, Large and Small

Women whose marriages have recently broken up show poorer immune system function than do married women. This observation is the latest in a series of reports linking distressing events in a person's life to depressed immune function (SN: 5/24/80, p. 335).

Recent research by behavioral scientist Janice Kiecolt-Glaser and immunologist Ronald Glaser of Ohio State University in Columbus has explored the relationship between marital status and immunity. Earlier epidemiologic studies had indicated that separated and divorced women have increased mortality rates for some diseases.

The Ohio scientists have examined immune function in two groups of 38 women each. The women in one group were married; those in the other group had separated from their husbands during the last six years. The groups were matched for a variety of socioeconomic factors.

On several different measures of how well immune system cells are functioning, the separated and divorced women showed lower responses than the married women, Kiecolt-Glaser reported this week in Philadelphia at the annual meeting of the American Association for the Advancement of Science. Within the group of separated and divorced women, those with a continued feeling of attachment to the husband or ex-husband — whether it was persistent anger or longing — reported greater feelings of depression and showed poorer immune system function.

Among the married women, those who reported dissatisfaction with their marriages showed a poorer response on three out of six measures of immune function than did women who rated their marriages more favorably. The less happily married women also reported more feelings of depression.

Life's stresses do not have to be as great as the breakup of a marriage to af-

fect the immune system. Kiecolt-Glaser also reports immune system changes occurring among medical students during the school year. In five different studies, employing 20 different assays, she and her colleagues have shown a decrease in immune system activity during medical school final examinations.

Among the immune functions suppressed during exams is natural killer cell activity. This activity is thought to be important as a defense against viruses and cancer. In addition, production of interferon, which stimulates natural killer cells, plummeted during final exams. Kiecolt-Glaser reports that she and her colleagues have also found that these periods of stress-related immunosuppression among medical students are associated with episodes of infectious disease.

"The heightened distress regularly found in our medical student samples during examinations is probably quite comparable to that elicited by everyday events that are frequently experienced [by the general population] — for example, the several days of frenzied activity that frequently precede vacations," she says. "If emotional distress in these situa-

tions is comparable to that of medical students during examinations, then similar immunologic changes may be expected."

Among both the separated and divorced women and the anxious medical students, changes in eating and sleeping habits do not explain the observed changes in immune function, Kiecolt-Glaser says.

If distress interferes with immune function, reductions in distress might enhance immunity, the Ohio scientists reasoned. In both medical students and an elderly population, they have observed that relaxation exercises increase measurable aspects of immune function.

"Transient immunosuppression can be produced by heightened and sustained distress," Kiecolt-Glaser concludes. But whether this condition leads to disease depends on factors including psychological resources, prior health and exposure to infectious diseases. She suggests that distress-related immunosuppression has its most important consequences in elderly individuals and others who have preexisting deficiencies in immune function. — J.A. Miller

AIDS research yields hormonal look-alike

Antibodies against a human hormone that stimulates the immune system also inhibit test-tube replication of the virus associated with AIDS, report researchers from the National Cancer Institute in Bethesda, Md., and George Washington University in Washington, D.C. Why antibodies to the hormone would work against the AIDS virus remains to be discovered, but the results suggest a new path to an AIDS vaccine, the researchers write in the May 30 SCIENCE.

The hormone target is thymosin alpha₁, which promotes the activity of helper T cells, the prime victims of the AIDS virus. The researchers suspected a thymosin-AIDS connection because children with a genetic inability to produce thymosin develop an AIDS-like disease. In a computer match-up of the viral and hormone proteins, they found that about half the components along a short stretch of thymosin are identical to an inner-core AIDS protein.

They injected thymosin alpha₁ into rabbits and added the resultant antibodies to a human cell line infected with the AIDS virus. "We found we could protect cells [in culture] by adding the antibody," says Prem S. Sarin of the National Cancer Institute. The researchers

are now searching for a vaccine that will stimulate humans to produce their own antibodies against the AIDS core protein.

AIDS vaccine work has focused predominantly on the proteins that surround the viral core, on the presumption that the "envelope" proteins are more exposed to the immune system. But these outer proteins vary from strain to strain of the AIDS virus, complicating the search for a single vaccine.

In contrast, the core protein — which George Washington's Allan L. Goldstein calls "the Achilles' heel of the virus" — apparently remains stable. "We feel we have solved one of the major obstacles to vaccine development — namely, genetic drift," says Goldstein.

For an immune reaction to occur, the core protein must be exposed to antibodies at some point. This may happen, Goldstein suggests, when the virus injects itself into the cell, or if the antibody enters infected cells, or if the core protein is in the envelope as well.

Whether the structural similarity between the virus and the hormone is simply coincidental or has a functional explanation, Sarin says, remains an open question. — J. Silberner

McTague leaves OSTP

Acting Presidential Science Adviser John P. McTague resigned last week to become executive director of research at the Ford Motor Co. in Dearborn, Mich. Formerly a deputy director of the White House Office of Science and Technology Policy (OSTP), McTague was elevated to the temporary posts of presidential science adviser and OSTP director when George A. Keyworth II resigned last Dec. 31 (SN: 12/7/85, p. 358). OSTP Assistant Director Richard Johnson will assume both posts until a permanent successor for Keyworth is named. □