**Group 1:**

Compare R1810 to R1811 day 0 HIF control to HIF induced tissue

Compare R1812 to R1813 day 7 HIF control to HIF induced tissue

Non-regenerative SW mice were treated with a compound to stabilize HIF and induce regenerative wound healing.

We are expecting miRs that would upregulate HIF-1a and down regulate p21CipWaf. We would also expect miRs that might be involved with de-differentiation or stem cell marker expression.

BTW – you have the RNA seq data for this if there is a way to compare -

**Group 2:**

Compare R1814 to R1608 day 0 MRL to B6

Compare R1816 to R1609 day 7 MRL to B6

MRL is a regenerative mouse, B6 is not. We would expect that HIF-1a would be up-regulated, PHDs would be inhibited, VHL would be inhibited. We would also expect that p21Cip/Waf would be down-regulated.

You also have the RNA-seq data for this to compare to

**Group 3:**

Compare R1589 to R1590 day 0 MRL to B6 HFD

Compare R1596 to R1598 day 7 MRL t0 B6 HFD

And then compare Group 3 to Group 2.

As before, MRL is a regenerative strain and B6 is a non-regenerative strain.

We found that healing was better when mice were fed a high fat diet.

So we would like to compare Group 2, those 2 strains on a regular diet to Group 3, those animals on a HFD.

We would expect that p21 would be down in MRLs compared to B6 and that HIF-1a would be up so the same principles as stated above.

We also genetically mapped this HFD effect to 5 genes: LARS1; DIAPH1; HDAC3, FGF1, and ADAMTS16. We expect that they would be involved and it would be interesting to know what miRs might be involved.

Group 1

* HIF-1a: mir-31, mir199a, mir-103
* P21CipWaf (Symbol: CDKN1A): Mir21 [Mir21a] or mir-21

Group 2

* HIF-1a: mir-31, mir199a, mir-103
* PHD
* P21 (17-92 family): 106a, 106b, 130b, 302a, 302b, 302c, 302d, 512-3p, 515-3p, 17, 20a, 18a (18 available), 19a, 19b, 92a (92-1, 92, 92-2 available)