The IFNL3 mutation associated with hepatitis C virus is also associated with the severity and frequency of reactivation of orofacial herpes simplex virus type 1 infection, which highlights two major concepts: first, that there are genetic variants that confer susceptibility to multiple pathogens; and second, that there are likely to be genotype-determined comorbidities that have not yet been observed and reported. Influenza A virus is a pathogen of global importance and a genetic polymorphism in IFITM3 is strongly associated with risk of severe influenza in human beings. If the clinical impact of this mutation can be quantified, and genotyping can be made both rapid and cost effective, then patients at high risk of life-threatening disease could be identified early and treated aggressively. The mortality associated with infective endocarditis is substantial and surgery can be lifesaving, but current guidelines recognise that indications for surgery are not supported by strong evidence. Staphylococcus aureus is the most common pathogen causing infective endocarditis and a single nucleotide polymorphism in the IFNL3 gene is associated with a sustained virological response to treatment, and this can be clinically tested to predict response to therapy. By contrast, oncologists successfully and routinely apply knowledge of both patient’s and tumour’s genotypes to allocate patients to ever-smaller groups for whom optimum therapeutic strategies have been found. For example, HER2-positive breast cancer responds to trastuzumab, and lung cancers carrying EGFR mutations respond to oral EGFR tyrosine kinase inhibitors.

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