

Perspective

Functional transcriptomics in the post-ENCODE era

1961^{OA}

Jonathan M. Mudge, Adam Frankish, and Jennifer Harrow

Research

The influence of genomic context on mutation patterns in the human genome inferred from rare variants

1974

Valerie M. Schaibley, Matthew Zawistowski, Daniel Wegmann, Margaret G. Ehm, Matthew R. Nelson, Pamela L. St. Jean, Gonçalo R. Abecasis, John Novembre, Sebastian Zöllner, and Jun Z. Li

Derived variants at six genes explain nearly half of size reduction in dog breeds

1985^{OA}

Maud Rimbault, Holly C. Beale, Jeffrey J. Schoenebeck, Barbara C. Hoopes, Jeremy J. Allen, Paul Kilroy-Glynn, Robert K. Wayne, Nathan B. Sutter, and Elaine A. Ostrander

Identifying multiple causative genes at a single GWAS locus

1996

Michael J. Flister, Shirng-Wern Tsaih, Caitlin C. O'Meara, Bradley Endres, Matthew J. Hoffman, Aron M. Geurts, Melinda R. Dwinell, Jozef Lazar, Howard J. Jacob, and Carol Moreno

The shared genomic architecture of human nucleolar organizer regions

2003^{OA}

Ioanna Floutsakou, Saumya Agrawal, Thong T. Nguyen, Cathal Seoighe, Austen R.D. Ganley, and Brian McStay

Disclosing the crosstalk among DNA methylation, transcription factors, and histone marks in human pluripotent cells through discovery of DNA methylation motifs

2013

Phuc-Loi Luu, Hans R. Schöler, and Marcos J. Araújo-Bravo

DNA methylation profiling in human B cells reveals immune regulatory elements and epigenetic plasticity at *Alu* elements during B-cell activation

2030

Anne Y. Lai, Deepak Mav, Ruchir Shah, Sara A. Grimm, Dhiral Phadke, Katerina Hatzi, Ari Melnick, Cissy Geigerman, Steve E. Sobol, David L. Jaye, and Paul A. Wade

Analysis of variable retroduplications in human populations suggests coupling of retrotransposition to cell division

2042^{OA}

Alexej Abyzov, Rebecca Iskow, Omer Gokcumen, David W. Radke, Suganthi Balasubramanian, Baikang Pei, Lukas Habegger, The 1000 Genomes Project Consortium, Charles Lee, and Mark Gerstein

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^{OA} Open Access paper	



Cover A “genetic circuit” represents the complex heritability at a single disease locus. For the past century, it has been widely acknowledged that independently segregating loci contribute to the overall risk of complex diseases, but the possible presence of multiple causative alleles at a single disease locus has been largely unexplored. In this issue, a single GWAS locus for hypertension is shown to harbor multiple alleles that contribute to overall disease risk and susceptibility to end-organ damage. (Cover illustration by Erin Fassbender, erin.fassbender.design, efassbender@sbcglobal.net. [For details, see Flister et al., pp. 1996–2002.]