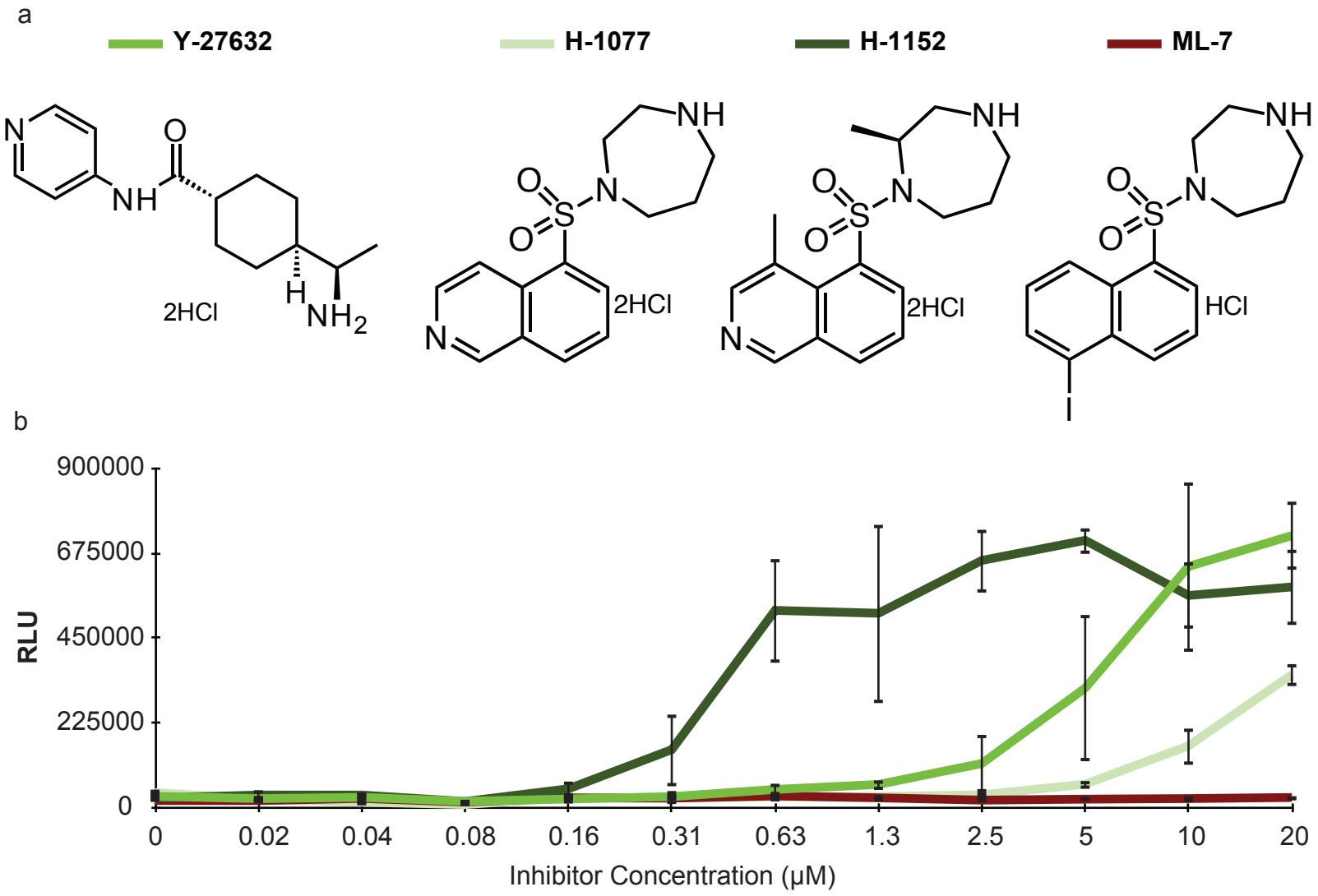
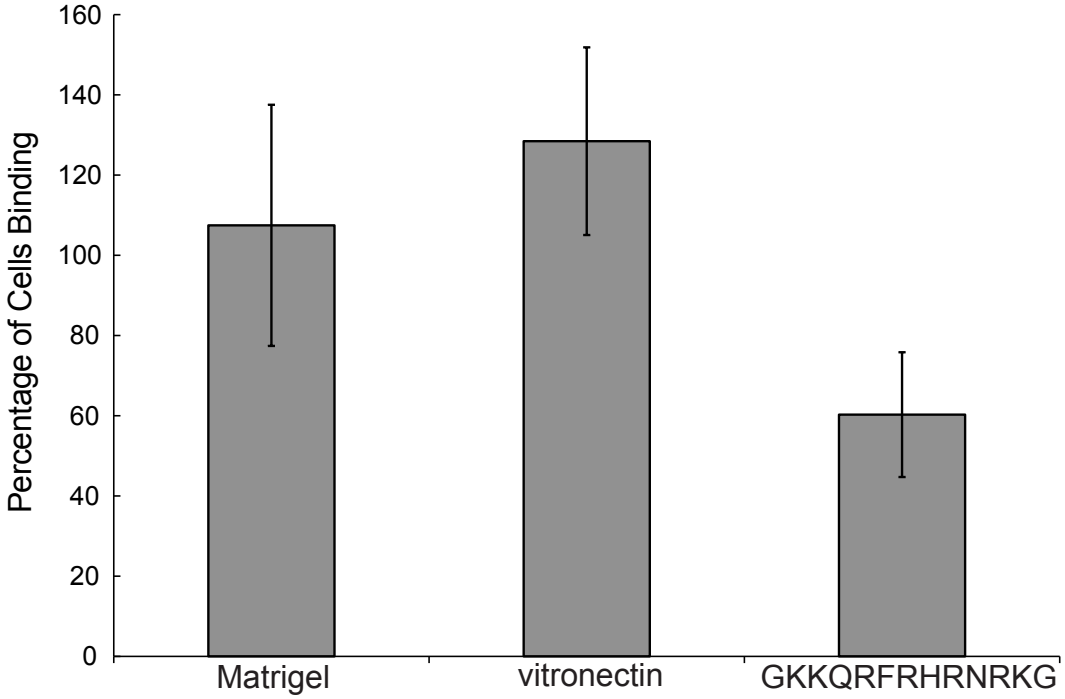
Supplementary Figure 1. The effect of ROCK inhibitors on cellular adhesion.



Supplementary Figure 1. The effect of ROCK inhibitors on cellular adhesion. (a) Structures of inhibitors tested. These compounds vary in their ROCK inhibitory potency (Y-27632 Ki=0.14 μM, H-1077 Ki=0.33 μM, and H-1152 Ki=0.0016) (1). To control for off-target effects, the strucutrally similar myosin light chain kinase inhibitor ML-7 was employed. This compound has no reported activity against ROCK. (b) Human ES cells (H14) were dissociated and applied to streptavidin-coated surfaces presenting biotinylated GKKQRFRHRNRKG in the presence of an inhibitor (Y-27632, HA-1077, H-1152, or ML-7) at varying concentrations. After 12 hours, surfaces were washed, and the cells were lysed in RIPA buffer containing 10% Cell Titer Glo (Promega), which is a homogenous and sensitive method to determine the number of viable cells in culture based on the presence of ATP. The luminescence was measured on an Ultra 384 plate reader from Tecan. Data represent the average of three wells ± the standard deviation. As the concentration of inhibitor increased, more cells attached to the surface. In the absence of inhibitor or in the presence of ML-7, very few cells attached to the surface.

