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S.R. Rosenbaum, N.A. Wilski, and A.E. Aplin

## RESEARCH BRIEFS
**Somatic HLA Class I Loss Is a Widespread Mechanism of Immune Evasion Which Refines the Use of Tumor Mutational Burden as a Biomarker of Checkpoint Inhibitor Response** .................. 282

**Précis:** Across cancer types, loss of heterozygosity at the locus encoding HLA-I was an independent prognostic factor for response to immune checkpoint blockade, and its predictive power improved when combined with measures of tumor mutation burden.

**Lower Airway Dysbiosis Affects Lung Cancer Progression** ........ 293
Précis: Lower airway dysbiosis characterized by excessive colonization with oral commensal microbes, particularly *Veillonella parvula*, was a predictor of poor prognosis in lung cancer, and in vivo experiments supported a role for *V. parvula* colonization in decreasing survival.

See commentary, p. 224
Timed Ang2-Targeted Therapy Identifies the Angiopoietin-Tie Pathway as Key Regulator of Fatal Lymphogenous Metastasis .......................... 424
Précis: A hindrance to the study of metastasis via lymphatic vessels with lymph nodes as intermediate sites was overcome using a genetically engineered mouse model-derived allograft model, the use of which uncovered the Ang2–Tie2 pathway as a critical dependency.

Netrin G1 Promotes Pancreatic Tumorigenesis through Cancer-Associated Fibroblast-Driven Nutritional Support and Immunosuppression ....................... 446
Précis: The glutamatergic presynaptic protein Netrin-G1 was found to be a major contributor to the metabolic and immunosuppressive tumor microenvironment in pancreatic ductal adenocarcinoma, and blocking Netrin-G1 in vivo suppressed tumorigenesis.

See commentary, p. 230

Correction

Correction: Impact of PD-1 Blockade on Severity of COVID-19 in Patients with Lung Cancers ....................... 520

The risk of skin cancers such as basal cell carcinoma and squamous cell carcinoma varies greatly across the body, and it is known that healthy aged skin cells often harbor cancer driver mutations, but whether these observations are connected has not been determined. Fowler and colleagues discovered that the mutation density in healthy human skin differed from location to location across the body. Common sites for skin cancer, such as the forearm, bore more mutations than uncommon sites, and the mutation profile suggested UV radiation as a cause. Interestingly, not only was there selection for cancer-associated mutations in healthy skin, but the DNA repair mechanisms used varied based on site. For more information, see the article by Fowler and colleagues on page 340.