Immunodeficiency — Continued

Editorial Note: Each reported female patient developed immunodeficiency during a close relationship, including repeated sexual contact, with a male who had AIDS. Patient 1 fits the CDC case definition of AIDS used for epidemiologic surveillance (*1*). Patient 2 does not meet this definition, but her persistent, generalized lymphadenopathy and cellular immunodeficiency suggest a syndrome described among homosexual men (*2*). The epidemiologic and immunologic features of this "lymphadenopathy syndrome" and the progression of some patients with this syndrome to Kaposi's sarcoma and opportunistic infections suggest it is part of the AIDS spectrum (*3,4*). Other than their relationships with their male sexual partners, neither patient had any apparent risk factor for AIDS. Both females specifically denied IV drug abuse.

Epidemiologic observations increasingly suggest that AIDS is caused by an infectious agent. The description of a cluster of sexually related AIDS patients among homosexual males in southern California suggested that such an agent could be transmitted sexually or through other intimate contact (*5*). AIDS has also been reported in both members of a male homosexual couple in Denmark (*6*). The present report supports the infectious-agent hypothesis and the possibility that transmission of the putative "AIDS agent" may occur among both heterosexual and male homosexual couples.

Since June 1981, CDC has received reports of 43 previously healthy females who have developed PCP or other opportunistic infections typical of AIDS. Of these 43 patients, 13 were reported as neither Haitians nor IV drug abusers. One of these 13 females is described in case 1; another four, including two wives, are reported to be steady sexual partners of male IV drug abusers. Although none of the four male partners has had an overt illness suggesting AIDS, immunologic studies of blood specimens from one of these males have shown abnormalities of lymphoproliferative response (*7*). Conceivably, these male drug abusers are carriers of an infectious agent that has not made them ill but caused AIDS in their infected female sexual partners.

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Formaldehyde Exposures in a Gross Anatomy Laboratory — Colorado

A recent study by the National Institute for Occupational Safety and Health (NIOSH) found significant exposures to formaldehyde in a gross anatomy laboratory at a medical school in Colorado (*1*).

In early December 1981, at the request of medical and dental students at the university,

Formaldehyde Exposures — Continued

NIOSH investigators conducted an environmental evaluation of the anatomy laboratory and a medical evaluation of the students using it. They collected 55 personal breathing-zone air samples from students dissecting cadavers (25 for determination of formaldehyde exposure and 30 for determination of phenol exposure). In addition, they performed pulmonary function tests on 23 students.

Results of the environmental sampling showed formaldehyde levels ranging from less than 0.02 mg/M^3 to 3.3 mg/M^3 (0.02 parts per million [ppm] to 2.7 ppm), indicating exposures sufficient to cause symptoms of irritation in most of the exposed students. Phenol levels ranged from less than 0.01 mg/M^3 to 12.2 mg/M^3, all within the NIOSH-recommended maximum occupational exposure level of 19.0 mg/M^3 (*2*).

Pulmonary function tests on the 23 students were normal. However, one student, tested after exposure, showed clinically significant decreases of 13.0% in forced vital capacity and 10.7% in forced expiratory volume in 1 second. This student had a history of adverse reactions to formaldehyde, presumably an allergic basis for these findings. Eleven other students who participated in post-exposure pulmonary function tests showed no significant decreases in pulmonary function.

Reported by the Hazard Evaluations and Technical Assistance Br, Div of Surveillance, Hazard Evaluations, and Field Studies, NIOSH, CDC.

Editorial Note: The sharp odor of formaldehyde can be detected at very low levels (less than 1 ppm). Exposure to concentrations ranging from 0.1 to 5.0 ppm can cause burning of the eyes, tearing, and general irritation to the upper respiratory passages. Low levels (0.3-2.7 ppm) have also been found to disturb sleep and to be irritating to some persons (*3*). Higher levels (10-20 ppm) may produce coughing, tightening in the chest, a sense of pressure in the head, and palpitations (*4-6*). Exposures of 50-100 ppm and above can cause serious injury, including pulmonary edema, pneumonitis, or death (*7*).

Dermatitis due to formaldehyde solutions or formaldehyde-containing resins is a well-recognized problem (*8*). After a few days of exposure, a sudden inflammatory skin reaction may develop on the eyelids, face, neck, scrotum, and flexor surfaces of the arms. Other surfaces of the body may also be involved, sometimes after years of repeated exposure.

Formaldehyde has been shown to induce a rare form of nasal cancer in both Fischer 344 rats and B6C3F1 mice (*9*) and may induce the same type of cancer in Sprague-Dawley rats. Although humans and animals may differ in their susceptibility to specific chemical compounds, any substance producing cancer in experimental animals, particularly in more than one species, should be viewed as a potential cancer-causing agent in humans. Formaldehyde has also demonstrated mutagenic activity in several test systems.