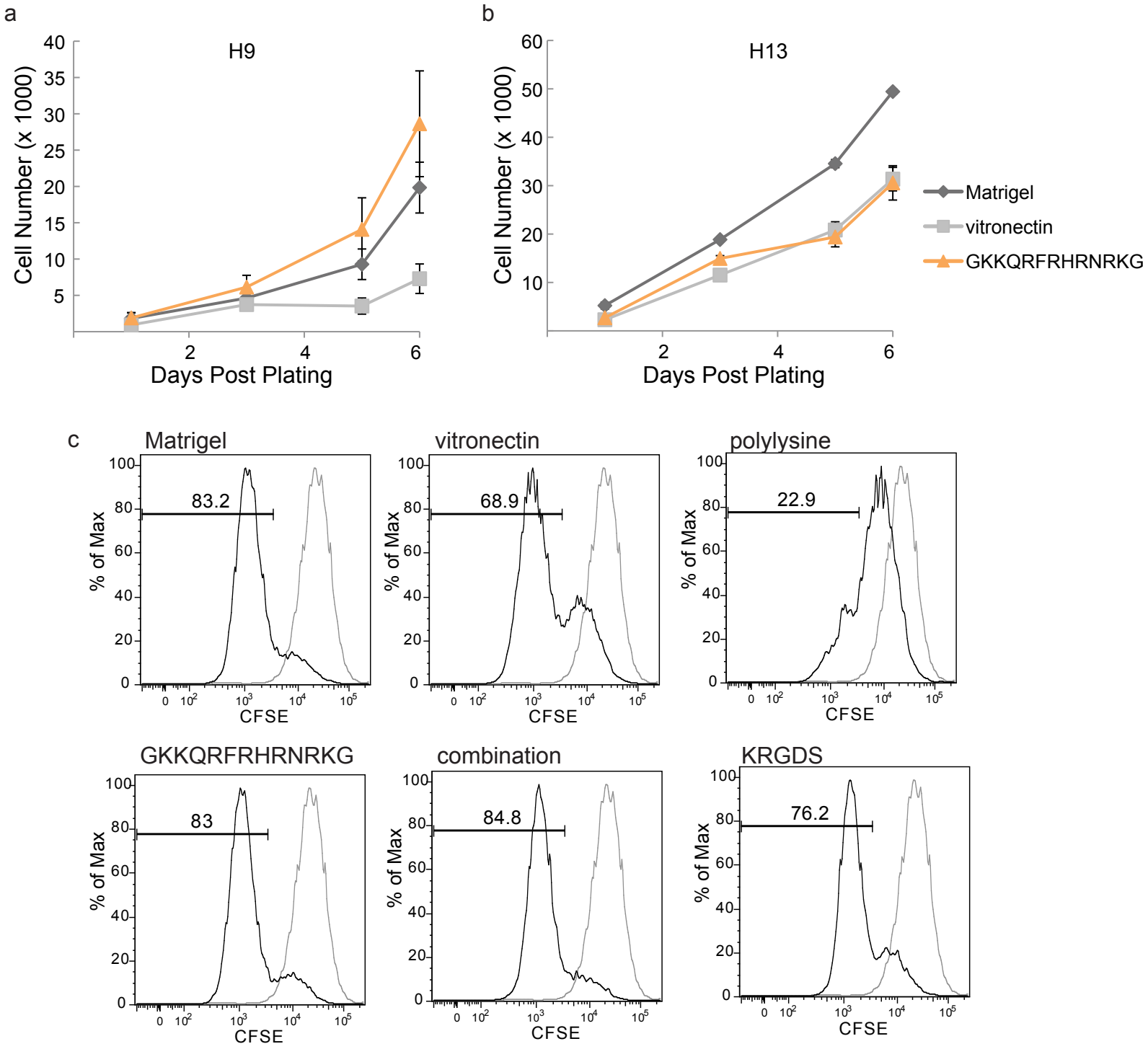
1. Yasuharu, S. (2003) New Aspects of Neurotransmitter Release and Exocytosis: Rho-Kinase-Dependent Myristoylated Alanine-Rich C-Kinase Substrate Phosphorylation and Regulation of Neurofilament Structure in Neuronal Cells. *J Pharmacol Sci* 93(1):35-40.

Supplementary Figure 2. Treatment of hES cells with enzymes that hydrolyze GAGs inhibits adhesion to surfaces presenting the heparin-binding peptide GKKQRFRHRNRKG.



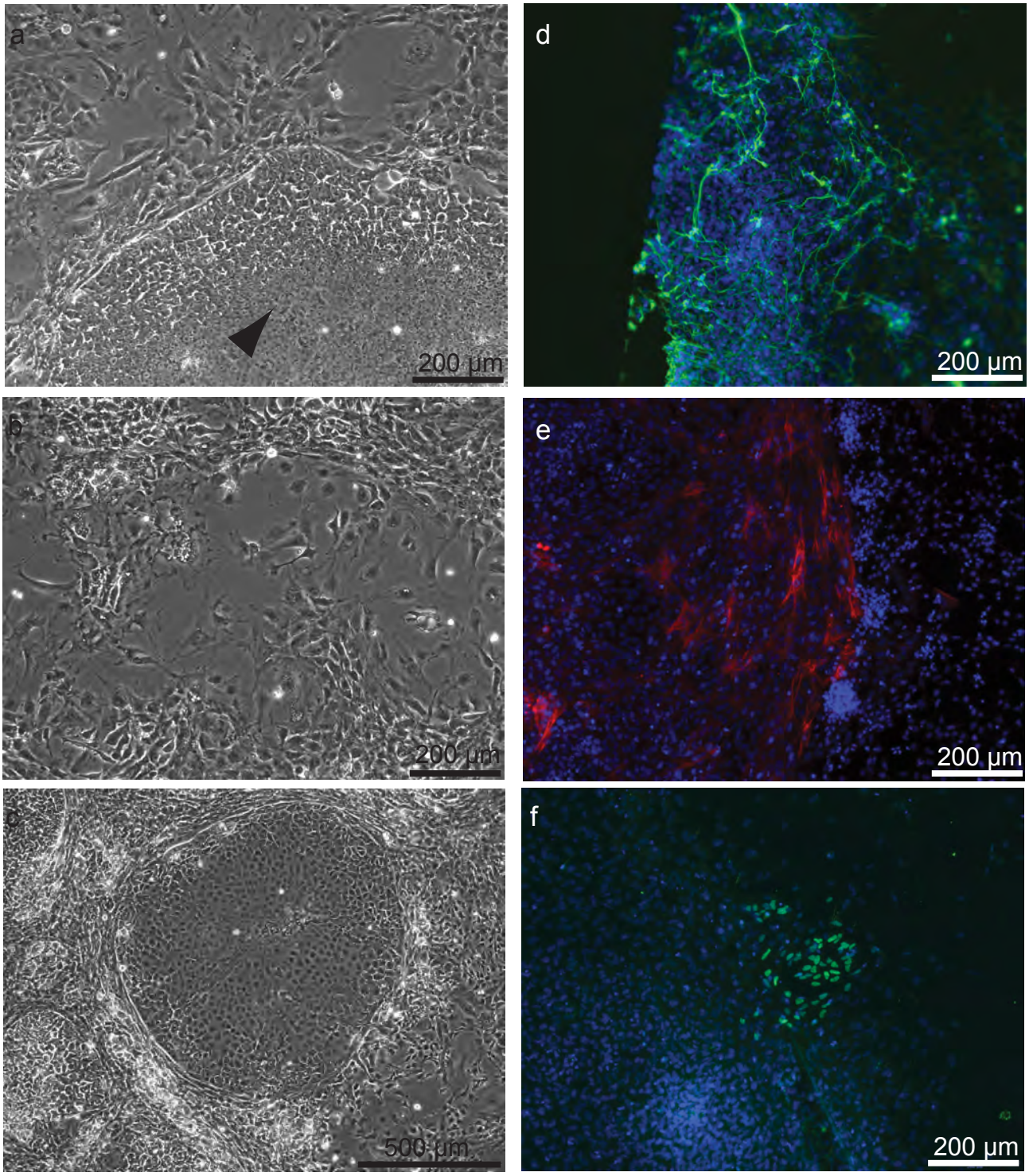
Supplementary Figure 2. Treatment of hES cells with enzymes that hydrolyze GAGs inhibits adhesion to surfaces presenting the heparin-binding peptide GKKQRFHRNRKG. Human ES cells (H9) cultured on Matrigel were dissociated using an enzyme-free buffer. Cells were resuspended in DMEM/F12, or DMEM/F12 supplemented with 2 units/mL chondroitinase ABC. Cells were incubated for 1 hour in suspension at 37 °C. Subsequently, cells were seeded on to Matrigel-coated surfaces, recombinant vitronectin-coated surfaces, or SAMs presenting the peptide GKKQRFRHRNRKG at a 5% surface density. After 1 hour, surfaces were washed and the cells were lysed. The cell lysate was used to approximate the cell number using Cell Titer Glo (Promega). Percentage of cells binding represents the mean luminescence of the cell lysates plated in the presence of chondroitinase ABC divided by the mean luminescence of cell lysates for the untreated cells ± the standard deviation.

