This week in therapeutics

Indication	Target/marker/ pathway	Summary	Licensing status	Publication and contact information
Autoimmune disease				
Psoriasis	<i>TRAF3 interacting protein 2 (TRAF3IP2; ACT1); inducible nitric oxide synthase 2 (NOS2; iNOS); F-box and leucine-rich repeat protein 19 (FBXL19); nuclear factor of κ light polypeptide gene enhancer in B cells inhibitor α (NFKBIA); proteasome subunit α-type 6 (PSMA6); endoplasmic reticulum aminopeptidase 1 (ERAP1); pituitary tumor-transforming gene 1 (PTTG1); CUB and sushi multiple domains 1 (CSMD1); gap junction protein β2, 26kDa (GJB2); serpin peptidase inhibitor clade B member 8 (SERPINB8); zinc finger protein 816A (ZNF816A)</i>	Four separate genomewide association studies identified susceptibility loci for psoriasis that could help diagnose the disease or lead to new treatments. In a genomewide association study of German patients with psoriasis vulgaris, two SNPs at the <i>TRAF3IP2</i> locus, rs132104247 and rs33980500, were associated with the disease ($p=2.36 \times 10^{-10}$ and $p=1.24 \times 10^{-16}$, respectively). The locus was also associated with psoriatic arthritis. In a genomewide association study in German patients with psoriatic arthritis, rs13190932 at <i>TRAF3IP2</i> was associated with psoriasis ($p=8.56 \times 10^{-17}$). In the third study, a meta-analysis of psoriasis patients from Michigan, Toronto, Newfoundland and Germany showed that rs4795067 at <i>NOS2</i> , rs10782001 at <i>FBXL19</i> and rs12586317 at <i>PSMA6-NFKBIA</i> were significantly associated with psoriasis ($p=4 \times 10^{-11}$, $p=9 \times 10^{-10}$ and $p=2 \times 10^{-8}$, respectively). The loci also were associated with psoriatic arthritis. The final study included psoriasis patients from China and showed that <i>ERAP1</i> , <i>PTTG1</i> , <i>CSMD1</i> , <i>GJB2</i> , <i>SERPINB8</i> and <i>ZNF816A</i> were all significantly associated with the disease ($p < 5 \times 10^{-8}$). Several of the loci also were associated with psoriasis in German and U.S. populations. Next steps could include replicating the findings of each susceptibility locus in larger cohorts.	Patent and licensing status unavailable	<p>Ellinghaus, E. <i>et al. Nat. Genet.</i>; published online Oct. 17, 2010; doi:10.1038/ng.689 Contact: Andre Franke, University of Kiel, Kiel, Germany e-mail: a.franke@mucosa.de</p> <p>Hüffmeier, U. <i>et al. Nat. Genet.</i>; published online Oct. 17, 2010; doi:10.1038/ng.688 Contact: André Reis, University of Erlangen-Nuremberg, Germany e-mail: reis@humgenet.uni-erlangen.de</p> <p>Stuart, P.E. <i>et al. Nat. Genet.</i>; published online Oct. 17, 2010; doi:10.1038/ng.693 Contact: James T. Elder, University of Michigan, Ann Arbor, Mich. e-mail: jelder@umich.edu</p> <p>Sun, L.-D. <i>et al. Nat. Genet.</i>; published online Oct. 17, 2010; doi:10.1038/ng.690 Contact: Xue-Jun Zhang, Anhui Medical University, Hefei, China e-mail: ayzxj@vip.sina.com</p>
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