SUPPLEMENTAL MATERIAL

Table S1. Raw Ct values (Mean±SEM) for each miR examined in this study.

Small RNA Control	Control	Small TAAs	Medium TAAs	Large TAAs
snRNAU6	16.2±0.6	16.1±0.9	18.8±0.9	16.9±1.0
miR-1	24.0±0.7	25.1±1.7	28.8±1.8	29.6±1.7
miR-21	18.6±0.9	18.7±1.5	21.8±1.6	21.8±1.7
miR-29a	15.2±0.4	17.8±1.7	21.8±1.7	21.9±1.6
miR-133a	18.8±0.4	20.5±1.3	22.8±1.4	23.2±1.2
miR-486-5p	21.7±0.5	23.6±1.3	24.8±1.7	24.7±1.0
miR-760	24.1±0.3	24.5±0.5	26.0±0.7	26.6±0.5

SUPPLEMENTAL FIGURE LEGENDS

Figure S1. Alterations in miR expression in clinical TAA specimens as compared to normal aorta. Representative plots of raw cycling data depicting the difference in ΔCt values on the x-axis and the change in relative fluorescence values on the y-axis, between normal aorta (solid line) and TAA aorta (dashed line) from quantitative real-time PCR results.

Figure S2. Functional characteristics of the 37 differentially expressed miRs between Aneurysm (n=4) and Normal (n=4) aorta. The functions for the 37 differentially expressed miRs were estimated based on published manuscripts reported in PubMed.; 27% target proliferation pathways, 16% target growth arrest pathways, 8% target ECM structure/function, 5% (each) target apoptosis, migration, and angiogenesis, 3% (each) target drug resistance, immune response, autophagy, and cell adhesion pathways, and 22% to date have no reported function.

Figure S3. Transduction of human primary aortic vascular smooth muscle cells with a non-targeting mismatch control virus. **A.** Cells were exposed to a non-targeting mismatch control lentivirus, containing a bicistronic copy of green fluorescent protein (GFP), or to the transduction reagent alone. Five days post-transduction the cells were harvested and cell homogenates were examined by gelatin zymography and immunoblotting. The results demonstrated no change in latent (72 kDa) or active (64 kDa) MMP-2 with lentiviral transduction of a non-targeting sequence (*top*).

Immunoblotting for GFP (*middle*) and β-actin (*bottom*) confirmed lentiviral transduction of the mismatch control (GFP), and equal lane loading (β-actin) respectively (representative blots shown, n=3). **B.** Quantitation of total MMP-2 protein abundance following viral transduction. The results demonstrated that overexpression of a non-targeting sequence had no effect on total MMP-2 protein levels (n=3; p=0.6047).

Figure S1.

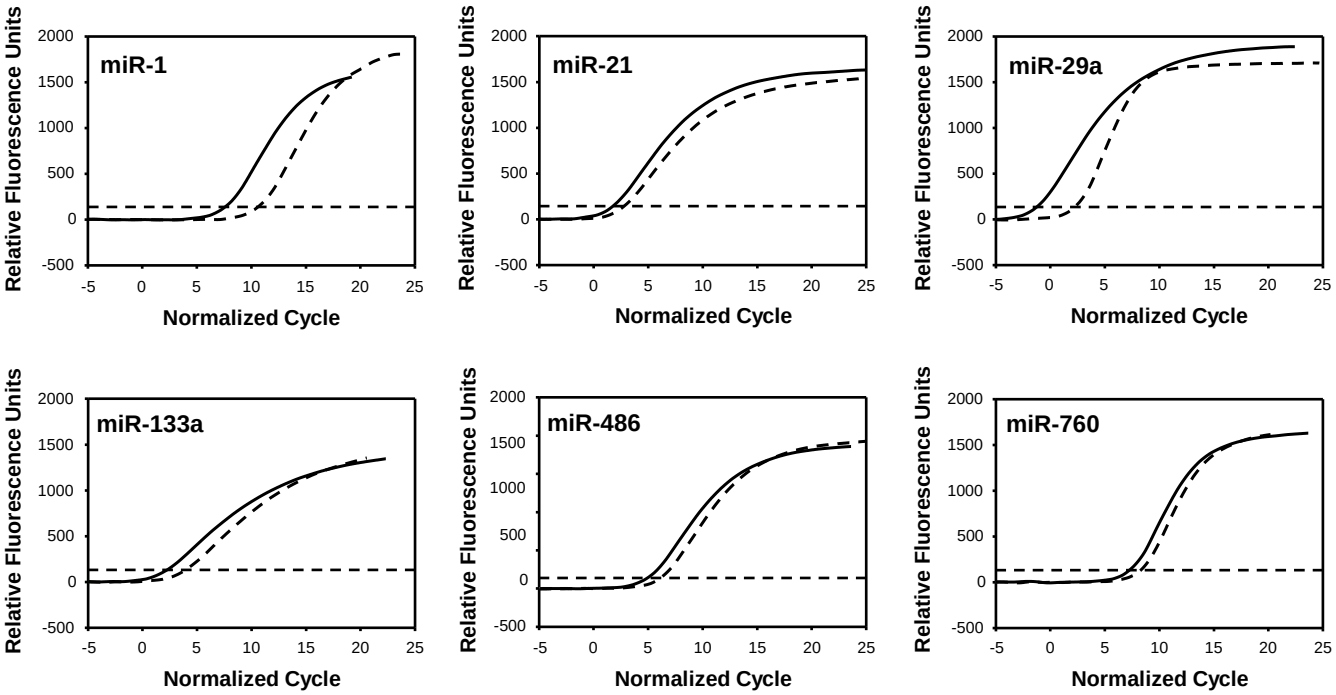

miR-1
miR-21
miR-29a
miR-133a
miR-486
miR-760
Relative Fluorescence Units
Normalized Cycle
2000
1500
1000
500
0
-500
-5
0
5
10
15
20
25

Figure S2.

