GSE10010_series_matrix.txtimp_info.txt

IL-22 r is 2

Found treated in !Series_overall_design "Control or IL-22-treated mouse colon in triplicate."

GSE10029-GPL6328_series_matrix.txtimp_info.txt

Vav2 r is 5

Found induced in !Series_summary "Tumor necrosis factor alpha (TNFalpha) plays an important role in immune regulation, inflammation, and autoimmunity. Chronic TNFalpha exposure has been shown to down-modulate T cell responses. In a mouse T cell hybridoma model, TNFalpha attenuated T cell receptor (TCR) signaling. We have confirmed that chronic TNFalpha and anti-TNFalpha exposure suppressed and increased T cell responses in BDC2.5 CD4+ T cells, respectively. The goal of this study is to analyze global transcriptional alterations resulting from TNFalpha treatment on TCR signaling pathways using cDNA microarrays. We found that genes involved in functional categories including T cell signaling, cell cycle, proliferation, ubiquitination, cytokine synthesis, calcium signaling, and apoptosis were modulated. Genes such as ubiquitin family genes, cytokine inducible SH2-containing genes, cyclin-dependent kinase inhibitors p21, p57, calmodulin family genes (calmodulin -V1, -V2, and ?V3) and calcium channel voltage- dependent, N type alpha1B subunit (CaV2.2) were induced by TNFalpha, while Vav2, Rho GTPase activating protein, calcium channel voltage dependent, L type alpha 1C subunit (CaV1.2), interleukin-1 (IL) receptor-associated kinase 1, and -V2 (IRAK-1and -2) and IL enhancer binding factor 3 were reduced by TNFalpha. Genes such as CaV1.2 and proliferating cell nuclear antigen, repressed by TNFalpha, were induced by anti-TNF treatment. Further, we showed that chronic TNFalpha exposure impaired NF-kappaB and AP-1 transactivation activity, leading to T cell unresponsiveness. Thus, our results present a detailed picture of transcriptional programs affected by chronic TNFalpha exposure, and provide candidate target genes, which may function to mediate TNFalpha induced T cell unresponsiveness."

GSE10029-GPL6329_series_matrix.txtimp_info.txt

Vav2 r is 5

Found induced in !Series_summary "Tumor necrosis factor alpha (TNFalpha) plays an important role in immune regulation, inflammation, and autoimmunity. Chronic TNFalpha exposure has been shown to down-modulate T cell responses. In a mouse T cell hybridoma model, TNFalpha attenuated T cell receptor (TCR) signaling. We have confirmed that chronic TNFalpha and anti-TNFalpha exposure suppressed and increased T cell responses in BDC2.5 CD4+ T cells, respectively. The goal of this study is to analyze global transcriptional alterations resulting from TNFalpha treatment on TCR signaling pathways using cDNA microarrays. We found that genes involved in functional categories including T cell signaling, cell cycle, proliferation, ubiquitination, cytokine synthesis, calcium signaling, and apoptosis were modulated. Genes such as ubiquitin family genes, cytokine inducible SH2-containing genes, cyclin-dependent kinase inhibitors p21, p57, calmodulin family genes (calmodulin -V1, -V2, and ?V3) and

calcium channel voltage- dependent, N type alpha1B subunit (CaV2.2) were induced by TNFalpha, while Vav2, Rho GTPase activating protein, calcium channel voltage dependent, L type alpha 1C subunit (CaV1.2), interleukin-1 (IL) receptor-associated kinase 1, and -V2 (IRAK-1and -2) and IL enhancer binding factor 3 were reduced by TNFalpha. Genes such as CaV1.2 and proliferating cell nuclear antigen, repressed by TNFalpha, were induced by anti-TNF treatment. Further, we showed that chronic TNFalpha exposure impaired NF-kappaB and AP-1 transactivation activity, leading to T cell unresponsiveness. Thus, our results present a detailed picture of transcriptional programs affected by chronic TNFalpha exposure, and provide candidate target genes, which may function to mediate TNFalpha induced T cell unresponsiveness."

GSE10029-GPL6330_series_matrix.txtimp_info.txt

Vav2 r is 5

Found induced in !Series_summary "tumor necrosis factor alpha (TNFalpha) plays an important role in immune regulation, inflammation, and autoimmunity. Chronic TNFalpha exposure has been shown to down-modulate T cell responses. In a mouse T cell hybridoma model, TNFalpha attenuated T cell receptor (TCR) signaling. We have confirmed that chronic TNFalpha and anti-TNFalpha exposure suppressed and increased T cell responses in BDC2.5 CD4+ T cells, respectively. The goal of this study is to analyze global transcriptional alterations resulting from TNFalpha treatment on TCR signaling pathways using cDNA microarrays. We found that genes involved in functional categories including T cell signaling, cell cycle, proliferation, ubiquitination, cytokine synthesis, calcium signaling, and apoptosis were modulated. Genes such as ubiquitin family genes, cytokine inducible SH2-containing genes, cyclin-dependent kinase inhibitors p21, p57, calmodulin family genes (calmodulin -V1, -V2, and ?V3) and calcium channel voltage- dependent, N type alpha1B subunit (CaV2.2) were induced by TNFalpha, while Vav2, Rho GTPase activating protein, calcium channel voltage dependent, L type alpha 1C subunit (CaV1.2), interleukin-1 (IL) receptor-associated kinase 1, and -V2 (IRAK-1and -2) and IL enhancer binding factor 3 were reduced by TNFalpha. Genes such as CaV1.2 and proliferating cell nuclear antigen, repressed by TNFalpha, were induced by anti-TNF treatment. Further, we showed that chronic TNFalpha exposure impaired NF-kappaB and AP-1 transactivation activity, leading to T cell unresponsiveness. Thus, our results present a detailed picture of transcriptional programs affected by chronic TNFalpha exposure, and provide candidate target genes, which may function to mediate TNFalpha induced T cell unresponsiveness."

GSE10134_series_matrix.txtimp_info.txt

estrogen receptor r is 2

Found expressing in !Series_summary "We employed a gene complementation strategy combined with microarray screening to identify miRNAs involved in the formation of erythroid (red blood) cells. To search for GATA-1-regulated erythroid miRNAs, we used the Gata-1– erythroblast line G1E. These cells proliferate in culture as immature erythroid precursors and undergo terminal maturation when GATA-1 activity is restored. G1E-ER4 is a sub-line stably expressing an estrogen-activated form of GATA-1 (GATA-1 fused to the ligand binding domain of the estrogen receptor). Treatment of G1E-ER4 cells with

estradiol induces a GATA-1-regulated program of gene expression with concomitant cellular maturation. We used a microarray to evaluate the expression of 292 different miRNAs in G1E-ER4 cells at 0 versus 24 hours after GATA-1 activation. Affymetrix gene expression profiling has previously been deposited (GEO accession no. GSE628)."

GSE10162_series_matrix.txtimp_info.txt

Clc5 r is 3

Found knockout in !Series_summary	"Dent disease has multiple defects attributed to proximal tubule malfunction including low molecular weight proteinuria, aminoaciduria, phosphaturia and glycosuria. In order to understand the changes in kidney function of the Clc5 transporter gene knockout mouse model of Dent disease, we examined gene expression profiles from proximal tubules of mouse kidneys."

GSE10162_series_matrix.txtimp_info.txt

Clcn5 r is 1

Found knockout in !Series_summary	"Overall 720 genes are expressed differentially in the proximal tubules of the Dent Clcn5 knockout mouse model compared to those of control wild type mice. The fingerprint of these gene changes may help us to understand the phenotype of Dent disease."

GSE10162_series_matrix.txtimp_info.txt

Clcn5 r is 4

Found knockout in !Series_summary	"Keywords: gene knockout, mouse, Clcn5, Dent's disease"

GSE10162_series_matrix.txtimp_info.txt

Clcn5 r is 1

Found knockout in !Series_overall_design	"Renal proximal tubules were dissected from wild type and Clcn5 knockout mice. Mice were anesthetized with halothane, the abdominal aorta of each animal was accessed and the left kidney was perfused with an ice-cold salt. Proximal tubule dissection was performed in an ice-cold salt solution. After dissection of approximately 80-100 segments of 2 mm in length per kidney, the RNA for 3-4 mice was combined to have enough RNA per chip."

GSE10166_series_matrix.txtimp_info.txt

aryl hydrocarbon receptor r is 3

Found activation in !Series_summary	"Over-activation of the aryl hydrocarbon receptor by TCDD in mice leads among other phenotypes to a severe thymic atrophy accompanied by immunosuppression. TCDD causes a block in thymocyte maturation and a preferential emigration of immature CD4-CD8- DN thymocytes (recent thymic emigrants) into the periphery. As part of this study gene expression profiles from DN thymocytes and thymic emigrants were generated from TCDD and solvent control mice"

GSE10167_series_matrix.txtimp_info.txt

Tcof1 r is 1

Found haploin-sufficiency in !Series_summary "The object of this study was to identify genes transcriptionally upregulated and downregulated in response to Tcof1 haploin-sufficiency during mouse embryogensis"

GSE10168_series_matrix.txtimp_info.txt

aryl hydrocarbon receptor r is 3

Found activation in !Series_summary "Over activation of the aryl hydrocarbon receptor (AhR) by TCDD results ampng other phenotypes in severe thymic atrophy accompanied by immunosuppression. The link between thymic atrophy, skewed thymocyte differntiation and immunosuppression is still not fully resolved. This study investigates the TCDD elicted exprssion changes in the ET, cortical thymus epithelial cell line."

GSE10175_series_matrix.txtimp_info.txt

Tcfap2c r is 1

Found mutant in !Series_title "Comparison of gene expression in the epidermis of Tcfap2c mutant and control skin at embryonic day 16.5"

GSE10175_series_matrix.txtimp_info.txt

Ly6/Plaur domain containing 1 r is 6

Found mutant in !Series_summary "The development of the epidermis, a stratified squamous epithelium, is dependent on the regulated differentiation of keratinocytes. Differentiation begins with the initiation of stratification, a process tightly controlled through proper gene expression. AP-2γ is expressed in skin and previous research suggested a pathway where p63 gene induction results in increased expression of AP-2γ which in turn is responsible for induction of K14. This study uses a conditional gene ablation model to further explore the role of AP-2γ in skin development. Mice deficient for AP-2γ exhibited delayed expression of p63, K14, and K1, key genes required for development and differentiation of the epidermis. In addition, microarray analysis of E16.5 skin revealed delayed expression of additional late epidermal differentiation genes: filaggrin, repetin and secreted Ly6/Plaur domain containing 1, in mutant mice. The genetic delay in skin development was further confirmed by a functional delay in the formation of an epidermal barrier. These results document an important role for AP-2γ in skin development, and reveal the existence of regulatory factors that can compensate for AP-2γ in its absence."

GSE10175_series_matrix.txtimp_info.txt

secreted Ly6/Plaur domain containing 1 r is 7

Found mutant in !Series_summary "The development of the epidermis, a stratified squamous epithelium, is dependent on the regulated differentiation of keratinocytes. Differentiation begins with the initiation of stratification, a process tightly controlled through proper gene expression. AP-2γ is expressed in skin and previous research suggested a pathway where p63 gene induction results in increased expression of AP-2γ which in turn is responsible for induction of K14. This study uses a conditional gene ablation model to further explore the role of AP-2γ in skin development. Mice deficient for AP-2γ exhibited delayed expression of p63, K14, and K1, key genes required for development and differentiation of the epidermis. In addition, microarray analysis of E16.5 skin revealed delayed expression of additional late epidermal differentiation genes: filaggrin, repetin and secreted Ly6/Plaur domain containing 1, in mutant mice. The genetic delay in skin development was further confirmed by a functional delay in the formation of an epidermal barrier. These results document an important role for AP-2γ in skin development, and reveal the existence of regulatory factors that can compensate for AP-2γ in its absence."

GSE10182_series_matrix.txtimp_info.txt

Nod2 r is 2

Found induced in !Series_summary "The transcriptome induced via Nod2 stimulation is greatly expanded in TLR2-tolerant macrophages."

GSE10182_series_matrix.txtimp_info.txt

Nod2 r is 1

Found stimulation in !Series_summary "The transcriptome induced via Nod2 stimulation is greatly expanded in TLR2-tolerant macrophages."

