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Human endogenous retroviruses and diseases: Pathogens within human genome interplay with environmental factors

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The twentieth century brought efficient preventive and therapeutic solutions to diseases that had long been without effective treatment. Major progresses in medical practice were provided by the discovery of environmental microbes, of the first antibiotic molecule and by the previous demonstration of the efficacy of vaccination. Diseases characterized by complex clinical expression and multifactorial pathogenic mechanisms, most of which have a long-term chronic evolution still remain poorly understood. It appears that the very effective knowledge learned from the environmental microbial causation of infectious disease cannot provide a solution to the equation posed by such diseases. A new category of pathogenic players, normally dormant within human genomes may interact with environmental agents and become pathogenic: human endogenous retroviruses (HERVs). They represent approximately 8% of the human genome and environmental viruses have reproducibly been shown to trigger their expression. The resulting production of envelope proteins from HERV-W and HERV-K families appear to engage pathophysiological pathways that have now been shown to reproduce the pathognomonic features of multiple sclerosis (MS) or amyotrophic lateral sclerosis (ALS). Such proteins act as a common pathway between environmental triggers and downstream physiopathological cascades, which make them potential therapeutic targets representing a unique "bottleneck" upstream multiple pathogenic effects in MS or in ALS. Pathogenic HERV elements activated in human DNA following exogenous infections may thus provide a missing link for the complete understanding of these complex and multifactorial diseases. This presentation will review and give an update on (i) the peculiar genetics of HERVs, (ii) the molecular mechanisms involved in their epigenetic dysregulation and their transactivation by viruses, (iii) the pathogenic effects of abnormally expressed envelope proteins from two HERV families, (iv) their sustained expression in pathognomonic lesions of MS and ALS and (v) ongoing clinical trials targeting these pathogenic drivers.

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