

In Appreciation

To the reviewers of 2018, who gave of their time to constructively assess the articles sent to them

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Capsule

The structures of the inner ear develop in response to serial signaling by growth factors

The human inner ear, required for hearing and balance, begins development as a thickening on the cranial ectoderm called a placode. The placode expresses fibroblast growth factors 3 and 10 (FGF3 and FGF10). **Umess** et al. used gene ablation and a ligand trap in mice to knock out FGF3 and FGF10 function after formation of the otic placode but before morphogenesis into complex structures. As the placode develops, it produces its own FGFs, which direct elaboration of the diverse shapes

and cell types required by this sensory system. Analysis of genes expressed differentially in response to FGF signaling identified both known and potentially new genetic drivers of deafness and indicates that FGF signaling keeps some genes silent until the right moment in the developmental program.

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Eitan Israeli

Capsule

Rescuing T cell glycolysis

One reason T cells in the tumor microenvironment (TME) become dysfunctional is that they compete with cancer cells for nutrients, particularly glucose. **Gemta** et al. studied the glucose metabolism of CD8+ T cells in the TME of mouse B16 and human melanomas. They found that the activity of enzyme enolase 1 was impaired. Enolase 1 catalyzes the synthesis of phosphoenolpyruvate, which is dephosphorylated to generate pyruvate, the end product of glycolysis. In vitro,

provision of pyruvate considerably improved the effector functions of CD8+ T cells isolated from murine melanomas. Pinning down enolase 1 as the rate-limiting step in glucose metabolism of tumor-infiltrating T cells may suggest that targeting enolase 1 activity could be used to improve cancer immunotherapy.

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Eitan Israeli