GSE10192_series_matrix.txtimp_info.txt

peroxisome proliferator-activated receptor gamma r is 8

Found activation in !Series_summary "Rosiglitazone (Rosi), a member of the thiazolidinedione class of drugs used to treat type 2 diabetes, activates the adipocyte-specific transcription factor peroxisome proliferator-activated receptor gamma (PPARg). This activation causes bone loss in animals and humans, at least in part due to suppression of osteoblast differentiation from marrow mesenchymal stem cells (MSC). In order to identify mechanisms by which PPARg2 suppresses osteoblastogenesis and promotes adipogenesis in MSC, we have analyzed the PPARg2 transcriptome in response to Rosi. A total of 4,252 transcriptional changes resulted when Rosi (1 uM) was applied to the U-33 marrow stromal cell line, stably transfected with PPARg2 (U-33/g2), as compared to non-induced U-33/g2 cells. Differences between U-33/g2 and U-33 cells stably transfected with empty vector (U-33/c) comprised 7,928 transcriptional changes, independent of Rosi. Cell type-, time- and treatment-specific gene clustering uncovered distinct patterns of PPARg2 transcriptional control of MSC lineage commitment. The earliest changes accompanying Rosi activation of PPARg2 included adjustments in morphogenesis, Wnt signaling, and immune responses, as well as sustained induction of lipid metabolism. Expression

signatures influenced by longer exposure to Rosi provided evidence for distinct mechanisms governing the repression of osteogenesis and stimulation of adipogenesis. Our results suggest interactions that could lead to the identification of a “master” regulatory scheme controlling osteoblast differentiation. "

GSE10210_series_matrix.txtimp_info.txt

VE-cadherin r is 1

Found expressing in !Series_title "Gene expression analysis of embryonic stem cells expressing VE-cadherin (CD144) during endothelial differentiation"

GSE10210_series_matrix.txtimp_info.txt

CD41 r is 6

Found expressing in !Series_overall_design "We identified four populations of cells; cells expressing VEGF-R2 (day 2.5), CD41 expressing cells (day 3.5), cells expressing CD144 (VE-Cadherin, day 3.5), and cells expressing CD144 (day 6.5). In addition to this, we have also obtained the negative control cells at each time such as VEGF-R2 (day 2.5) negative, CD41 negative (day 3.5), CD144 negative (VE-Cadherin, day 3.5), and negative CD144 (day 6.5). RNA for the microarray experiments were obtained in duplicate from two separately conducted experiments using the murine embryonic stem cells.."

GSE10210_series_matrix.txtimp_info.txt

R2 r is 2

Found expressing in !Series_overall_design "We identified four populations of cells; cells expressing VEGF-R2 (day 2.5), CD41 expressing cells (day 3.5), cells expressing CD144 (VE-Cadherin, day 3.5), and cells expressing CD144 (day 6.5). In addition to this, we have also obtained the negative control cells at each time such as VEGF-R2 (day 2.5) negative, CD41 negative (day 3.5), CD144 negative (VE-Cadherin, day 3.5), and negative CD144 (day 6.5). RNA for the microarray experiments were obtained in duplicate from two separately conducted experiments using the murine embryonic stem cells.."

GSE10216_series_matrix.txtimp_info.txt

Emx2 r is 1

Found KO in !Series_overall_design "The epithelial cells of the gonadal primordium were obtained by Laser Microdissection System. The specimens prepared from three individuals were mixed as one pool. There are three experimental replicates in each genotype, 3-pools of wild-type and 3-pools of Emx2 KO mouse."

GSE10218_series_matrix.txtimp_info.txt

K5 r is 2

Found deletion in !Series_title "Keratinocyte specific Fos-deletion in K5-Sos-F mouse tumor model"

GSE10218_series_matrix.txtimp_info.txt

K5 r is 5

Found deletion in !Series_summary "Keywords: Fos-deletion, Fos-floxed, K5-SOS-F mouse tumor model, skin papilloma, global gene expression, microarray, Fos target in skin carcinogenesis"

GSE10235_series_matrix.txtimp_info.txt

NF-kappaB r is 3

Found inhibition in !Series_title "Transgenic inhibition of astroglial NF-kappaB in experimental autoimmune encephalomyelitis"

GSE10250_series_matrix.txtimp_info.txt

estrogen receptor r is 6

Found expressing in !Series_overall_design "Initially, we compared RNA from Ad-Cre infected Hdac3FL/+ and Hdac3FL/- MEFs. In this analysis, RNA isolated from 64hr and 72hr post Ad-Cre infection were used. We extended the analysis to Hdac3FL/- MEFs expressing Cre-ER (tamoxifen responsive Cre-estrogen receptor fusion protein). In this analysis, two different MEF preparations (repl1 and repl2), two different time points (64hr and 72hr) were used for array analysis with tamoxifen-treated MEFs. For vehicle (ethanol) treated MEFs, two different MEF preparations (repl1 and repl2), and a single timepoint (64hr) was used."

GSE10273_series_matrix.txtimp_info.txt

IRF-4 r is 5

Found lacking in !Series_summary "Productive rearrangement of the immunoglobulin heavy chain locus triggers a major developmental checkpoint that promotes limited clonal expansion of pre-B cells, culminating in cell cycle arrest and rearrangement of the kappa (κ) or lambda (λ) light-chain loci. B lineage cells lacking the related transcription factors IRF-4 and IRF-8 undergo a developmental arrest at the cycling pre-B cell stage and are blocked for light-chain recombination. Using Irf-4,8-/- pre-B cells we demonstrate that two pathways converge to synergistically drive light-chain rearrangement, a process that is not simply activated by cell cycle exit. One pathway is directly dependent on IRF-4, whose expression is elevated by pre-BCR signaling. IRF-4 targets the κ 3' and λ enhancers to increase locus accessibility and positions a kappa allele away from pericentromeric heterochromatin. The other pathway is triggered by attenuation of IL-7 signaling and results in activation of the κ intronic enhancer via binding of the transcription factor, E2A. Intriguingly, IRF-4 regulates the expression of CXCR4 and promotes the migration of pre-B cells in response to the chemokine CXCL12. We propose that IRF-4 coordinates the two pathways regulating light-chain recombination by positioning pre-B cells away from IL-7 expressing stromal cells."

GSE10273_series_matrix.txtimp_info.txt

IRF-8 r is 5

Found lacking in !Series_summary "Productive rearrangement of the immunoglobulin heavy chain locus triggers a major developmental checkpoint that promotes limited clonal expansion of pre-B cells, culminating in cell cycle arrest and rearrangement of the kappa (κ) or lambda (λ) light-chain loci. B lineage cells lacking the related transcription factors IRF-4 and IRF-8 undergo a developmental arrest at the cycling pre-B cell stage and are blocked for light-chain recombination. Using Irf-4,8-/- pre-B cells we demonstrate that two pathways converge to synergistically drive light-chain rearrangement, a process that is not simply activated by cell cycle exit. One pathway is directly dependent on IRF-4, whose expression is elevated by pre-BCR signaling. IRF-4 targets the κ 3' and λ enhancers to increase locus accessibility and positions a kappa allele away from pericentromeric heterochromatin. The other pathway is triggered by attenuation of IL-7 signaling and results in activation of the κ intronic enhancer via binding of the transcription factor, E2A. Intriguingly, IRF-4 regulates the expression of CXCR4 and promotes the migration of pre-B cells in response to the chemokine CXCL12. We propose that IRF-4 coordinates the two pathways regulating light-chain recombination by positioning pre-B cells away from IL-7 expressing stromal cells."

GSE10318_series_matrix.txtimp_info.txt

Tbx3 r is 2

Found activation in !Series_overall_design "Nppa-Cre4 (Cre4) mice were crossed with CT mice to obtain efficient activation of Tbx3 in atria of double transgenic Cre4-CT mice, as previously described (Hoogaars et al., 2007). To investigate the gene expression profile of atria of Cre4-CT mice, we performed whole genome microarray analysis using Sentrix Mouse-6 oligonucleotide beadchips."

GSE10318_series_matrix.txtimp_info.txt

Tbx3 r is 1

Found expressing in !Series_summary "Methods and Results: We analyzed mice ectopically expressing Tbx3 in the atrial myocardium by genome-wide microarray and expression analysis. We found a prominent role for Tbx3 in defining the nodal phenotype by repressing working myocardial genes (sarcomeric, mitochondrial, fast conduction) and cell proliferation regulators, and in inducing node-associated genes. Moreover, there was a striking induction of genes associated with endocardial cushions and mesenchyme. Using gain-of-function models, we found that in the developing heart both Tbx2 and Tbx3 induce ectopic Bmp2 and Tgfb2 expression and endocardial cushion formation. Analysis of compound Tbx2/Tbx3 mutant embryos revealed that upon loss of more than two functional alleles, expansion of the AV myocardium does not occur and AV cushions fail to form."

GSE10341_series_matrix.txtimp_info.txt

tyrosine hydroxylase r is 2

Found null in !Series_overall_design "The first aim was to compare gene expression of E12.5 mouse fetuses between wild type (Th+/+) versus tyrosine hydroxylase null (Th-/-) animals from normoxic dams. This resulted in 6 arrays."

GSE10341_series_matrix.txtimp_info.txt

tyrosine hydroxylase r is 2

Found null in !Series_overall_design "The second aim compared wild type and tyrosine hydroxylase null E12.5 fetuses from dam exposed to hypoxia (8% oxygen) for 6 hours prior to sacrifice at E12.5 of gestation. This resulted in 6 arrays."

GSE10386_series_matrix.txtimp_info.txt

RIP140 r is 1

Found knockout in !Series_overall_design "for macrophages from RIP140 knockout or wild-type mice. "

GSE10389-GPL81_series_matrix.txtimp_info.txt

beta c r is 2

Found stimulated in !Series_summary "STAT5A and STAT5B proteins belong to the family of signal transducers and activators of transcription. They are encoded by 2 separate genes with 91% identity in their amino acid sequences. Despite their high degree of conservation, STAT5A and STAT5B exert non-redundant functions, resulting at least in part from differences in target gene activation. To better characterize the differential contribution of STAT5A and STAT5B in gene regulation, we performed single or double knock-down of STAT5A and STAT5B using small interfering RNA. Subsequent gene expression profiling and RT-qPCR analyses of IL-3-stimulated Ba/F3-beta cells led to the identification of putative novel STAT5 target genes. Chromatin immunoprecipitation assays analyzing the corresponding gene loci identified unusual STAT5 binding sites compared to conventional STAT5 responsive elements. Some of the STAT5 targets identified are upregulated in several human cancers, suggesting that they might represent potential oncogenes in STAT5-associated malignancies."

GSE10389-GPL82_series_matrix.txtimp_info.txt

beta c r is 2

Found stimulated in !Series_summary "STAT5A and STAT5B proteins belong to the family of signal transducers and activators of transcription. They are encoded by 2 separate genes with 91% identity in their amino acid sequences. Despite their high degree of conservation, STAT5A and STAT5B exert non-redundant functions, resulting at least in part from differences in target gene activation. To better characterize the differential contribution of STAT5A and STAT5B in gene regulation, we performed single or double knock-down of STAT5A and STAT5B using small interfering RNA. Subsequent gene expression profiling and RT-qPCR analyses of IL-3-stimulated Ba/F3-beta cells led to the identification of putative

novel STAT5 target genes. Chromatin immunoprecipitation assays analyzing the corresponding gene loci identified unusual STAT5 binding sites compared to conventional STAT5 responsive elements. Some of the STAT5 targets identified are upregulated in several human cancers, suggesting that they might represent potential oncogenes in STAT5-associated malignancies."

GSE10389-GPL83_series_matrix.txtimp_info.txt

beta c r is 2

Found stimulated in !Series_summary "STAT5A and STAT5B proteins belong to the family of signal transducers and activators of transcription. They are encoded by 2 separate genes with 91% identity in their amino acid sequences. Despite their high degree of conservation, STAT5A and STAT5B exert non-redundant functions, resulting at least in part from differences in target gene activation. To better characterize the differential contribution of STAT5A and STAT5B in gene regulation, we performed single or double knock-down of STAT5A and STAT5B using small interfering RNA. Subsequent gene expression profiling and RT-qPCR analyses of IL-3-stimulated Ba/F3-beta cells led to the identification of putative novel STAT5 target genes. Chromatin immunoprecipitation assays analyzing the corresponding gene loci identified unusual STAT5 binding sites compared to conventional STAT5 responsive elements. Some of the STAT5 targets identified are upregulated in several human cancers, suggesting that they might represent potential oncogenes in STAT5-associated malignancies."

GSE10403_series_matrix.txtimp_info.txt

granzyme B r is 4

Found expressing in !Series_summary "IL-2 and IL-21 are closely related cytokines that might have arisen by gene duplication. Both cytokines promote the function of effector CD8+ T cells, but their distinct effects on antigen-driven differentiation of naïve CD8+ T cells into effector CD8+ T cells are not clearly understood. We found that antigen-induced expression of eomesodermin and maturation of naïve CD8+ T cells into granzyme B and CD44 expressing effector CD8+ T cells was enhanced by IL-2, but, unexpectedly, suppressed by IL-21. Furthermore, IL-21 repressed expression of IL-2Ra and inhibited IL-2-mediated acquisition of a cytolytic CD8+ T cell phenotype. Despite its inhibitory effects, IL-21 did not induce anergy, but instead potently enhanced the capacity of cells to mediate tumor regression upon adoptive transfer. In contrast, IL-2, surprisingly, impaired the subsequent anti-tumor function of transferred cells. Gene expression studies revealed a distinct IL-21-program that was characterized phenotypically by increased expression of L-selectin and functionally by enhanced anti-tumor immunity that was not reversed by secondary in vitro stimulation with antigen and IL-2. Thus, the efficacy of CD8+ T cells for adoptive immunotherapy can be influenced by opposing differentiation programs conferred by IL-2 and IL-21, a finding with important implications for the development of cellular cancer therapies."

