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When two worlds (meningococcal and gonococcal) collide

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The pathogenic *Neisseria*, *N. meningitidis* (the meningococcus) and *N. gonorrhoea* (the gonococcus), are genetically very closely related but differ considerably in many other ways. The meningococcus is a leading cause of meningitis and septicaemia, yet harmlessly inhabits the nasopharynx of approximately ten percent of the population. The gonococcus is a sexually transmitted obligate human pathogen that causes gonorrhoea and associated genitourinary diseases. Antibiotic resistance is prevalent among gonococci but relatively rare, thus far, in meningococci. Pathogenic meningococci typically possess an outer capsule, gonococci are acapsulate. Several effective vaccines exist against invasive meningococcal disease, for gonorrhoea there are none. Despite these and many other differences, gonococci occasionally cause invasive disease whilst meningococci have, over many decades, been documented as an occasional cause of genitourinary disease. The meningococci responsible, however, were notably diverse. Since the early 2000s, a particular serogroup C-associated strain of the ST-11 clonal complex (lineage 11.2/ET-15) has caused numerous outbreaks among men who have sex with men (MSM) in several Western countries. Early suggestions of possible adaptation to the genitourinary niche were supported by the discovery that corresponding isolates expressed nitrite reductase enabling growth in the anaerobic conditions of the GU tract. In 2015 a distinct lineage 11.2 strain was reported to be causing urethritis among mainly heterosexual males. This strain had independently acquired an efficient gonococcal nitrite reductase whilst also dispensing with the ability to express a capsule. Despite the lack of a capsule, a gonococcus-like trait proposed to enhance adhesion within the GU tract, this strain was reported to have caused several cases of IMD in the USA. Further concerns arising from this situation include the heightened potential for the acquisition of antibiotic resistance by GU tract-associated lineage 11.2 strains from gonococci. Along with the potential lack of a vaccine against the acapsulate urethritis-associated strain, or even yet-to-emerge GU-associated serogroup B strains, this may pose a particular threat to complement-deficient individuals taking long term antibiotic prophylaxis. Efficient transfer of resistance from GU-associated meningococci to the wider population of invasive meningococci is another worry, as is the potential emergence of novel GU-associated lineages with enhanced virulence/pathogenicity.