In view of the fact that over 33 (in some estimates 43) million people worldwide are infected with HIV-AIDS, this now constitutes the most important infectious disease in which osteoporosis can occur. Recent reports have shown that HIV infection is an additional risk factor for osteoporosis and pathologic fractures.

Immobilisation, gastrointestinal infections, lipodystrophy, hepatitis and hormone deficiencies are all further risk factors for bone loss. Highly active antiretroviral therapy (HAART) has also been shown to accelerate bone loss in HIV-infected patients and is therefore a potent inducer of osteoporosis in these patients. The hypothesis that the systemic activation of T lymphocytes leads to an osteoprotegerin ligand-mediated increase in active osteoclasts and loss of bone may in part explain the interaction of HIV infection and bone resorption. Risk factors such as nutrition, insufficient physical activity and other lifestyle influences also play a part in the skeletal changes listed above. With widespread introduction of treatment to delay progression of AIDS, early attention should be paid to these potential complications, in order to avoid them as much as possible.

Other chronic infections may also influence the bones, especially when the patient’s physical ability is impaired and long-term therapy is required, which may be the case in, for example, tuberculosis. The situation is even worse if drug resistance develops. It has been suggested that adjuvant therapies, such as L-arginine and vitamin D, could stimulate mycobactericidal and immunomodulatory actions against the infection, and thereby shorten the duration of therapy. It is of note that patients with tuberculosis are reported to have deficient vitamin D levels. Studies of the effects of vitamin D therapy in these patients have not yet been reported.