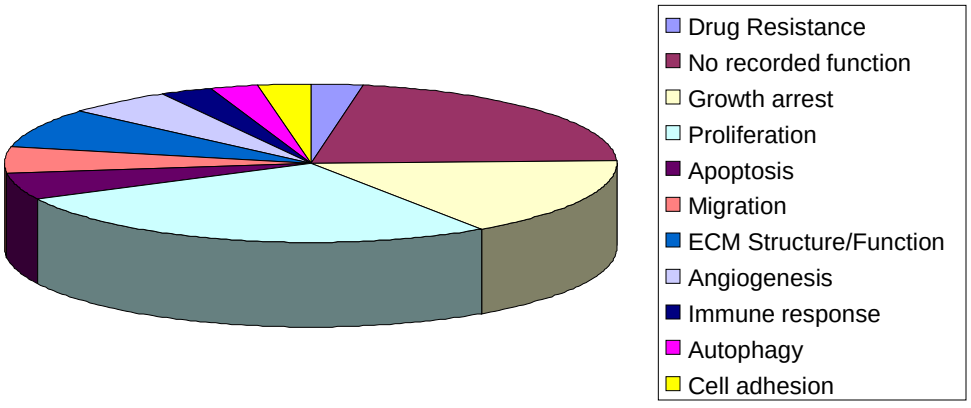
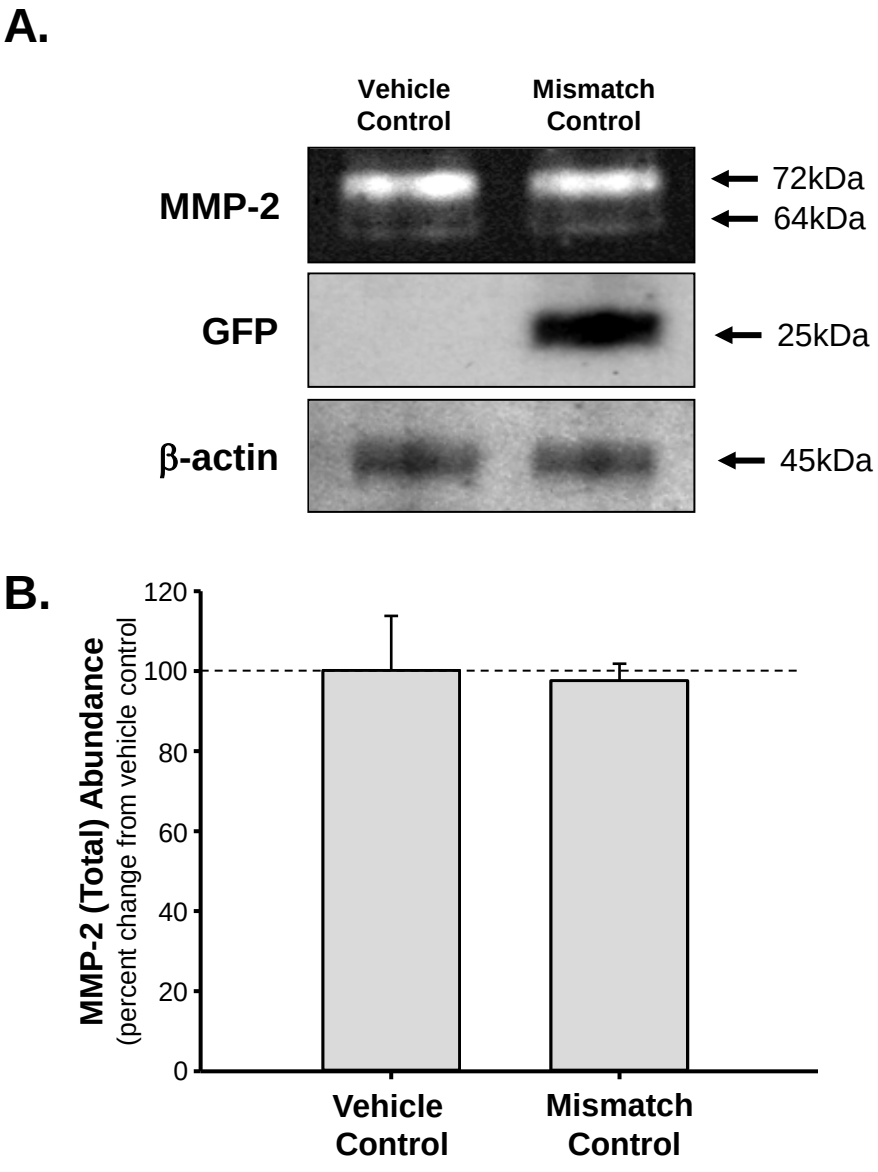

Drug Resistance
No recorded function
Growth arrest
Proliferation
Apoptosis
Migration
ECM Structure/Function
Angiogenesis
Immune response
Autophagy
Cell adhesion

Figure S3.


A.
Vehicle Control
Mismatch Control
MMP-2
72kDa
64kDa
GFP
25kDa
β-actin
45kDa
B.
MMP-2 (Total) Abundance
(percent change from vehicle control
120
100
80
60
40
20
0
Vehicle Control
Mismatch Control