Supplementary Figure 3. Growth characteristics of hES cells cultured on natural and synthetic substrata.



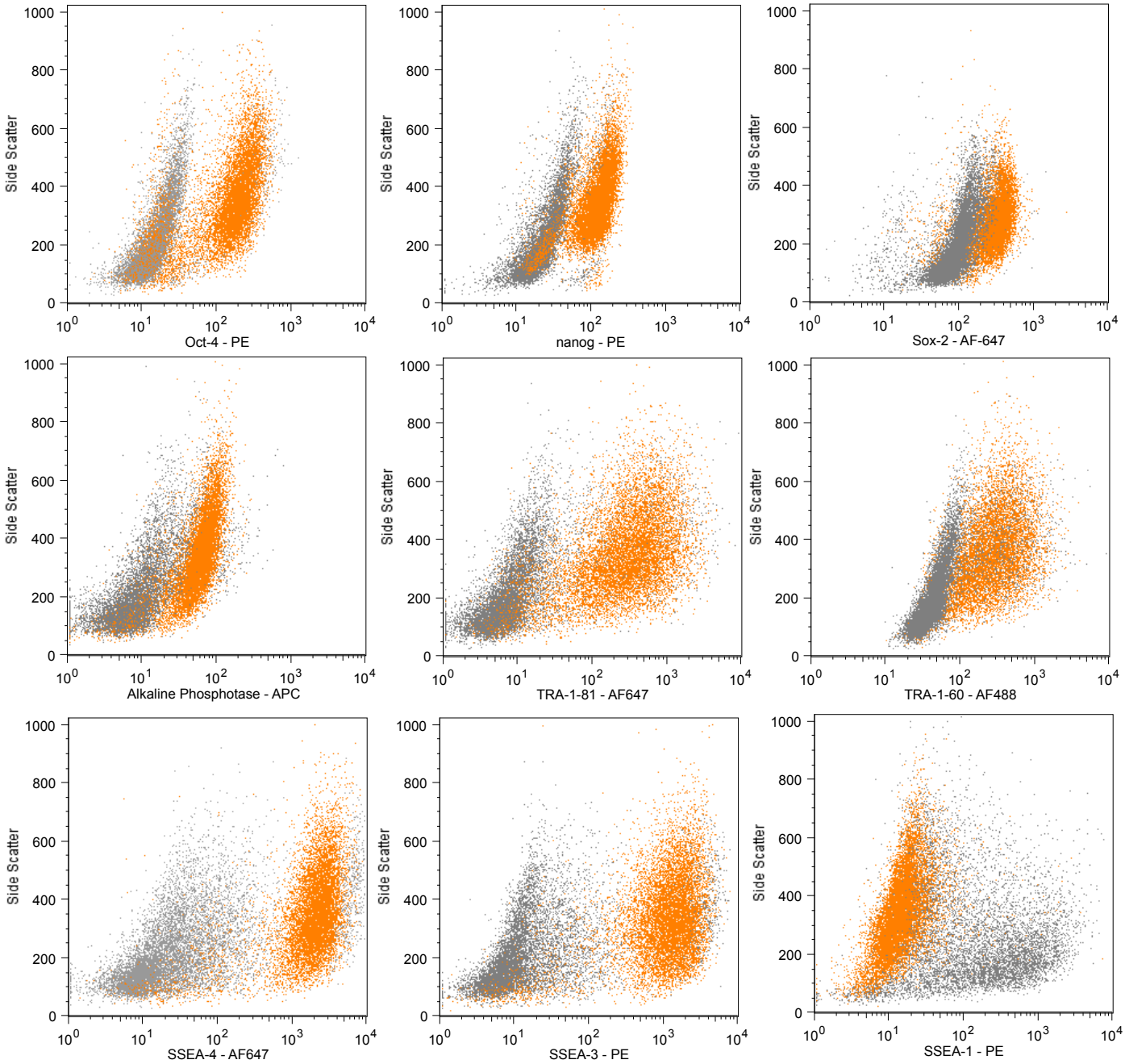
Supplementary Figure 3. Growth characteristics of hES cells cultured on natural and synthetic substrata. (a-b) Growth curves for the second passage of hES cells (H9, a, and H13, b) cultured on Matrigel, vitronectin, or GKKQRFRHRNRKG in mTeSR media supplemented with 5 μM ROCK inhibitor. For each time point, cell counts are reported from triplicate wells. Cell counts were determined using a standard curve generated with the Cell Counting Kit-8 (Dojindo), which allows for the colorimetric detection of viable cells in culture based on their dehydrogenase activity. Culturing cells on Matrigel, vitronectin, or surfaces presenting the heparin-binding peptide GKKQRFRHRNRKG afforded an increase in the number of cells. (c) Cell division analysis using the fluorescent cell staining dye, carboxyfluoresceindiacetate (CFSE). Human ES cells (H9) were labeled with CFSE, which allows for cell divisions to be monitored by a decrease in fluorescence. After labeling, cells were either immediately fixed for later analysis or distributed to the indicated surfaces. After 72 hours of culture in mTeSR media supplemented with 5 μM ROCK inhibitor, cells were analyzed using flow cytometry. The gray lines represent the parent population, and the black lines represent the cells cultured on the various surfaces. A gate was drawn between two discreet populations of cells to compare cell divisions on the indicated surfaces. The numbers indicate the percentage of cells in the population with lower fluorescence.

Supplementary Figure 4. Cells cultured on RGD presenting surfaces differentiate into a variety of cell types.



Supplementary Figure 4. Cells cultured on RGD presenting surfaces differentiate into a variety of cell types. (a-c) Human ES cells (H1) were cultured in mTeSR + Ri on synthetic surfaces presenting the integrin binding peptide KGRGDS for 15 days. In addition to the undifferentiated cell morphology (a, arrowhead), a variety of cell morphologies was observed. (d-f) Human ES cells (H7) were cultured in mTeSR + Ri on synthetic surfaces presenting the integrin binding peptide KGRGDS for 22 days and then immunostained for markers of differentiation. Immunoreactivity for β-III tubulin (d), α-smooth muscle actin (e), and FoxA2 (f) indicate the presence of ectoderm, mesoderm, and endoderm, respectively.