GSE10421_series_matrix.txtimp_info.txt

Smad4 r is 3

Found deficient in !Series_summary "Results: Among 1419 transcripts significantly modulated by the dietary iron content, four were regulated similarly to the hepcidin genes Hamp1 and Hamp2. They are coding for Bmp6, the regulator of Bmp/Smad signal transduction Smad7, the negative regulator of basic helix-loop-helix (bHLH) proteins Id1, and a protein with a bHLH domain, Atoh8. The iron overload developed by Smad4 and Hamp1-deficient mice also increased Bmp6 transcription. Body iron stores influence Smad1/5/8 phosphorylation and, as shown by analysis of mice with liver-specific disruption of Smad4, the binding partner for the receptor-activated Smads is necessary for activation of Smad7, Id1, and Atoh8 transcription by iron."

GSE10422_series_matrix.txtimp_info.txt

Traf3 r is 3

Found knockout in !Series_title "Traf2 and Traf3 B cell knockout mice and Baff tg mice - gene expression in lymph node B cells"

GSE10422_series_matrix.txtimp_info.txt

Traf2 r is 3

Found knockout in !Series_overall_design "Lymph node B cells were purified from Traf2 B cell knockout mice, Traf3 B cell knockout mice, Baff-tg mice and respective controls. RNA was extracted and hybridised to Affymetrix 430 2.0 Mouse Genome Arrays. Samples were processed and hence analysed on three spearate days. Day 1 two control mice: Traf2lox/lox pool and CD19-cretg were compared to two knockout mice: Traf2DB 80 and Traf3DB 94. On Day 2 three control mice: Traf2lox/lox 77, Traf2lox/lox 79 and Traf3lox/lox 97 were compared to two knockout mice: Traf2DB 76 and Traf3DB 01. On Day 3 three control mice: WT33, WT34, WT35 were compared to three Baff-tg mice: Baff-tg 99, Baff-tg 100, Baff-tg 101."

GSE10422_series_matrix.txtimp_info.txt

Traf3 r is 3

Found knockout in !Series_overall_design "Lymph node B cells were purified from Traf2 B cell knockout mice, Traf3 B cell knockout mice, Baff-tg mice and respective controls. RNA was extracted and hybridised to Affymetrix 430 2.0 Mouse Genome Arrays. Samples were processed and hence analysed on three spearate days. Day 1 two control mice: Traf2lox/lox pool and CD19-cretg were compared to two knockout mice: Traf2DB 80 and Traf3DB 94. On Day 2 three control mice: Traf2lox/lox 77, Traf2lox/lox 79 and Traf3lox/lox 97 were compared to two knockout mice: Traf2DB 76 and Traf3DB 01. On Day 3 three control mice: WT33, WT34, WT35 were compared to three Baff-tg mice: Baff-tg 99, Baff-tg 100, Baff-tg 101."

GSE10424-GPL81_series_matrix.txtimp_info.txt

F10 r is 2

Found + in !Series_summary "The Differentiation is leaded by removing the Proliferation Medium (Ham's F10 Medium + growth factors) and feeding with Differentiation Medium (DMEM hg + 2.5% Horse serum)."

GSE10424-GPL82_series_matrix.txtimp_info.txt

F10 r is 2

Found + in !Series_summary "The Differentiation is leaded by removing the Proliferation Medium (Ham's F10 Medium + growth factors) and feeding with Differentiation Medium (DMEM hg + 2.5% Horse serum)."

GSE10424-GPL83_series_matrix.txtimp_info.txt

F10 r is 2

Found + in !Series_summary "The Differentiation is leaded by removing the Proliferation Medium (Ham's F10 Medium + growth factors) and feeding with Differentiation Medium (DMEM hg + 2.5% Horse serum)."

GSE10430-GPL81_series_matrix.txtimp_info.txt

p6 r is 7

Found -/- in !Series_summary "Note that the Triplicate1 sample = MyoD-/-1999; Triplicate 2 sample = MyoD-/-p6; Triplicate 3 sample = MyoD-/-p10."

GSE10430-GPL81_series_matrix.txtimp_info.txt

F10 r is 2

Found + in !Series_summary "The Differentiation is leaded by removing the Proliferation Medium (Ham's F10 Medium + growth factors) and feeding with Differentiation Medium (DMEM hg + 2.5% Horse serum)."

GSE10430-GPL82_series_matrix.txtimp_info.txt

p6 r is 7

Found -/- in !Series_summary "Note that the Triplicate1 sample = MyoD-/-1999; Triplicate 2 sample = MyoD-/-p6; Triplicate 3 sample = MyoD-/-p10."

GSE10430-GPL82_series_matrix.txtimp_info.txt

F10 r is 2

Found + in !Series_summary "The Differentiation is leaded by removing the Proliferation Medium (Ham's F10 Medium + growth factors) and feeding with Differentiation Medium (DMEM hg + 2.5% Horse serum)."

GSE10430-GPL83_series_matrix.txtimp_info.txt

p6 r is 7

Found -/- in !Series_summary "Note that the Triplicate1 sample = MyoD-/-1999; Triplicate 2 sample = MyoD-/-p6; Triplicate 3 sample = MyoD-/-p10."

GSE10430-GPL83_series_matrix.txtimp_info.txt

F10 r is 2

Found + in !Series_summary "The Differentiation is leaded by removing the Proliferation Medium (Ham's F10 Medium + growth factors) and feeding with Differentiation Medium (DMEM hg + 2.5% Horse serum)."

GSE10467_series_matrix.txtimp_info.txt

myeloid leukemia r is 8

Found induced in !Series_summary "Mammalian microRNAs (miRNAs) are emerging as key regulators of the development and function of the immune system. Here, we report a strong but transient induction of miR-155 in mouse bone marrow after injection of bacterial lipopolysaccharide (LPS) correlated with granulocyte/monocyte (GM) expansion. Demonstrating the sufficiency of miR-155 to drive GM expansion, enforced expression in mouse bone marrow cells caused GM proliferation in a manner reminiscent of LPS treatment. However, the mir-155-induced GM populations displayed pathological features characteristic of myeloid neoplasia. Extending possible relevance to human disease, miR-155 was overexpressed in the bone marrow of patients with acute myeloid leukemia (AML). Furthermore, miR-155 repressed a subset of genes implicated in hematopoietic development and disease. These data implicate miR-155 as a contributor to physiological GM expansion during inflammation and to certain pathological features associated with AML, emphasizing the importance of proper miR-155 regulation in developing myeloid cells during times of inflammatory stress."

GSE10476_series_matrix.txtimp_info.txt

Ring1B r is 5

Found KO in !Series_overall_design "We observed gene expression of Ring1B single and Ring1A/B double KO cells using Affymetrix chip: MOE 430 2.0."

GSE10476_series_matrix.txtimp_info.txt

Ring1B r is 4

Found double KO in !Series_overall_design "We observed gene expression of Ring1B single and Ring1A/B double KO cells using Affymetrix chip: MOE 430 2.0."

GSE10503_series_matrix.txtimp_info.txt

P17 r is 6

Found loss of in !Series_title "Identification of transcriptional changes due to loss of Hdac3 in liver at P17 and P28"

GSE10503_series_matrix.txtimp_info.txt

Hdac3 r is 2

Found loss of in !Series_title "Identification of transcriptional changes due to loss of Hdac3 in liver at P17 and P28"

GSE10503_series_matrix.txtimp_info.txt

Hdac3 r is 2

Found knockout in !Series_summary "Keywords: Genetic knockout of Hdac3 specifically in liver, and RNA analyzed from postnatal day 17 or 28 livers for transcriptional changes related to the given phenotypes"

GSE10503_series_matrix.txtimp_info.txt

Hdac3 r is 4

Found expressing in !Series_overall_design "Postnatal day 17 or 28 mice were used for the microarray analysis. Total RNA was extracted from livers of either Hdac3 wild-type/heterozygous mice expressing Albumin-Cre or Hdac3-null livers, and pooled together in groups of 5. One pool of control or Hdac3-null liver RNA from both time points was used in technical replicates, meaning each sample was run in duplicate on separate lots of microarray chips to determine reproducibility of the Applied Biosystems ABI1700 chips. The second set of RNA pools were used as biological replicates for each group at each time point."

GSE10516_series_matrix.txtimp_info.txt

Lmx1b r is 2

Found lacking in !Series_summary "A control vs. genetic knockout experiment aimed at determining what RNAs are upregulated or downregulated in e11.5 mouse proximal limb tissue lacking the Lmx1b gene. Because Lmx1b is required for dorsal-ventral patterning of the limb, this screen gives insight into what putative downstream targets of Lmx1b contribute to dorsal-ventral patterning."

GSE10519_series_matrix.txtimp_info.txt

Dnmt1 r is 1

Found KO in !Series_overall_design "Gene expression in Dnmt1-KO cells were observed using Affymetrix chip: MOE 430 2.0."

GSE10534_series_matrix.txtimp_info.txt

Gata6 r is 1

Found overexpression in !Series_title "Gene expression of mouse ES cells in Gata6 overexpression experiments"

GSE10555_series_matrix.txtimp_info.txt

Slc39a13 r is 1

Found knockout in !Series_title "Comparision of expression profile between wild-type and Slc39a13 knockout osteoblasts"

GSE10556_series_matrix.txtimp_info.txt

Slc39a13 r is 1

Found knockout in !Series_title "Comparision of expression profile between wild-type and Slc39a13 knockout chondrocytes"

GSE10573-GPL1261_series_matrix.txtimp_info.txt

Ring1A r is 6

Found KO in !Series_overall_design "ChIP-chip experiments (GSM266076 and GSM266077) revealed the Ring1B-binding sites in promoter regions in ES cells. Microarray experiments were also performed (GSM265040 and GSM265041 for Ring1B KO, GSM265042 and GSM265043 for Ring1A and Ring1B double KO, and GSM265044, GSM265045 and GSM266115 for Oct3/4 KO)."

GSE10573-GPL1261_series_matrix.txtimp_info.txt

Ring1A r is 3

Found double KO in !Series_overall_design "ChIP-chip experiments (GSM266076 and GSM266077) revealed the Ring1B-binding sites in promoter regions in ES cells. Microarray experiments were also performed (GSM265040 and GSM265041 for Ring1B KO, GSM265042 and GSM265043 for Ring1A and Ring1B double KO, and GSM265044, GSM265045 and GSM266115 for Oct3/4 KO)."

GSE10573-GPL4128_series_matrix.txtimp_info.txt

Ring1A r is 6

Found KO in !Series_overall_design "ChIP-chip experiments (GSM266076 and GSM266077) revealed the Ring1B-binding sites in promoter regions in ES cells. Microarray experiments were also performed (GSM265040 and GSM265041 for Ring1B KO, GSM265042 and GSM265043 for Ring1A and Ring1B double KO, and GSM265044, GSM265045 and GSM266115 for Oct3/4 KO)."

GSE10573-GPL4128_series_matrix.txtimp_info.txt

Ring1A r is 3

Found double KO in !Series_overall_design "ChIP-chip experiments (GSM266076 and GSM266077) revealed the Ring1B-binding sites in promoter regions in ES cells. Microarray experiments were also performed (GSM265040 and GSM265041 for Ring1B KO, GSM265042 and GSM265043 for Ring1A and Ring1B double KO, and GSM265044, GSM265045 and GSM266115 for Oct3/4 KO)."

GSE10587_series_matrix.txtimp_info.txt

Slc26a4 r is 1

Found lacking in !Series_title "Gene expression in stria vascularis of mice lacking Slc26a4 and heterzygous controls before the onset of hearing."

GSE10589_series_matrix.txtimp_info.txt

Slc26a4 r is 1

Found lacking in !Series_title "Comparison of gene expression between the thyroid of mice lacking Slc26a4 and heterzygous controls."

GSE10628_series_matrix.txtimp_info.txt

Foxj3 r is 1

Found KO in !Series_summary "Myoblasts harvested from a postnatal day 2 WT and Foxj3 KO litter."

GSE10628_series_matrix.txtimp_info.txt

Foxj3 r is 1

Found mutant in !Series_summary "We used Affymetrix microarrays to identify dysregulated transcripts in Foxj3 mutant myoblasts."

GSE10659_series_matrix.txtimp_info.txt

RFC1 r is 4

Found inactivation in !Series_summary "The reduced folate carrier (RFC1) is an integral membrane protein and facilitative anion exchanger that mediates delivery of 5-methyltetrahydrofolate into mammalian cells. Adequate maternal-fetal transport of folate is necessary for normal embryogenesis.

Targeted inactivation of the murine RFC1 gene results in post-implantation embryo lethality, but daily folic acid supplementation of pregnant dams prolongs survival of homozygous embryos until mid-gestation. At E10.5 RFC1-/- embryos are developmentally delayed relative to wildtype littermates, have multiple malformations, including neural tube defects, and die due to failure of chorioallantoic fusion. The mesoderm is sparse and disorganized, and there is a marked absence of erythrocytes in yolk sac blood islands. Affymetrix microarray analysis and quantitative RT-PCR validation of the relative gene expression profiles in E9.5 RFC1-/- vs. RFC1+/+ embryos indicates a dramatic downregulation of multiple genes involved in erythropoiesis, and upregulation of several genes that form the cubilin-megalin multiligand endocytic receptor complex. Megalin protein expression disappears from the visceral yolk sac of RFC1-/- embryos, and cubilin protein is widely misexpressed. Inactivation of RFC1 impacts the expression of several ligands and interacting proteins in the cubilin-amnionless-megalin complex that are involved in the maternal-fetal transport of folate, vitamin B12, and other nutrients, lipids and morphogens required for normal embryogenesis."

