

CCR5–delta32: a very beneficial mutation

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Cysteine-cysteine chemokine receptor 5 (CCR5) is found in the cell membranes of many types of mammalian cells, including nerve cells and white blood cells.^{1,2} The role of CCR5 is to allow entry of chemokines into the cell³—chemokines are involved in signaling the body's inflammation response to injuries.⁴

The gene that codes for CCR5 is situated on human chromosome 3. Various mutations of the CCR5 gene are known that result in damage to the expressed receptor. One of the mutant forms of the gene is *CCR5–delta32*, which results from deletion of a particular sequence of 32 base-pairs. This mutant form of the gene results in a receptor so damaged that it no longer functions. But surprisingly, this does not appear to be harmful:

“It's highly unusual,” says Dr. Stephen J. O'Brien of the National Institutes of Health in Washington D.C. “Most genes, if you knock them out, cause serious diseases like cystic fibrosis or sickle cell anemia or diabetes. But *CCR5–delta32* is rather innocuous to its carriers. The reason seems to be that the normal function of CCR5 is redundant in our genes; that several other genes can perform the same function.”⁴

Moreover, this mutation can be advantageous to those individuals who carry it. The virus HIV normally enters a cell via its CCR5 receptors, especially in the initial stage of a person becoming infected.⁵ But in people with receptors crippled by the *CCR5–delta32* mutation, entry of HIV by this means is blocked, providing immunity to AIDS for homozygous carriers and greatly slowing progress of the disease in heterozygous carriers.^{6–8}

There has also been research suggesting that *CCR5–delta32* hampers development of cerebral malaria from

Plasmodium infection,⁹ and that it may slow progression of Multiple Sclerosis.^{10,11}

With the advantage of providing full or partial immunity to certain diseases, and with no apparent disadvantages, *CCR5–delta32* can be considered a prime example of a beneficial mutation—a mutation that decreases the information content of the genome and degrades the functionality of the organism, yet provides a tangible benefit.¹²

To date over 10,000 specific disease-causing mutations of the human genome have been identified.¹³ In contrast, only a handful of beneficial mutations have been discovered, none of which involve an increase in genetic information as required by evolution. All this is highly consistent with the biblical account of a very good creation¹⁴ followed by the Fall,¹⁵ and a subsequent six millennia¹⁶ of cumulative physical degeneration.¹⁷ However, it clashes irreconcilably with the evolutionary view that the accumulation of mutations over time brings about upward evolution (increasing functional complexity).

In God's original creation, before the Fall and the Curse, the CCR5 receptor would not have constituted an entryway for pathogens. It may be that infectious agents like HIV only became pathogenic after degeneration from their original ‘very good’ created state. Or it may be that humans did not live in the same environment as such pathogens and so were just not exposed to them. Perhaps both these scenarios apply (see Origin of the Bubonic Plague on p. 7). We look forward to God's promised Restoration, when there will be no more mutation, disease or suffering.¹⁸

References

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18. Revelation 21:4; 22:3