**CLN8: an Atorvastatin-specific marker for common myalgia**

1Richard Seip, Ph.D., 2Paul D. Thompson, M.D., 3John P. Kane, 3Clive R. Pullinger, 4Alan H.B. Wu, Ph.D., 1Gualberto Ruaño, M.D., Ph.D.  
1Genetics Research Center, Hartford Hospital, Hartford, CT 06106, USA  
2Hartford Hospital, Hartford, CT  
3Cardiovascular Research Institute, University of California, San Francisco, CA  
4Department of Laboratory Medicine, University of California, San Francisco and the San Francisco General Hospital, San Francisco, CA

**Background:** Statin therapy is highly successful for the treatment of hypercholesterolemia and prevention of cardiovascular disease. Side effects including muscle pain (myalgia), weakness and/or increased serum CK activity (myositis) often disrupt treatment, with no unifying hypothesis to explain them. We used genome-wide association to investigate whether genetic links to myalgia may be statin-specific. **Methods:** We genotyped 812 statin-treated patients with an array of 865,483 SNPs. There were 328 patients on atorvastatin, 119 on simvastatin; 154 on rosuvastatin, 213 on other statins. Myalgia index was scored as 1 for myalgia presence in 377 patients and 0 for no myalgia in 416. **Results:** The SNP rs7014327 of the ceroid lipofuscinosis, neuronal 8 (CLN8) gene was associated with myalgia at a significance of \( p < 2 \cdot 10^{-7} \) (\( R^2 = 5.2\% \)) in patients receiving atorvastatin only. In all patients the SNP was unassociated (\( p < 10^{-5} \), \( R^2 = 2.6\% \)). **Conclusion:** We propose a new candidate for myalgia, CLN8, which has an effect only in atorvastatin patients. This drug dependent association supports the hypothesis of statin-specific pathways for statin myopathy.

**Figure legend.** Left: CLN8 genomic locus on chr 8p23 and effect on myalgia (all patients). Log-scores of p values for SNPs within 200 kb of the index SNP (red circle). Right: Effect of the SNP on myalgia index in patient subgroups taking simvastatin, atorvastatin, or rosuvastatin.