GSE10726_series_matrix.txtimp_info.txt

beta-catenin r is 2

Found mutant in !Series_title "Expression data from skin of epithelial activated beta-catenin mutant mouse embryo"

GSE10727_series_matrix.txtimp_info.txt

beta-catenin r is 2

Found mutant in !Series_title "Expression data from dermis of epithelial activated beta-catenin mutant mouse embryo"

GSE10728_series_matrix.txtimp_info.txt

beta-catenin r is 2

Found mutant in !Series_title "Expression data from epidermis of epithelial activated beta-catenin mutant mouse embryo"

GSE10733_series_matrix.txtimp_info.txt

beta-catenin r is 2

Found mutant in !Series_title "Expression data from skin, dermis, and epidermis of epithelial activated beta-catenin mutant mouse embryo"

GSE10740_series_matrix.txtimp_info.txt

Slc9a3 r is 2

Found deficient in !Series_title "Expression data from the colon of wild-type and Slc9a3 (NHE3)-deficient mice"

GSE10740_series_matrix.txtimp_info.txt

NHE3 r is 1

Found deficient in !Series_overall_design "Whole colon was dissected from 6-8 week old NHE3-deficient mice and their wild-type littermates and total RNA isolated for microarray analysis using Affymetrix murine MOE430 2.0 arrays"

GSE10743_series_matrix.txtimp_info.txt

RP3 r is 5

Found expressing in !Series_summary "The RET/PTC3 (RP3) fusion gene is the most frequent mutation found in radiation-induced papillary thyroid cancers (PTC). Several studies suggest that the RET/PTC rearrangement is an initiating event in tumorigenesis. E7 is an oncoprotein derived from the Human Papilllomavirus 16 (HPV16) responsible for most cervical carcinoma in women. We studied here the sequence of events leading to thyroid cancer in Tg-RP3 and Tg-E7 mice expressing the transgene exclusively in the thyroid under the control of thyroglobulin (Tg) promoter. Both transgenic mice develop thyroid hyperplasia followed by solid differentiated carcinoma in older animals. To understand the different steps leading to carcinoma, we analyzed thyroid gene expression in both strains at different ages (2, 6, 10 months) by microarray technology. Important biological processes were differentially regulated in the two tumor types. In E7 thyroids cell cycle was the most upregulated process; observation consistent with the huge size of these tumors. In RP3 thyroids immunity was the most significantly regulated process, as previously observed in microarray data on human PTC. Interestingly, other human PTC characteristics were also observed in RP3 but not in E7 mouse tumors: similar regulation of several human PTC markers, upregulation of many EGF-like growth factors and finally significant regulation of angiogenesis and extracellular matrix remodeling-related genes. In summary we showed that RP3 contrary to E7 mouse tumors share several important genotypic characteristics with human PTC, observation reinforcing the validity of this model to study human thyroid tumorigenesis. "

GSE10745_series_matrix.txtimp_info.txt

histone H3 r is 6

Found treated in !Series_summary "Methodology/Principal Findings: By chromatin immunoprecipitation, we detected the same heterochromatin marks in homozygous mice carrying a (GAA)230 repeat in the first intron of the mouse frataxin gene (KIKI mice). These animals have decreased frataxin levels and, by microarray analysis, show significant gene expression changes in several tissues. We treated KIKI mice with a novel histone deacetylase inhibitor, compound 106, which substantially increases frataxin mRNA levels in cells from Friedreich ataxia individuals. Treatment increased histone H3 and H4 acetylation in chromatin near the GAA repeat and restored wild-type frataxin levels in the

nervous system and heart, as determined by quantitative RT-PCR and semiquantitative western blot analysis. No toxicity was observed. Furthermore, most of the differentially expressed genes in KIKI mice reverted towards wild-type levels. "

GSE10765_series_matrix.txtimp_info.txt

IRAK-1 r is 1

Found -/- in !Series_title "Expression data from MALP-2-stimulated macrophages from wild-type, IRAK-2-/- and IRAK-1-/IRAK-2-/- mice"

GSE10765_series_matrix.txtimp_info.txt

IRAK-2 r is 1

Found -/- in !Series_title "Expression data from MALP-2-stimulated macrophages from wild-type, IRAK-2-/- and IRAK-1-/IRAK-2-/- mice"

GSE10765_series_matrix.txtimp_info.txt

IRAK-1 r is 1

Found -/- in !Series_overall_design "Peritoneal macrophages from wild-type, IRAK-2-/- and IRAK-1/IRAK-2 mice were stimulated with MALP-2 for 0, 2, 4, and 8 hours, followed by RNA extraction. Then hybridization on affymetrix microarrays was performed."

GSE10765_series_matrix.txtimp_info.txt

IRAK-2 r is 1

Found -/- in !Series_overall_design "Peritoneal macrophages from wild-type, IRAK-2-/- and IRAK-1/IRAK-2 mice were stimulated with MALP-2 for 0, 2, 4, and 8 hours, followed by RNA extraction. Then hybridization on affymetrix microarrays was performed."

GSE10765_series_matrix.txtimp_info.txt

IRAK-1 r is 6

Found stimulated in !Series_title "Expression data from MALP-2-stimulated macrophages from wild-type, IRAK-2-/- and IRAK-1-/IRAK-2-/- mice"

GSE10765_series_matrix.txtimp_info.txt

IRAK-2 r is 6

Found stimulated in !Series_title "Expression data from MALP-2-stimulated macrophages from wild-type, IRAK-2-/- and IRAK-1-/IRAK-2-/- mice"

GSE10796_series_matrix.txtimp_info.txt

leukemia inhibitory factor r is 7

Found stimulated in !Series_summary "During development of the mammalian central nervous system (CNS), neurons and glial cells (astrocytes and oligodendrocytes) are generated from common neural precursor cells (NPCs). However, neurogenesis precedes gliogenesis, which normally commences at later stages of fetal telencephalic development. Astrocyte differentiation of mouse NPCs at embryonic day (E) 14.5 (relatively late gestation) is induced by activation of the transcription factor STAT3, whereas at E11.5 (mid-gestation) NPCs do not differentiate into astrocytes even when stimulated by STAT3-activating cytokines such as leukemia inhibitory factor (LIF). This can be explained in part by the fact that astrocyte-specific gene promoters are highly methylated in NPCs at E11.5, but other mechanisms are also likely to play a role. We therefore sought to identify genes involved in the inhibition of astrocyte differentiation of NPCs at midgestation. We first examined gene expression profiles in E11.5 and E14.5 NPCs, using Affymetrix GeneChip analysis, applying the Percellome method to normalize gene expression level. We then conducted in situ hybridization analysis for selected genes found to be highly expressed in NPCs at midgestation. Among these genes, we found that N-myc and high mobility group AT-hook 2 (Hmga2) were highly expressed in the E11.5 but not the E14.5 ventricular zone of mouse brain, where NPCs reside. Transduction of N-myc and Hmga2 by retroviruses into E14.5 NPCs, which normally differentiate into astrocytes in response to LIF, resulted in suppression of astrocyte differentiation. However, sustained expression of N-myc and Hmga2 in E11.5 NPCs failed to maintain the hypermethylated status of an astrocyte-specific gene promoter. Taken together, our data suggest that astrocyte differentiation of NPCs is regulated not only by DNA methylation but also by genes whose expression is controlled spatio-temporally during brain development."

GSE10817_series_matrix.txtimp_info.txt

Mll5 r is 2

Found -/- in !Series_overall_design "Bone marrow cells were pooled from 6 pairs of eight weeks old Mll5-/- mice and sex matched wild type littermates, and then were stained with a cocktail of biotinylated anti-mouse lineage antibodies to CD3, CD4, CD5, CD8, B220, CD11b, Gr-1 and Ter119 (eBioscience). For detection and sorting, we used Streptavidin conjugated with PE-Cy7, anti-Sca-1-APC and c-Kit-APC-Alexa F750 antibodies (eBioscience). 2.5 × 105 LSK (Lin-, Sca-1+, c-kit+) cells were flow sorted. Total RNA (320 ng) was isolated by using RNeasy Mini kit (Qiagen) following the manufactures instruction. RNA amplification and array hybridization were performed in UCSF Shared Microarray Core Facilities. Briefly, total RNA quality was assessed using a Pico Chip on an Agilent 2100 Bioanalyzer (Agilent Technologies, Palo Alto, CA). RNA was amplified and labeled with Cy3-CTP using the Agilent low RNA input fluorescent linear amplification kits following the manufacturers protocol (Agilent). Labeled cRNA was assessed using the Nandrop ND-100 (Nanodrop Technologies, Inc., Wilmington DE), and equal amounts of Cy3 labeled target were hybridized to Agilent whole mouse genome 4x44K Ink-jet arrays (Agilent). Hybridizations were performed for 14 hours, according to the manufacturers protocol

(Agilent). Arrays were scanned using the Agilent microarray scanner (Agilent) and raw signal intensities were extracted with Feature Extraction v9.1 software (Agilent,)."

GSE10823-GPL81_series_matrix.txtimp_info.txt

beta s r is 3

Found inhibition in !Series_summary "TCE is a non-genotoxic hepatocarcinogen in mouse, but not in rat or human. Extrapolation of data from laboratory animals to humans is difficult due to species-specific differences. To identify molecular pathways and biological changes responsible for species-specific differences in hepatocarcinogenesis, we analyzed gene expression profiles of livers from B6C3F1 mice and SD rats administered TCE by oral gavage once or repeatedly every 24 hrs for 14 days. Gene expression analysis revealed distinct clusters of transcriptional profiles in single- and repeated-dose mice and rats. Pathway analysis showed differences in biological pathways between single- and repeated-dose mice and rats. Activation of the MAPK signaling cascade and ubiquitin-proteasome inhibitory function, as well as inhibition of TGF-beta signaling, were specific to mice and suggest a role in hepatocyte proliferation. Although pathological analysis showed no evidence of apoptosis, gene expression analysis revealed changes in apoptosis-related genes. In addition to the previously reported suppression of apoptosis, results in repeated-dose mice showed that toxicity induced by TCE in turn induces apoptosis."

GSE10866_series_matrix.txtimp_info.txt

Mlh1 r is 3

Found -/- in !Series_overall_design "RNA was extracted from uterine segments with either CAH or invasive carcinoma from Pten+/-;Mlh1-/- , Pten+/-;Mlh1+/+ and Wild type female mice. The RNA was hybridized to Affymetrix mouse 430A chip in order to determine changes in global gene expression patterns"

GSE10895_series_matrix.txtimp_info.txt

multifunctional protein 2 r is 3

Found deficient in !Series_summary "Study on gene expression in multifunctional protein 2 deficient mice. Liver samples of two days old mice in normal conditions are used. In total 8 arrays were hybridized corresponding to 4 KO mice and 4 WT mice Results: Cholesterol synthesis is induced and ppar alpha targets also differentially expressed between KO and WT."

GSE10902_series_matrix.txtimp_info.txt

cyclin D1 r is 6

Found activation in !Series_summary "The LIM-only protein FHL2 acts as a transcriptional modulator that positively or negatively regulates multiple signaling pathways. We recently reported that FHL2 cooperates with CBP/p300 in the activation of ß-catenin/TCF target gene cyclin D1. In this paper, we

demonstrate that FHL2 is associated with the cyclin D1 promoter at the TCF/CRE site, providing evidence that cyclin D1 is a direct target of FHL2. We show that deficiency of FHL2 greatly reduces the proliferative capacity of spontaneously immortalized mouse fibroblasts which is associated with decreased expression of cyclin D1 and p16INK4a, and hypophosphorylation of Rb. Reexpression of FHL2 in FHL2-null fibroblasts efficiently restores cyclin D1 levels and cell proliferative capacity, indicating that FHL2 is critical for cyclin D1 activation and cell growth. Moreover, ectopic cyclin D1 expression is sufficient to override growth inhibition of immortalized FHL2-null fibroblasts. Gene expression profiling revealed that FHL2 deficiency triggers a broad change of the cell cycle program that is associated with downregulation of several G1/S and G2/M cyclins, E2F transcription factors and DNA replication machinery, thus correlating with reduced cell proliferation. This change also involves downregulation of the negative cell cycle regulators, particularly INK4 inhibitors, which could counteract the decreased expression of cyclins, allowing cells to grow. Our study illustrates that FHL2 can act on different aspects of the cell cycle program to finely regulate cell proliferation."

