Clin Infect Dis. 2010 Aug 1;51(3):259-65.

Quantification and Spread of *Pneumocystis jirovecii* in the surrounding air of patients with *Pneumocystis* pneumonia.

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Background

For many years, *Pneumocystis* pneumonia was thought to be due to reactivation of latent infection, but several lines of evidence are now in favor a de novo acquisition of the fungus from an exogenous source. An animal reservoir for *P. jirovecii* is improbable because *Pneumocystis* organisms infecting each mammalian species are host specific. These data strongly suggest that *Pneumocystis* infection in humans is an anthroponosis with humans as a reservoir for *P. jirovecii*.

It was clearly established in animal models that host-to-host transmission occurs via the airborne route. These experiments, combined with the fact that *P. jirovecii* has a high tropism for the human lungs, support the hypothesis that acquisition of *P. jirovecii* organisms by humans also takes place via the airborne route. Because *Pneumocystis* organisms multiply at the alveolar surface, they are exhaled during ventilation by infected patients.

P. jirovecii complementary DNA has been evidenced in air samples from rooms of patients with *Pneumocystis* pneumonia, suggesting that the detected airborne stage of *P. jirovecii* was still viable and therefore potentially infectious.

However, information concerning burdens of *Pneumocystis jirovecii* (human-derived *Pneumocystis*) in exhaled air from infected patients is lacking. Our objective is to evaluate *P. jirovecii* air diffusion in patients with *Pneumocystis* pneumonia.

Methods

Patients admitted with *Pneumocystis* pneumonia were prospectively enrolled from 9 January 2008 to 21 July 2009. Air samples (1.5 m^3) were collected on liquid medium with a commercial sampler at 1-, 3-, 5-, and 8-m distances from patients' heads. Air control samples were collected away from *Pneumocystis* pneumoniae patient wards and outdoors. Samples were examined for *P. jirovecii* detection and quantification using a real-time polymerase chain reaction assay targeting the mitochondrial large subunit ribosomal RNA gene.

Results

Forty patients were diagnosed as having *Pneumocystis* pneumonia. Air sampling was performed in the environment for 19 of them. At a 1-m distance from patients' heads, *P. jirovecii* DNA was detected in 15 (79.8%) of 19 patients, with fungal burdens ranging from 7.5×10^3 to 4.5×10^6 gene copies/m3. These levels decreased with distance from the patients ($P<.002$). Nevertheless, 4 (33.3%) of the 12 samples taken at 8 m, in the corridor adjacent to their room, were still positive. Forty control samples were collected and remained negative.

Conclusion

This study provides the first quantitative data on the spread of *P. jirovecii* in exhaled air from infected patients. It sustains the risk of *P. jirovecii* direct transmission in close contact with patients with *Pneumocystis* pneumonia and leads the way for initiating a quantitative risk assessment for airborne transmission to *P. jirovecii*.