Response to “Unfair to ignore long-established line of published research”

Vera L. G. Calich¹ and Adriana Pina

Departamento de Imunologia do Instituto de Ciências Biomédicas, Universidade de São Paulo, São Paulo, Brazil

Unfortunately, Dr. Goihman-Yahr took our last publication in the Journal of Leukocyte Biology [1] as disrespectful to his work on the role of PMN leukocytes in paracoccidioidomycosis (PCM) because his previous articles on the subject were not cited. No offense was intended by not mentioning his important contribution about the interaction between polymorphonuclear phagocytes and Paracoccidioides brasiliensis. The main objective of our paper was to study the immunoprotective and immunomodulatory role of PMN cells in pulmonary PCM developed by genetically resistant and susceptible mice to P. brasiliensis infection [2, 3]. We verified that PMN cells are more important to susceptible than to resistant mice since in vivo PMN depletion at an early phase of the infection induced mortality, more severe lesions, higher fungal loads, and a huge fungal dissemination to organs in susceptible but not in resistant mice. PMN depletion induced only a transitory CFU increase in the resistant strain that had lately recovered their usual resistant behavior. Based on Dr. Goihman-Yahr’s letter, it became evident that he concluded that our results showed PMN cells to be more important to resistant mice, a fact that our data does not support. This misunderstanding probably led him to compare our resistant hosts with normal individuals that, according to his investigation, do not develop overt PCM and present normal ability to digest (and kill?) P. brasiliensis yeasts [4]. His publication [4] also showed that PMN cells from PCM patients—which in his opinion are equivalent to susceptible individuals—presented a defective ability to digest P. brasiliensis yeasts, but a normal ability to kill and digest Candida albicans. This PMN defect, not confirmed by other authors [5, 6], was interpreted as a “specific” failure to P. brasiliensis cells. In another report [7], Dr. Goihman-Yahr’s investigation team found no correlation between the killing and digestive abilities of patients’ PMN cells, and to our knowledge, this particular PMN defect had not been elucidated so far. Thus, it is quite difficult to compare our data with his previous investigations inasmuch as our data led to opposite conclusions on the role of PMN cells in resistance to PCM.

Another important reason for not citing Dr. Goihman-Yahr’s papers can be attributed to the differing objectives of his and our studies. We did not study the fungicidal and digestive ability of PMN leukocytes on fungal cells for our publication in the Journal of Leukocyte Biology [1]. Furthermore, our work showed that neutrophils play a very important role in the innate immunity of susceptible hosts, a conclusion that opposes Dr. Goihman-Yahr’s findings (if we could take the liberty to transpose data from an in vivo experimental model to in vitro studies with human cells). Nevertheless, we would also like to emphasize that Dr. Goihman-Yahr’s report [4] was cited in our previous work which was also published in the Journal of Leukocyte Biology [8] and which aimed to evaluate the activation and killing ability of PMN cells obtained following infection of air pouches from resistant and susceptible mice.

In the last decades, several relevant papers on the function of PMN cells in PCM were published [9–12]. However, the role of these cells in human and experimental PCM is far from being totally understood. More recently, after a period of neglect, this important cell that links innate and adaptive immunity has been the subject of reinvigorated investigations [13] due to its polyvalent ability as modulator of several cell types through its capacity to secret pro- and anti-inflammatory cytokines, reactive nitrogen and oxygen intermediates, hydrolytic enzymes, and microbicidal peptides, among other components and activities [14–18].

We would like to note that the restricted space available to each manuscript in specialized journals, the huge amount of information available, and the allied publication costs may limit the number of references in papers.

We would like very much to express our respect for Dr. Goihman-Yahr and his relevant scientific contribution to PCM; but we would also like to emphasize that we do not believe that we were unfair. We reaffirm the originality of our findings, since the role played by PMN cells in an in vivo model of PCM and the influence of the host genetic pattern in this phenomenon had not been described previously.

REFERENCES


1 Correspondence: Departamento de Imunologia, Instituto de Ciências Biomédicas da Universidade de São Paulo, Av. Prof. Lineu Prestes 1730, São Paulo CEP 05508-900, SP, Brazil. E-mail: vlcalicoh@icb.usp.br
Received December 20, 2006; accepted December 20, 2006. doi: 10.1189/jib.12006740