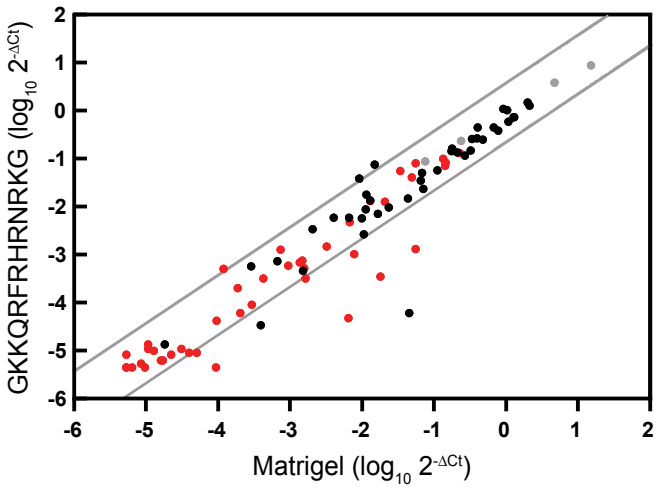
Supplementary Figure 5. Human ES cells cultured long-term on a synthetic surface maintain markers of pluripotency.



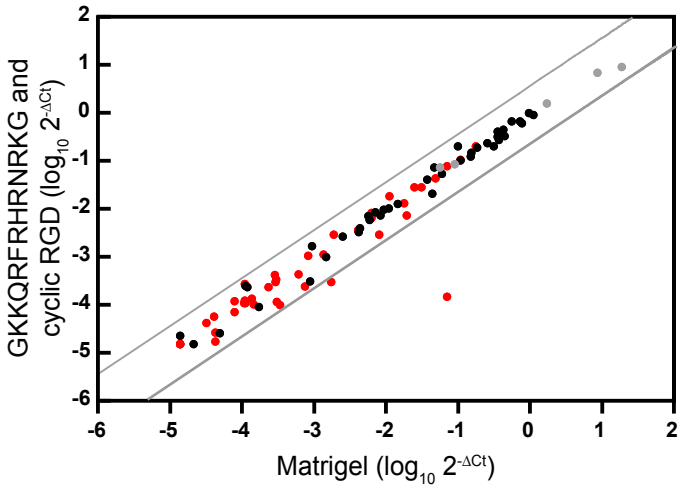
Supplementary Figure 5. Human ES cells cultured long-term on a synthetic surface maintain markers of pluripotency. (f) After three months (16 passages), cells (H14) cultured in mTeSR + Rock inhibitor (Ri) on a surface displaying the peptide GKKQRFRHRNRKG were analyzed by flow cytometry for markers of pluripotency. For comparison, differentiated cells were stained to serve as a biological negative. A biological negative is superior to an isotype control since their background staining will depend upon their specificity, concentration, degree of aggregation, and fluorophore:antibody ratio. Compared to differentiated cells (gray dot plots), cells cultured on the synthetic surface (orange dot plots) maintained high levels of markers of pluripotency and low levels of the marker of differentiation SSEA-1.

Supplementary Figure 6. Gene expression differences between hES cells cultured on defined surfaces presenting GKKQRHRNRKG and cells cultured on Matrigel.

a



c



• Lineage-specific Genes • Genes Associated with Pluripotency • Housekeeping Genes

b

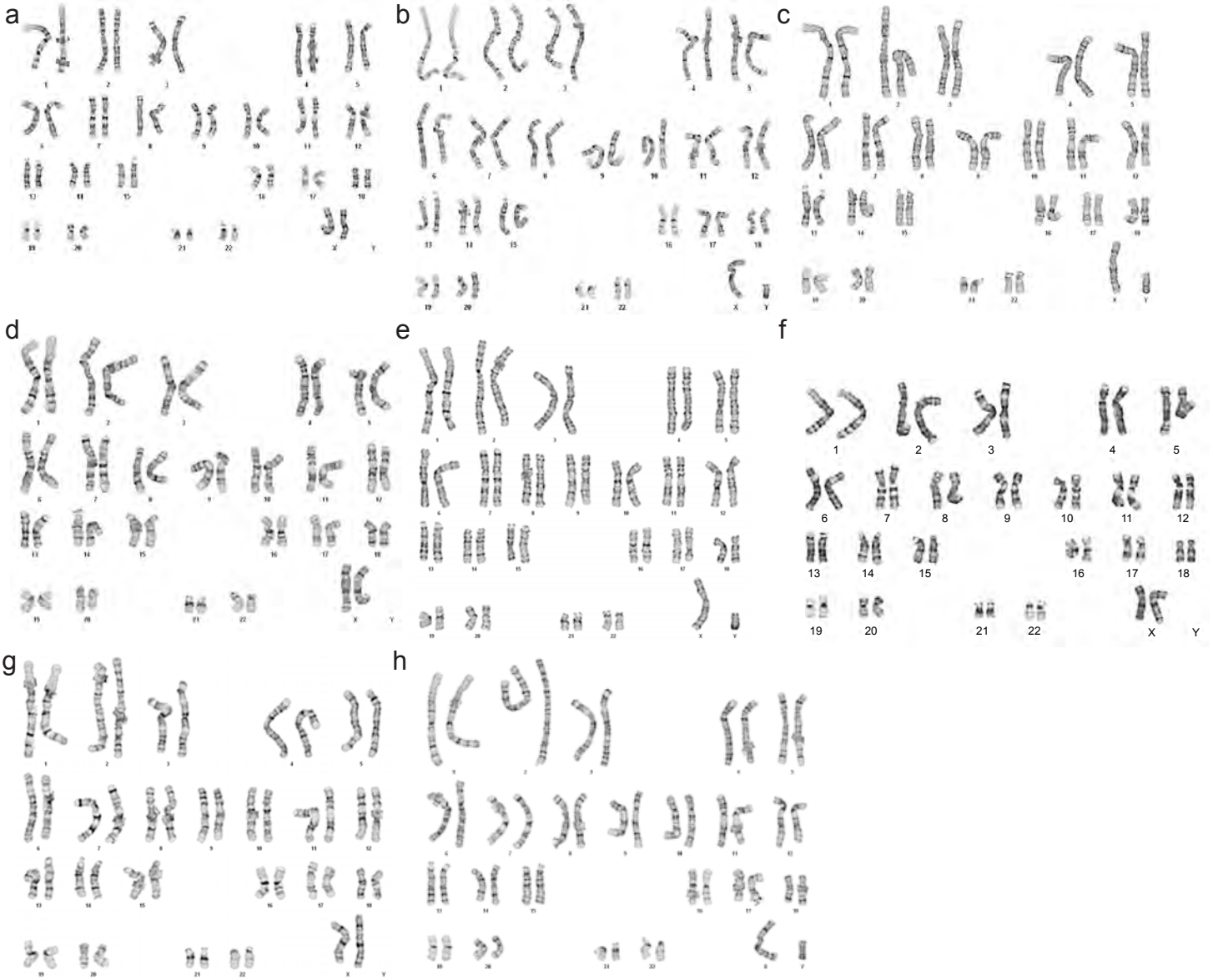
Genes Under-expressed		Genes Over-expressed	
Gene Symbol	Fold Regulation	Gene Symbol	Fold Regulation
AFP	-6.3	IAPP	4.9
CDX2	-117.1	LEFTY1	5.9
EDNRB	-626.5	UTF1	4.9
EOMES	-44.2		
GATA6	-9.9		
NEUROD1	-17.5		
PAX6	-35.8		
SOX17	-4.6		
T	-4.4		

