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SEX AND GENDER IN SPORT: FALLACY OF THE "LEVEL PLAYING FIELD"

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There is controversy regarding participation in high profile competitive events by female athletes perceived as inappropriately "masculine" by competitors and others. Over 50 years, international sports bodies such as the International Olympic Committee (IOC) and the International Association of Athletics Federations (IAAF) employed - and ultimately discarded - various procedures to ensure feminity. Though ostensibly to detect male imposters, in practice these identified women with genetic Disorders of Sex Development (DSD), such as androgen insensitivity syndrome, complete or partial; 5 alpha-reductase deficiency; mixed gonadal dysgenesis. Further, increased participation in women's sport has vastly enlarged the competitive field and the probability of athletes with a DSD competing.

Do female athletes with a DSD have any "unfair" competitive advantage, especially since specific genetic endowment provides advantage to excel in specific events? This "endowment" can include recognized medical disorders such as height in Marfan Syndrome and delayed maturation and short stature in gonadal dysgenesis, even simple genetic variation such as more fast or slow twitch muscle fibers. A Finnish athlete with exceptional success in endurance Nordic skiing was found to have high hemoglobin and increased oxygen carrying capacity due to an inherited mutation in the erythropoietin receptor; similar mutations occur world wide. Even longer toes are associated with greater "lift-off" and success in sprint events.

Thus the ideal of a "level playing field" is illusory and fundamentally women with some DSDs have no more competitive advantage than other elite athletes with favorable genetic characteristics.

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ANALYSIS OF GENETIC PREDISPOSITION IN LARGE THREE-GENERATION FAMILY WITH MANIFESTATION OF MULTIPLE AUTOIMMUNE DISEASES

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The aim of study was genetic analysis in 24 members of three-generation family strongly affected by coeliac disease (CD). Excepting this one, other autoimmune diseases (AD) such as type 1 diabetes (T1D) and systemic lupus erythematoses (SLE) were diagnosed within the family. The key role of genetic predisposition play alleles encoding the HLA-molecules (HLA-DQA1*0501 and HLA-DQB1*02 encoding HLA-DQ2 molecule, HLA-DQA1*0301 and HLA-DQB1*0302 encoding HLA-DQ8 molecule, both associated with CD and T1D, HLA-DRB1*0301 and HLA-DRB1*1501 associated with SLE). We studied a correlation between presence of HLA-alleles and manifestation of these AD.

Methods: Peripheral blood DNA was used for detection of HLA alleles by the PCR-SSP technique. For statistical verification was used Fisher's exact test with 95% confidence interval.

Results: We confirmed a linkage between HLA-DQ2 molecule and manifestation of CD and T1D ($P=0.0003$, $RR=0.1765$, $CI=0.06318-0.4929$) with the strongest effect of HLA-DQB1*0201 allele ($P=0.0119$, $RR=0.2059$, $CI=0.05966-0.7105$). We have not confirmed HLA-DRB1*0301 and HLA-DRB1*1501 alleles in member with SLE.

Conclusion: The prevalence of CD in family is strongly associated with carriage of HLA-DQ2 molecule. Only one member of the family carrying this molecule has manifested T1D. With regard to preponderant manifestation of CD in family we suppose in this person influence of some other genes shifting manifestation of autoimmunity towards T1D. The unknown genetic factors are probably responsible for case of SLE, thus proving existence of complex genetic predisposition to