Abstract

Acne is the most common skin disorder. While usually transient, severe acne would leave behind disfiguring scars. Acne could be successfully treated, but as most drugs have serious side effects while not preventing scarring, new therapeutic options are needed. Novel insights in acne pathogenesis and potential therapeutic targets could be identified by studying rare monogenetic acne-associated diseases. The Acne and Sebaceous Gland Programme (ASGP) studies acne-associated genes by knocking them out in zebrafish. Being genetically similar to humans, this vertebrate poses a representative model for human disorders. ASGP has created a zebrafish knock-out line for *przl*, a gene that, when mutated, causes an acne-associated syndrome in humans. Adult *przl*$^{\Delta/\Delta}$ mutants portray skeletal abnormalities accompanied by stiff, presumably fibrotic, fins. We hypothesized that this fish could be used as a preclinical model for acne-related scarring. We aimed to document the *przl*$^{\Delta/\Delta}$ phenotype formally and to test whether the fibrosis, i.e., abnormal collagen deposition, is inducible through the wounding of larval and adult zebrafish. Our results showed that the *przl*$^{\Delta/\Delta}$ mutants were smaller compared to their wild type siblings. Furthermore, histological analysis revealed that their fins were indeed fibrotic. Larval tail fins showed high variability in collagen staining intensity without a significant difference between wounded and unwounded fins. Although the effort was made to optimize adult tail fin collagen staining, the optimized protocol failed to produce reproducible results upon wounding. Future studies should explore alternative methods, such as flank wounding, to further test inducibility of fibrosis in the *przl*$^{\Delta/\Delta}$ fish.