d

Genes Under-expressed	
Gene Symbol	Fold Regulation
HBZ	-398.93
FOXA2	-4.9

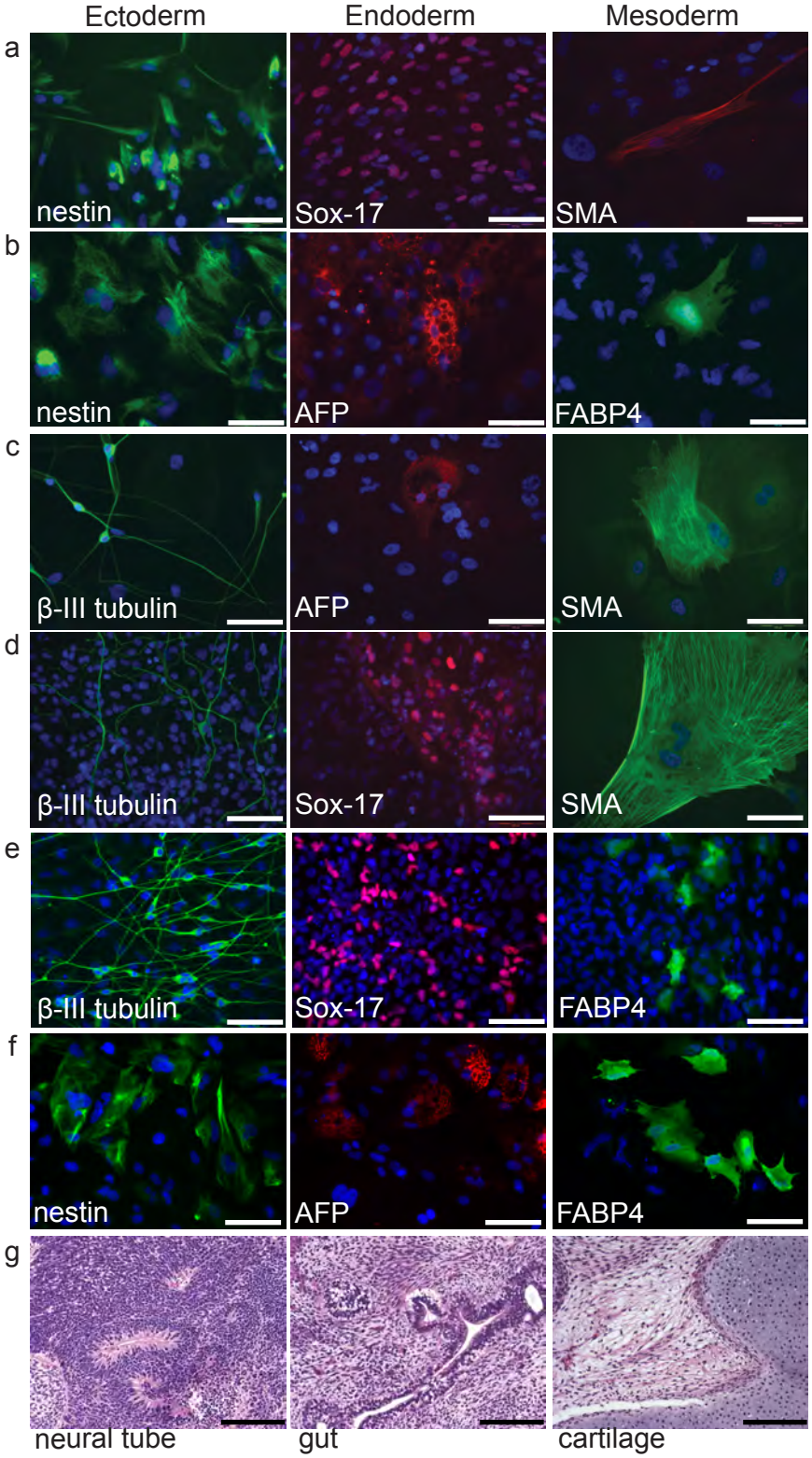
Supplementary Figure 6. Gene expression differences between hES cells cultured on defined surfaces presenting GKKQRFRHRNRKG and cells cultured on Matrigel. (a-b) Total RNA from hES cells (H9) cultured long-term in mTeSR + Ri on SAM surfaces presenting the peptide GKKQRFRHRNRKG or Matrigel was characterized using the human embryonic stem cell RT2 Profiler PCR array. Data represents the average of two technical replicates. (a) The relative expression levels for each gene in the two samples are plotted against each other in the scatter plot. The majority of genes including the markers of pluripotency (black dots) are expressed at similar levels between the two samples (gray lines represent a four-fold change). (b) Genes expression differences (> 4 fold) are listed in the table. These genes are typically associated with differentiation (AFP, CDX2, EOMES, NEUROD1, PAX6, SOX17, and T). These data suggest that the population of cells cultured on our simple, synthetic surfaces is more homogenous with fewer differentiated cells than the population cultured on Matrigel. (c-d) Total RNA from hES cells (SA02) cultured for 1 month in mTeSR on streptavidin-coated surfaces presenting the peptide GKKQRFRHRNRKG and cyclic RGD or Matrigel was characterized using the human embryonic stem cell RT2 Profiler PCR array. (c) The relative expression levels for each gene in the two samples are plotted against each other in the scatter plot. The majority of genes including the markers of pluripotency (black dots) are expressed at similar levels between the two samples (gray lines represent a four-fold change). (d) Genes expression differences (> 4 fold) are listed in the table.

Supplementary Figure 7. Human pluripotent stem cells cultured on surfaces presenting GKKQRHRNRKG maintain a normal karyotype.



