



***Pneumocystis jirovecii* DNA detection**

Pneumocystis jirovecii (previously known as *P. carinii*) is an unusual fungus; lacking ergosterol in its cell wall it cannot be treated with conventional anti-fungals and until recently, was thought to be most closely related to protozoal species. Found ubiquitously in the environment, this organism has three developmental stages: the trophic form, the sporocyte, and the spore form. Acquisition is through the airborne route and in a susceptible human host, infection may present as *Pneumocystis* pneumonia (PCP)¹ or less commonly, extra-pulmonary disease. Once inhaled, the trophic form of the fungus attaches to the alveolar type I cell and undergoes proliferation. In healthy individuals this will be asymptomatic, however in immunocompromised individuals, respiratory symptoms may be severe.

Prophylaxis in immunocompromised patients may prevent *Pneumocystis* pneumonia and its use, particularly in patients with AIDS, has been successful in reducing infections.

Asymptomatic colonisation of *P. jirovecii* has been described in immunocompromised patients with primary acute and chronic respiratory disorders including bacterial pneumonia, lung fibrosis, transplant patients and lung edema^{2,3,4}. The detection of *P. jirovecii* in these groups of patients suggests that lung tissue damage may favour colonisation of this organism². Consequently, colonisation may represent a reservoir for person-to-person transmission in these patients^{2,4}.

P. jirovecii cannot be cultured *in vitro* and therefore laboratory diagnosis for many years relied upon cytological staining or immunofluorescent assay. Molecular detection of this organism provides a sensitive and specific alternative for rapid diagnosis, particularly in non-HIV infected immunocompromised patients where symptoms may be less well defined.

Generally, very few organisms are present within the upper respiratory tract and thus lower respiratory tract specimens such as BAL and induced sputa (in conjunction with other clinical indicators) are usually required for the definitive diagnosis of PCP. Detection of *P. jirovecii* in whole blood samples has a high positive predictive value for the diagnosis of infection but reduced sensitivity compared to the analysis of lower respiratory samples which show better sensitivity and higher negative predictive values and may be more appropriate for patients with respiratory symptoms.

Clients may wish to send us accredited specimen types for this assay which are sputum, BAL, and NPA. Other clinically relevant sample types may be tested and reported along with an appropriate caveat stating that the sample type provided is not accredited. EDTA whole blood is a validated but unaccredited sample type.

References

1. Wakefield AE. 2002. *Pneumocystis carinii*. Br Med Bull. Vol 61:175-88.
2. Sing A *et al.*, 1999. *Pneumocystis carinii* carriage in immunocompetent patients with primary pulmonary disorders as detected by single or nested PCR. J Clin. Micro. Vol 37. p3409–10.
3. Sing A *et al.*, 2000. Evaluation of diagnostic value and epidemiological implications of PCR for *Pneumocystis carinii* in different immunosuppressed and immunocompetent patient groups. J Clin Microbiol. Vol 38 (4). P 1461-7.
4. Jarboui M. 2010. Molecular diagnosis of *Pneumocystis jiroveci* pneumonia in immunocompromised patients. Mycoses. Vol 53 (4). P 329-33.