GSE10902_series_matrix.txtimp_info.txt

CBP/p300 r is 3

Found activation in !Series_summary "The LIM-only protein FHL2 acts as a transcriptional modulator that positively or negatively regulates multiple signaling pathways. We recently reported that FHL2 cooperates with CBP/p300 in the activation of ß-catenin/TCF target gene cyclin D1. In this paper, we demonstrate that FHL2 is associated with the cyclin D1 promoter at the TCF/CRE site, providing evidence that cyclin D1 is a direct target of FHL2. We show that deficiency of FHL2 greatly reduces the proliferative capacity of spontaneously immortalized mouse fibroblasts which is associated with decreased expression of cyclin D1 and p16INK4a, and hypophosphorylation of Rb. Reexpression of FHL2 in FHL2-null fibroblasts efficiently restores cyclin D1 levels and cell proliferative capacity, indicating that FHL2 is critical for cyclin D1 activation and cell growth. Moreover, ectopic cyclin D1 expression is sufficient to override growth inhibition of immortalized FHL2-null fibroblasts. Gene expression profiling revealed that FHL2 deficiency triggers a broad change of the cell cycle program that is associated with downregulation of several G1/S and G2/M cyclins, E2F transcription factors and DNA replication machinery, thus correlating with reduced cell proliferation. This change also involves downregulation of the negative cell cycle regulators, particularly INK4 inhibitors, which could counteract the decreased expression of cyclins, allowing cells to grow. Our study illustrates that FHL2 can act on different aspects of the cell cycle program to finely regulate cell proliferation."

GSE10902_series_matrix.txtimp_info.txt

D1 r is 7

Found activation in !Series_summary "The LIM-only protein FHL2 acts as a transcriptional modulator that positively or negatively regulates multiple signaling pathways. We recently reported that FHL2 cooperates with CBP/p300 in the activation of ß-catenin/TCF target gene cyclin D1. In this paper, we demonstrate that FHL2 is associated with the cyclin D1 promoter at the TCF/CRE site, providing evidence that cyclin D1 is a direct target of FHL2. We show that deficiency of FHL2 greatly reduces the

proliferative capacity of spontaneously immortalized mouse fibroblasts which is associated with decreased expression of cyclin D1 and p16INK4a, and hypophosphorylation of Rb. Reexpression of FHL2 in FHL2-null fibroblasts efficiently restores cyclin D1 levels and cell proliferative capacity, indicating that FHL2 is critical for cyclin D1 activation and cell growth. Moreover, ectopic cyclin D1 expression is sufficient to override growth inhibition of immortalized FHL2-null fibroblasts. Gene expression profiling revealed that FHL2 deficiency triggers a broad change of the cell cycle program that is associated with downregulation of several G1/S and G2/M cyclins, E2F transcription factors and DNA replication machinery, thus correlating with reduced cell proliferation. This change also involves downregulation of the negative cell cycle regulators, particularly INK4 inhibitors, which could counteract the decreased expression of cyclins, allowing cells to grow. Our study illustrates that FHL2 can act on different aspects of the cell cycle program to finely regulate cell proliferation."

GSE10904_series_matrix.txtimp_info.txt

Pdss2 r is 1

Found knockout in !Series_title "Expression data from wildtype and alb/cre liver-conditional Pdss2 knockout mutant mice"

GSE10904_series_matrix.txtimp_info.txt

Pdss2 r is 3

Found knockout in !Series_summary "Utilizing M. musculus as a model of mitochondrial dysfunction provides insight into cellular adaptations which occur as a consequence of genetic alterations causative of human disease. We characterized genome-wide expression profiles of liver-conditional knockout mice for Pdss2 compared with loxP controls."

GSE10904_series_matrix.txtimp_info.txt

Pdss2 r is 2

Found mutant in !Series_title "Expression data from wildtype and alb/cre liver-conditional Pdss2 knockout mutant mice"

GSE10941_series_matrix.txtimp_info.txt

Mxi1 r is 1

Found deficient in !Series_title "Confirmation of the gene expression pattern in Mxi1-deficient mouse (time course)"

GSE10941_series_matrix.txtimp_info.txt

Mxi1 r is 1

Found KO in !Series_summary "For final aim to completes network map between gene that is concerned in cystogenesis, verifies meaning genes connected with cyst formation analyzing gene pattern in time-dependent Mxi1 KO mouse that have phenotype of polycystic kidney disease. Monitoring meaning genes through microarray analysis, and confirm the function of these genes. Also, find pathway including meaning genes and new pathway related with PKD. Completes network map concerned in time-dependent cystogenesis through integrates knowing pathway and predicting pathway. Develope diagnostic of cyst disease and cure target through such completed network map, and wish to clear new role of cyst formation connection gene through in vivo model and examine closely control system of cyst formation mechanism. "

GSE10989_series_matrix.txtimp_info.txt

Fh1 r is 1

Found knockout in !Series_summary "Fumarate hydratase (FH) mutations cause hereditary leiomyomatosis and renal cell cancer (HLRCC). We have conditionally inactivated the murine ortholog (Fh1) in renal tubular epithelial cells in order to generate an in vivo model of HLRCC. Fh1 knockout mice recapitulates important aspects of HLRCC including the development of renal cysts that overexpress hypoxia inducible factor alpha (Hifa) and Hif-target genes."

GSE10989_series_matrix.txtimp_info.txt

Fh1 r is 1

Found knockout in !Series_summary "We used microarrays to detail the global programme of gene expression underlying cyst development in Fh1 knockout mice and identified distinct classes of up-regulated genes during this process."

GSE10989_series_matrix.txtimp_info.txt

Fh1 r is 1

Found knockout in !Series_overall_design "Renal epithelial tissue was macro-dissected from Fh1 knockout mice and sex-matched litter mate control disease-free animals for RNA extraction and hybridization on Affymetrix microarrays."

GSE11005_series_matrix.txtimp_info.txt

CD40 ligand r is 2

Found knock-out in !Series_summary "Pneumocystis is a pathogen of immunocompromised hosts but can also infect healthy hosts, in whom infection is rapidly controlled and cleared. To better understand the immune mechanisms contributing to clearance of infection, microarray methods were used to examine differential gene expression in the lungs of C57BL/6 and CD40 ligand knock-out (CD40L-KO) mice over time following exposure to Pneumocystis. Immuncompetent C57BL/6 mice, which control and clear infection efficiently, showed a robust response to infection characterized by the upregulation of

349 primarily immune-response associated genes. Temporal changes in the expression of these genes suggested that there was an early (week 2) primarily innate response, that waned without controlling infection; this were followed by primarily adaptive immune responses that peaked at week 5 and successfully cleared the infection. In conjunction with the latter, there was an increased expression of B cell associated (immunoglobulin) genes at week 6 that persisted through 11 weeks. In contrast, CD40L-KO mice, which are highly susceptible to developing severe Pneumocystis pneumonia, showed essentially no upregulation of immune-response associated genes at days 35 to 75. Immunohistochemical staining supported these observations by demonstrating an increase in CD4+, CD68+, and CD19+ cells in C57BL/6 but not CD40L-KO mice. Thus, the healthy host demonstrates a robust biphasic response to infection by Pneumocystis; CD40 ligand is an essential upstream regulator of the adaptive immune responses that efficiently control infection and prevent development of progressive pneumonia."

GSE11035_series_matrix.txtimp_info.txt

5HTT r is 8

Found loss of in !Series_overall_design "Comparison of whole lung total RNA from pools of wild-type, heterozygote, and knockout 5HTT mice to determine the pulmonary effects of loss of 5HTT expression."

GSE11035_series_matrix.txtimp_info.txt

5HTT r is 1

Found knockout in !Series_title "Effect of 5HTT knockout and heterozygosity in whole mouse lung"

GSE11035_series_matrix.txtimp_info.txt

5HTT r is 1

Found knockout in !Series_overall_design "Comparison of whole lung total RNA from pools of wild-type, heterozygote, and knockout 5HTT mice to determine the pulmonary effects of loss of 5HTT expression."

GSE11035_series_matrix.txtimp_info.txt

5-HTT r is 2

Found knock-out in !Series_summary "Rationale: While modulation of the serotonin transporter (5HTT) has shown to be a risk factor for pulmonary arterial hypertension for almost 40 years, there is a lack of in vivo data about the broad molecular effects of pulmonary inhibition of 5HTT. Previous studies have suggested effects on inflammation, proliferation, and vasoconstriction. The goal of this study was to determine which of these were supported by alterations in gene expression in serotonin transporter knockout mice. Methods: Eight week old normoxic mice with a 5-HTT knock-out (5HTT-/-) and their heterozygote(5HTT+/-) or wild-type(5HTT+/+) littermates had right ventricular systolic pressure(RVSP) assessed, lungs collected for RNA, pooled, and used in duplicate in Affymetrix array analysis.

Representative genes were confirmed by quantitative RT-PCR and western blot. Results: RVSP was normal in all groups. Only 124 genes were reliably changed between 5HTT-/- and 5HTT+/+ mice. More than half of these were either involved in inflammatory response or muscle function and organization; in addition, some matrix, heme oxygenase, developmental, and energy metabolism genes showed altered expression. Quantitative RT-PCR for examples from each major group confirmed changes seen by array, with an intermediate level in 5HTT+/- mice. Conclusions: These results for the first time show the in vivo effects of 5HTT knockout in lungs, and show that many of the downstream mechanisms suggested by cell culture and ex vivo experiments are also operational in vivo. This suggests that the effect of 5HTT on pulmonary vascular function arises from its impact on several systems, including vasoreactivity, proliferation, and immune function."

GSE11035_series_matrix.txtimp_info.txt

5HTT r is 2

Found inhibition in !Series_summary "Rationale: While modulation of the serotonin transporter (5HTT) has shown to be a risk factor for pulmonary arterial hypertension for almost 40 years, there is a lack of in vivo data about the broad molecular effects of pulmonary inhibition of 5HTT. Previous studies have suggested effects on inflammation, proliferation, and vasoconstriction. The goal of this study was to determine which of these were supported by alterations in gene expression in serotonin transporter knockout mice. Methods: Eight week old normoxic mice with a 5-HTT knock-out (5HTT-/-) and their heterozygote(5HTT+/-) or wild-type(5HTT+/+) littermates had right ventricular systolic pressure(RVSP) assessed, lungs collected for RNA, pooled, and used in duplicate in Affymetrix array analysis. Representative genes were confirmed by quantitative RT-PCR and western blot. Results: RVSP was normal in all groups. Only 124 genes were reliably changed between 5HTT-/- and 5HTT+/+ mice. More than half of these were either involved in inflammatory response or muscle function and organization; in addition, some matrix, heme oxygenase, developmental, and energy metabolism genes showed altered expression. Quantitative RT-PCR for examples from each major group confirmed changes seen by array, with an intermediate level in 5HTT+/- mice. Conclusions: These results for the first time show the in vivo effects of 5HTT knockout in lungs, and show that many of the downstream mechanisms suggested by cell culture and ex vivo experiments are also operational in vivo. This suggests that the effect of 5HTT on pulmonary vascular function arises from its impact on several systems, including vasoreactivity, proliferation, and immune function."

GSE11053_series_matrix.txtimp_info.txt

Mxi1 r is 1

Found deficient in !Series_title "Confirmation of the gene expression pattern in Mxi1-deficient mouse"

GSE11053_series_matrix.txtimp_info.txt

Mxi1 r is 1

Found KO in !Series_summary "For final aim to completes network map between gene that is concerned in cystogenesis, verifies meaning genes connected with cyst formation analyzing gene pattern in Mxi1 KO mouse that have phenotype of polycystic kidney disease. Monitoring meaning genes through microarray analysis, and confirm the function of these genes. Also, find pathway including meaning genes and new pathway related with PKD. Completes network map concerned in cystogenesis through integrates knowing pathway and predicting pathway. Develope diagnostic of cyst disease and cure target through such completed network map, and wish to clear new role of cyst formation connection gene through in vivo model and examine closely control system of cyst formation mechanism. "

GSE11098-GPL1261_series_matrix.txtimp_info.txt

p21 r is 1

Found knockout in !Series_overall_design "Livers from adult wildtype, Fah or Fah, p21 knockout mice were analyzed either after continuous treatment (ON) with NTBC or after NTBC withdrawal for 14 days (OFF)."

GSE11098-GPL339_series_matrix.txtimp_info.txt

p21 r is 1

Found knockout in !Series_overall_design "Livers from adult wildtype, Fah or Fah, p21 knockout mice were analyzed either after continuous treatment (ON) with NTBC or after NTBC withdrawal for 14 days (OFF)."

GSE11116_series_matrix.txtimp_info.txt

Atf4 r is 1

Found knockout in !Series_overall_design "The low expressing Ttr::Fv2E-Perk transgene (#58) was bred into the Atf4 knockout strain and the derivative compound heterozygous mice (in the mixed FvB/n; Swiss Webster background) were backcrossed to the Atf4+/- parental stock and Ttr::Fv2E-PERK positive siblings with Atf4+/+ and Atf4-/- genetypes were analyzed."

GSE11116_series_matrix.txtimp_info.txt

Atf4 r is 2

Found -/- in !Series_title "ISR target genes in the liver of mock-injected and AP20187-treated mice of wildtype and Atf4-/- genotype"

GSE11116_series_matrix.txtimp_info.txt

Atf4 r is 5

Found treated in !Series_title "ISR target genes in the liver of mock-injected and AP20187-treated mice of wildtype and Atf4-/- genotype"

GSE11147_series_matrix.txtimp_info.txt

protein G r is 4

Found overexpressing in !Series_summary "Post-transcriptional mechanisms play an important role in the control of gene expression. RNA-binding proteins are key players in the post-transcriptional control of many neural genes and they participate in multiple processes, from RNA splicing and mRNA transport to mRNA stability and translation. Our laboratory has developed the first mouse model overexpressing a RNA-binding protein, the ELAV-like protein HuD, in the CNS under the control of the CaMKinII alpha promoter. Initial behavioral characterization of the mice revealed that they had significant learning deficits together with abnormalities in prepulse inhibition (PPI). At the molecular level, we found that the expression of the growth-associated protein GAP-43, one of the targets of HuD, was increased in the hippocampus of HuD transgenic mice. To characterize these mice further and to evaluate the utility of these animals in understanding human diseases, we propose to use DNA microarray methods."