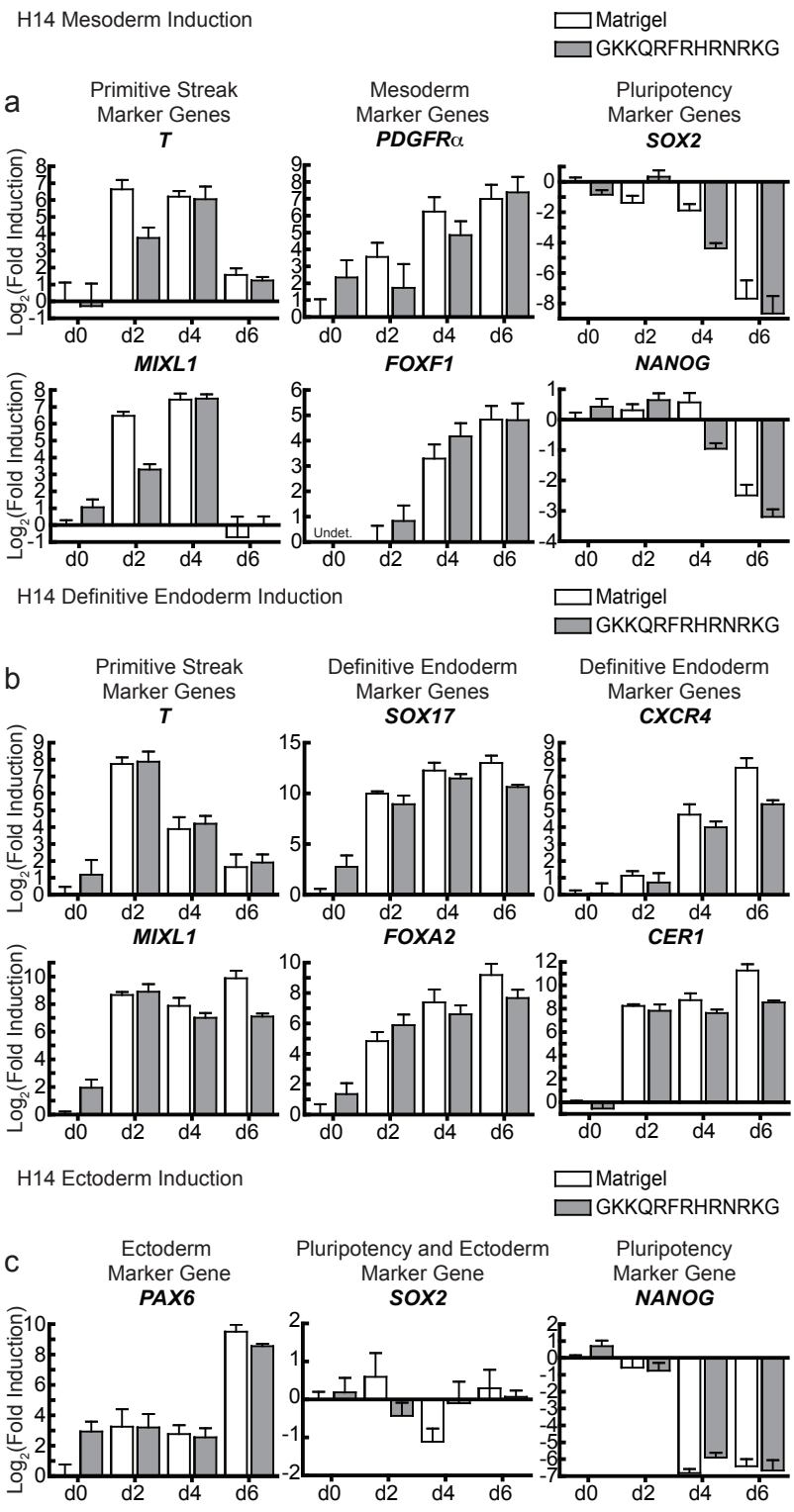
Supplementary Figure 7. Human pluripotent stem cells cultured on surfaces presenting GKKQRHRNRKG maintain a normal karyotype. (a) The karyotype of human ES cells (H7) cultured in mTeSR + Ri on SAMs displaying the peptide GKKQRFRHRNRKG for 1 month (6 passages) was analyzed by standard G banding. (b) Karyotype of human ES cells (H1) cultured in mTeSR + Ri on SAMs displaying the peptide GKKQRFRHRNRKG for 1 month (6 passages) was determined by standard G banding. (C) Karyotype of human ES cells (H13) cultured in mTeSR + Ri on SAMs displaying the peptide GKKQRFRHRNRKG for 2 months (14 passages) as determined by standard G banding. (D) Karyotype of induced pluripotent stem cells (IMR-90-1) cultured in mTeSR + Ri on SAMs displaying the peptide GKKQRFRHRNRKG for 1 month (7 passages) as determined by standard G banding. (e) No clonal abnormalities were detected by standard G banding for human ES cells (H14) cultured in mTeSR + Ri on surfaces displaying the peptide GKKQRFRHRNRKG for 3 months (20 passages). After muliptle multiple cryopreservations however, one out of forty cells examined had three copies of chromose 1$\bar{7}$. Trisomy 17 is a recurrent clonal abnormality in human ES cells. (f) Karyotype of human ES cells (H9$\bar{}$) cultured in mTeSR + Ri on SAMs displaying the peptide GKKQRFRHRNRKG for 3 months (17 passages) as determined by standard G banding. (g) Karyotype of human ES cells (H9) cultured in mTeSR on streptavidin-coated surfaces displaying a combination of GKKQRFRHRNRKG and cyclic RGD for 2 months as determined by standard G banding. (g) Karyotype of human ES cells (H14) cultured in mTeSR on streptavidin-coated surfaces displaying a combination of GKKQRFRHRNRKG and cyclic RGD for 1 month as determined by standard G banding.

Supplementary Figure 8. Pluripotent stem cells cultured on a synthetic surface maintain the ability to differentiate into many different cell types.



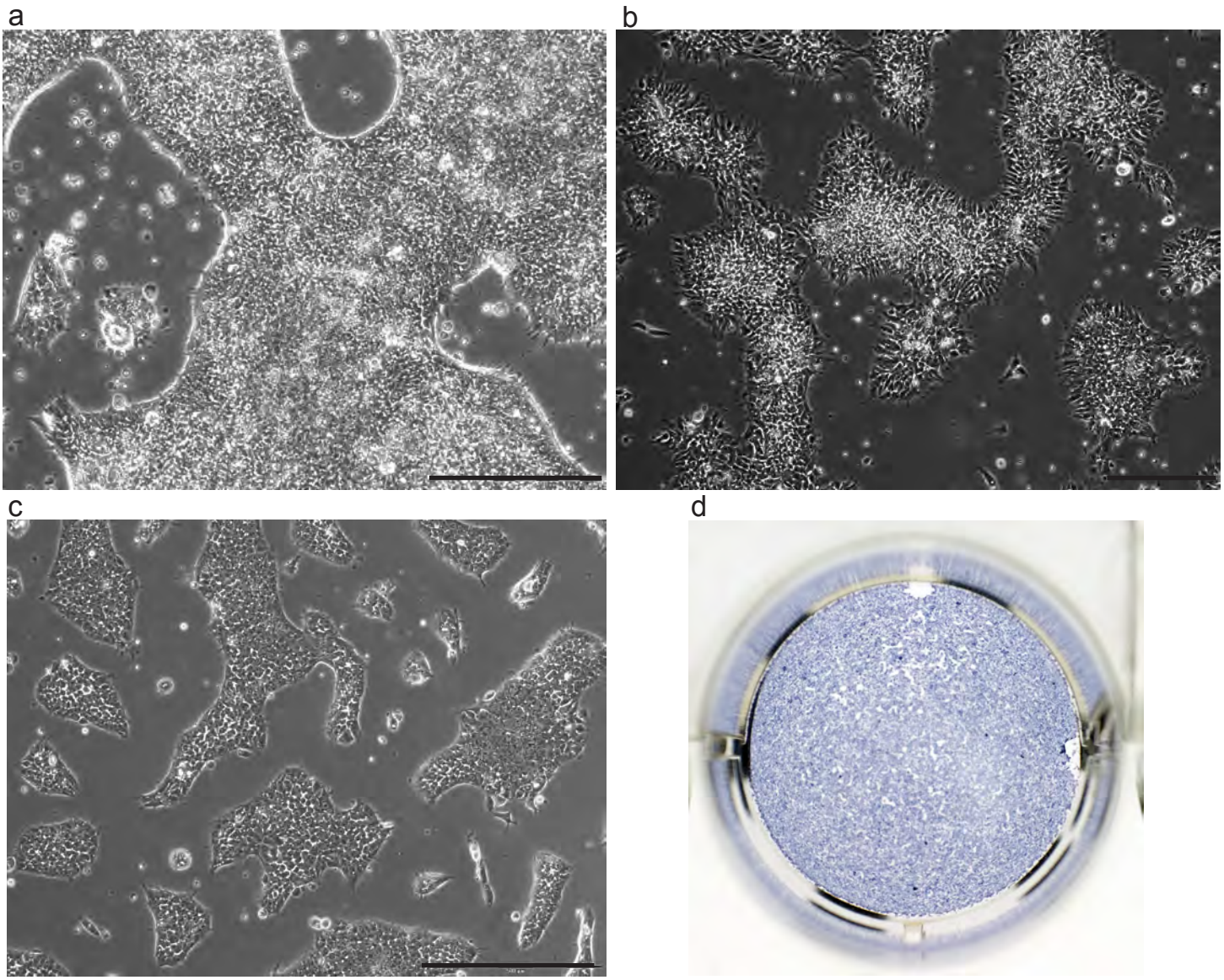
Supplementary Figure 8. Differentiation of pluripotent stem cells cultured on synthetic surfaces. (a-f) In vitro differentiation assay results. Differentiated cells were stained for markers of ectoderm (β-III tubulin, nestin), endoderm (Sox17, AFP), and mesoderm (SMA, FABP-4) and counterstained with DAPI (blue). (a) Results from H1 cells maintained for 1 month in mTeSR + Ri on SAMs presenting the heparin-binding peptide GKKQRFRHRNRKG. (b) Results from H7 cells cultured for 1 month in mTeSR + Ri on SAMs presenting the heparin-binding peptide GKKQRFRHRNRKG. (c) Results from iPS cells (IMR-90-1) maintained in mTeSR + Ri on SAMs presenting the heparin-binding peptide GKKQFRHRNRKG for 1 month. (d) Results from H13 cells maintained in mTeSR + Ri on SAMs presenting the heparin-binding peptide GKKQFRHRNRKG for 2 months. (e) Results from H14 cells maintained in mTeSR + Ri on SAMs presenting the heparin-binding peptide GKKQR-FRHRNRKG for 3 months. (f) Results from H9 cells maintained in mTeSR + Ri on SAMs presenting the heparin-binding peptide GKKQFRHRNRKG for 3 months. Scale bars, 100 μm. (g) Teratoma formation assay for H1 cells maintained in mTeSR + Ri on SAMs presenting the heparin-binding peptide GKKQRFRHRNRKG for 1 month (6 passages). Teratomas contained a mixture of tissues resembling the neural tube, the gut, and cartilage. Scale bars, 200 μm.

