among noncolonized children. The OR among the enrolled case-patients ($OR_{case\text{-}patient}$) becomes r_n/r_c, which is the reciprocal of the risk ratio (RR) of developing the disease ($RR = r_c/r_n$) (online Technical Appendix). Usually, RR is >1 for diseased children and <1 for healthy children. Therefore, even in this independent colonization scenario, $OR_{case\text{-}patient}$ becomes <1 ("pseudo-competitive associations") in diseased children, and $OR_{case\text{-}patient}$ becomes >1 ("pseudo-synergistic associations") in healthy children. This is probably what the authors have observed in the study.

We cannot infer an association of multiple bacterial colonizations in a population despite an observed association in the diseased (or healthy) children, and this association is widely misunderstood (*2–4*). The authors' discussion regarding a potential emergence of *H. influenzae*, associated with AOM, after the introduction of pneumococcal conjugate vaccine is thus unjustifiable.

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In Response: The point made by Suzuki et al. (*1*) is an interesting one, but it is not directly relevant to our conclusions (*2*). To summarize, given bacterium 1 and bacterium 2, suppose OR[pop] is the odds ratio (OR) between bacterium 1 and bacterium 2 in the population. If r[c] and r[n] are the risk of enrollment among colonization-positive and colonization-negative children, respectively, then OR[case] = (r[n]/r[c]) OR[pop], so that OR[case] <1 can be attributed to a higher risk among colonized children.

The underlying assumption of the analysis is that enrollment risk is constant for both bacteria (alone or in combination). In fact, differential risk does exist, and it cannot be separated from the issue of the relative aggressiveness of the bacterium. This can be seen by considering the ORs in the Table.

When we compared the ORs between bacteria pairs for all subjects and children with acute otitis media (AOM), we found a large decrease for pairs involving nontypeable *Haemophilus influenzae* (NTHi), in contrast to the remaining pair, *Streptococcus pneumoniae*/ *Moraxella catarrhailis* (*Spn*/*Mcat*). We do not believe that these values are explainable by the effect described by Suzuki et al., especially when (as is done in that analysis) we assume a colonization-positive risk of enrollment r[c] that is independent of bacterium distribution. We also point out that these estimates are instructive, but not sufficient, because each pairwise comparison may depend on interactions with the third bacterium. We therefore used a statistical model (*2,3*) that permits the isolation of third-order effects by modeling co-occurrence rates of 2 bacteria while controlling for a third. This allowed us to reach our conclusion, which is primarily concerned with the specific role played by NTHi, and follows from the existence of a pattern in the reported ORs, rather than the absolute value of any single OR.

It is also instructive to examine the colonization rates for AOM versus number of AOM events (nAOM) subjects: p(*Spn*-nAOM) = 0.30; p(*Spn*-AOM) = 0.53; p(NTHi-nAOM) = 0.12; p(NTHi-AOM) = 0.48; p(*Mcat*-nAOM) = 0.36; p(*Mcat*-AOM) = 0.43. As we would expect, colonization rates of each bacterium are higher for AOM patients. What is of interest is that the colonization distribution is different for children with AOM, which suggests that NTHi is more aggressive than other bacteria in some sense, and this effect is made more precise by the statistical model we used. The essential point is that the issue of competitive association cannot be isolated from differential enrollment risks, which is what our analysis reports.

Table. Comparison of the odds ratios between bacterium pairs for all subjects and children with acute otitis media*

	Odds ratios, p values	
Bacteria pair	All subjects	Acute otitis media
NTHi/*Mcat*	0.94, p = 0.6935	0.47, p = 0.0006
NTHi/*Spn*	1.58, p = 0.0004	0.50, p = 0.001
Spn/*Mcat*	1.71, p<0.0001	1.53, p = 0.05

*NTHi, nontypeable *Haemophilus influenzae*; Mcat, *Moraxella catarrhailis*; Spn, *Streptococcus pneumoniae*.

