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.

We declare that we have no conflicts of interest.

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Is MERS another SARS?

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In September, 2012, two fatal cases of a novel coronavirus (CoV) infection were reported: a Saudi patient who was diagnosed in Saudi Arabia, and a Qatari patient who was diagnosed in the UK.[1,2] Symptoms of this transmissible respiratory disease—known as Middle East respiratory syndrome (MERS)—are severe. In *The Lancet Infectious Diseases*, Abdullah Assiri and colleagues[3] provide a clinical synopsis of 47 cases of MERS-CoV infection identified between September, 2012, and June, 2013, in Saudi Arabia. This work enables us to compare MERS with severe acute respiratory syndrome (SARS), at least from a clinical perspective.

Almost all individuals with MERS-CoV infection had fever as the main symptom on admission. However, occurrence of fever is not surprising in (mostly) self-reporting patients; in studies of the clinical features of SARS, with a few exceptions, equivalent selection biases were noted.[4,5] Furthermore, similar to SARS, only a few people with MERS had upper-respiratory-tract symptoms such as sore throat and rhinorrhoea, providing a means to discriminate MERS from the common cold in adults.

A striking difference to SARS is the high rate of underlying comorbidity in patients with MERS. A virus not yet fully adapted to human infection might be more likely to cause illness in people weakened by pre-existing disease. However, caution is necessary when interpreting comorbidity data, because we should compare rates in affected patients with those in the exposed population. In a study of more than 6000 adults attending an outpatient department in Riyadh, 30% had diabetes overall, including 63% of those older than 50 years.[6] In Assiri and colleagues' report, 32 (68%) of 47 patients (most of whom were older than 50 years) had diabetes, a prevalence that does not seem high in view of the background rate. Furthermore, about half the patients included were from an outbreak centred around a haemodialysis unit.[7] The rates of chronic kidney disease (49%) and hypertension (34%) noted would, therefore, be expected in this overall context. Since community-based studies are unavailable for comparison, we have no reason to regard MERS as a disease restricted to people with underlying disorders.

An unfortunate finding from Assiri and colleagues' study is the rapid progression to respiratory failure and intubation in individuals with MERS, occurring about 1 week after onset of symptoms, up to 5 days earlier than in SARS.[5] This finding accords with the high rate of haemoptysis seen in patients with MERS, suggesting severe lung injury. Data of a preliminary infection study in lung explants indeed indicate that MERS-CoV reaches higher replication levels and shows broader cell tropism in the lower human respiratory tract than does SARS-CoV.[8] Even capillary endothelial cells of the lung became infected. Post-mortem analyses and further experimental studies are needed to understand why

MERS-CoV can induce lung failure so rapidly and with such severity.

Use of recommended real-time RT-PCR assays[9,10] in Assiri and colleagues' study means we can compare virological features of MERS-CoV infection between cohorts. This situation is much different from the SARS experience, during which the diversity of home-brewed RT-PCR formulations made cross-study comparisons very difficult. A key question is whether we can use upper-respiratory-tract samples for RT-PCR-based diagnostics? On the basis of our experience,[9,10] we would judge eight of the 37 upper-respiratory-tract samples obtained in Assiri and colleagues' study difficult to interpret, namely those with a cycle threshold (Ct) value greater than 37 (Ct is a technical surrogate for viral load; higher numbers indicate lower viral loads). This rate is better than that noted in patients with SARS: only a third of laboratory-confirmed cases yielded virus in upper-respiratory-tract samples.[5] The values reported by Assiri and colleagues also show that viral loads in the upper respiratory tract are higher than in patients with MERS who were treated overseas and, thus, who were investigated later in the disease course.[2,11,12] Of note, in seven of 37 upper-respiratory-tract swabs (Ct <24), we estimate that viral loads could exceed five million viral genome copies per sample, implying efficient viral shedding from the upper respiratory tract in only some patients, as assumed in mathematical projections of MERS epidemics.[13]

To ascertain relevant data for MERS epidemiology, we need to develop serological assays using samples from well defined groups of patients, such as described here. Population-based antibody testing could establish the extent of MERS-CoV infection, instead of only seeing the tip of the iceberg represented by cases admitted, such as those summarised in this important paper.

Christian Drosten
Institute of Virology, University of Bonn Medical Centre, Bonn, Germany
drosten@virology-bonn.de

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Cephalosporin resistance in gonorrhoea

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In *The Lancet Infectious Diseases*, Catherine Ison and colleagues[1] describe recent dissemination (2007–11) of the *penA* mosaic gene in *Neisseria gonorrhoeae* in England and Wales, particularly among men who have sex with men. Their findings indicate a creeping resistance to cefixime—the former first-line treatment for gonorrhoea—and much slower development of ceftriaxone resistance, both with breakpoints of 0·25 mg/L. This decreased susceptibility has been reported previously in the USA,[2] most typically in men who have sex with men and with similar relation to the mosaic *penA* resistance gene. Isolated cases of resistance to these drugs have also been recorded in France and Spain.[3] In Japan, although cefixime was not used as standard treatment until the mid 2000s, a sporadic case of resistance was noted in a man in 2001.[4] However, in general, the prevalence of ceftriaxone