SUPPLEMENTARY TEXT

Supplementary Figure Legends

Supplementary Figure 1. Ectopic expression of GRHL2 in mesenchymal-like cancer cells. A. GRHL2 mRNA is over-expressed to similar levels in all three cell lines. **B.** GRHL2 protein localizes to the nucleus in all three cell lines.

Supplementary Figure 2. Combined expression of miR-200s and GRHL2 induces an MET-like phenotype in RD cells. A. GRHL2 and miR-200s induce EpCAM and **B.** TJP1 expression at cell-cell contacts (white arrows).

Supplementary Figure 3. GRHL2 is dispensible for MET induction in MDA-MB-231 breast cancer cells. A. The EMT transcription factors ZEB1 and SNAIL are expressed at almost equal levels in MDA-MB-231 cells. **B.** E-cadherin is upregulated upon ectopic expression of miR-200s, with a modest upregulation of E-cadherin by GRHL2 alone. Combined expression of GRHL2 and miR-200s did not further enhance E-cadherin expression. **C.** E-cadherin mRNA levels are increased in miR-200-transfected cells; GRHL2 alone had a small (2.7 fold), but significant effect on E-cadherin mRNA. **D.** ZEB1 is downregulated in miR-200-expressing cells, but not in GRHL2-expressing cells.

Supplementary Figure 4. GRHL2 and miR-200s induce MET in BT-549 cells. A. BT549 cells express high levels of Twist relative to other known EMT master regulators. **B.** Ectopic expression of GRHL2 in BT549 cells. **C.** GRHL2 mRNA is upregulated upon ectopic over-expression, with a further increase of GRHL2 mRNA with miR-200 expression. **D.** ZEB1 is downregulated with GRHL2 and miR-200 expression, with a further downregulation upon combined GRHL2 and miR-200 expression. **E.** Epithelial mRNAs are upregulated with GRHL2 and miR-200 over-expression. Epithelial gene expression is predominantly driven by miR-200s. **F.** E-cadherin is upregulated upon ectopic expression of miR200s, with a modest upregulation of E-cadherin by GRHL2 alone. Combined GRHL2 and miR-200 expression led to a further increase in E-cadherin expression compared to miR-200s or GRHL2 alone.

Supplementary Figure 5. GRHL2 and miR-200s induce MET in U2-OS cells. A. U2-OS cells express high levels of ZEB1 relative to other known EMT master regulators. **B.** Ectopic expression of GRHL2 in U2-OS cells. **C.** GRHL2 mRNA is upregulated upon ectopic over-expression, with a further increase of GRHL2 mRNA with miR-200 expression. **D.** ZEB1 is downregulated with GRHL2 and miR-200 expression. **E.** Epithelial mRNAs are upregulated with GRHL2 and miR-200 over-expression. Epithelial gene expression is predominantly driven by miR-200s.

Supplementary Figure 6. GRHL2 and miR-200s induce MET in SK-LMS cells. A. SK-LMS cells express high levels of Twist relative to other known EMT master regulators. **B.** Ectopic expression of GRHL2 in SK-LMS cells. **C.** GRHL2 mRNA is upregulated upon ectopic over-expression of miR-200s or GRHL2. **D.** ZEB1 is downregulated with GRHL2 and miR-200 expression, with a further downregulation upon combined GRHL2 and miR-200 expression. **E.** A morphological change consistent with MET was observed in SK-LMS cells with combined

expression of GRHL2 and miR-200s. Cells displayed a rounded morphology, with increased cell-cell contacts. **F.** Epithelial mRNAs are upregulated with GRHL2 and miR-200 over-expression. Epithelial gene expression is predominantly driven by miR-200s.

Supplementary Figure 7. Relationship between GRHL2 methylation and EMT status across different cancer types.

Supplementary Figure 8. Prognostic significance of GRHL2 and ZEB1 in osteosarcomas. A. B. Kaplan Meier curves of metastasis-free survival (**A**) and overall survival (**B**) based on GRHL2 expression. **C. D.** Kaplan Meier curves of metastasis-free survival (**C**) and overall survival (**D**) based on ZEB1 expression.

