subjective, so it is understandable that bacterial counts continue to have a role in endpoint definitions. Hopefully, the new endpoints[4] will provide a somewhat more representative picture of a novel drug's true efficacy, although it is already clear that this new picture will stay limited to early effects (the primary timepoint remains 5 days after treatment cessation) and will not capture resistance outcomes.

We urgently need new antibiotics with activity against multiresistant Gram-negative organisms, and cefiderocol holds enormous promise. I too am rooting for this cleverly designed drug and will be thrilled when promise is replaced by even more proof.

I declare no competing interests.

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A case of sepsis in a 17th century man from Porto Ercole

We read with interest the Correspondence by Michel Drancourt and colleagues[1] concerning the identification of a case of *Staphilococcus aureus* sepsis in the skeletal remains of an early 17th century man exhumed at Porto Ercole (Italy). These remains have been confidently attributed by Drancourt and colleagues to Michelangelo Merisi, also known as Caravaggio. We wish to point out several inconsistencies in Caravaggio's identification, place of death, and cause of his death.

Caravaggio did not father children, nor did his brother, Giovanni Battista, who was a Catholic priest.[2] Therefore, no contemporary pre-sumed patrilineal descendants of Caravaggio were available for direct Y chromosome comparison.[1,3] No detailed accompanying data were provided, such as DNA typing methods or biostatistics, which might have supported the identification of Caravaggio through the combination of genetic analysis and surname information, including the actual Merisi–Merisio Y-STR haplotype matching that was obtained from skeletal remains.[1,3]

The presence of high levels of lead in the bones of the 17th century skeleton from Porto Ercole does not support the attribution of the remains to Caravaggio either. From antiquity to the Renaissance, exposure to heavy metals (ie, lead, mercury, or arsenic) through dietary intake and medicinal uses has contributed to absorption of these toxins in bones. Exposure to heavy metals also occurred with the use of pewter and other lead-bearing cooking utensils, tableware, and pottery. Similarly, the use of lead water pipes and ingestion of foods and beverages adulterated with lead-based additives contributed to chronic lead poisoning.[4]

Lastly, historical sources indicate that Caravaggio was assaulted and severely disfigured in Naples in late September, 1609, 10 months before his demise.[2,5] After having recovered, he went back to work and, between Oct 20–24, 1609, and July 18, 1610, he painted several masterpieces, including *David and Goliath* (1610).[1] The hypothesis of a secondary sepsis due to superinfection of healed facial wounds appears, therefore, to be unfounded. Finally, both the place of death (Porto Ercole) and the authenticity of the death register[1,3] are still a matter of debate among art historians.[2]

We agree with Drancourt and colleagues that the presence of *S aureus* and the osteomyelitis lesions in the male skeleton exhumed at Porto Ercole might indicate that this man died of sepsis. However, more focused historical and biological research is needed before these remains are unequivocally attributed to Caravaggio.

We declare no competing interests.

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Author's reply

I thank Antonio Perciaccante and colleagues for their comments on our Correspondence[1] reporting on the remains of a man who died from *Staphylococcus aureus* sepsis, who

we considered to be Caravaggio. Of course, history is always subject to multiple questions and there is always a proportion of uncertainty. Regarding Caravaggio, the conjunction of his description at arrival in Porto Ercole together with the finding of a skeleton that had his size and age, whose date of burial and carbon 14 dating corresponded to that of Caravaggio, and in whom we identified a haplotype Y-STR that is substantially more common in contemporary people with the Merisi surname, are all concordant elements. Genetic relationships do not require direct descendants, and the Y chromosome, except in special conditions, is the marking of the surname, and thus translates into a family relationship.[2,3] Here, the Y chromosome shows a link between the skeleton and the Merisi surname.

The ensemble of evidence leaves little doubt about the nature of the skeleton, except for the hypothesis that another Merisi of the same age, with wound infection resulting in osteomyelitis, and 1·65 m in height was also buried in Porto Ercole at the same time, which seems unlikely statistically. The taste of mystery that surrounds brilliant artists is not satisfied with simple answers. Finally, our hypothesis is that the source of *S aureus* sepsis was an osteomyelitis of the leg, the trace of which was found, and which probably dates from an older wound. To conclude, like the investigation of cold cases in general, there is always some uncertainty, but the data are so convergent that outside the principle of doubt that animates any scientist, it is difficult not to conclude that this was indeed Caravaggio's skeleton.

I declare no competing interests.

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For Correspondence by Petit and colleagues see *Lancet Infect Dis* 2018; **18:** 1310

Scabies outbreaks in care homes for the elderly

Authors' reply

Antoine Petit and colleagues agree with a major finding of our prospective study:[1] diagnosing scabies in elderly people is challenging because clinical presentation differs from textbook descriptions. However, they express concern about rates of mite visualisation by dermatoscopy and confirmatory microscopy of skin scrapings. We reported positive dermatoscopy in seven (11%) of 61 individuals diagnosed with scabies, two of which cases were crusted. Three (43%) of seven had positive microscopy (done 1 day after sampling), including one crusted case. Thus, most diagnoses were based on clinical signs rather than visual confirmation of mite presence, a stated limitation.

As we did, Petit and colleagues contrast our study with two others.[2,3] A look at these studies' characteristics might in part explain differences in visualisation rates, whilst results show they are not as different as implied regarding confirmatory microscopy of positive dermatoscopy. Petit and colleagues cite their hospital-based study[2] of dermatoscopy and immediate skin scraping microscopy in individuals (mean age 33 years, SD 18) suspected of having scabies by a dermatologist. In 122 (51%) of 236 patients, dermatoscopy took more than 5 min. Despite this, 35 (26%) of 133 patients with negative microscopy on initial scraping had positive dermatoscopy. Walter and colleagues[3] did a study in Brazil (median age 14 years, range 2–72). Dermatoscopy was limited to 5 min duration and microscopy was done within 3 h. 18 (51%) of 35 dermatoscopy positive individuals diagnosed with scabies had negative miscroscopy. Our study's proportion was four (57%) of seven dermatoscopy positive cases. A further 39 (53%) of 74 dermatoscopy positive individuals were deemed to be false positives by Walter and colleagues. The populations, settings, methods, and limiting factors in both studies were very different to our study of scabies outbreaks in care homes for elderly people (median age 87 years, IQR 82–92; 157 (68%) of 230 had dementia; 22 (10%) of 230 were bedbound). Time available to do dermatoscopy influences results. Unfortunately, very prolonged skin and dermatoscopy examinations are not always possible in this population during outbreaks.

Our case definitions attempted to define infestation likelihood as definite, probable, or possible. These case definitions appeared to do well because at scabies treatment follow-up, all but three examined cases had improved. The definitions are further supported by their similarity to subsequently developed international consensus criteria.[4] Our work demonstrates how seriously we believe in careful, thorough clinical examination and we emphasised that the value of dermatoscopy in outbreaks needs further assessment, including predictive values affected by prevalence of infestation.

The role of mass treatments for scabies outbreaks needs further research. We agree with Petit and colleagues that treating only those infested would be ideal. Yet, without robust diagnostic tests, identification of all such individuals is unlikely, some of whom should be expected to be asymptomatic. Our study, and other evidence, suggest piecemeal or delayed approaches to scabies outbreaks in care homes prolongs transmission.[5,6]