

**The “HIV-retrovirus” and its characteristics: Product of adverse effects of antibiotics, heavy metals, herbicides and pesticides and their mitochondrial toxicity?**

**Jean Luc Montagnier:**

**Twenty five years of research on AIDS Lessons and prospects for cure and vaccination**

[http://nobelprize.org/nobel\\_prizes/medicine/laureates/2008/montagnier\\_slides.pdf](http://nobelprize.org/nobel_prizes/medicine/laureates/2008/montagnier_slides.pdf)

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**Analysis and commentary on Montagnier's scientific publications in regard to the 2008 Nobel Prize in Medicine**

**The Nobel Committee has been made aware of the files on this webpage**

<http://www.theperthgroup.com/montagniernobel.html>

**Felix de Fries**

**AIDS, an autoimmune Reaction: The Role of Heavy Metals and Other Environmental Toxins**

[http://ummafrapp.de/skandal/felix/autoimmun/aids\\_an\\_autoimmune\\_reaction.html](http://ummafrapp.de/skandal/felix/autoimmun/aids_an_autoimmune_reaction.html)

**Regulation of HIV-2 long terminal repeats by Interaction of C/EBP (NF-IL6) and NF-kB/Rel transcription factors**

<http://www.jbc.org/cgi/reprint/271/37/22479>

**DNA damaging agents increase the stability of interleukin-1 alpha, interleukin-1 beta, and interleukin-6 transcripts and the production of the relative proteins.**

<http://www.jbc.org/cgi/reprint/269/21/14899>

**The human immunodeficiency virus type 1 long terminal repeat is activated by monofunctional and bifunctional DNA alkylating agents in human lymphocytes.**

<http://www.jbc.org/cgi/reprint/268/35/26719>

**An NF-kappaB site in the 5'-untranslated leader region of the human immunodeficiency virus type 1 enhances the viral expression in response to NF-kappaB-activating stimuli.**

<http://www.jbc.org/cgi/reprint/271/34/20820>

**Physical and functional interaction of HIV-1 Tat with E2F-4, a transcriptional regulator of mammalian cell cycle**

<http://www.jbc.org/cgi/reprint/277/35/31448>

**HIV-1 Tat induces the expression of the interleukin-6 (IL6) gene by binding to the IL6 leader RNA and by interacting with CAAT enhancer-binding protein beta (NF-IL6) transcription factors.**

<http://www.jbc.org/cgi/reprint/272/23/14883>