Supplementary Figure 9. TCGA analysis of epithelial and mesenchymal biomarkers in soft tissue sarcomas. A. RNA-Seq data from soft tissue sarcomas shows a range of EMT biomarker expression, with a subset of more epithelial-like samples expressing relatively low levels of mesenchymal markers and relatively high levels of epithelial biomarkers. **B.** Normalization of soft tissue sarcoma samples to all cancers in TCGA (PANCAN normalized) reveals a strongly mesenchymal biomarker profile compared to all cancers.

Supplementary Table S1. List of antibody information.

Antibody	Company	Catalog number	Assay	Dilution
Anti-E-cadherin	BD Transduction Laboratories	610181	Western blotting	1:1,000
Anti-GRHL2	Sigma	HPA004820	Immunofluorescence	1:1,000
Anti-β-actin	Santa Cruz Biotech.	sc-47778	Western blotting	1:2,000
Anti-EpCAM	ABD Serotec	MCA1870G	Immunofluorescence	1:500
Anti-TJP1	Biogenesis	9910-1006	Immunofluorescence	1:500
Anti-GAPDH	Santa Cruz Biotech.	sc-25778	Western blotting	1:2,000
Anti-ZEB1	Santa Cruz Biotech.	sc-25388	Immunofluorescence	1:500

Supplementary Methods

Regulatory circuit details and mathematical model formulation

(a) Theoretical framework for microRNA-based circuits

The theoretical framework for microRNA-based circuits is derived and presented in detail in our previous work [4]. A microRNA is usually 22nt long, and the seed sequence it recognizes on mRNA is 7~8 nt long. microRNA and mRNA can bind and unbind at a rate much faster than

production and degradation of proteins, and a microRNA can both sequester the mRNA by forming a complex, or degrade it.

In brief, the translational rate of mRNA is given by $mL(\mu)=\sum_{i=0}^{n} l_i C_n^i[m_i]=m\sum_{i=0}^{n} l_i C_n^i M_n^i(\mu)$, where l_i is the translation rate of an mRNA when bound to *i* microRNAs. The degradation of the mRNA due to binding with the microRNA is described by $mY_m(\mu)=\sum_{i=0}^{n}\gamma_{mi}C_n^i[m_i]=m\sum_{i=0}^{n}\gamma_{mi}C_n^i M_n^i(\mu)$, where γ_{mi} is the individual degradation rate of mRNA bound to *i* molecules of miRNA. Similarly, the degradation rate of microRNA due to binding to many mRNAs is $mY_\mu(\mu)=\sum_{i=0}^{n} i\gamma_{\mu i}C_n^i[m_i]=m\sum_{i=0}^{n} i\gamma_{\mu i}C_n^i M_n^i(\mu)$, where $\gamma_{\mu i}$ is the individual degradation rate for a microRNA molecule. For a small number of binding sites on the mRNA for miRNA (~2), the miRNA-mediated regulation can be approximated as a Hill function [4].

Transcriptional regulation is denoted by shifted Hill functions $H^S(X,\lambda)$ – a weighted sum of positive and negative Hill functions – as defined by $H^S(X,\lambda)=H^-(X)+\lambda H^+(X)$, where λ is the weight factor that represents fold-change in production rate from its basal level due to the binding of regulatory factor. For activation, $\lambda>1$ and shifted Hill function is denoted by H^{S+}; for repression, $0<\lambda<1$ and shifted Hill function is denoted by H^{S-}; and for no change, λ=1. $\lambda_{X,Y}$ denotes the effect of X on Y[4].

(b) Initial model

The model depicted in Figure 1A consists of six species – microRNA (miR)200 (μ_{200}), ZEB mRNA (m_Z), ZEB protein (Z), GRHL2 mRNA (m_G), GRHL2 protein (G), and E-cadherin (E) – each with innate production and degradation rates. The effect of miR-200 on ZEB are

captured by both the degradation of mRNA by miRNAs (depicted by $Y_m(\mu)$) and the inhibition of translation by miRNAs (depicted by $L(\mu)$). Also, the miRNAs that bind to mRNAs can be degraded after forming a complex with mRNAs (depicted by $Y_\mu(\mu)$). The dynamics of miR-200 (μ_{200}) can be described by the following equation:

$$\frac{d\mu_{200}}{dt} = g_{\mu_{200}} H^{S-}\left(Z, \lambda_{Z,\mu_{200}}\right) H^{S-}\left(S, \lambda_{S,\mu_{200}}\right) - m_Z Y_\mu\left(\mu_{200}\right) - k_{\mu_{200}}\mu_{200} \cdots\cdots (1)$$

