formed Embryoid Bodies from the iPSC’s to investigate the expression of markers of craniofacial and eye development in order to understand the potentially shared role of ALX1 in both developmental processes.

**RESULTS:** We identified a missense L165F variant in the homeodomain of ALX1 leading to a loss-of-function of this transcription factor. This mutation was found to be heterozygous in the parents (ALX1+/−) and homozygous in the children born with FND (ALX1−/−). Using CRISPR, we generated mutant alx1−/− zebrafish which exhibited reduced gene expression and anomalies of the craniofacial cartilage of the median palate, corresponding to the frontonasal oblique facial cleft phenotype of the subjects. The alx1−/− zebrafish also formed smaller Meckel’s cartilage, analogous to human micrognathia. We also found that ALX1 mutant zebrafish displayed strong increases in the expression of ALX3 and ALX4a at early developmental stages relative to their wild-type counterparts, likely related to a redundancy of these genes. The reprogramming process into iPSC’s from the patient’s samples as well as those of the father and the control showed no differences pointing toward a role in later development of ALX1. Through the analysis of the EB’s developed from the iPSC’s, we found that ALX1 loss of function resulted in altered expression of genes relevant to neural crest differentiation, as well as PAX6 and its target gene SIX6, both key genes of eye development.

**CONCLUSION:** We found that ALX1 plays a vital role in the development of both facial and ocular structures, through regulation of cranial neural crest progenitors that contribute to facial and eye structures of the frontonasal process. Identifying the genetic basis of developmental disorders such as FND allows for both a deeper understanding of molecular processes that regulate midface development. This work also highlights translation of surgical care to mechanistic discovery, by applying stem cell and CRISPR gene editing approaches, underscoring the unique advantage of craniofacial surgeon-scientists.

---

**The University of Toronto, Toronto, ON, Canada, 2 The Hospital for Sick Children, Toronto, ON, Canada**

**PURPOSE:** The cornea is one of the most densely innervated tissues in the body, and in the absence of corneal sensory innervation, patients develop Neurotrophic Keratopathy (NK). NK is a disease characterized by corneal epithelial breakdown that can lead to progressive corneal scarring and ultimately, vision loss. Corneal neurotization uses nerve autografts to restore sensory innervation in the NK cornea.1,2 In our rat model of NK, we previously demonstrated that corneal neurotization with common peroneal and sural nerve grafts reduces epithelial breakdown, prevents scarring, and improves the rate of epithelial healing in the cornea1. Central corneal epithelial thinning is present in corneal denervation and NK. In the present study, we evaluate the effect of corneal neurotization on corneal epithelial and stromal thickness in a rat model of NK.

**METHODS:** Previously validated models of NK and corneal neurotization were used in this study. The experimental groups were rats with i) NK (negative control) and ii) NK treated with corneal neurotization (treatment), and the control group consisted of rats with normally innervated corneas. In the experimental groups, NK was achieved with stereotactic ablation of the ophthalmic nerve. Four weeks after ablation, affected corneas (n=5 per group) were harvested. Three 10μm cross-sections from each cornea, sampled from representative areas of the cornea, were stained with Hematoxylin and Eosin for analysis. Each section was imaged with bright-field microscopy at 200X magnification. The epithelial and stromal thicknesses of each section were measured using ImagePro. Mean thicknesses were compared using the Kruksal-Wallis test and unpaired t-tests.

**RESULTS:** The central epithelial thickness of the NK corneas (15.83±1.62) was significantly decreased compared to that of the central epithelium in the treated (25.07±3.89, p < 0.05) and normally innervated corneas (24.75±2.46, p < 0.05), the treated and normally innervated corneas not being significantly different. In contrast, the stromal thicknesses were insignificantly different in all three groups.

**CONCLUSIONS:** Corneal neurotization rescues the NK cornea from central epithelial thinning, suggesting the reinervating axons of the inserted graft restore corneal epithelial integrity. We are pursuing further research with the same rat models of NK and corneal neurotization to elucidate the underlying mechanisms.
Change is Happening: An Evaluation of Gender and Race Disparities in Academic Plastic Surgery

Brandon T. Smith, MS, Francesco M. Egro, MBChB, MSc, MRCS, Carolyn P. Murphy, BA, Alex G. Stavros, BA, Elizabeth M. Kenny, BS, Vu T. Nguyen, MD

University of Pittsburgh Medical Center, Pittsburgh, PA, USA

PURPOSE: Gender and race disparities in academic plastic surgery are known, but in recent years a change in culture has been promoted by professional societies. This study aims to evaluate the effects of these changes at faculty and leadership positions.

METHODS: A cross-sectional study was conducted in June 2018 to evaluate minority representation among academic plastic surgery faculty of programs accredited by the Accreditation Council for Graduate Medical Education. A search of the ACGME program listings identified current accredited plastic surgery training programs (n = 140). Institutions identified to have either an integrated residency or an independent plastic surgery fellowship or both were unified under a single listing with duplications removed (n = 100). Corresponding institution websites were located for all but one of the listed programs (n = 99). Faculty directories on these websites were utilized to identify the cohort of the study, which included clinical, adjunct, and tenure- and non-tenure-track plastic surgery faculty belonging to each institution. Faculty without plastic surgery training, emeritus professors, and research faculty without medical degrees were excluded from the analysis. Online faculty profiles, Doximity, LinkedIn, private-practice, and public records websites were used to obtain additional faculty data. Group comparisons were made using student’s t-test and Chi-squared test, where appropriate.

RESULTS: Overall, women represented 19.8% of all academic plastic surgeons. Female academic plastic surgeons were significantly more likely to be an assistant professor [OR:2.19, (95%CI:1.58–3.05)], and significantly less likely to be a full professor [OR:0.20, (95%CI:0.11–0.35)] or program chair [OR:0.32, (95%CI:0.16–0.65)]. After adjustment for years of post-residency experience, only disparities at the full professor position remained [OR:0.34; (95%CI:0.16–0.47)], highlighting the importance of cohort experience.

Non-white plastic surgeons held 25.1% of all academic positions. Non-white plastic surgeons were significantly less likely to hold the full professor position [OR:0.60, (95%CI:0.14–0.88)], despite being more likely to have had prior fellowship training [OR:1.62, (95%CI:1.16–2.26)] and microsurgery fellowship training [OR:1.78, (95%CI:1.24–2.54)]. Programs with a non-white chair had a significantly greater proportion of non-white faculty (40.5% versus 20.5%; p<0.0001) and more equitable career outcomes for non-white faculty (Non-white chair=22.7% increase in non-white full-professors; p<0.0001).

CONCLUSION: As these faculty cohorts mature, a more equitable distribution of career outcomes may occur. However, the unequal appointment and advancement of faculty by program leadership indicates there are still issues which must be addressed.