GSE11178_series_matrix.txtimp_info.txt

Fbw7 r is 1

Found deficient in !Series_overall_design "Four samples were analyzed: wild-type (WT) control and Fbw7-deficient (FBW7) Lin-ckit+Sca1+ (LSK) cells, as well as Lin-ckit+Sca1- myeloid progenitor (MP) cells, which served as a control for LSK-enriched/specific genes. Total bone marrow cells were pooled from three WT and three FBW7 mice before sorting LSK and MP populations."

GSE11178_series_matrix.txtimp_info.txt

Fbw7 r is 8

Found -/- in !Series_summary "Ubiquitination is a post-translational mechanism of control of diverse cellular processes. We focus here on the ubiquitin ligase Fbw7, a recently identified hematopoietic tumor suppressor that can target for degradation several important oncogenes including Notch1, c-Myc and cyclin E. We have generated conditional Fbw7 knock-out animals and inactivated the gene in hematopoietic stem cells (HSC) and their differentiated progeny. Deletion of Fbw7 specifically and rapidly affects the HSC compartment in a cell-autonomous manner. Fbw7-/- HSCs show defective maintenance of quiescence, leading to impaired self-renewal and a severe loss of competitive repopulating capacity. Furthermore, Fbw7-/- HSC are unable to colonize the thymus leading to a profound depletion of T cell progenitors. Deletion of Fbw7 in bone marrow stem cells and progenitors leads to the stabilization of c-Myc, a transcription factor previously implicated in HSC self-renewal. On the other hand, neither Notch1 nor cyclin E are stabilized in the bone marrow of Fbw7 deficient mice. Genome-wide transcriptome studies of Fbw7-/- HSC and hematopoietic progenitors indicate that Fbw7

controls, through the regulation of HSC cell cycle entry, the global transcriptional “signature” that is associated with the quiescent, self-renewing HSC phenotype."

GSE11178_series_matrix.txtimp_info.txt

ubiquitin ligase r is 6

Found -/- in !Series_summary "Ubiquitination is a post-translational mechanism of control of diverse cellular processes. We focus here on the ubiquitin ligase Fbw7, a recently identified hematopoietic tumor suppressor that can target for degradation several important oncogenes including Notch1, c-Myc and cyclin E. We have generated conditional Fbw7 knock-out animals and inactivated the gene in hematopoietic stem cells (HSC) and their differentiated progeny. Deletion of Fbw7 specifically and rapidly affects the HSC compartment in a cell-autonomous manner. Fbw7-/- HSCs show defective maintenance of quiescence, leading to impaired self-renewal and a severe loss of competitive repopulating capacity. Furthermore, Fbw7-/- HSC are unable to colonize the thymus leading to a profound depletion of T cell progenitors. Deletion of Fbw7 in bone marrow stem cells and progenitors leads to the stabilization of c-Myc, a transcription factor previously implicated in HSC self-renewal. On the other hand, neither Notch1 nor cyclin E are stabilized in the bone marrow of Fbw7 deficient mice. Genome-wide transcriptome studies of Fbw7-/- HSC and hematopoietic progenitors indicate that Fbw7 controls, through the regulation of HSC cell cycle entry, the global transcriptional “signature” that is associated with the quiescent, self-renewing HSC phenotype."

GSE11186_series_matrix.txtimp_info.txt

Bmal1 r is 1

Found mutant in !Series_summary "Hair follicles undergo recurrent cycling of controlled growth (anagen), regression (catagen), and relative quiescence (telogen) with a defined periodicity. Taking a genomics approach to study gene expression during synchronized mouse hair follicle cycling, we discovered that, in addition to circadian fluctuation, CLOCK-regulated genes are also modulated in phase with the hair growth cycle. During telogen and early anagen, circadian clock genes are prominently expressed in the secondary hair germ, which contains precursor cells for the growing follicle. Analysis of Clock and Bmal1 mutant mice reveals a delay in anagen progression, and the secondary hair germ cells show decreased levels of phosphorylated Rb and lack mitotic cells, suggesting that circadian clock genes regulate anagen progression via their effect on the cell cycle. Consistent with a block at the G1 phase of the cell cycle, we show a significant upregulation of p21 in Bmal1 mutant skin. While circadian clock mechanisms have been implicated in a variety of diurnal biological processes, our findings indicate that circadian clock genes may be utilized to modulate the progression of non-diurnal cyclic processes."

GSE11197_series_matrix.txtimp_info.txt

miR-146a r is 7

Found treated in !Series_summary "microRNA (miRNA), recently identified, non-coding, small RNA, are emerging as key regulators in homeostasis of the immune system. Therefore, aberrant expression of

miRNA may be linked to immune dysfunction, such as in chronic inflammation and autoimmunity. In this study, we investigated the potential role of miRNA in estrogen-mediated regulation of innate immune responses, as indicated by upregulation of LPS-induced IFNg, inducible nitric oxide synthase (iNOS), and nitric oxide in splenic lymphocytes from estrogen-treated mice. We found that miR-146a, a negative regulator of Toll-like receptor (TLR) signaling, was decreased in freshly-isolated splenic lymphocytes from estrogen-treated mice compared to placebo controls. Increasing the activity of miR-146a significantly inhibited LPS-induced IFNg and iNOS expression in mouse splenic lymphocytes. Further, miRNA microarray and Real-time RT-PCR analysis revealed that estrogen selectively upregulates/downregulates the expression of miRNA in mouse splenic lymphocytes. miR-223, which is highly upregulated by estrogen, regulates LPS-induced IFNg, but not iNOS or nitric oxide in splenic lymphocytes. Inhibition of miR-223 activity decreased LPS-induced IFNg in splenic lymphocytes from estrogen-treated mice. Our data are the first demonstrating selective regulation of miRNA expression in immune cells by estrogen and are indicative of an important role of miRNA in estrogen-mediated immune regulation. "

GSE11197_series_matrix.txtimp_info.txt

miR-223 r is 7

Found treated in !Series_summary "microRNA (miRNA), recently identified, non-coding, small RNA, are emerging as key regulators in homeostasis of the immune system. Therefore, aberrant expression of miRNA may be linked to immune dysfunction, such as in chronic inflammation and autoimmunity. In this study, we investigated the potential role of miRNA in estrogen-mediated regulation of innate immune responses, as indicated by upregulation of LPS-induced IFNg, inducible nitric oxide synthase (iNOS), and nitric oxide in splenic lymphocytes from estrogen-treated mice. We found that miR-146a, a negative regulator of Toll-like receptor (TLR) signaling, was decreased in freshly-isolated splenic lymphocytes from estrogen-treated mice compared to placebo controls. Increasing the activity of miR-146a significantly inhibited LPS-induced IFNg and iNOS expression in mouse splenic lymphocytes. Further, miRNA microarray and Real-time RT-PCR analysis revealed that estrogen selectively upregulates/downregulates the expression of miRNA in mouse splenic lymphocytes. miR-223, which is highly upregulated by estrogen, regulates LPS-induced IFNg, but not iNOS or nitric oxide in splenic lymphocytes. Inhibition of miR-223 activity decreased LPS-induced IFNg in splenic lymphocytes from estrogen-treated mice. Our data are the first demonstrating selective regulation of miRNA expression in immune cells by estrogen and are indicative of an important role of miRNA in estrogen-mediated immune regulation. "

GSE11197_series_matrix.txtimp_info.txt

E2 r is 8

Found induced in !Series_title "Suppression of LPS-induced IFNg and NO in splenocytes by select E2-regulated miRNA: A novel mechanism of immune mod."

GSE11197_series_matrix.txtimp_info.txt

inducible nitric oxide synthase r is 3

Found induced in !Series_summary "microRNA (miRNA), recently identified, non-coding, small RNA, are emerging as key regulators in homeostasis of the immune system. Therefore, aberrant expression of miRNA may be linked to immune dysfunction, such as in chronic inflammation and autoimmunity. In this study, we investigated the potential role of miRNA in estrogen-mediated regulation of innate immune responses, as indicated by upregulation of LPS-induced IFNg, inducible nitric oxide synthase (iNOS), and nitric oxide in splenic lymphocytes from estrogen-treated mice. We found that miR-146a, a negative regulator of Toll-like receptor (TLR) signaling, was decreased in freshly-isolated splenic lymphocytes from estrogen-treated mice compared to placebo controls. Increasing the activity of miR-146a significantly inhibited LPS-induced IFNg and iNOS expression in mouse splenic lymphocytes. Further, miRNA microarray and Real-time RT-PCR analysis revealed that estrogen selectively upregulates/downregulates the expression of miRNA in mouse splenic lymphocytes. miR-223, which is highly upregulated by estrogen, regulates LPS-induced IFNg, but not iNOS or nitric oxide in splenic lymphocytes. Inhibition of miR-223 activity decreased LPS-induced IFNg in splenic lymphocytes from estrogen-treated mice. Our data are the first demonstrating selective regulation of miRNA expression in immune cells by estrogen and are indicative of an important role of miRNA in estrogen-mediated immune regulation. "

GSE11210_series_matrix.txtimp_info.txt

Atf4 r is 1

Found knockout in !Series_overall_design "bred into the Atf4 knockout strain and the derivative compound heterozygous mice (in the mixed FvB/n; Swiss Webster background) were backcrossed to the Atf4+/- parental stock and Ttr::Fv2E-PERK positive siblings with Atf4+/+ and Atf4-/- genetypes were analyzed."

GSE11226_series_matrix.txtimp_info.txt

Nkx2-1 r is 2

Found null in !Series_overall_design "DNA microarray was performed using RNAs isolated from 4 day-organ cultured Nkx2-1-null lungs with and without SCGB3A2 treatment."

GSE11229_series_matrix.txtimp_info.txt

Dicer1 r is 1

Found deficient in !Series_summary "We identify the miR-290 cluster as being downregulated in Dicer1-deficient cells and show that it silences Rbl2, thereby controlling Dnmt expression. These results identify a pathway by which miR-290 directly regulates Rbl2-dependent Dnmt expression, indirectly affecting telomere-length homeostasis."

GSE11229_series_matrix.txtimp_info.txt

Rbl2 r is 7

Found deficient in !Series_summary "We identify the miR-290 cluster as being downregulated in Dicer1-deficient cells and show that it silences Rbl2, thereby controlling Dnmt expression. These results identify a pathway by which miR-290 directly regulates Rbl2-dependent Dnmt expression, indirectly affecting telomere-length homeostasis."

GSE11229_series_matrix.txtimp_info.txt

retinoblastoma-like 2 r is 5

Found overexpression in !Series_summary "Dicer initiates RNA interference by generating small RNAs involved in various silencing pathways. Dicer participates in centromeric silencing, but its role in the epigenetic regulation of other chromatin domains has not been explored. Here we show that Dicer1 deficiency in Mus musculus leads to decreased DNA methylation, concomitant with increased telomere recombination and telomere elongation. These DNA-methylation defects correlate with decreased expression of Dnmt1, Dnmt3a and Dnmt3b DNA methyltransferases (Dnmts), and methylation levels can be recovered by their overexpression. We identify the retinoblastoma-like 2 protein (Rbl2) as responsible for decreased Dnmt expression in Dicer1-null cells, suggesting the existence of Dicer-dependent small RNAs that target Rbl2."

GSE11229_series_matrix.txtimp_info.txt

Dnmt1 r is 1

Found overexpressing in !Series_title "Dicer-null cell lines vs. wt ES cells and Dicer-null cell lines vs. Dicer-null cells overexpressing Dnmt1 or Dnmt3a/3b"

GSE11240_series_matrix.txtimp_info.txt

Tip60 r is 8

Found knockdown in !Series_summary "Proper regulation of chromatin structure is necessary for the maintenance of cell type-specific gene expression patterns. The embryonic stem cell (ESC) expression pattern governs self-renewal and pluripotency. Here, we present an RNAi screen in mouse ESCs of 1008 loci encoding chromatin proteins. We identified 68 proteins that exhibit diverse phenotypes upon knockdown (KD), including seven subunits of the Tip60-p400 complex. Phenotypic analyses revealed that Tip60-p400 is necessary to maintain characteristic features of ESCs. We show that p400 localization to the promoters of both silent and active genes is dependent upon histone H3 lysine 4 trimethylation (H3K4me3). Furthermore, the Tip60-p400 KD gene expression profile is enriched for developmental regulators and significantly overlaps with that of the transcription factor Nanog. Depletion of Nanog reduces p400 binding to target promoters without affecting H3K4me3 levels. Together, these data indicate that Tip60-p400 integrates signals from Nanog and H3K4me3 to regulate gene expression in ESCs."