where $g_{\mu_{200}}$ and $k_{\mu_{200}}$ are the innate production and degradation rates of miR-200 respectively. $H^{S-}\left(Z, \lambda_{Z,\mu_{200}}\right)$ represents the transcriptional inhibition of miR-200 by ZEB and $H^{S-}\left(S, \lambda_{S,\mu_{200}}\right)$ represents transcriptional inhibition of miR-200 by SNAIL. $Y_\mu\left(\mu_{200}\right)$ represents the degradation rate of miR-200 due to forming a complex with ZEB mRNAs.

When considering the effect of an external activating signal Ex on miR-200, an additional shifted Hill function $H^{S+}\left(Ex, \lambda_{Ex,\mu_{200}}\right)$ is multiplied with the production rate of miR-200. The updated equation is:

$$\frac{d\mu_{200}}{dt} = g_{\mu_{200}} H^{S-}\left(Z, \lambda_{Z,\mu_{200}}\right) H^{S-}\left(S, \lambda_{S,\mu_{200}}\right) H^{S+}\left(Ex, \lambda_{Ex,\mu_{200}}\right) - m_Z Y_\mu\left(\mu_{200}\right) - k_{\mu_{200}}\mu_{200} \cdots\cdots (1\_1)$$

The dynamics of ZEB mRNA (m_Z) and ZEB protein (Z) are described by the following equations:

$$\frac{dm_Z}{dt} = g_{m_Z} H^{S-}\left(G, \lambda_{G,m_z}\right) H^{S-}\left(E, \lambda_{E,m_z}\right) H^{S+}\left(Z, \lambda_{Z,m_Z}\right) H^{S+}\left(S, \lambda_{S,m_Z}\right) - m_Z Y_m\left(\mu_{200}\right) - k_{m_Z} m_Z \ldots\ldots\ldots (2)$$

$$\frac{dZ}{dt} = g_Z m_Z L\left(\mu_{200}\right) - k_Z Z \cdots\cdots (3)$$

where g_{m_Z} and g_Z are the innate production rates of ZEB mRNA and ZEB protein respectively, k_{m_Z} and k_Z are their respective innate degradation rates. $H^{S+}\left(Z, \lambda_{Z,m_Z}\right)$ denotes transcriptional self-activation of ZEB, and $H^{S+}\left(S, \lambda_{S,m_Z}\right)$ denotes transcriptional activation of ZEB by SNAIL.

$Y_m(\mu_{200})$ represents the degradation of ZEB mRNA due to forming mRNA-miRNA complexes with miR-200, $L(\mu_{200})$ denotes the translational inhibition of ZEB by miR-200, and $H^{S-}(G,\lambda_{G,m_z})$ and $H^{S-}(E,\lambda_{E,m_z})$ represent the inhibition from GRHL2 and E-cadherin respectively.

The dynamics of GRHL2 mRNA (m_G) and GRHL2 protein (G) are described by these equations:

$$\frac{dm_G}{dt} = g_{m_G} H^{S-}(Z,\lambda_{Z,m_G}) - k_{m_G} m_G \text{.....}(4)$$

$$\frac{dG}{dt} = g_G m_G - k_G G \text{.....}(5)$$

g_{m_G} and g_G are innate production rates of GRHL2 mRNA and protein respectively; k_{m_G} and k_G are their respective degradation rates. $H^{S-}(Z,\lambda_{Z,m_G})$ represents transcriptional inhibition of GRHL2 by ZEB.

When considering an external activation Ex on GRHL2, a shifted Hill function $H^{S+}(Ex,\lambda_{Ex,\mu_{200}})$ is multiplied with its production rate; hence the equation is given by:

$$\frac{dm_G}{dt} = g_{m_G} H^{S-}(Z,\lambda_{Z,m_G}) H^{S+}(Ex,\lambda_{Ex,m_G}) - k_{m_G} m_G \text{.....}(4\_1)$$

The dynamics of E-cadherin are given by:

$$\frac{dE}{dt} = g_E H^{S-}(Z,\lambda_{Z,E}) H^{S+}(G,\lambda_{G,E}) - k_E E \text{.....}(6)$$

Where g_E and k_E denote the innate production and degradation terms for E-cadherin protein, $H^{S-}(Z,\lambda_{Z,E})$ and $H^{S+}(G,\lambda_{G,E})$ represent the transcriptional inhibition by ZEB1 and activation by GRHL2 respectively.

