

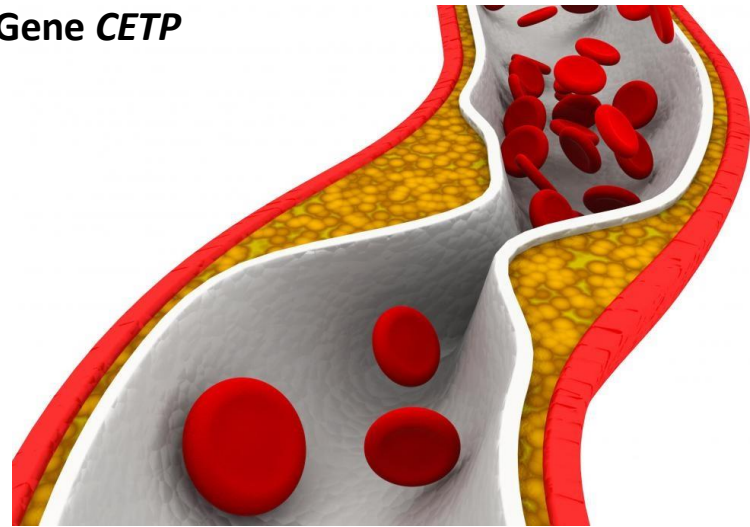
Candidate SNP markers of atherosclerosis reliably altering the affinity of TATA-binding protein for human gene promoters point to stabilizing natural selection as sum of neutral drift spurring atherogenesis and directional natural selection preventing it

Rasskazov D, Chadaeva I, Ponomarenko M, Sharypova E, Drachkova I, Nazarenko[#] M

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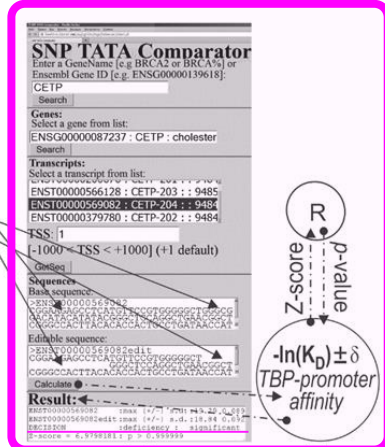
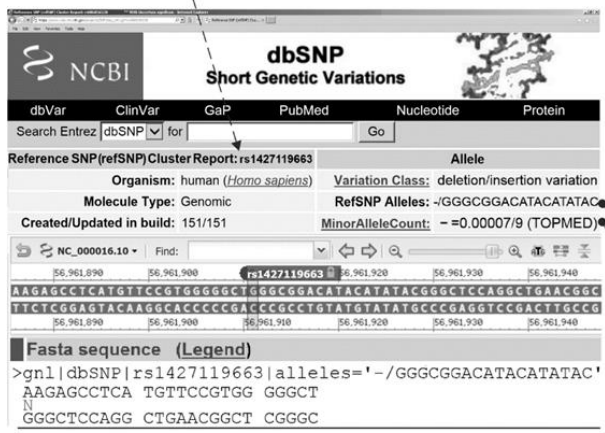
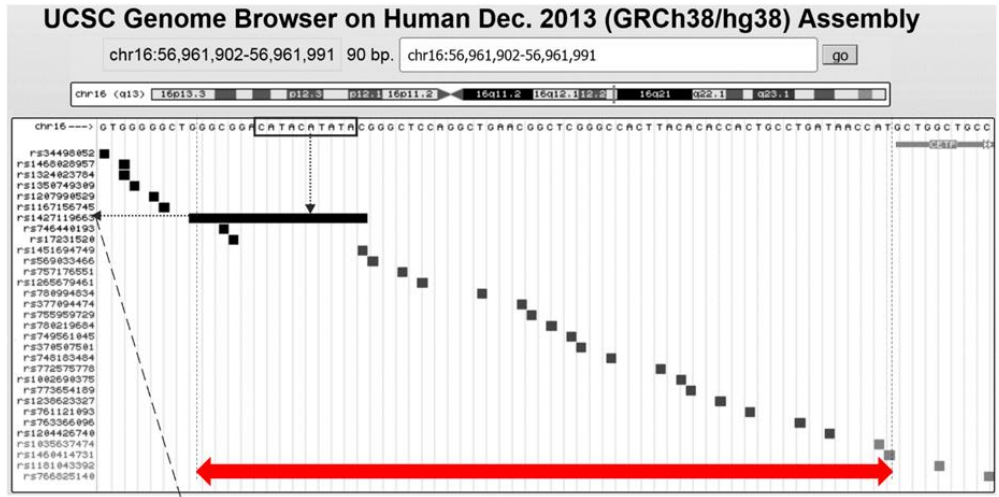
We made a genome-wide prognosis of candidate SNP markers for atherogenesis, because atherosclerosis provokes myocardial infarction and stroke as the two most common causes of death in humans, as resumed by the World Health Organization

