Global antibiotic resistance: of contagion, confounders, and the COM-B model

Antibiotic resistance is an inevitable consequence of selective pressure imposed by antibiotic use in human beings, (food) animals, agriculture, and aquaculture. It generally results from overuse and indiscriminate use of antibiotics in developed countries and underuse and misuse of them in developing countries. When a bacterial population is exposed to an antibiotic, susceptible organisms are eradicated whereas resistant ones persist and pass on their resistant genes to their offspring by replication (vertical transfer) or to other species through conjugation, transformation, and transduction (horizontal gene transfer) via mobile genetic elements such as plasmids, transposons, and integrons.1 Antibiotic resistance thus occurs in an individual human being or animal but it is perpetuated by diverse socioeconomic risk factors and maintained within environments as a result of poor hygiene and sanitation and suboptimal infection prevention and control.

In *The Lancet Planetary Health*, Peter Collignon and colleagues2 set out to quantify the relationship between antibiotic consumption and antimicrobial resistance and other potential contributing factors using an ecological (aggregated-data design) analysis. The authors make the distinction between antibiotic consumption as a driver for the evolution of resistance and several governance, socioeconomic, and health-systems drivers that facilitate contagion—ie, the dissemination and subsequent maintenance of antibiotic resistance levels in human health.

Several epidemiological studies have shown that the type and frequency of resistance mechanisms varies between countries, with such differences commonly attributed to the qualitative and quantitative differences in antibiotic use3,4 and exposure in human beings, animals, fish, plants, and the environment. This well accepted causal relationship is confounded by governance, socioeconomic, and health-systems drivers. The authors noted a counterintuitive finding of an inverse correlation between antibiotic consumption and resistance that was partly attributed to the ecological design of the study (ie, its susceptibility to ecological fallacy), the absence of a temporal lag between exposure and outcome, limitations of data sources, incomplete datasets, aggregated indices of antimicrobial resistance, and limitations in the data processing and statistical methods. Nevertheless, they reiterate the need for the engagement of the whole of society for the prevention and containment of antibiotic resistance, requiring behavioural and organisational changes ranging from national governments and policy makers to individual patients and civil society, as articulated in a global action plan on antimicrobial resistance.5 A conceptual framework is presented in a figure in the appendix that aligns the findings of Collignon and colleagues2 to this plan.

Comprehensively addressing antibiotic resistance requires more than just financial investment in adequate human, operational, and infrastructure resources; it requires capability, opportunity, and motivation to elicit behavioural or organisational changes6,7 in long-standing government policies and government and individual practices—as described by the COM-B model. The COM-B model stipulates three pre-conditions for behaviour change: capability (ie, knowledge and skills), opportunity (physical and social), and motivation.6,7 The model covers the behavioural and social sciences, which include, but are not limited to, psychology, anthropology, sociology, economics, and political science;6 sociology, economics, and political science are all pertinent to the contagion of antibiotic resistance. Illustrative examples of mitigating governance, socioeconomic, and health-systems confounders in the context of antibiotic resistance, the global action plan on antimicrobial resistance, and the COM-B model are in a table in the appendix. The prevention and containment of antibiotic resistance clearly requires reduced and rational antibiotic use in tandem with mitigating the risk factors for contagion.

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