viation, and presumably also inhibits the phagocytosis of platelets in the spleen.

**Conclusions.** The authors concluded that IVIG inhibits autoantibody-mediated thrombocytopenia by inducing inhibitory FcγRIIB receptors on macrophages. The balance of inhibitory (FcγRIIB) and activating (FcγRIII) Fc receptors is likely to be a critical factor in the regulation of macrophage phagocytosis. These results suggest that autoantibody-triggered inflammatory diseases could be treated by new medications that inhibit macrophage phagocytosis.

**Reviewer’s Comments.** IVIG is an effective treatment for many diseases caused by autoantibodies, but its utility is limited by high cost, administration by prolonged intravenous infusion, and the occasional occurrence of side effects such as aseptic meningitis and renal insufficiency. This work suggests that new approaches could be developed that would utilize the same protective mechanism as IVIG, but be more easily administered and potentially have fewer side effects. This research also suggests that defects in the FcγRIIB inhibitory mechanism could underlie antibody-mediated autoimmunity. Although additional work is needed to determine whether the findings in this mouse model apply to human disease, these results are cause for optimism.

**HUMAN IMMUNODEFICIENCY VIRUS**

**LYPODYSTROPHY IN HIV-INFECTED CHILDREN IS ASSOCIATED WITH HIGH VIRAL LOAD AND LOW CD4+ LYMPHOCYTE COUNT AND CD4+ LYMPHOCYTE PERCENTAGE AT BASELINE AND USE OF PROTEASE INHIBITORS AND STAVUDINE**


**Purpose of the Study.** Maldistribution of body fat often associated with abnormalities in lipid and insulin metabolism (“lypodystrophy”) has been reported in human immunodeficiency virus (HIV)-infected adults. This study was initiated to determine whether similar abnormalities occur in children with HIV.

**Methods.** Twenty-eight prepubertal HIV-infected children were studied. Total and regional body fat mass was measured by dual energy x-ray absorptiometry (DEXA). Lypodystrophy was defined by both a decrease in arm and leg fat (extremity lypoatrophy) and an increase in truncal fat as measured by repeat DEXA. Baseline and follow-up characteristics of children with an without lypodystrophy were compared and factors associated with lypodystrophy were identified using odds ratios and appropriate statistical analyses.

**Results.** Eight of the twenty-eight children (29%) experienced lypodystrophy as defined. Children with lypodystrophy had significantly higher levels of HIV RNA, and lower CD4+ T-cell counts. Lypodystrophy was associated with the use of protease inhibitors and stavudine (d4T).

**Conclusions.** In this longitudinal observational study, 29% of children experienced morphologically defined lypodystrophy. This finding strongly suggests that children are also likely at risk for elevation in atherogenic lipid levels and insulin resistance as previously described in adults with HIV and lypodystrophy.

**Reviewer’s Comments.** Lypodystrophy is one of several emerging complications seen in HIV-infected children. These complications are likely related to the complex interplay of intrinsic host lipid metabolism, the presence of chronic HIV infection, and the specific drugs used to treat HIV. Lypodystrophy may be associated with significant cosmetic alterations in body habitus and consequently social and emotional disturbances. The long-term impact of the changes associated with lypodystrophy are yet to be defined in children but are likely to involve cardiovascular complications. The improved duration and quality of life of HIV patients is associated with significant long-term metabolic complications.

**POLIO VACCINE SAMPLES NOT LINKED TO AIDS**


**ANALYSIS OF ORAL POLIO VACCINE CHAT STOCKS**


**MOLECULAR ANALYSES OF ORAL POLIO VACCINE SAMPLES**


**Purpose of the Studies.** The origin of human immunodeficiency virus (HIV) has been controversial. It is now generally accepted that HIV is a zoonosis, which occurred when simian immunodeficiency virus specific for chimpanzees (SIVcpz) was transmitted to humans. This likely occurred on several occasions in the past. It has been suggested in the lay literature that chimpanzee kidney cultures may have been used in the preparation of oral polio vaccine stocks used in Africa during the late 1950s and so could have introduced the virus into humans.

**Methods.** Molecular analysis of archived polio vaccines were studied independently by 3 groups.

**Results.** All 3 studies fail to find either chimpanzee components or HIV/SIV sequences in the polio vaccine stocks. Further, macaque monkey sequences were found, demonstrating that the technique for detection was appropriate, and that the source of the kidney cells used for vaccine development were macaque monkeys and not chimpanzees.

**Conclusion.** Polio vaccines did not transmit HIV to humans.

**Reviewer’s Comments.** Human vaccine development is complex, and the use of animal tissue cultures for vaccine production may allow contamination by microbes in the source material. The preparation of oral polio vaccines in the 1950s was done under conditions that would not meet current specifications for purity and safety. In 1999, Edward Hooper promulgated a theory about the transmission of HIV by early batches of oral polio vaccine supposedly grown in chimpanzee kidney cell cultures and tested in Africa. This theory had great social and political implications for the testing of new medical agents in developing countries. The articles reviewed demonstrate the vaccine stocks tested in Africa were not contaminated with HIV, were not prepared in chimpanzee cells, and were not responsible for the epidemic of HIV now ravaging the continent.

**MOLECULAR ANALYSES OF ORAL POLIO VACCINE SAMPLES**

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Pediatrics 2002;110;468

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