(c) Revised model

Our experimental results indicated that the interplay between ZEB1 and GRHL2 might be cell line- or lineage-dependent. ZEB1 and GRHL2 have been reported to form a mutually inhibitory feedback loop in MDA-MB-231 cells, while in RD and 143B cells, our data indicated that ZEB1 might not inhibit GRHL2 transcriptionally (or vice-versa); rather GRHL2 activity is inhibited by potentially restricting its binding to the E-cadherin promoter. Based on these observations, we refined our model to include these regulations. The dynamics of miR-200 is still given by equation (1) and that of ZEB protein by equation (3), but the dynamics of ZEB mRNA were given by:

$$\frac{dm_Z}{dt} = g_{m_Z} H^{S-}\left(E, \lambda_{E,m_z}\right) H^{S+}\left(Z, \lambda_{Z,m_Z}\right) H^{S+}\left(S, \lambda_{S,m_Z}\right) - m_Z Y_m\left(\mu_{200}\right) - k_{m_Z} m_Z \text{.....} (7)$$

We introduce two species G_u and G_b instead to represent the unbound and bound forms of GRHL2 to the E-cadherin promoter. The equation for bound form of GRHL2 (G_b) is given by:

$$\frac{dG_b}{dt} = g_{G_b} H^{S-}(Z, \lambda_{Z,G_b}) H^{S+}(Ex1, \lambda_{Ex1,G_b}) - k_{G_b} G_b \text{.....} (8)$$

Where g_{G_b} denotes the production of bound form of GRHL2, and k_{G_b} denotes degradation or unbinding of the bound form. Since binding/unbinding of GRHL2 to E-cadherin promoter is much faster than the half-life of protein, g_{G_b} captures steady state levels of GRHL2 multiplied by binding rate. $H^{S-}(Z, \lambda_{Z,G_b})$ represents the inhibition by ZEB on GRHL2's binding to the E-cadherin promoter, $H^{S+}(Ex1, \lambda_{Ex1,G_b})$ denote the increase in levels of GRHL2 bound due to an external signal such as activation of GRHL2 (overall).

The equation for E-cadherin is given by: $\frac{dE}{dt} = g_E H^{S-}\left(Z, \lambda_{Z,E}\right) H^{S+}\left(G_b, \lambda_{G_b,E}\right) - k_E E \text{.....} (9)$

where $H^{S+}\left(G_b, \lambda_{G_b,E}\right)$ denotes the activation of E-cadherin promoter by the bound form of GRHL2.

(d) Parameter estimation

The levels of miRNAs, mRNAs, and proteins are estimated according to their canonical concentrations in eukaryotic cells. Of course, we recognize that these values can change dramatically for specific genes and under different conditions; however, we use these estimates to provide a broad, robust model framework. Typically, the volume of a mammalian cell is 100-10000 um^3 and the concentration for a single protein is 10nM-1μM [39]. 1μM protein concentration amounts to around 6 million molecules ($6.02\times10^{23}\times10^{-6}\times\left(10000\times\left(10^{-5}\right)^3\right)$).

The ratio of protein/mRNA of one gene is about 2800 [40], hence the number of mRNA molecules for one gene should be around 1000. The number of microRNA molecules in a cell are around 10000 molecules [41], hence μ_0 =10000 molecules. A general translation rate is around 140 proteins per mRNA per hour [40], and we chose translation rates accordingly. Innate degradation rates of miRNAs, mRNAs, and proteins were selected based on their half-lives from experimental data. Typically, the half-life of mammalian proteins is about 10 hours [42]; therefore we selected 0.1 $hour^{-1}$ as the innate degradation rate for proteins ZEB and GRHL2. The half-life of mRNA is a few hours [43], so we chose 0.5 $hour^{-1}$ as the innate degradation rate for ZEB mRNA and GRHL2 mRNA. The innate degradation rate of miR-200 was selected as 0.05 $hour^{-1}$ because miRNAs are generally more stable than mRNAs [44,45]. For transcriptional regulation, the weight factors for shifted Hill function vary from 5 to 10 for activation, and from 0.5 to 0.1 for repression. Tables S2-S4 show the parameter values.

n (# of miRNA binding sites)	0	1	2	3	4	5	6
l_i **(hour^{-1})**	1	0.6	0.3	0.1	0.05	0.05	0.05
γ_{mi} **(hour^{-1})**	0	0.04	0.2	1	1	1	1
$\gamma_{\mu i}$**(hour^{-1})**	0	0.005	0.05	0.5	0.5	0.5	0.5

Table S2. The parameters used in Y(u), Y(m) and L functions.

