**List of Cancer Papers (reverse chronological order)**

Khurana, E. *et al.* Integrative Annotation of Variants from 1092 Humans: Application to Cancer Genomics. *Science* **342,** 1235587–1235587 (2013a).

Kittler, R. *et al.* A Comprehensive Nuclear Receptor Network for Breast Cancer Cells. *Cell Reports* **3,** 538–551 (2013).

Khurana, E., Fu, Y., Chen, J. & Gerstein, M. Interpretation of Genomic Variants Using a Unified Biological Network Approach. *PLoS Computational Biology* **9,** e1002886 (2013b).

Lin, P.-C. *et al.* Epigenetic Repression of miR-31 Disrupts Androgen Receptor Homeostasis and Contributes to Prostate Cancer Progression. *Cancer Research* **73,** 1232–1244 (2012).

Abyzov, A. *et al.* Somatic copy number mosaicism in human skin revealed by induced pluripotent stem cells. *Nature* **492,** 438–442 (2012).

**Summary**

In the past year, we have worked on developing the Function-based Prioritization of Sequence Variants (FunSeq) tool for the prioritization of somatic cancer variants on the basis of their overlap with important functional elements (Khurana *et al.* 2013a). We have also investigated interaction network disruptions in cancer, including the transcription factor (TF) and nuclear receptor (NR) regulatory network’s role in breast cancer (Kittler *et al.* 2013), as well as the place of essential and loss-of-function (LoF) tolerant genes in a Multinet consisting of protein-protein interaction (PPI), phosphorylation, metabolic, signaling, genetic, and regulatory network data (Khurana *et al.* 2013b). Furthermore, we have explored the interaction between miR-31 expression and androgen receptor regulation in prostate cancer (Lin *et al.* 2012). Finally, we have investigated somatic copy number mosaicism in primary human skin fibroblasts (Abyzov *et al*. 2012). This work has brought attention to the extent of somatic variation in human cells, which is relevant to understanding the rise of genetic diseases such as cancer. In the future, we have plans to develop a companion tool to FunSeq called Large-scale Analysis of Recurrent Variants in Annotations (LARVA), for the discovery of functional elements that are recurrently mutated across many cancer patients.