



**Between the Lines of Genetic Code: Chapter  
Seven. Gene-Gene and Gene-Environment  
Interactions in Defining Risk and Spectrum of  
Phenotypes in Idiopathic Inflammatory  
Myopathies**

*Robert G Cooper, Hector Chinoy, Ingrid E. Lundberg*

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The idiopathic inflammatory myopathies (IIMs) represent a complex spectrum of overlapping connective tissue diseases which have skeletal muscle involvement as one of their major components. While recent immunogenetic research has shown that myositis-specific and myositis-associated autoantibodies (MSAs/MAAs) are clearly associated with and thus define many clinical IIM subsets, future research is needed to clarify whether these antibodies (Abs) are just a secondary epiphenomenon, i.e., representing surrogate markers for underlying human leukocyte antigen (HLA) genes and or genotypes, or whether they play some prime role in the immune processes characterizing the IIM. There is increasing evidence suggesting that the IIM disease subtype an individual will get following any environmental triggering event is likely predetermined by their genotype at HLA, and that epistatic genetic interactions in this region critically determine overall disease phenotype, including disease severity and treatment outcomes. It seems clear in inclusion body myositis (IBM), but not yet in other IIM, that HLA gene dosage determines disease severity, again highlighting the importance of epistatic genetic interactions in determining disease characteristics, including outcome. As for rheumatoid arthritis (RA), it appears that HLA genes and the environment interact to modify IIM disease susceptibility. That a specific IIM subtype (necrotizing myopathy characterized by infiltrating macrophages without the usual T and B cells) can be induced in some genetically susceptible individuals by the specific environmental trigger, i.e., the use of certain lipid-lowering statin drugs, suggests that this may represent a potential human model to further study IIM disease-induction mechanisms.

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