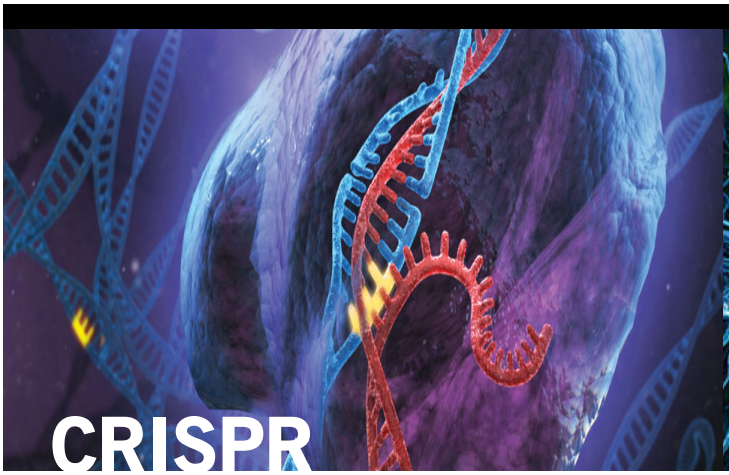

CRISPR

CRISPR GOES VIRAL

JIM XIE

In the 1980s, scientists discovered that bacterial genomes held arrays of repeating DNA sequences with unique intermittent strings of viral DNA. They named this configuration Clustered Regularly Interspaced Short Palindromic Repeats (CRISPR) and later found to it to work in combination with CRISPR-associated (Cas) enzymes to edit viral genetic information.

Since then, scientists have adapted Cas9 enzymes from bacteria to cut and edit the genome of virtually any organism. What makes the CRISPR-Cas9 pair unique is its unrivaled precision, efficiency, and flexibility. Researchers now have an extremely accurate means of targeting and studying particular DNA sequences in vast genomes. Experiments using genetically-engineered organisms that traditionally take a year or more to complete can now be accomplished in just a few months with CRISPR. Furthermore, modifications to CRISPR, such as the disruption of Cas9 enzymes, have opened up expansive paths of study in various subfields such as epigenetics.[1]

CRISPR has already been proven to prevent HIV infection in human cells.[2] Now it is entering clinical trials as a form of immunotherapy for cancer patients.[3] Although CRISPR is currently a mere first-generation tool, it will undoubtedly continue to transform the fields of biology and medicine.

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MULTIPLE SCLEROSIS

S1PR1 MODULATOR AND MULTIPLE SCLEROSIS

DEVIN ROSHAN

Multiple sclerosis (MS) is an autoimmune disorder that destroys the myelin sheath, a critical insulator surrounding axons, in the central nervous system.[1,2] While MS affects more than 2.3 million individuals globally, current therapeutic options are limited.[2,3]

Discovery of the G-protein coupled receptor sphingosine 1-phosphate receptor 1 (S1PR1) evoked interest in the scientific community as a possible therapeutic target for MS and other autoimmune disorders.[4,5] S1PR1 activation has been linked to myelin production, astrocyte migration, neurite outgrowth, and neurogenesis.[4,5] The link between S1PR1 and myelin-producing oligodendrocytes is of particular interest because new oligodendrocytes could potentially reverse early demyelination.[2]

A study recently published in *Lancet Neurology* discusses amiselimod, a novel oral selective modulator of the S1PR1 receptor.[4] In the double-blind phase II clinical trial, patients from Europe and Canada with relapsing MS were randomly assigned to a range of daily doses of amiselimod or placebo for 24 weeks.[4] From weeks 8-24, researchers monitored axonal damage and used advanced magnetic resonance to locate lesions.[4]

Doses of 0.2 mg and 0.4 mg of amiselimod were found to significantly reduce the number of lesions.[3] Amiselimod was safe, well-tolerated, and efficacious, reducing disease activity in a dose-dependent manner.[4] The selective S1PR1 modulator is the first to be investigated for treatment of MS in phase II studies without adverse cardiac events.[4] However, amiselimod warrants further investigation to determine if it is a feasible long-term treatment for MS and other autoimmune-mediated diseases.[4]

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