exhibited dramatic defects in bone density, dysmorphology of maxillary and mandibular elements, disorganized relationship between bones, and pathogenic fractures. Formation, maturation and morphology of the dentition were also disrupted.

Conclusions: We discovered that rspo3 is a target gene of irf6, and is expressed in perichondrial cells that are juxtaposed to the oral epithelium and cartilage structures. We show that rspo3 function is required in a non-cell autonomous way to regulate osteogenesis of the chondrogenic cells, and that rspo3 is also required for bone homeostasis. In contrast to the many genes known to affect embryonic craniofacial development, this study highlights the key function of rspo3 and Wnt signaling in bone and tooth homeostasis. We are now interrogating the genetic interaction between rspo3 with Wnt genes such as wls, wnt9a and gpcs. Taken together, this work uncovered the transcriptional activation of rspo3 by irf6 in craniofacial morphogenesis and integrates rspo3 function and Wnt signaling during palatogenesis.

QS6
Multi-institutional Testing Of A 3D Printed Cleft Lip Model For Plastic Surgical Training

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Purpose: Surgical simulation has garnered increasing interest and become an adjunct for surgical training in recent years. For rare surgical pathologies, simulation allows trainees to increase their confidence, understanding and technical skills. Yet, quantitative data on the efficacy of surgical simulation is fairly sparse. We investigated the impact of a cleft lip repair simulation course using a 3D-printed model on plastic surgery training.

Methods: A 3D-printed, unilateral, complete cleft lip and palate model was developed and incorporated into a proctored simulation laboratory course at two plastic surgery training programs. Examinations were distributed before and after the laboratory to assess residents’ confidence and surgical knowledge. Sub-group analyses were performed based on training level, the number of prior cleft cases performed, and the number of operative and non-operative hours training on cleft repairs.

Results: Trainees collectively exhibited significant improvements in their overall knowledge (p<0.001), confidence describing (p=0.02), and confidence performing (p<0.001) the operation. Upon sub-group analysis, significant improvements in subjective measures of confidence were observed in all experience levels. The residents with the least experience with cleft lip repairs demonstrated the largest increases in overall knowledge, equalizing the differences in knowledge compared to their more experienced cohorts following completion of the laboratory.

Conclusion: A surgical simulation lab utilizing a 3D-printed, unilateral complete cleft lip and palate model significantly improved confidence describing and performing the procedure as well as overall knowledge. The significant improvements in knowledge among less experienced trainees suggest that simulation may be a useful method for equalizing discrepancies in experience.

QS7
Mechanical Signaling Critically Drives Human Foreign Body Response To Biomedical Implants.

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**Purpose:** An estimated $170 billion is spent annually on biomedical devices including pacemakers, implants for reconstructive surgery and biosensors around the world. Implantable materials used in biomedical devices elicit a host response termed foreign body response (FBR). FBR begins as a wound healing response but progresses into a fibrotic reaction resulting in the formation of a fibrous capsule, which isolates the implant from the surrounding microenvironment leading to implant failure. As advances are made in materials sciences, electronics, and design of sophisticated biomedical devices, modulating FBR remains the final frontier in developing durable man-machine interfaces. One key component of fibrosis that is often overlooked in the study of FBR is mechanical signaling. We have previously shown that fibrotic wound healing does not occur under normal conditions in mice, but when healing wounds are subject to high levels of mechanical stress, they produce human-like fibrotic scar tissue. Since wound healing and FBR are very closely related pathologies concluding in a fibrotic reaction characterized by increased collagen deposition, we hypothesized that mechanical signaling is a critical component of FBR to implantable materials.

**Methods:** FBR capsules from humans (breast implants, pacemakers, and neurostimulator batteries) and mice were analyzed using immunohistochemistry. Subsequently, we quantified the local mechanical stress patterns that emerge at the implant-tissue interface in both mice and humans using computational finite element modeling. To further test our hypothesis, we developed vibration-enabled implants (VEIs), which recapitulate human levels of mechanical stress in mice via in situ vibration of silicone implants. We then compared FBR in control mice, VEI model, and humans. Next, we used mass spectrometry to identify the mechanotransduction pathways that are upregulated in the VEI model. Finally, experiments using WT and IQGAP1 KO mice verified the critical role of mechanotransduction in FBR. Single cell sequencing experiments and immunostaining of FBR tissue were employed to study FBR in WT and KO mice.

**Results:** We demonstrate that the differences in FBR between mice and humans is a result of differential mechanical stress at the implant-tissue interface. Applying human levels of mechanical stress around murine implants using VEIs results in human-like robust FBR, which proves that mechanical stress is a critical component of human FBR. Proteomics analyses revealed that IQGAP1, which is a scaffolding protein involved in multiple mechanotransduction pathways is upregulated in VEI capsules as compared to control subcutaneous tissue. Here we show that IQGAP1 KO mice reverse the effect of mechanically enhanced FBR. Finally, single cell sequencing experiments identified critical mechanoresponsive subpopulations of macrophages and fibroblasts that were underrepresented in the KO mice, further confirming that targeting mechanotransduction is a viable means to reduce FBR to biomedical implants.

**Conclusion:** Here we demonstrate the central role of mechanical signaling at the implant-tissue interface in human FBR. Further studies are underway to develop therapeutic strategies to limit FBR. These findings reveal the importance of modulating the mechanical environment to improve the in vivo biocompatibility of biomedical devices.

**QS8**

**A Prospective Analysis Of Opioid Prescription, Consumption, And Psychometric Correlations In Outpatient Plastic Surgery Procedures**

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**Purpose:** The number of deaths due to opioid overdose is increasing annually in North America. Plastic Surgeons prescribe opioids after outpatient surgery based on anecdotal training rather than evidence; thus, excess opioid tablets may be available for diversion and abuse. The primary purpose of this study is to determine the factors predicting the decision to prescribe an opioid, and the secondary purpose is to determine the factors which predict the use of opioid tablets after outpatient Plastic Surgery procedures.

**Methods:** Data was collected prospectively using two surveys: (1) one pre-operative and (2) post-operative at approximately day 14. Participants were included if they were over the age of 16, could provide their own consent, and underwent an outpatient procedure. The primary outcome was type of prescription given (opioid versus non-opioid). The secondary outcome was the number of opioid tablets after outpatient Plastic Surgery procedures.

**Results:** We demonstrate that the differences in FBR between mice and humans is a result of differential mechanical stress at the implant-tissue interface. Applying human levels of mechanical stress around murine implants using VEIs results in human-like robust FBR, which proves that mechanical stress is a critical component of human FBR. Proteomics analyses revealed that IQGAP1, which is a scaffolding protein involved in multiple mechanotransduction pathways is upregulated in VEI capsules as compared to control subcutaneous tissue. Here we show that IQGAP1 KO mice reverse the effect of mechanically enhanced FBR. Finally, single cell sequencing experiments identified critical mechanoresponsive subpopulations of macrophages and fibroblasts that were underrepresented in the KO mice, further confirming that targeting mechanotransduction is a viable means to reduce FBR to biomedical implants.

**Conclusion:** Here we demonstrate the central role of mechanical signaling at the implant-tissue interface in human FBR. Further studies are underway to develop therapeutic strategies to limit FBR. These findings reveal the importance of modulating the mechanical environment to improve the in vivo biocompatibility of biomedical devices.