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Sex has another benefit: It makes humans less prone to disease over time

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Mixing our genes through sex helps purge us of disease mutations

For decades, theories on the genetic advantage of sexual reproduction had been put forward, but none had ever been proven in humans, until now.

Researchers at the University of Montreal and the Sainte-Justine University Hospital Research Centre in Montreal, Canada have just shown how humanity's predispositions to disease gradually decrease the more we mix our genetic material together. This discovery was finally made possible by the availability in recent years of repositories of biological samples and genetic data from different populations around the globe.

What we already knew

As humans procreate, generation after generation, the exchange of genetic material between man and woman causes our species to evolve little by little. Chromosomes from the mother and the father recombine to create the chromosomes of their child (chromosomes are the larger building blocks of genomes). Scientists have known for some time, however, that the parents' genomes don't mix together in a uniform way. Chromosomes recombine frequently in some segments of the genome, while recombination is less frequent in others. These segments of low-frequency recombination will eventually recombine like others do but it

will take many, many generations.

The findings

More specifically, the team of Canadian researchers led by Dr. Philip Awadalla discovered the following: the segments of the human genome that don't recombine as often as others also tend to carry a significantly greater proportion of the more disease-enabling genetic mutations. Until chromosome recombination eventually occurs, these segments accumulate more and more bad mutations. In other words, as far as susceptibility to disease is concerned, our genetic material actually worsens, before it gets better. Thankfully, disease-enabling mutations are eventually shuffled off our genetic code through sexual reproduction. "But since these mutations rest on less dynamic segments of our genome, the process can potentially take many hundreds of generations," explains Dr. Awadalla.

Why these findings are significant

"This discovery gives us a better understanding of how we, as humans, become more or less at risk of developing or contracting diseases," says Dr. Awadalla. It also tells scientists more precisely where to look in the human genome to find disease-enabling mutations, he adds, which should speed up the discovery and identification of mutations associated with specific diseases. Researchers and health authorities will in turn be able to apply this new information to develop more effective treatments and prevention programs.

The science behind the findings

Dr. Awadalla and his team studied the sequenced genomes of hundreds of individuals from Canada's CARTaGENE genetic data repository and

the multinational 1000 Genomes Project. They found that the proportion of mutations associated with disease was significantly higher in low recombining segments known as "coldspots" relative to highly recombining regions, and that the bad mutations in these coldspots were generally more damaging than the mutations in the highly recombining segments.

Through the 1000 Genomes and CARTaGENE programs, the team was able to compare this phenomenon across four present-day population basins: Africans, Asians, Europeans and Canadians of French descent. Each of these genetic groups exhibit the above behaviour to varying degrees. African individuals showed the smallest relative proportion of disease-associated mutations on their genome's coldspots, with Western Europeans showing the largest.

The complete scientific paper was published by *Nature Genetics* and can be found online at DOI: 10.1038/ng.3216" target="_blank">www.nature.com/articles/DOI: 10.1038/ng.3216

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