Gene CETP



Deficiency → hyperalphalipoproteinemia (atheroprotector)

rs1427119663 causing CETP deficiency is the only clinical SNP-marker of atherogenesis, which locales within TATA binding protein binding site (Plengpanich et al., 2011)



Using the only clinical SNP marker rs1427119663 of atherogenesis we confirmed an adequacy of the candidate SNP markers for this disease predicted by our Web-service SNT_TATA_Comparator (Ponomarenko et al., 2015)

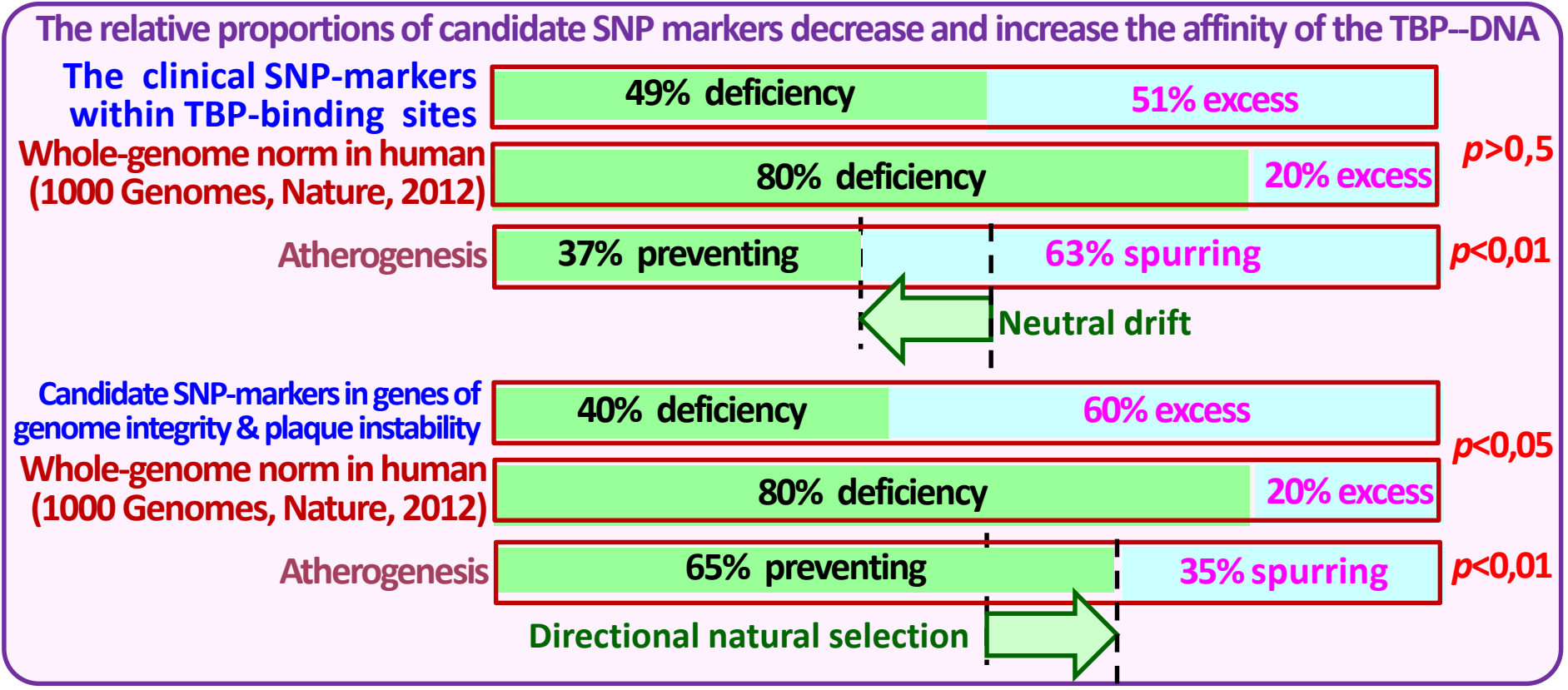
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We tested all 1189 SNPs in question within promoters of 34 atherogenesis-related genes on the human genome that yielded 238 SNPs altering significantly expression of these genes.