Description	Fold change	Value	# binding sites	Value	Threshold	Molecules
Inhibition of ZEB by GRHL2	λ_{G,m_Z}	0.1	n_{G,m_Z}	1	$G^0_{m_Z}$	25000
Inhibition of GRHL2 by ZEB	λ_{Z,m_G}	0.5	n_{Z,m_G}	3	$Z^0_{m_G}$	10000
Self-activation of ZEB	λ_{Z,m_Z}	7.5	n_{Z,m_Z}	2	$Z^0_{m_z}$	25000
Inhibition of miR-200 by ZEB	$\lambda_{Z,\mu_{200}}$	0.1	$n_{Z,\mu_{200}}$	3	$Z^0_{\mu_{200}}$	220000
Activation of ZEB by SNAIL	λ_{S,m_Z}	10	n_{S,m_Z}	2	$S^0_{m_z}$	180000
Inhibition of miR-200 by SNAIL	$\lambda_{S,\mu_{200}}$	0.1	$n_{S,\mu_{200}}$	2	$S^0_{\mu_{200}}$	180000
External activation signal on GRHL2	λ_{Ex,m_G}	4	n_{Ex,m_G}	2	$Ex^0_{m_G}$	4000
Activation of E-cad by GRHL2	$\lambda_{G,E}$	2	$n_{G,E}$	1	G^0_E	25000
Inhibition of E-cad by ZEB	$\lambda_{Z,E}$	0.1	$n_{Z,E}$	2	Z^0_E	100000
Inhibition of ZEB by E-cad	λ_{E,m_Z}	0.7	n_{E,m_Z}	1	$E^0_{m_Z}$	80000
Inhibition of GRHL2 binding to E-cad by ZEB	λ_{Z,G_b}	0.05	n_{Z,G_b}	2	$Z^0_{G_b}$	50000

Activation of E-cad by GRHL2 bound	$\lambda_{G_b,E}$	5	$n_{G_b,E}$	1	$G^0_{b_E}$	10000
External activation on miR-200	$\lambda_{Ex,\mu_{200}}$	4	$n_{Ex,\mu_{200}}$	2	$Ex^0_{\mu_{200}}$	4000
External activation on GRHL2 (bound)	λ_{Ex1,G_b}	5	n_{Ex1,G_b}	2	$Ex1^0_{G_b}$	4000

Table S3. The parameters used in different Hill functions for different circuits

Species	**Production rates (molecules/Hour)**		**Degradation rates (Hour^{-1})**	
miR-200	$g_{\mu_{200}}$	2100	$k_{\mu_{200}}$	0.05
ZEB mRNA	g_{m_Z}	11	k_{m_Z}	0.5
ZEB protein	g_Z	100	k_Z	0.1
GRHL2 mRNA	g_{m_G}	22	k_{m_G}	0.5
GRHL2 protein	g_G	200	k_G	0.1
GRHL2 bound	g_{G_b}	5000	k_{G_b}	0.5

Table S4. The production and degradation rates of different species in the circuits

(e) Parameter Sensitivity analysis

To test the sensitivity of the predictions of the revised model based on the parameters listed above, we conduct parameter sensitivity analyses by varying each parameter at one time. All the parameters – production rates, degradation rates, thresholds and weight factors in the shifted Hill functions are varied by +/-20%. The number of binding sites for the different interactions have been kept fixed as most of them are directly determined from experimental

data. Sensitivity of the model is measured by how changes in the parameters affect the steady state fold-change in levels of E-cadherin when (a) miR-200 is overexpressed, (b) GRHL2 is overexpressed, and (c) both miR-200 and GRHL2 are overexpressed, at a fixed SNAIL level. We find that the model is quite robust to parameter changes (Table S5), and the fold-change of E-cadherin changes largely only when parameters those alter the innate production rate of E-cadherin – such as g_E (innate production rate of E-cadherin), k_E (innate degradation rate for E-cadherin), and $\lambda_{G_b,E}$ (fold-change in E-cadherin levels due to binding of GRHL2 to its promoter) – are altered.

