Mannose-binding lectin (MBL) is a plasma protein, which plays an important role in the innate immune system by recognizing pattern recognition molecules in infectious agents' surfaces. Mutations in the coding region of the mbl2 gene affect its serum levels and low concentrations had been associated with recurrent infection and autoimmune diseases. HIV gp120 is extensively glycosylated, and appears to be an excellent target for interaction with MBL. Several reports were carried out to detect a possible influence of MBL in AIDS development and progression, leading to conflicting conclusions. There are few studies describing the possible influence of MBL in HIV vertical transmission. We decided to evaluate the role of MBL gene in HIV mother-to-child infection transmission associating laboratorial and clinical characteristics. Serum plasma and DNA samples from 79 HIV-sera positive and negative children, and their HIV-sera positives mothers were collected. Patients were divided in two groups: HIV-sera positive children (C+) and their mothers (TM) (n=18); and HIV-sera negative children (C-) and their mothers (NTM) (n=61). Hemolitic tests were performed to evaluate the integrity of the classic and alternative pathways. The capacity of formation of the C5b-9 from the complement cascade was evaluated with ELISA. The MBL serum levels and the functional activity of the MBL were verified with ELISA, detected by monoclonal anti-MBL antibodies, and by the C4 consume respectively. mbl2 gene was analyzed by a real-time polymerase chain reaction performed on a LightCycler. No impairment of classical and alternative pathway activation was observed except for the MNT group, witch values of CH50 was higher then the MT group (p<0.01). One HIV positive child showed undetectable CH50 with normal other analyzes. There was no significant statistical difference of the MBL serum level and the MBL function between the groups. It was found a weak correlation between the MBL serum level and the MBL function. The distribution of the MBL haplotypes found was: **NTM**: YA 57.3% (70/122), XA 9.0% (11/122), B 18.0% (18/122), C 13.1% (16/122) and D 2.2% (3/122); **C-**: YA 58% (71/122), XA 13% (16/122), B 16.4% (20/122), C 9.8% (12/122) and D 2.4% (3/122); **TM**: YA 66.6% (24/36), XA 8.3%
(3/36), B 16.6% (6/36), C 5.5% (2/36) and D 2.7% (1/36); C+ YA 69.4% (25/36), XA 2.7% (1/36), B 16.6% (6/36), C 5.5% (2/36) and D 5.5% (2/36) and didn’t show significant difference between the groups. Viral load distribution was not statistically different in the mother’s groups and in C+. The MBL serum level and haplotypes were not different in groups treated and not treated with HAART. Our data did not show correlation between MBL haplotype and HIV mother-to-child transmission.

Keywords: 1. MANNAN-BINDING LECTIN/deficiency 2. MANNAN-BINDING LECTIN/genetics 3. MANNAN-BINDING LECTIN/immunology 4. COMPLEMENT/deficiency 5. HIV ENVELOPE PROTEIN gp120 6. HIV-1 7. VERTICAL DISEASE TRANSMISSION.