NIOSH recommends that formaldehyde be handled in the workplace as a potential occupational carcinogen (*3*). Safe levels of exposure to carcinogens have not been demonstrated, but decreasing exposure should reduce the possibility of developing cancer. The extent of cancer risk from exposure to formaldehyde levels at or below the current Occupational Safety and Health Administration (OSHA) standard of 3 ppm (*10*) has not yet been determined. As a prudent public health measure, NIOSH recommends that engineering controls and stringent work practices reduce occupational exposure to the lowest possible levels.

To minimize formaldehyde exposure in gross anatomy laboratories, NIOSH recommends the following:

1. Students and instructors should be aware of the potential health hazards of formaldehyde.
2. Persons handling formalin or preparing dilute formalin solutions should wear protective equipment, including rubber gloves, protective aprons, and eye and face protection.
3. Ventilation should provide a minimum of five air changes per hour to help lower formaldehyde concentrations.

Formaldehyde Exposures — Continued

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Acquired Immune Deficiency Syndrome (AIDS) in Prison Inmates — New York, New Jersey

CDC has received reports from New York and New Jersey of 16 prison inmates with the acquired immune deficiency syndrome (AIDS).

New York: Between November 1981 and October 1982, ten AIDS cases (nine with *Pneumocystis carinii* pneumonia [PCP] and one with Kaposi's sarcoma [KS]) were reported among inmates of New York State correctional facilities. The patients had been imprisoned from 3 to 36 months (mean 18.5 months) before developing symptoms of these two diseases.

All ten patients were males ranging in age from 23 to 38 years (mean 29.7 years). Four were black, and of the six who were white, two were Hispanic. Four of the nine patients with PCP died; the patient with KS is alive. All nine patients with PCP also developed oral candidiasis. None of the patients was known to have an underlying illness associated with immunosuppression, and no such illness was found at postmortem examination of the four patients who died. PCP was diagnosed in all nine cases by means of transbronchial or open-lung biopsy, while KS was diagnosed by biopsy of a lesion on the leg.

Evidence of cellular immune dysfunction was present in the nine patients with PCP: eight were lymphopenic, and all nine were anergic to multiple cutaneous recall antigens. An abnormally low ratio of T-helper to T-suppressor cells was present in six of seven patients tested, and in vitro lymphocyte proliferative responses to a variety of mitogens and antigens were significantly depressed or negative in the six patients tested. The one patient with KS had cutaneous anergy and a decreased proportion of T-cells in his peripheral blood. The ratio of T-helper to T-suppressor cells was normal; studies of lymphoproliferative response were not done.

All ten patients reported that they were heterosexual before imprisonment; one is known to have had homosexual contacts since confinement. However, the nine patients with PCP were regular users of intravenous (IV) drugs (principally heroin and cocaine) in New York City

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MORBIDITY AND MORTALITY WEEKLY REPORT

Epidemiologic Notes and Reports

Immunodeficiency among Female Sexual Partners of Males with Acquired Immune Deficiency Syndrome (AIDS) — New York

CDC has received reports of two females with cellular immunodeficiency who have been steady sexual partners of males with the acquired immune deficiency syndrome (AIDS).

Case 1: A 37-year-old black female began losing weight and developed malaise in June 1982. In July, she had oral candidiasis and generalized lymphadenopathy and then developed fever, non-productive cough, and diffuse intestitial pulmonary infiltrates. A transbronchial biopsy revealed *Pneumocystis carinii* pneumonia (PCP). Immunologic studies showed elevated immunoglobulin levels, lymphopenia, and an undetectable number of T-helper cells. She responded to antimicrobial therapy, but 3 months after hospital discharge had lymphadenopathy, oral candidiasis, and persistent depletion of T-helper cells.

The patient had no previous illnesses or therapy associated with immunosuppression. She admitted to moderate alcohol consumption, but denied intravenous (IV) drug abuse. Since 1976, she had lived with and had been the steady sexual partner of a male with a history of IV drug abuse. He developed oral candidiasis in March 1982 and in June had PCP. He had laboratory evidence of immune dysfunction typical of AIDS and died in November 1982.

Case 2: A 23-year-old Hispanic female was well until February 1982 when she developed generalized lymphadenopathy. Immunologic studies showed elevated immunoglobulin levels, lymphopenia, decreased T-helper cell numbers, and a depressed T-helper/T-suppressor cell ratio (0.82). Common infectious causes of lymphadenopathy were excluded by serologic testing. A lymph node biopsy showed lymphoid hyperplasia. The lymphadenopathy has persisted for almost a year; no etiology for it has been found.

The patient had no previous illnesses or therapy associated with immunosuppression and denied IV drug abuse. Since the summer of 1981, her only sexual partner has been a bisexual male who denied IV drug abuse. He developed malaise, weight loss and lymphadenopathy in June 1981 and oral candidiasis and PCP in June 1982. Skin lesions, present for 6 months, were biopsied in June 1982 and diagnosed as Kaposi's sarcoma. He has laboratory evidence of immune dysfunction typical of AIDS and remains alive.

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