Notes on *Pneumocystis carinii*

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A symposium at the June congress of the International Society for Human and Animal Mycology highlighted not only recent advances from research into Pneumocystis carinii *but also major gaps in our understanding of its epidemiology.*

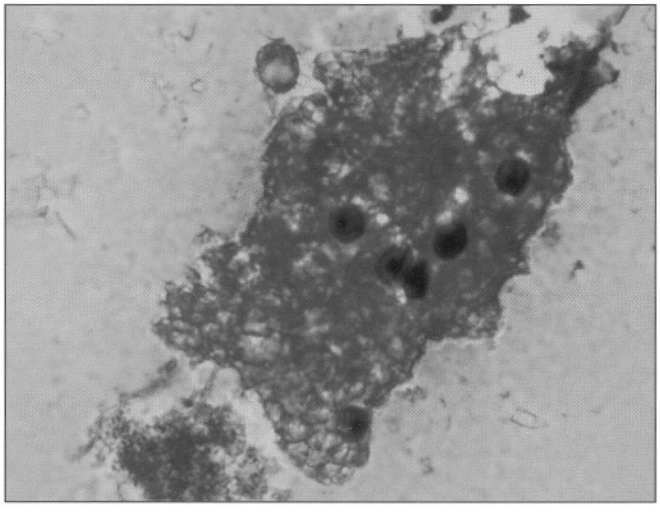
History

Pneumocystis carinii (Figure 1) was discovered in 1909 by Chagas, who mistakenly identified it as a trypanosome. Several years later it was classified as a protozoon and named *P. carinii*, in honour of Dr Carini, an early worker in the field [1].

In the 1960s it became appreciated that *P. carinii* is an important cause of pneumonia in immunocompromised patients. The first Australian case of *P. carinii* pneumonia was diagnosed in the mid-60s in Australia's first heart transplant patient at St Vincent's Hospital, Darlinghurst by Doctor Justin Raby, formerly director of microbiology in our laboratory [2].

Prior to 1988, *P. carinii* was considered a protozoon, an assumption based on the fact that *P. carinii* pneumonia (PCP) did not respond to amphotericin B and could be treated with drugs that were active against protozoa. In 1988, using molecular biological techniques, it was found that a molecular sequence in the small ribosomal subunit encoding RNA was much more similar to fungal sequences than to protozoa [3].

Figure 1. Pneumocystis carinii.



Over the next 5 years, scientific evidence supported the proposal that *P. carinii* was a fungus, and there is now compelling evidence to support the change in classification [4]. However, there is no defined taxon*. The organism has not yet been cultured *in vitro*; hence, its lifecycle is unknown. Pneumocystosis is not classed as a zoonosis, and transmission is by the inhalation of airborne organisms.

P. carinii has been found in samples of ambient air collected in rural Oxfordshire. These air samples were screened for *P. carinii* – specific DNA sequences by DNA amplification. On cloning and sequencing, the majority of recombinants had sequences identical to those of *P. carinii* [5].

Laboratory diagnosis

The diagnosis of *P. carinii* is a major challenge to laboratories. The two factors that play a significant role in its diagnosis are the type of specimen and the staining method used.

Even with the advent of more sensitive diagnostic techniques, such as polymerase chain reaction (PCR), proper specimen collection remains the cornerstone for diagnosis, with the quality and type of respiratory specimen influencing the result. Lung tissue biopsy produces the preferred specimen; however, the majority of those received in this laboratory are induced sputa, bronchial washings and, occasionally, bronchoalveolar lavage (BAL). In the literature, BAL specimens have a sensitivity of over 80 per cent when compared to ordinary brushings or washings, in which the sensitivity is just over 50 per cent. Induced sputa can be examined but the sensitivity depends on the organism burden. This accounts for the wide range of sensitivity, which in the literature ranges from 15 to 99 per cent [6].

Staining techniques commonly used include Gomori's methenamine silver, toluidine blue O – used for the detection of cysts – and a modified Wright-Giemsa to detect trophozoites

and intracystic bodies. (Note that microscopic terminology has lagged behind generic classification.) This laboratory uses the inexpensive Gomori stain and the Wright-Giemsa stain. Since these are non-specific, experience is required to distinguish *P. carinii* from other yeasts. To overcome this problem, the microbiology department is currently evaluating the fluorescent monoclonal antibody stain. Although the fluorescent stain is more expensive it is specific for cell walls, trophic forms and intracystic bodies.

Isolation precautions

Standard infection control precautions are sufficient for patients infected with *P. carinii*. However, consideration should be given to avoiding their placement in the same room as an immunocompromised patient.

Unanswered questions

There are many conundra surrounding *P. carinii*, including the following.

- How genetically similar are the isolates in Australia to those in England?
- What is the distribution of this organism in the environment in Australia?
- What are the biochemical reactions which account for the effectiveness of apparently inappropriate 'antifungal' agents, including co-trimoxazole (trimethoprim – sulphamethoxazole) and pentamidine?

* ***Editor's note***: *'taxon' is the name given to a particular level or grouping in a systematic classification of living things or organisms – taxonomy.*

References

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