The BAF enhances the work/productivity of PIs by:

- Providing a centralized setting for services and instrumentation in ‘omics’: genomics, transcriptomics, proteomics, lipidomics, metabolomics, and molecular interaction. BAF provides expertise in experimental design, cost-effective data collection, data analysis, and post-experiment processing for manuscripts and grants.
- Giving investigators locally to a wide variety of services that can determine qualitative and quantitative changes at the cellular level. The consolidated organization of the facility allows for researchers to perform many of these experiments on the same or related samples.
- Being a local repository of “omics” knowledge and instrumentation and offering a strategic and economical advantage for UVA.

Identification of RhoC as a Target of RhoGDI2 for Prevention of Lung Colonization of Bladder Cancer (Proteomics)
David L. Brautigan, Ph.D.

Further analysis showed knockdown of RhoGDI2 increased RhoC activation in response to serum stimulation. Overexpression of RhoGDI2 decreased RhoC activation.

RhoC promoted bladder cancer cell growth and invasion. RhoC knockdown increased cell doubling time, decreased invasion through Matrigel, and decreased colony formation in soft agar.

RhoC knockdown reduced in vivo lung colonization by bladder cancer cells in immunocompromised mice.

Unbiased transcriptome analysis revealed a set of genes regulated by RhoGDI2 overexpression and RhoC knockdown in bladder cancer cells.

Funding — CA919935, R01CA143971-05

http://www.coloradocancerblogs.org/study-pinpoints-rhogid2-suppresses-bladder-cancer-metastasis/

microRNA-34a Promotes DNA Damage and Mitotic Catastrophe (DNA Sciences)
James Larner, M.D. and Roger Abounader, M.D., Ph.D.

- miR-34a, a potent tumor suppressor, influences a large set of p53-regulated genes and contributes to p53-mediated apoptosis.
- Using tel- inducible miR-34a-expressing human p53 wild-type and R273H p53 mutant GBM cell lines, we found that miR-34a influences the breadth of 53BP1-mediated DNA damage responses.
- It escalates both post-irradiation and endogenous DNA damage, aggravates radiation-induced G2/M arrest and increases the number of irradiated cells undergoing mitotic catastrophe. Also, miR-34a downregulates 53BP1 and inhibits its recruitment to the site of DNA double-strand breaks.
- These properties of miR-34a can potentially be exploited for DNA damage-effecting therapies of malignancies.

DNA Section staff worked very closely with the investigators from the conception of the project in providing consultation in the experimental design and protocol selection for optimisation of extraction of microRNA from cells and tissues and in gene expression of miRNA and microRNAs. For Tagman quantitative real time PCR, the core staff provided assay design and validation.

- The figure shows that miR-34a increases DNA damage. Back bars indicate miR-34a-over expressing cells; stars * indicate statistical significance of P < 0.05; X-ray irradiation dose, 4 G.

Funding – 1R01CA192669-01 (PI: Larner) and 5R01CA134843-05 (PI: Abounader)