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China bans colistin as a feed additive for animals

Last year, the first mobile mechanism of colistin resistance, termed MCR-1, was reported in China.[1] Subsequently, MCR-1 has been reported in more than 30 countries or regions, spanning four continents.[2-4] Jianzhong Shen and colleagues,[5] in a retrospective study, reported that MCR-1 was present in *Escherichia coli* as far back as the mid-1980s, and suggested that the use of colistin in animal feed has probably accelerated the dissemination of MCR-1 in animals and, subsequently, human beings. The predominance of MCR-1 on farms and in livestock, compared with in normal human flora and hospital infections, supports this theory (unpublished data).

Previous reporting of antibiotic resistance mechanisms, such as NDM-1, has, rightly or wrongly, caused international consternation.[6] We have engaged with the Chinese Government to forewarn them of the international interest in MCR-1 and any subsequent criticism of China's status quo on the use of colisitin in animal feeds. In early November, 2015, Shen and colleagues drafted a report for the Bureau of Veterinary Medicine, Ministry of Agriculture, to present data on MCR-1, and suggested that the risk assessment of colisitin used in animal feeds should be commenced immediately. The Ministry of Agriculture gave this report serious consideration, and responded positively by hosting two assessment meetings regarding the use of colistin as an animal feed additive. During this period, one of us (TRW) met with officials from both the China National Center for Food Safety Risk Assessment, Ministry of Heath, and the China Institute of Veterinary Drug Control, Ministry of Agriculture, to discuss and exchange ideas on the risks and impact of MCR-1 on both colistin use in animals, and human beings in China. On July 26, the formal Ministry of Agriculture announcement (number 2428) regarding the cessation of colistin as a growth promoter (feed additive) in animals was released, and the mandate in the revised document regarding colsitin use for disease treatment will be put into effect from Nov 1, 2016. This seminal event will lead to withdrawal of more than 8000 tonnes of colistin as a growth promoter from the Chinese veterinary sector, which will be replaced by other non-human antibiotics supplemented by traditional Chinese medicines.[7]

This unprecedented action has been comparatively swift and its implementation is unequivocally anticipated to be immediate and absolute, and will be supported by constant governmental monitoring. This example of a top-down approach is in vivid contrast to other countries' approaches to tackling antibiotic resistance, for which governmental support is at best negligible, if not completely absent, as in the case of the Chennai Declaration.[8]

In light of the rapid international reporting of MCR-1, in June, 2016, the European Medicines Agency (EMA) re-evaluated their advice on the use of colistin in European veterinary practices and forwarded a position paper.[9] However, although we welcome further discussion on the use of colistin, our data on human carriage and infections is contrary to the EMA's value of 1 for vertical transmission of resistance genes (unpublished).[1] Equally, our data on the coexistence of NDM-1 and MCR-1 is at odds with the value of 1 for coselection of resistance (unpublished). Therefore, we hope that the EMA will deliberate further on these criteria and upgrade both from 1 to 3. To permit the use of colistin for the treatment of individual animals might seem ethically prudent, but the temptation to abuse this ethos for metaphylactic purposes might prove too much in some countries.[10-12] The EMA report clearly indicates that Italy, Spain, and Portugal need drastic reductions in colistin use to meet their new

recommendations (5 mg per population correction unit). Accordingly, in both Europe and China, constant monitoring and implementation will need unbridled political commitment.

The global media was predictably excitable following the press release regarding the first report of MCR-1 in China,[1] which ensued a worldwide debate on how efforts can be internationally co-ordinated to forebear a headlong plunge into the global abyss of the pre-antibiotic era. The treatment of clinical infections is not only beset by the increasing emergence of pan-drug resistance, but also by a drug discovery pipeline that is woefully depleted. Colistin still remains a vital treatment option for highly resistant Gram-negative bacteria and therefore efforts must be unified and fortified, nationally and internationally, to prolong its clinical longevity.

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A hepatitis B-free generation in China: from dream to reality

In China, chronic hepatitis B virus infection remains a public health threat and is associated with about 60% of cirrhosis cases and 80% of liver cancer cases.[1] Since 1992, the Chinese Government has given high priority to immunoprophylaxis to newborn babies, with the high immunisation coverage in China successfully reducing the infection prevalence rate from 9·75% of the general population in 1992, to as little as 0·32% of children aged 1–4 years in 2014.[2] However, more than 50 000 newborn infants are infected with hepatitis B virus every year and might go on to become chronic virus carriers;[3] a rate related to high failure rates of the immuoprophylaxis (up to 15%) in mothers with a high viral load.[4] To eradicate hepatitis B virus in China, greater efforts should be made to completely prevent mother-to-child transmission (MTCT), one of the most common transmission routes in China.

With the advent of potent antiviral drugs in pregnancy category B, the MTCT prevention strategy of short-term antiviral therapy during the late stage of pregnancy was proposed to reduce virus transmission through maternal-fetal transfusion during the peripartum period or direct contact with infected secretions and blood from the mother during labour. A previous study had demonstrated a linear correlation between maternal hepatitis B virus DNA levels and MTCT rates.[5] Mothers with hepatitis B virus DNA less than 6 $\log_{10}$ copies per mL at birth had the lowest rate of MTCT (0%). However, over the past decade, clinical trials of the use of antiviral drugs for MTCT prevention[6–11] showed inconsistent results. Hence, until now, none of the international guidelines had strong recommendation on the use of antiviral therapies during pregnancy.