Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

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Comments on "Impact of bronchoalveolar lavage multiplex polymerase chain reaction on microbiological yield and therapeutic decisions in severe pneumonia in intensive care unit"

Dear Editor,

The article by Sircar et al [1] on the use of multiplex polymerase chain reaction (M-PCR) for therapeutic decisions in severe pneumonia in intensive care unit is very interesting. The article suffers from the following shortcomings.

1) Comparing 2 different M-PCR with standard culture of a nonsterile sample like bronchoalveolar lavage is like comparing apples and oranges. The M-PCR is very sensitive, picks up many organisms including colonizers, does not give the colony-forming unit which is critical to determine if the isolate is significant [2], picks up viruses and anaerobes which are usually not investigated in conventional cultures, and last but not the least, does not pick up pathogens like *Geotrichum*, *Mucor*, *Burkholderia cepacia*, *Serratia* spp, and *Stenotrophomonas maltophilia* mentioned in the study. Therefore, the study design itself is faulty. The authors need to include organisms that are picked up by both the tests to have a head-to-head comparison.
2) *Enterococcus* and *Candida* rarely cause pneumonia in most patients. They are considered colonizers unless concomitantly isolated from blood or histopathologically proven [2,3].
3) The authors have never attempted to define conventional cultures of bronchoalveolar lavage. Does it include anaerobic, viral, fungal, and *Mycobacterium tuberculosis* cultures. They could have briefly described how the isolates were identified and susceptibility testing was done.
4) As expected 70% (41/58) of the samples showed more than 3 organisms in M-PCR. How were the authors able to decide which were pathogens and which were colonizers. In the absence of quantitative/semiquantitative methods, it is very difficult to differentiate between both. The M-PCR will always lead to overdiagnosis and overkill.
5) How was reporting time for conventional cultures determined? Presumptive identification of most of the pathogens like *Escherichia coli, Klebsiella* spp, *Acinetobacter* spp, *Staphylococcus aureus, B cepacia*, *Pseudomonas* spp, *Enterococcus* spp, and so on, can be made in 18 to 24 hours using automated identification systems [4–6]. Sensitivity, however, would take another 12 to 18 hours, which was not the objective of the study. Therefore, reporting time for cultures should be till identification and not till generation of susceptibility report. The authors have reported a very high reporting time of 62.96 ± 26.32 hours, which needs clarification.
6) The authors also fail to explain why M-PCR took 36.41 ± 10.79 hours, which by any standards is too long. That the M-PCR was done in a laboratory which was 2121 km away from the hospital where the study was done needs to be mentioned.
7) Table 1 in the supplementary material lists out the pathogens identified by the 2 M-PCRs. I really doubt if *M tuberculosis* and *Cryptococcus neoformans* were part of the posttransplant infection M-PCR by Xcyton Diagnostics (Bangalore, Karnataka, India). Therefore, I am curious to know how the authors could identify 2 cases of *M tuberculosis* using M-PCR.
8) Moreover, both the M-PCR panels used in this study do not have the RNA viruses like influenza, RSV, rhinoviruses, coronaviruses, human metapneumoviruses, and so on, which predominantly cause pneumonia. Therefore, the authors need to justify why such an expensive test was ordered when it did not pick most of the relevant viruses.

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