Supplementary Figure 9. Human ES cells cultured long-term on surfaces presenting GKKQR-FRHRNRKG can be directed to differentiate into each of the primary germ layers.



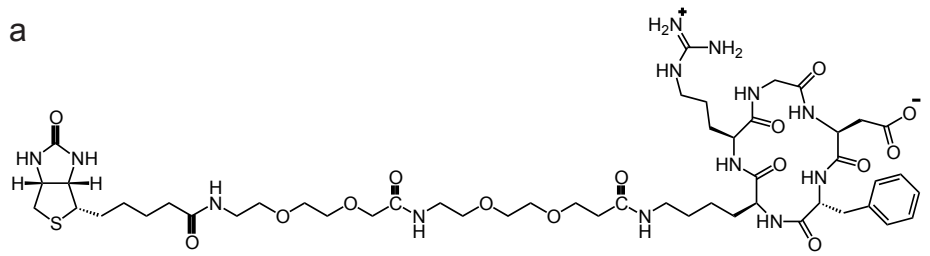
Supplementary Figure 9. Human ES cells cultured long-term on surfaces presenting GKKQR-FRHRNRKG can be directed to differentiate into each of the primary germ layers. (a) To induce mesoderm differentiation, cells (H14) were treated with Activin A for 1 day followed by BMP-4 for 5 days. Markers of the primitive streak (T and MIXL1) showed transient activiation from day 2 to 4. Expression of mesoderm markers (PDGFRα and FOXF1) increased through day 6, and pluripotency markers (SOX2 and NANOG) decreased. (b) To induce definitive endoderm differentiation, cells were treated for 6 days with Activin A . Expression of markers of the primitive streak (T and MIXL1) increased beginning on day 2. Expression of definitive endoderm markers (SOX17, FOXA2, CXCR4, and CER1) increased steadily beginning on day 2 or day 4. (c) To induce ectoderm differentiation, cells were treated with SB431542 and Noggin for 6 days to inhibit SMAD signaling. A marker for ectoderm (PAX6) was induced on day 6. A marker for pluripotency (NANOG) decreased beginning day 4. Cells continued to express SOX2, a marker common to both ectoderm and pluripotent cells. For each differentiation protocol, cells cultured long-term on GKKQRFRHRNRKG responded similarly to those cultured on Matrigel. Gene expression was analyzed via RT-qPCR, and error bars represent the standard deviation of triplicate reactions.

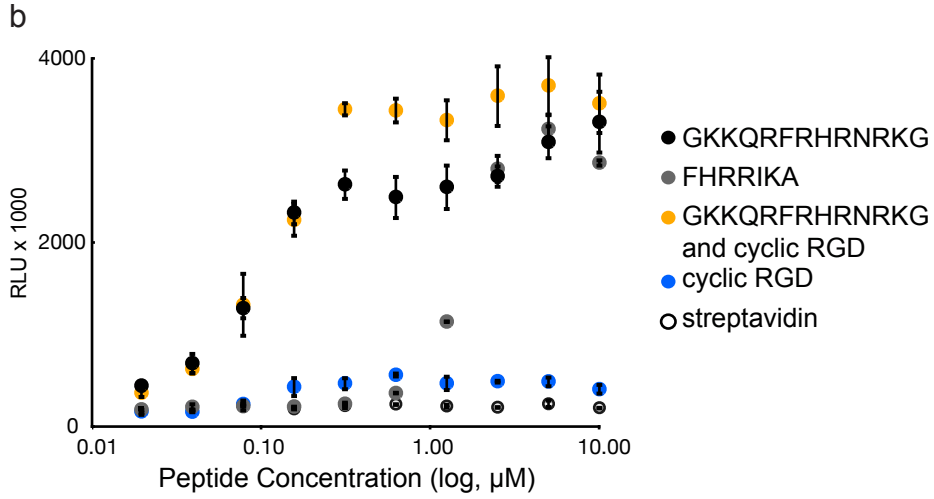
Supplementary Figure 10. Presentation of the heparin-binding peptide GKKQRFRHRNRKG on a variety of materials provides substances that can support pluripotent stem cell propagation.

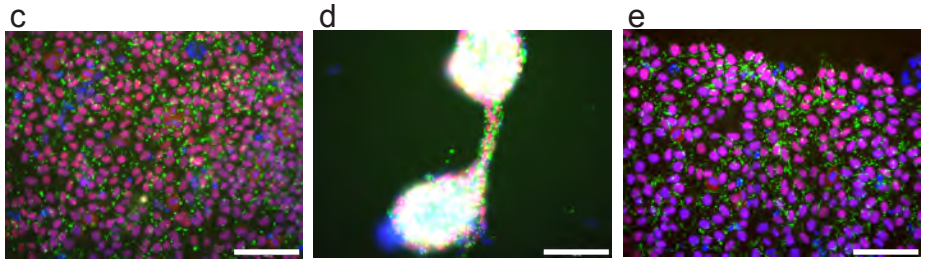


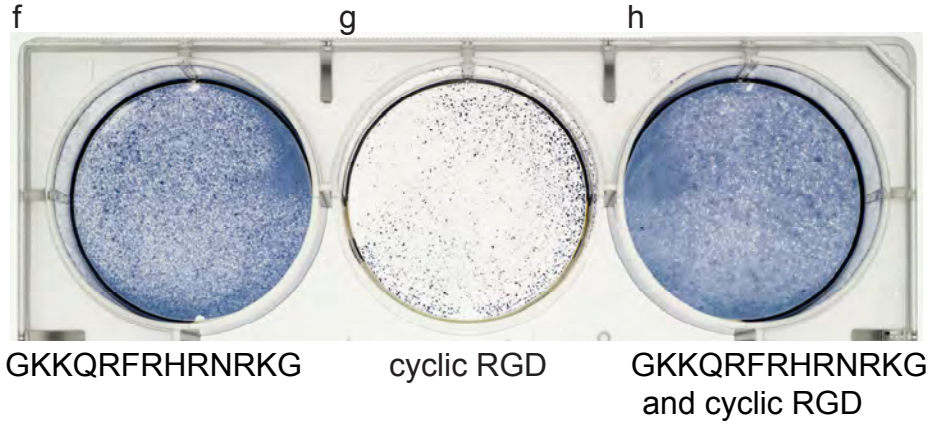
Supplementary Figure 10. Presentation of the heparin-binding peptide GKKQRFRHRNRKG on a variety of materials provides substances that can support pluripotent stem cell propagation. (a-b) Human ES cells (H9, a, H7, b) cultured (H9-12 days, H7-4 days) in mTeSR + Ri on surfaces generated by coupling CGKKQRFRHRNRKG to activated glass coverslips. (c) Induced pluripotent stem cells (IMR-90-1) cultured (8 days) on streptavidin-coated surfaces presenting biotinylated GKKQRFRHRNRKG. (d) Alkaline phosphatase staining (Vector Labs) for iPS cells (DF19-9 Clone 7T) cultured for three passages in mTeSR + Ri on streptavidin-coated surfaces presenting biotinylated GKKQRFRHRNRKG. Image represents one well of a 12-well plate. Scale bars, 200 μm, (a,c), 500 μm, (b). Streptavidin-coated surfaces presenting biotinylated peptides represent a versatile method for fabricating pluripotent stem cell culture surfaces of any size.

