CYREX® Laboratories

CLINICAL SPECIFICATIONS

MYCOPLASMAS

Pathogen Type:

Mycoplasma refers to a genus of bacteria that lack a cell wall, which makes them immune to common antibiotics such as penicillin. Array 12 assesses immune reactivity to *Mycoplasma pneumoniae*, *Mycoplasma arthritidis* and *Ureaplasma*.

Associated With:

Arthritis[1, 2]
Stevens-Johnson syndrome[reviewed in 3]
Guillain-Barré syndrome[4]

Known Cross-Reactions: *Acholeplasma laidlawii*;[5,6] brain tissue, lung tissue, cardiolipin[7]

Clinical Significance:

The detection of antibodies to Mycoplasmas indicates the patient has increased risk of lupus, arthritis and anti-phospholipid syndrome. Mycoplasmas rely on the host to provide nutrients since they are deficient in many biochemical pathways. Its cell membrane contains two layers of lipid family lipoproteins, which are highly bio-reactive. The lack of rigid cell wall allows mycoplasma to have direct and intimate contact with the cytoplasmic membrane of the host cell. Mycoplasmas release a superantigen or mycoplasma-derived mitogen (MDM) which is also pathogenic. Mycoplasmas by themselves can cause acute and chronic diseases at multiple sites with wide-ranging complications and have been implicated as cofactors in disease.[8] Predominantly a trigger of respiratory disorders, *Mycoplasma* can contribute to extra-pulmonary conditions. Central nervous system manifestations are the most frequent extra-pulmonary complications including encephalitis, meningoencephalitis, polyradiculitis and aseptic meningitis.[reviewed in 3] Additional extra-pulmonary disorders include Hematological manifestations (autoimmune hemolytic anemia, autoimmune thrombocytopenia and disseminated intravascular coagulation), musculo-skeletal manifestations (myalgias, arthralgias and polyarthropathies), gastrointestinal manifestations may include abdominal pain, vomiting, diarrhea and loss of apetite.[reviewed in 3] Cytokine release due to *Mycoplasma* includes tumor necrosis factor-α, interferon-γ, and interleukins, interleukin-1β [IL-1β], IL-2, IL-4, IL-5, IL-6, IL-8, IL-10, and IL-18.[reviewed in 9]

This array tests for IgG immune reactivity associated with Mycoplasmas. This is not a measurement of acute infection. Equivocal or out-of-range results indicate IgG antibody reactivity to the tested antigen. We tested 288 blood donor sera against *Mycoplasma* antigens at optimal dilution, 13% of these donors were IgG reactive.

References:

1. Cole, et al. Arthritis of mice induced by *Mycoplasma arthritidis*. Ann Rheum Dis, 1976; 35:14-22.
2. Kirchhoff, et al. Pathogenetic mechanisms in the *Mycoplasma arthritidis* polyarthritis of rats. Rheumatol Int, 1989; 9(3-5):193-196.
3. Kashyap, Sarkar M. *Mycoplasma pneumonia*: Clinical features and management. Lung India, 2010; 27(2):75-85.
4. Steele, et al. *Mycoplasma pneumoniae* as a determinant of the Guillain-Barré syndrome. Lancet, 1969; 2:710.
5. Madsen, et al. 1986. Species-specific monoclonal antibody to a 43,000-molecular weight membrane protein of *Mycoplasma pneumoniae*. J Clin Microbiol, 1986; 24:680-683.
6. Cimolai, et al. Immunological cross-reactivity of a *Mycoplasma pneumoniae* membrane-associated protein antigen with *Mycoplasma genitalium* and *Acholeplasma laidlawii*. J Clin Microbiol, 1987; 25(11):2136-2139.
7. Biberfeld. Antibodies to brain and other tissues in cases of *Mycoplasma pneumoniae* infection. Clin Exp Immunol, 1971; 8:319-333.
8. Baseman and Tully. Mycoplasmas: sophisticated, reemerging, and burdened by their notoriety. Emerg Infect Dis, 1997; 3(1):21-32.
9. Waites and Talkington. *Mycoplasma pneumoniae* and its role as a human pathogen. Clin Microbiol Rev, 2004; 17(4):697-728.