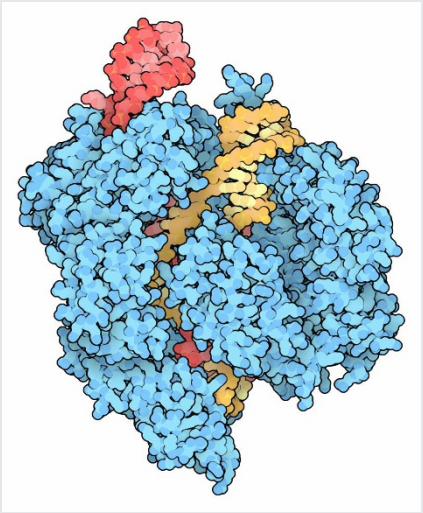
NEWS BRIEFS

Antidotes to CRISPR may help control its actions



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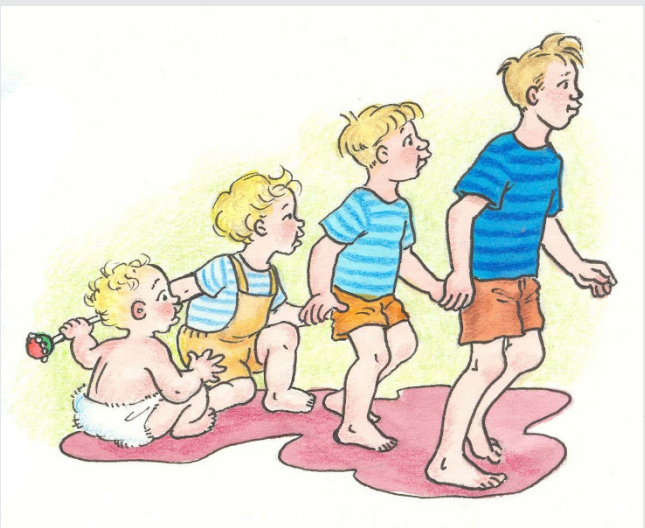
One of the main concerns about gene editing with the CRISPR/Cas9 protein complex has been maintaining control over its actions in unintended targets. Fortunately for scientists, microbes have acquired anti-CRISPR nucleases of their own to defend against its actions. In a series of research studies, investigators independently discovered natural inhibitor proteins in mobile genetic elements that insert themselves into bacterial genomes. Microbiologist Joseph Bondy-Denomy and his colleagues at University of California, San Francisco, reported in the 12 January 2017, issue of *Cell* that they had isolated four different anti-CRISPR proteins that inactivate *Listeria monocytogenes* CRISPR/Cas9. Two of them also blocked the Cas9 variant used commercially for genome editing in bacterial and human cells. In a similar study published 15 December 2016, also in *Cell*, Alan Davidson of the University of Toronto and colleagues reported three families of anti-CRISPR proteins in *Neisseria meningitidis*. Because they potently halt the activity of Cas9, the authors suggest these natural Cas9-specific "anti-CRISPRs" could be developed into tools to regulate genome editing of human cells. In a third anti-CRISPR technique, reported in the 12 December 2016 issue of *Proceedings of the National Academy of Sciences*, David Edgell and colleagues at Western University, London, Ontario, modify the Cas9 protein to precisely control how target DNA gets cut. Genome editing with CRISPR/Cas9 introduces a double-strand break at a target site that can be repaired by the host cell and lead to further cuts. The modified nuclease (TevCas9) helps control breaks by creating cuts that can't be rejoined into new target sites. The hope is that eliminating new target sites will lead to better control over the targeted gene editing process.—*Karyn Hede, News Editor*

Timing matters in genetic risk factors for autism and schizophrenia

Genes involved in communication exert influence over the risk for both autism spectrum disorders and schizophrenia, but at different times during childhood and adolescence, according to a new study. A quantitative analysis of several large genome-wide data sets showed an overlap in genes conferring disease risk, but the risk varied during developmental stages. Genes known to be associated with social communication problems during childhood overlapped with genes conferring risk for autism. Genes affecting social skills conferred increased risk for schizophrenia, but only during adolescence. The genetic risks mirrored the onset of clinical symptoms for each disorder. Researchers plumbed several of the largest publicly available genome-wide data sets for autism and schizophrenia, as well as the Avon Longitudinal Study of Parents and Children (ALSPAC) Genome-wide Complex Trait Analysis. The findings appeared in *Molecular Psychiatry* on 3 January 2017. The finding helps clarify the complex genetics underlying these psychiatric disorders. Overall, the study underscores the fact that these disorders result from the combined influence of small effects involving many genetic variants. Particularly since these genetic risk factors involve communication skills that emerge at different times during development, they exert their influence on disease risk at different times, according to the study. "This study has shown convincingly how the measurement of social communicative competence in childhood is a sensitive indicator of genetic risk," said David Skuse, of University College London, in a media statement. "Our greatest challenge now is to identify how genetic variation influences the development of the social brain."—*Karyn Hede, News Editor*



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