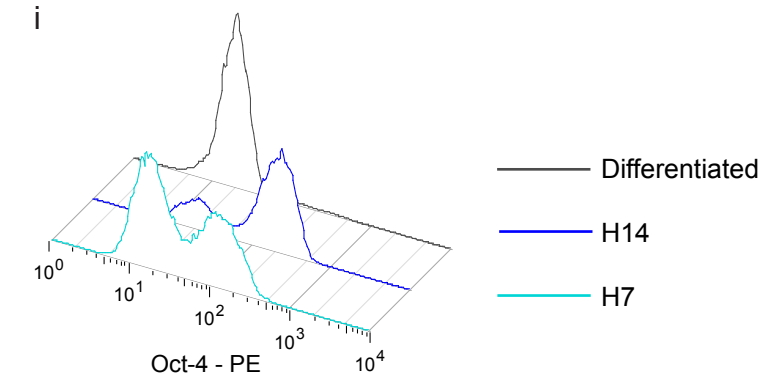
Supplementary Figure 11. Comparison of surfaces presenting cyclic RGD to those presenting GAG-binding peptides.











Supplementary Figure 11. Comparison of surfaces presenting cyclic RGD to those presenting GAG-binding peptides. (a) Structure of biotinylated cyclic RGD (Peptides International). (b) The effect of peptide concentration on hES cell adhesion. Human ES cells (H7) were dissociated using an enzyme-free buffer. Cells were seeded in mTeSR + Ri on to streptavidin-coated surfaces presenting biotinylated peptides. After 12 h, surfaces were washed and the cells were lysed. The cell lysate was used to approximate the cell number using Cell Titer Glo (Promega). Surfaces presenting the peptide GKKQRFRHRNRKG supported adhesion of the greatest number of cells and at concentrations lower than the other peptides tested. Error bars represent the average relative luminescence units of triplicate wells ± the standard deviation. (c-h) Human ES cells (SA02) grown on streptavidin-coated surfaces presenting biotinylated GKKQRFRHRNRKG (c,f), cyclic RGD (d,g), or a 7:3 combination of the two peptides (e,h) in mTeSR + Ri. After the formation of colonies, Y-27632 was removed. After 2 days, cells were immunostained for Oct-4 (green), SSEA-4 (red), and counter-stained with DAPI (blue) (c-e), or stained for alkaline phosphatase (f-h). Cells cultured on surfaces presenting GAG-binding peptides (c,e,f,h). Cells cultured on cyclic RGD alone form loosely bound, multilayered aggregates that detach in the absence of Ri (d,g). Scale bars, 100 μm (c-e). (i) Human ES cells (H7 and H14) cultured on streptavidin-coated surfaces presenting cyclic RGD for 3 passages (21 days) in mTeSR media supplemented with 5 μM Y-27632 were stained for Oct-4 and analyzed by flow cytometry. On surfaces presenting cyclic RGD, most H14 cells maintained Oct-4 levels (77% positive) but the H7 cell line showed sustantially decreased levels of Oct-4 (43% positive).

Supplementary Table 1. Collection of bioactive peptides.

Peptide Sequence	Origin	Ref.	Observations
GGGEVYVVAENQQGKSKA	NCAM	[1]	Able to support hES cell adhesion but not self-renewal. Cannot replace soluble bFGF.
KIPKASSVPTELSAISTLYL	BMP-2	[2]	Inconsistent adhesion and unable to support hES cell self-renewal
LSSKMYHTKGQEGSVSLRSSD	Dkk-1	[3]	Inconsistent adhesion
ADSQLIHGGLRS	Phage Display	[4]	Unable to support hES cell adhesion
MHRMPSFLPTTL	Phage Display	(unpublished)	Inconsistent adhesion. Cannot replace soluble TGFβ.
INPISGQ	N-cadherin	[5]	Unable to support hES cell adhesion
GSQIYALCNQFYTPAATGLYVD	Occludin	[6]	Unable to support hES cell adhesion
ATYTLFSHAVSSNGNAV	E-cadherin	[7]	Inconsistent adhesion and unable to support hES cell self-renewal
GGSTVHEILSKLSLEG	Annexin	[8]	Unable to support hES cell adhesion
GGGKHIFSDDSSE	Combinatorial Chemistry	[9]	Unable to support hES cell adhesion
KPHSRN	Fibronectin	[10]	Able to support hES cell adhesion but not self-renewal
GKKQRFRHRNRKG	Vitronectin	[11]	Able to support hES cell adhesion and self-renewal
GWQPPRARI	Fibronectin	[12]	Able to support hES cell adhesion and self-renewal
GGPEILDVPST	Fibronectin	[13]	Unable to support hES cell adhesion
GSDPGYIGSR	Laminin	[14]	Inconsistent adhesion and unable to support hES cell self-renewal
FHRRIKA	Bone Sialoprotein	[15]	Able to support hES cell adhesion and self-renewal
KGRGDS	Fibronectin, Vitronectin	[16]	Able to support hES cell adhesion but not self-renewal
GGIKVAV	Laminin	[17]	Unable to support hES cell adhesion

Supplementary Table 1. Collection of bioactive peptides. Specifically, we chose to display peptides that were developed to mimic soluble signaling factors, mediate cell—cell interactions, or reside in other key ECM proteins. Alkanethiols displaying peptides with reported bioactivity were synthesized and used to form SAMs. Some amino acids were added or changed from original publications to provide additional distance from the surface, remove cysteines that could

interfere with peptide presentation, or to match the corresponding human residues. The major criteria for the screen was to identify peptides that could support hES cell adhesion. Combinations of peptides were tested, but no beneficial combinations were identified.

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Supplementary Table 2. Primer sets used for quantitative real-time PCR analysis.

Gene Symbol	Forward Primer (5'-3')	Reverse Primer (5'-3')
SRP72	AGAAGACACTGATGGGACTGAG	CTCCTCTGTTCGACCCTGAAG
T	TTTCAAAGCAGTGGAGGAGCACAC	ACTGCATCATCTCCACAGTTGGGT
MIXL1	AACCTTTGGCTAGGCCGGAGATTA	GCAGGCAGTTCACATCTACCTCAA
PDGFRα	GCAAAGGCATCACAATGCTGG	GCACATTCGTAATCTCCACTGTC
FOXF1	CGGCTTCCGAAGGAAATGC	CAAGTGGCCGTTCATCATGC
SOX2	GAGTGGAAACTTTTGTCCGAGA	GAAGCGTGTACTTATCCTTCTTCAT
NANOG	CAGAAGGCCTCAGCACCTAC	ATTGTTCCAGGTCTGGTTGC
SOX17	TATCTGCACTTCGTGTGCAAGCCT	AGTGTGTAACACTGCTTCTGGCCT
FOXA2	GGGTGATTGCTGGTCGTTTGTTGT	GCTCATGTACGTCTTCATGCCGTT
CXCR4	TACACCGAGGAAATGGGCTCA	AGATGATGGAGTAGATGGTGGG
CER1	TCACTGTTTGCCTGCCAAGTTCAC	AGCCAGCATGTAGGATGTGTCCAT
PAX6	GCCCAGCTTCACCATGGCAAATAA	ATCATAACTCCGCCCATTCACCGA
OCT4	CAGTGCCCGAAACCCACAC	GGAGACCCAGCAGCCTCAAA