Oropharyngeal colonization density of Kingella kingae

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EV (1.7% for both). However, it was significantly lower than observed for RV, RSV, HBoV, AdV and PIV (33%, 17%, 11%, 10% and 9%, respectively) \( (P < 0.001) \). In 17 of 19 (89%) HPeV-positive samples, other respiratory viruses were codetected. The frequency of HPeV codetection was substantially higher than for other respiratory viruses (RV, RSV, PIV, hMPV and EV) \( (P < 0.05) \). Furthermore, codetection frequencies with RSV and AdV were significantly higher than for HPeV-negative cases \( (P < 0.05) \).

HPeV were detected in children aged between 3 months and 4.5 years (mean age, 1.2 years). HPeV type 1b was the most frequent \((12/19, 63\%)\), followed by HPeV-6 \((6/19, 32\%)\) and HPeV-3 \((1/19, 5\%)\). The mean age of HPeV–infected children was significantly lower \((7.25 \pm \text{standard deviation} 2.59 \text{months})\) than in patients infected by HPeV-6 \((27.83 \pm \text{standard deviation} 16.15 \text{months})\) \( (P < 0.00042) \).

Most of the HPeV \((74\%)\) were detected in the autumn and the early winter, with another minor peak in March. Regarding clinical disease association, 11 \((58\%)\) HPeV-positive cases were diagnosed as recurrent wheezing, 5 \((26\%)\) as bronchiolitis, 1 as pneumonia, 1 as upper respiratory infection and diarrhea and 1 as a febrile syndrome. The 2 patients in whom HPeV was detected had no respiratory infection and were diagnosed as sepsis-like illness, where the HPeV-3 association has been firmly established.1

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In conclusion, as with other respiratory viruses, the etiology of HPeV in respiratory diseases is unclear.5 Further studies with more samples, epidemiological and clinical data could improve our understanding of causality between HPeV and childhood respiratory syndromes, as in neonatal sepsis-like illness, where the HPeV-3 association has been firmly established.1

Oropharyngeal Colonization Density of Kingella kingae:

To the Editors:

The pathogenesis of Kingella kingae invasive infections remains unclear, even if there is evidence that K. kingae first colonizes the oropharynx before penetrating the bloodstream and invading distant organs. The density of the oropharyngeal carriage may thus be suspected to play a role in the pathogenesis of invasive K. kingae infections, as it is the case with many other pathogens of the oropharyngeal flora, such as Streptococcus pneumoniae or Haemo-
philus influenzae.2–7 In the current study, we explored whether there was a modification in the density of K. kingae oropharyngeal bacterial load that might modify the risk of invasive infections during the first years of life in children.

We quantified the oropharyngeal colonization density of 117 positive oropharyngeal swabs, analyzed with a rtPCR assay targeting the toxin-encoding gene rtxB. As we have previously demonstrated that there is no difference in the density of oropharyngeal K. kingae colonization between children with osteoarticular infections and asymptomatic carriers,4 throat swabs of all children were considered together. All participants’ parents provided written consent, and the protocol was approved by the institutional ethics committee.

For baseline characteristics, variables are reported as mean ± standard deviation. Pearson coefficient was used to establish correlation between continuous variables.

Between January 2008 and December 2012, 117 children with positive K. kingae rtPCR assays on oropharyngeal swabs were included and investigated in this study (71 asymptomatic oropharyngeal carriers and 46 children with suspected or proven osteoarticular infections due to K. kingae). The mean age of the children was 22.8 ± 10.1 months (range: 8–48 months). The oropharyngeal colonization density was estimated by the number of cycles needed to detect the DNA target. The mean number of cycles for detection was 33 ± 4.5 cycles. The Pearson’s correlation coefficient \( r \) between oropharyngeal colonization density and age of the children was –0.050 \( (P = 0.592) \),

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Letters to the Editor

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