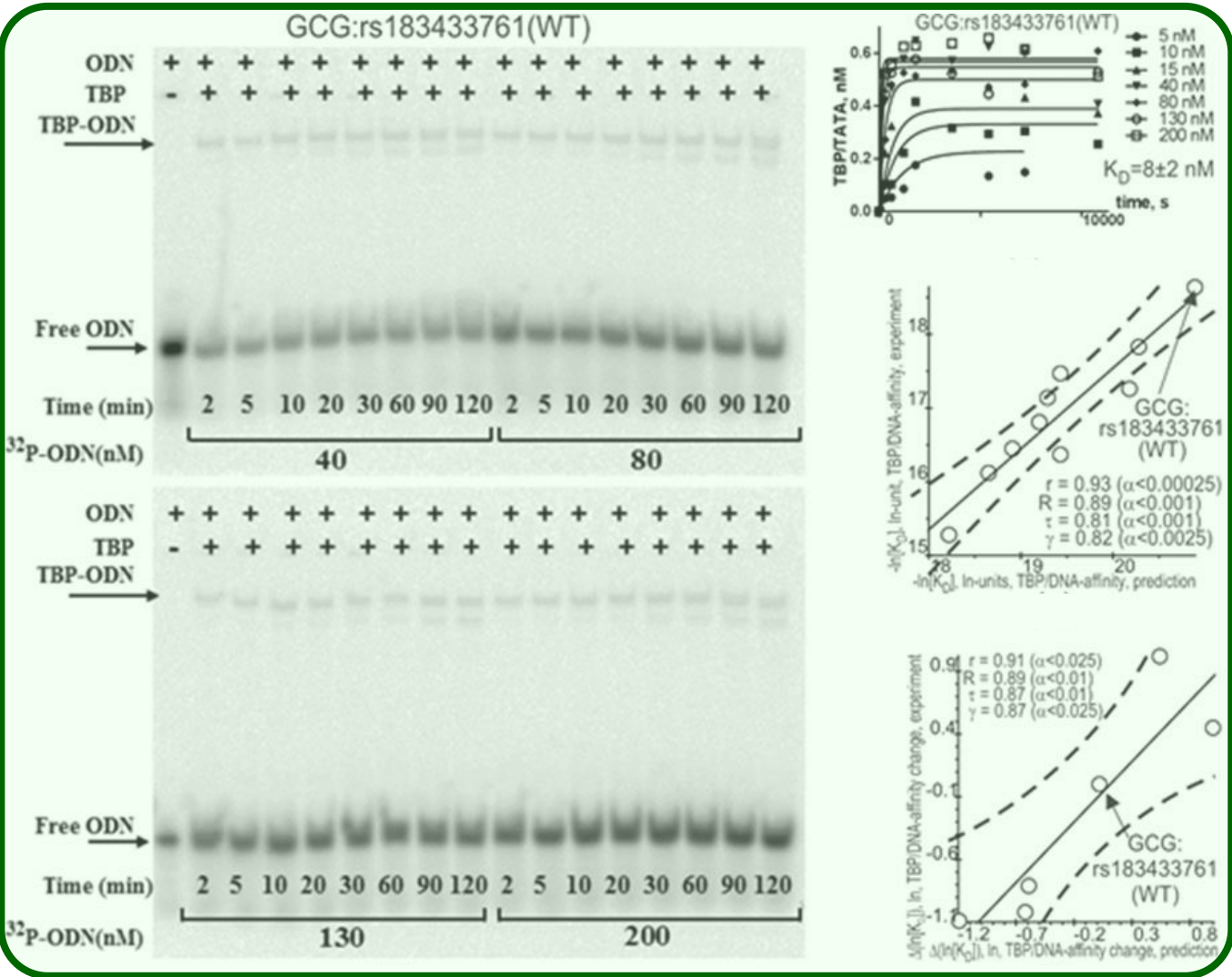
Stabilizing natural selection as sum of neutral drift spurring atherogenesis and directional natural selection preventing it

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Using the EMSA method, we have selectively confirmed our predictions of SNP_TATA_Comparator for the human genes associated with atherogenesis.

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