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Laboratory-acquired Buffalopox Virus Infection, India

To the Editor: In India, buffalopox virus (BPXV), a variant of vaccinia virus, is associated with severe disease outbreaks among buffaloes (*1,2*), cattle (*3*), and humans in contact with these animals (*1,4*). Most human BPXV infections occur in animal attendants and milkers (*1,4*). A similar type of vaccinia virus infection has also been reported from rural areas in Brazil (*5*). We report a case of laboratory-acquired infection with BPXV in a researcher in India. Clinical signs, symptoms, diagnosis, and management of this case highlight the need for observance and enforcement of strict biosafety measures within the laboratory.

A 28-year-old man (researcher) who was freeze-drying BPXV isolates in a laboratory in Hisar, India sustained a cut on his right palm through nitrile gloves by accidental piercing of shrapnel from a broken ampule. The virus being freeze-dried ($10^{5.5}$ 50% tissue culture infectious doses/mL) was isolated from a buffalo in Jalgaon, India, in 2010. The injured site on the palm was immediately cleaned with 70% ethanol and treated with povidone–iodine solution. No untoward reaction was observed ≤2 days postinjury. Erythema appeared at the injury site on postinjury day 3. Subsequently, a small vesicle developed on postinjury day 5 (Figure, panel A). This vesicle progressed into a pustule with a central area of necrosis by postinjury day 7 (Figure, panel B). On postinjury day 9, symptoms worsened (onset of high fever and general malaise and pain at the affected site), and the researcher sought medical care.

Physical examination showed high fever (104°F), unilateral axillary lymphadenopathy, and edema of the palm (Figure, panel C). Amoxicillin (500 mg, 2×/d), cephalexin (500 mg, 2×/d), and analgesic/antipyretic (paracetamol, 500 mg, 2×/d) were prescribed to control secondary complications caused by bacterial infection and pain.

The next day, the entire palm became cyanotic and edema increased (Figure, panel D). The researcher was then referred to a specialty hospital where the lesion was surgically excised on postinjury day 11 (Figure, panel E) under axial block anesthesia. Blood, necrotic tissue, pustular material, and swab specimens were obtained for laboratory examination. Postsurgery treatment included cleaning of the surgical site on alternate days and oral medication (amoxicillin/clavulanic acid, 625 mg; ibuprofen, 400 mg; paracetamol, 325 mg; and rabeprazole, 20 mg) for 5 days.

On postinjury day 19, the surgeon advised the patient to take cefuroxime (500 mg/d for 5 days) and use a topical ointment containing mupirocin to prevent a delay in healing (Figure, panel F). The lesion healed slowly, and by postinjury day 30, thickening and blackening of the skin was observed (Figure, panel G) that extended to a wider area by postinjury day 38, and the skin started to peel off by postinjury day 50. The entire skin of the palm sloughed off with complete healing by postinjury day 85, leaving a 20-mm blackened eschar over the area (Figure, panel H).

Clinical samples were subjected to laboratory examination. Virus was isolated from tissue samples in a Vero cell line during the first passage. BPXV infection was confirmed by PCR amplification of orthopoxvirus-specific A type inclusion gene (552 bp) and a BPXV-specific C18L gene (368 bp) from tissue material and the laboratory-isolated virus (BPXV/Human/Lab/11), according to procedures described by Singh et al. (*6*). Sequences of these 2 genes were submitted to GenBank under accession nos. JN653284.1 and JN653278.1, respectively. Phylogenetic analysis showed 95%–100% nt similarity of the laboratory isolate (BPXV/Human/Lab/11) with other BPXVs from India (online Technical Appendix Figure, wwwnc.cdc.gov/EID/article/20/2/13-0358-Techapp1.pdf). Antibodies against BPXV were detected in patient serum samples by using an indirect immunoperoxidase test and a 50% plaque-reduction neutralization test according to methods reported by Bera et al. (*7*). Serum of the infected patient showed 50% plaque-reduction neutralization test titers of 256 and 512 on postinjury days 11 and 28, respectively. These findings confirmed BPXV infection because the patient had not been vaccinated against smallpox.

Pre-freezing of the glass ampule to –80°C caused a hairline crack in