GSE11241-GPL6770_series_matrix.txtimp_info.txt

Tip60 r is 8

Found knockdown in !Series_summary "Proper regulation of chromatin structure is necessary for the maintenance of cell type-specific gene expression patterns. The embryonic stem cell (ESC) expression pattern governs self-renewal and pluripotency. Here, we present an RNAi screen in mouse ESCs of 1008 loci encoding chromatin proteins. We identified 68 proteins that exhibit diverse phenotypes upon knockdown (KD), including seven subunits of the Tip60-p400 complex. Phenotypic analyses revealed that Tip60-p400 is necessary to maintain characteristic features of ESCs. We show that p400 localization to the promoters of both silent and active genes is dependent upon histone H3 lysine 4 trimethylation (H3K4me3). Furthermore, the Tip60-p400 KD gene expression profile is enriched for developmental regulators and significantly overlaps with that of the transcription factor Nanog. Depletion of Nanog reduces p400 binding to target promoters without affecting H3K4me3 levels. Together, these data indicate that Tip60-p400 integrates signals from Nanog and H3K4me3 to regulate gene expression in ESCs."

GSE11241-GPL6771_series_matrix.txtimp_info.txt

Tip60 r is 8

Found knockdown in !Series_summary "Proper regulation of chromatin structure is necessary for the maintenance of cell type-specific gene expression patterns. The embryonic stem cell (ESC) expression pattern governs self-renewal and pluripotency. Here, we present an RNAi screen in mouse ESCs of 1008 loci encoding chromatin proteins. We identified 68 proteins that exhibit diverse phenotypes upon knockdown (KD), including seven subunits of the Tip60-p400 complex. Phenotypic analyses revealed that Tip60-p400 is necessary to maintain characteristic features of ESCs. We show that p400 localization to the promoters of both silent and active genes is dependent upon histone H3 lysine 4 trimethylation (H3K4me3). Furthermore, the Tip60-p400 KD gene expression profile is enriched for developmental regulators and significantly overlaps with that of the transcription factor Nanog. Depletion of Nanog reduces p400 binding to target promoters without affecting H3K4me3 levels. Together, these data indicate that Tip60-p400 integrates signals from Nanog and H3K4me3 to regulate gene expression in ESCs."

GSE11253_series_matrix.txtimp_info.txt

p130 r is 3

Found deficient in !Series_title "Expression data from Rb family (Rb, p130 and p107) deficient Hematopoietic stem Cells"

GSE11258_series_matrix.txtimp_info.txt

Npas4 r is 1

Found expressing in !Series_overall_design "We infected mouse hippocampal neurons with lentivirus expressing Npas4-RNAi or control-RNAi @ 3 DIV and depolarized the neurons @ 8 DIV with 50 mM of KCl for 0, 1, 3 or 6 hours. Neurons were lysed, mRNA isolated and hybridized to Affymetrix arrays. Data were collected from 3 independent experiments."

GSE11276_series_matrix.txtimp_info.txt

Nogo-A r is 2

Found KO in !Series_title "Comparison of adult intact naive C57Bl/6 Nogo-A KO versus WT spinal cord"

GSE11276_series_matrix.txtimp_info.txt

Nogo-A r is 2

Found KO in !Series_summary "Nogo-A localized on myelin adaxonal membrane in the adult CNS is well known for its role as neurite outgrowth inhibitor following a lesion. Nogo-A KO mice show enhanced regenerative/compensatory fiber growth following CNS lesion. However, changes undergoing in their intact CNS have not been studied. Moreover, Nogo-A in the intact adult CNS in also expressed in some neuronal subpopulations, e.g. in the hippocampus, olfactory bulbs and dorsal root ganglia."

GSE11276_series_matrix.txtimp_info.txt

Nogo-A r is 2

Found KO in !Series_summary "We compared the intact adult CNS (spinal cord) of Nogo-A KO mice in order to identify: potential compensating molecules which could be interesting new inhibitory neurite outgrowth candidates, possible molecules involved in the up to now not yet clarified downstream signalling pathway of Nogo-A, additional new functions for myelin or neuronal Nogo-A in the intact adult CNS."

GSE11276_series_matrix.txtimp_info.txt

Nogo-A r is 2

Found KO in !Series_summary "Keywords: gene expression, Nogo-A KO, spinal cord, adult, naive, unlesioned"

GSE11276_series_matrix.txtimp_info.txt

Nogo-A r is 2

Found KO in !Series_overall_design "Spinal cords from 3 adult C57Bl/6 wild type and Nogo-A KO mice have been explanted. Total RNA has been extracted and processed for hybridization on Mouse 430 2.0 Affymetrix GeneChips. Following scanning and first analysis with MAS 5.0, further analysis was performed by GeneSpring 7.2 (Silicon Genetics, Redwood City, CA). A present call filter (2 out of 3

present calls in at least one out of the different studied conditions) was applied. Normalization was run per chip as well as per gene to the median of the control replicates. Data were statistical restricted through a 1-way Anova (p=0.05). A final threshold of =1.2 folds of increase or decrease in the expression level of each single transcript was applied. Regulated transcripts have been assigned to functional categories according to GeneOntology as well as literature and database mining (Pubmed and Bioinformatics Harvester EMBL Heidelberg)."

GSE11287_series_matrix.txtimp_info.txt

Keap1 r is 1

Found knockout in !Series_title "Keap1-dependent gene expression determined in the liver using conditional Keap1 knockout mice vs. genetic control mice"

GSE11287_series_matrix.txtimp_info.txt

Keap1 r is 1

Found knockout in !Series_summary "To compare hepatic gene expression in conditional Keap1 knockout (Alb-Cre:Keap1(flox/-)) and genetic control mice. Disruption of Keap1-mediated repression of Nrf2 signaling was expected to result in increased expression of Nrf2-regulated genes."

GSE11287_series_matrix.txtimp_info.txt

Keap1 r is 1

Found knockout in !Series_overall_design "Hepatic gene expression was compared in conditional Keap1 knockout and genetic control mice (Alb-Cre:Keap1(flox/+)) mice. Male 9 week old mice were used, n=3/group."

GSE11314_series_matrix.txtimp_info.txt

FOG2 r is 8

Found -/- in !Series_summary "We have demonstrated previously that mammalian sexual differentiation requires both GATA4 and FOG2 transcription regulators to assemble the functioning testis. We have now determined that the sexual development of female mice is profoundly affected by the loss of GATA4-FOG2 interaction. We have also identified the Dkk1 gene, encoding a secreted inhibitor of canonical β-catenin signaling as a target of GATA4/FOG2 repression in the developing ovary. The tissue-specific ablation of the β-catenin gene in the gonads disrupts female development while in the Gata4ki/ki/Dkk1-/- or Fog2-/-/Dkk1-/- embryos the normal ovarian gene expression pattern is partially restored. Control of ovarian development by the GATA4/FOG2 complex presents a novel insight into the crosstalk of transcriptional regulation and extracellular signaling in ovarian development."

GSE11317_series_matrix.txtimp_info.txt

progesterone receptor r is 4

Found deficient in !Series_summary "The lymphatic system is a common avenue for the spread of breast cancer cells and dissemination through it occurs at least as frequently as hematogenous metastasis. Approximately 75% of primary breast cancers are estrogen receptor (ER) positive and the majority of these maintain receptor expression as lymph node (LN) metastases. However, it is unknown if ER function is equivalent in cancer cells growing in the breast and in the LNs. We have developed a model to assess estrogen responsiveness in ER(+) breast tumors and LN metastases. Fluorescent ER(+) MCF-7 tumors were grown in ovariectomized nude mice supplemented with estradiol. Once axillary LN metastasis arose, estradiol was withdrawn (EWD), for 1 or 4 weeks, or continued, to assess estradiol responsiveness. On EWD, proliferation rates fell similarly in tumors and LN metastases. However, estradiol-dependent ER down-regulation and progesterone receptor induction were deficient in LN metastases, indicating that ER-dependent transcriptional function was altered in the LN. Cancer cells from estradiol-treated and EWD primary tumors and matched LN metastases were isolated by laser capture microdissection. Global gene expression profiling identified transcripts that were regulated by the tissue microenvironment, by hormones, or by both. Interestingly, numerous genes that were estradiol regulated in tumors lost estradiol sensitivity or were regulated in the opposite direction by estradiol in LN metastases. We propose that the LN microenvironment alters estradiol signaling and may contribute to local antiestrogen resistance."

GSE11321_series_matrix.txtimp_info.txt

p53 r is 2

Found deletion in !Series_summary "Neural tube defects (NTDs) are one of the most common human birth defects, with a prevalence of approximately 1 in 1000 live births in the United States. In animal studies, deletion of p53 leads to a significant increase in embryos that exhibit exencephaly. Whereas several studies have closely investigated the morphological changes of p53-deficient embryos, there is no study that has reported the molecular-level alternations in p53-deficient embryos. Here we use microarray approach to find genes modified by deletion of p53 in day 8.5 mouse embryos to identify genes that may be involved in the mechanisms underlining NTDs and begin to define the developmental role of p53 in the etiology of NTDs. "

GSE11321_series_matrix.txtimp_info.txt

p53 r is 5

Found -/- in !Series_overall_design "Day 8.5 mouse embryos from p53 heterozygous crosses were collected, genotyped, and embryos of similar genotype (+/+; +/-; -/-) were pooled. Total RNA of biological quarduplicate samples were isolated each from p53 +/+, +/-, -/- embryos. Total RNA of each sample was obtained from 3-7 embryos. Gene expression differences between the three genotypes were examined."

GSE11322_series_matrix.txtimp_info.txt

Xbp1 r is 1

Found knockout in !Series_summary "XBP1 is a transcription factor that is induced by unconventional splicing associated with endoplasmic reticulum stress and plays a role in development of liver and plasma cells. We previously reported that brain derived neurotrophic factor (BDNF) leads to splicing of XBP1 mRNA in neurites, and that XBP1 is required for BDNF-induced neurite extension and branching. To search for the molecular mechanisms of how XBP1 plays a role in neural development, comprehensive gene expression analysis was performed in primary telencephalic neurons obtained from Xbp1 knockout mice at embryonic day 12.5. By searching for the genes induced by BDNF in wild type neurons but this induction was reduced in Xbp1 knockout mice, we found that upregulation of three GABAergic markers, somatostatin (Sst), neuropeptide Y (Npy), and calbindin (Calb1), were compromised in Xbp1 knockout neurons. Attenuated induction of Npy and Calb1 was confirmed by quantitative RT-PCR. In neurons lacking in Xbp1, upregulation of GABAergic markers was attenuated. Impaired BDNF-induced neurite extension in Xbp1 knockout neurons might be mediated by disturbed BDNF-induced differentiation of GABAergic interneurons."

GSE11322_series_matrix.txtimp_info.txt

Xbp1 r is 2

Found -/- in !Series_overall_design "Two female Xbp1 +/- mice were mated with male Xbp1 +/- mice, and at embryonic day 12.5 (E12.5) the embryos were dissected. Among the 25 embryos obtained from the 2 pregnant female mice, 6 were genotyped as Xbp1 -/- and 4 were Xbp1 +/+ by a rapid PCR assay using Z-Taq. Telencephalon was dissected from each embryo, and treated with collagenase and trypsin. Six Xbp1 -/- telencephalon samples and 4 Xbp1 +/+ samples were collected together, respectively. Each sample was divided into 15 aliquots, and the cells were subject to low density culture on plastic culture dishes. The neurons were maintained in a serum-free medium (Neurobasal medium supplemented with 0.5 mM glutamine and B27 supplement [Invitrogen]). On the third day in vitro (3 DIV) , neurons in 10 of 15 dishes in each group were stimulated with BDNF (100 ng/ml). On 4 DIV, neurons in 5 dishes stimulated with BDNF (24 hours BDNF treatment) and 5 dishes with no stimulation (0 hour) were lysed to extract total RNA. On 5 DIV, neurons in 5 dishes stimulated with BDNF (48 hours BDNF treatment) were lysed."

GSE11331_series_matrix.txtimp_info.txt

Rps6 r is 3

Found deletion in !Series_title "Ribosomal protein S6 Rps6 heterozygous null deletion effect on footpad epidermis"

GSE11331_series_matrix.txtimp_info.txt

Rps6 r is 2

Found deletion in !Series_summary "The tissue (footpad epidermis) is from a conditional heterozygous null deletion of Rps6. One copy of Rps6 was deleted from keratinocytes in the skin using the K5.Cre transgene."

GSE11331_series_matrix.txtimp_info.txt

Rps6 r is 2

Found null in !Series_title "Ribosomal protein S6 Rps6 heterozygous null deletion effect on footpad epidermis"

GSE11331_series_matrix.txtimp_info.txt

Rps6 r is 3

Found null in !Series_summary "The tissue (footpad epidermis) is from a conditional heterozygous null deletion of Rps6. One copy of Rps6 was deleted from keratinocytes in the skin using the K5.Cre transgene."

GSE11333_series_matrix.txtimp_info.txt

protein G r is 4

Found overexpressing in !Series_summary "Post-transcriptional mechanisms play an important role in the control of gene expression. RNA-binding proteins are key players in the post-transcriptional control of many neural genes and they participate in multiple processes, from RNA splicing and mRNA transport to mRNA stability and translation. Our laboratory has developed the first mouse model overexpressing a RNA-binding protein, the ELAV-like protein HuD, in the CNS under the control of the CaMKinII alpha promoter. Initial behavioral characterization of the mice revealed that they had significant learning deficits together with abnormalities in prepulse inhibition (PPI). At the molecular level, we found that the expression of the growth-associated protein GAP-43, one of the targets of HuD, was increased in the hippocampus of HuD transgenic mice."

