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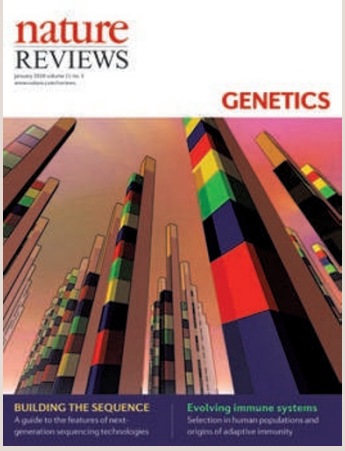

► **COVER:** 'Looming large' by Patrick Morgan, inspired by the Review on p31.


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Selection spares no corner of our genomes — and is visible in what it touches and in what it leaves alone. Articles in this issue highlight the genome innovations produced by selective forces, from 'big bang' genomic reorganizations to subtle nucleotide variation.

Pressure from pathogens shapes genomes on many levels. As Flajnik and Kasahara review on p47, the origin and diversification of our adaptive immune system was made possible by two catastrophic macroevolutionary events, the invasion of the RAG transposon and whole-genome duplications. The evolutionary legacy of our exposure to infectious diseases is also felt in the functional evolution of immune genes themselves, discussed by Barreiro and Quintana-Murci on p17.

The influence of selection on another vast family of genes is the subject of the Review by Lahiry and colleagues (p60). Their genotype–phenotype analysis of human kinases highlights the tissue-specific disease consequences of mutations in different gene regions.

At the level of gene architecture, Licatalosi and Darnell (p75) draw attention to the role of exon–intron structure in the functional diversification of mRNA, and an In Brief article (p7) describes the effect of the little explored phenomenon of intragenic duplications on architecture.

Underlying many of the effects above is selective pressure at individual nucleotides. In this regard, a Research Highlight (p8) reports that a surprising number of human SNPs segregate three alleles; the authors suggest that these alleles arise by a molecular mechanism that creates two new base variants simultaneously.

Studies of selection have been driven by advances in the ability to generate, analyse and compare sequence variation. The data, in turn, generate new hypotheses, and offer the possibility to address new questions.