Parameter	miR-200	GRHL2	GRHL2 + miR-200	miR-200	GRHL2	GRHL2 + miR-200
Control	**25.67**	**1.44**	**37.23**	**25.67**	**1.44**	**37.23**
$g_{\mu_{200}}$	25.73	1.45	37.28	25.56	1.43	37.10
$k_{\mu_{200}}$	25.59	1.44	37.14	25.74	1.45	37.29
g_{m_Z}	25.22	1.43	36.74	26.10	1.52	37.68
k_{m_Z}	25.81	1.48	37.37	25.51	1.43	37.05
g_Z	25.22	1.43	36.74	26.10	1.50	37.68
k_Z	26.03	1.48	37.61	25.11	1.43	36.61
g_E	30.85	1.73	44.71	20.51	1.15	29.75
k_E	21.37	1.20	30.99	32.15	1.80	46.59
$Z^0_{\mu_{200}}$	25.67	1.44	37.23	25.67	1.44	37.23
$\lambda_{Z,\mu_{200}}$	25.67	1.44	37.23	25.67	1.44	37.23
λ_{E,m_Z}	25.43	1.44	36.92	25.92	1.44	37.52

$E^0_{m_Z}$	25.64	1.44	37.19	25.72	1.44	37.26
$\lambda_{Z,E}$	25.67	1.69	37.22	25.67	1.19	37.22
Z^0_E	25.87	1.46	37.50	25.38	1.43	36.82
$\lambda_{G_b,E}$	29.92	1.59	44.36	21.43	1.29	30.09
$G^0_{b_E}$	24.07	1.35	36.14	27.65	1.55	38.39
$Z^0_{G_b}$	25.81	1.45	37.31	25.48	1.44	37.11
λ_{Z,G_b}	25.67	1.52	37.22	25.67	1.34	37.22
λ_{Z,m_Z}	25.65	1.43	37.20	25.70	1.52	37.25
$Z^0_{m_Z}$	25.71	1.44	37.26	25.61	1.44	37.16
$S^0_{m_Z}$	25.92	1.46	37.49	25.41	1.43	36.94
λ_{S,m_Z}	25.25	1.43	36.76	26.08	1.51	37.67
$S^0_{\mu_{200}}$	25.69	1.44	37.24	25.67	1.44	37.21
$\lambda_{S,\mu_{200}}$	25.73	1.45	37.29	25.58	1.44	37.10
λ_{Ex1,G_b}	25.67	1.53	38.17	25.68	1.34	35.94
$Ex1^0_{G_b}$	25.67	1.42	36.97	25.68	1.46	37.45
$\lambda_{Ex,\mu_{200}}$	25.73	1.44	37.28	25.57	1.44	37.11
$Ex^0_{\mu_{200}}$	25.66	1.44	37.21	25.69	1.44	37.24

Table S5. Fold-change in E-cadherin levels when (a) miR-200 is overexpressed, (b) GRHL2 is overexpressed, and (c) both GRHL2 and miR-200 are overexpressed; and each parameter is varied one at a time – either increased by 20% (first three columns) or decreased by 20% (last three columns), for a fixed level of SNAIL=250*10^3 molecules.

Rows labeled by red show the cases when the fold-change levels are relatively different from the control case (marked in green).

Among these three parameters, the one affected by GRHL2 is $\lambda_{G_b,E}$ (fold-change in E-cadherin levels due to binding of GRHL2 to its promoter). Therefore, we characterized how varying $\lambda_{G_b,E}$ and the levels of SNAIL alter the fold-change in the levels of E-cadherin (Table S6).

$\lambda_{G_b,E}$	*SNAIL (10^3 molecules)*	*miR-200*	*GRHL2*	*miR-200+GRHL2*
2.5	300	16.63	1.18	21.44
10	300	39.15	1.83	61.03
7.5	250	33.10	1.65	50.21
5	350	25.24	1.44	36.83
15	300	49.01	2.11	78.30

Table S6. Fold-change in E-cadherin levels on individual and combinatorial overexpression of miR-200 and GRHL2 for different values of SNAIL and $\lambda_{G_b,E}$

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