GSE11396_series_matrix.txtimp_info.txt

growth hormone receptor r is 1

Found mutant in !Series_title "Gene expression of transgenic knock-in mutant growth hormone receptor mice"

GSE11494_series_matrix.txtimp_info.txt

M1 r is 2

Found deletion in !Series_overall_design "C57BL/6 mice (6-10 weeks old), 4 per group, were infected intranasally with log-phase Streptococcus pyogenes, 2 to 4 x 10^8 CFU per 15 µl of pyrogen-free PBS. Sham-infected mice were administered 15 µl of the same PBS. Mice were infected with wild type strain 90-226 (Cue 1998), a 90-226 strain containing an in-frame deletion of M1 protein (90-226 delta emm1) (Zimmerlein 2005) or an attenuated 90-226 which lacks both M1 and SCPA proteins (90-226att). NALT was collected from mice at 24h after infection and stored in RNAlater until RNA could be purified)."

GSE11496_series_matrix.txtimp_info.txt

GCN2 r is 2

Found activation in !Series_summary "We use array analysis to determine the global mRNA shift into polysomes following a stress response, and to compare the translational response following activation of GCN2 versus PERK, two of the four eIF2alpha kinases."

GSE11498_series_matrix.txtimp_info.txt

Irg1 r is 7

Found induced in !Series_summary "Results. Eleven of the 12 most highly upregulated mRNAs related to innate immunity and inflammation. They included mRNAs encoding histidine decarboxylase (the enzyme that synthesizes histamine), interleukin (IL)-6, the cell surface receptors PUMA-g and TREM-1, and the polypeptides Irg1 and PROK-2. MSU crystals induced dramatic rises in these mRNAs in the pouch membrane within 3-8 hours after the surge in pro-inflammatory cytokine (IL-6, IL-1beta and TNFalpha) and immediate early gene (Egr-1) transcription, which occurred 1h after crystal injection. MSU crystals induced these mRNAs in cultured macrophages with similar kinetics but lower fold changes. In keeping with their downregulation by MSU crystals according to the microarrays, qPCR confirmed that TREM-2 and granzyme D mRNAs decreased 79% and 94%, respectively, in MSU crystal inflamed membranes."

GSE11507_series_matrix.txtimp_info.txt

Abcb4 r is 7

Found deficient in !Series_summary "Background. Abcb4 (-/-) mice secrete phosphatidylcholine-deficient bile and develop sclerosing cholangitis (SC). The cholangitis involves differential hepatic transcription of genes whose products govern inflammation, activation of hepatic stellate cells and fibrosis. This study was undertaken to test the hypothesis that several genes involved in regulation of tissue inflammation and fibrosis display transcription rates that reflect SC disease activity. Methods. Abcb4 (-/-) mice fed cholic acid (CA) display high SC activity and ursodeoxycholic acid (UDCA) fed mice display low SC activity. Differential hepatic transcription of genes was accordingly measured in abcb4 (-/-) mice maintained on CA- and UDCA-supplemented diets using cDNA microarrays. Abcb4 (+/+) mice served as controls. The differential transcription of selected genes was verified by real time polymerase chain reaction. Liver tissue pathology was quantified by histopathology scoring and immunohistochemistry to visualize bile duct cells and activated hepatic stellate cells. Results. Differential transcription of Ccl2, Ccl20, Cxcl10, Nfκb1, Nfκb2, Tgfβ1, Tgfβ2, Sparc, Ctgf, Lgals3, Elf3, Spp1, Pdgfa, Pdgfrb, Col1a1, Col1a2 and Col4a1 genes paralleled the differing SC activities of cholic acid- and UDCA-fed abcb4 (-/-) mice. Histopathology scores and immunohistochemistry showed greatly enhanced activation of hepatic stellate cells during high SC activity due to CA feeding. Conclusion. Differential transcription of several genes relating to tissue inflammation and hepatic stellate cell

activation parallels SC activity in abcb4 (-/-) mice. Data on their differential transcription may be used to gauge SC disease activity. "

GSE11507_series_matrix.txtimp_info.txt

Abcb4 r is 1

Found -/- in !Series_summary "Background. Abcb4 (-/-) mice secrete phosphatidylcholine-deficient bile and develop sclerosing cholangitis (SC). The cholangitis involves differential hepatic transcription of genes whose products govern inflammation, activation of hepatic stellate cells and fibrosis. This study was undertaken to test the hypothesis that several genes involved in regulation of tissue inflammation and fibrosis display transcription rates that reflect SC disease activity. Methods. Abcb4 (-/-) mice fed cholic acid (CA) display high SC activity and ursodeoxycholic acid (UDCA) fed mice display low SC activity. Differential hepatic transcription of genes was accordingly measured in abcb4 (-/-) mice maintained on CA- and UDCA-supplemented diets using cDNA microarrays. Abcb4 (+/+) mice served as controls. The differential transcription of selected genes was verified by real time polymerase chain reaction. Liver tissue pathology was quantified by histopathology scoring and immunohistochemistry to visualize bile duct cells and activated hepatic stellate cells. Results. Differential transcription of Ccl2, Ccl20, Cxcl10, Nfκb1, Nfκb2, Tgfβ1, Tgfβ2, Sparc, Ctgf, Lgals3, Elf3, Spp1, Pdgfa, Pdgfrb, Col1a1, Col1a2 and Col4a1 genes paralleled the differing SC activities of cholic acid- and UDCA-fed abcb4 (-/-) mice. Histopathology scores and immunohistochemistry showed greatly enhanced activation of hepatic stellate cells during high SC activity due to CA feeding. Conclusion. Differential transcription of several genes relating to tissue inflammation and hepatic stellate cell activation parallels SC activity in abcb4 (-/-) mice. Data on their differential transcription may be used to gauge SC disease activity. "

GSE11547_series_matrix.txtimp_info.txt

p53 r is 2

Found deletion in !Series_summary "Neural tube defects (NTDs) are one of the most common human birth defects, with a prevalence of approximately 1 in 1000 live births in the United States. In animal studies, deletion of p53 and hyperthermia leads to a significant increase in embryos that exhibit exencephaly. To evaluate the effects of hyperthermia on p53-deficient mouse embryos we used microarray approach to find genes modified by deletion of p53 and hyperthermia treatment in day 8.5 mouse embryos to identify genes that may be involved in the mechanisms underlining NTDs and begin to define the developmental role of p53 in the etiology of NTDs. "

GSE11547_series_matrix.txtimp_info.txt

p53 r is 4

Found treated in !Series_overall_design "After day 8.5 mouse embryos from p53 heterozygous crosses were treated in either control (38C) water bath or hot (43C) water bath for 10 minutes, they were collected, genotyped, and embryos of similar genotype (+/+; +/-; -/-) and treatment were pooled. Total RNA of biological quarduplicate samples were isolated each from p53 +/+, +/-, -/- embryos. Total RNA of

each sample was obtained from 3-7 embryos. Gene expression differences between the three genotypes were examined."

GSE11557_series_matrix.txtimp_info.txt

Evi-1 r is 2

Found deletion in !Series_title "Effect of Evi-1 deletion in hematopoietic stem cells"

GSE11591_series_matrix.txtimp_info.txt

Irgm1 r is 3

Found deficient in !Series_summary "To assess gene expression changes in Irgm1 (Lrg-47) deficient HSCs"

GSE11591_series_matrix.txtimp_info.txt

Irgm1 r is 2

Found -/- in !Series_title "Expression profiling of Irgm1-/- (Lrg-47) HSCs"

GSE11596_series_matrix.txtimp_info.txt

Mecp2 r is 1

Found null in !Series_title "Gene expression profiles of wild type and Mecp2-null mice in three different regions of the brain"

GSE11596_series_matrix.txtimp_info.txt

Mecp2 r is 1

Found null in !Series_overall_design "Comparative experiment: Mecp2-null (KO) mice vs. their corresponding age-mated wild type (WT) littermates (CONTROLS). Four couples of KO-WT animals are used and three different brain regions are studied from each couple; cortex, midbrain, and cerebellum."

GSE11628_series_matrix.txtimp_info.txt

D3 r is 8

Found treated in !Series_summary "The molecular processes underlying the properties of ESC are yet unknown even when it's well established that LIF/STAT3 is neccesary for the maintenance of pluripotency. Other pathways as Wnt are may be implicated in the regulation of the biological mechanisms in mESC. Work model: D3-ES cultivated with or without LIF and treated with chronic (7 days) low doses (50nM) of GSK3 β inhibitor (lithium)."

GSE11632_series_matrix.txtimp_info.txt

Tmprss6 r is 1

Found deficient in !Series_title "Transcriptional profiling of Tmprss6-deficient mouse liver"

GSE11660_series_matrix.txtimp_info.txt

Oct3/4 r is 3

Found treated in !Series_summary "Bone marrow stromal cells (BMSCs) are multipotent stem cells that preferentially differentiate into mesenchymal cells. If they can be dedifferentiated into embryonic stem cell-like cells, they will be a highly attractive source for cell therapy. Cell and egg extracts have been used in a few studies to evaluate nuclear reprogramming, but these have not examined cell pluripotency in any detail. In this study, we used a cell reversible permeabilization method to treat BMSC with Xenopus laevis mitotic egg extract. We observed an upregulation of the pluripotent protein Oct3/4 in BMSCs treated by this extract. We also further evaluated transcriptional changes with a focused stem cell oligonucleotide array. A number of genes involved in the Notch or Wnt signaling pathways were upregulated in BMSC exposed to Xenopus egg extract. In conclusion, our microarray data from BMSCs exposure to egg extracts may provide interesting clues regarding factors involved in nuclear reprogramming. Our approach is an alternative method towards dedifferentiation of cells without genetic modification, which is preferable in the clinical situation."

GSE11661_series_matrix.txtimp_info.txt

MuRF1 r is 3

Found lacking in !Series_summary "Muscle ring finger-1 (MuRF1) is a muscle-specific protein implicated in the regulation of cardiac myocyte size and contractility. MuRF2, a closely related family member, redundantly interacts with protein substrates, and hetero-dimerizes with MuRF1. Mice lacking either MuRF1 or MuRF2 are phenotypically normal whereas mice lacking both proteins develop a spontaneous cardiac and skeletal muscle hypertrophy indicating cooperative control of muscle mass by MuRF1 and MuRF2. In order to identify the role that MuRF1 plays in regulating cardiac hypertrophy in vivo, we created transgenic mice expressing increased amounts of cardiac MuRF1. Adult MuRF1 transgenic (Tg+) hearts exhibited a non-progressive thinning of the left ventricular wall and a concomitant decrease in cardiac function. Experimental induction of cardiac hypertrophy by trans-aortic constriction (TAC) induced rapid failure of MuRF1 Tg+ hearts. Microarray analysis identified that the levels of genes associated with metabolism (and in particular mitochondrial processes) were significantly altered in MuRF1 Tg+ hearts, both at baseline and during the development of cardiac hypertrophy. Surprisingly, ATP levels in MuRF1 Tg+ mice did not differ from wild type mice despite the depressed contractility following TAC. To explain this discrepancy between the ongoing heart failure and maintained ATP levels in MuRF1 Tg+ hearts, we compared the level and activity of creatine kinase (CK) between wild type and MuRF1 Tg+ hearts. Although mCK and CK-M/B protein levels were unaffected in MuRF1 Tg+ hearts, total CK activity was significantly inhibited. We conclude that MuRF1's inhibition of CK activity leads to increased susceptibility to heart failure following TAC, demonstrating for the first time that MuRF1 regulates cardiac energetics in vivo."

GSE11662_series_matrix.txtimp_info.txt

Akt1 r is 5

Found overexpression in !Series_summary "We explored the mechanistic involvement of the growth arrest and DNA damageinducible gene, GADD45a, in LPS- and ventilator-induced inflammatory lung injury (VILI). Multiple biochemical and genomic parameters of inflammatory lung injury indicated GADD45a-/- mice to be modestly susceptible to intratracheal LPS-induced lung injury and profoundly susceptible to high tidal volume ventilation-induced lung injury (VILI) with increases in microvascular permeability and levels of inflammatory cytokines in bronchoalveolar lavage. Expression profiling of lung tissues from GADD45a-/- mice revealed strong dysregulation in the B cell receptor signaling pathway suggesting involvement of PI3 kinase/Akt signaling components while the wild type controls depicted no observable changes. Western blot analyses of lung homogenates confirmed ~50% reduction in Akt protein levels in GADD45a-/- mice accompanied by marked increases in Akt ubiquitination. Electrical resistance measurements across human lung endothelial cell monolayers with either reduced GADD45a or Akt expression (siRNAs) revealed significant potentiation of LPS-induced human lung endothelial barrier dysfunction which was attenuated by overexpression of a constitutively active Akt1 transgene. These studies validate GADD45a as a novel candidate gene in inflammatory lung injury and a significant participant in vascular barrier regulation via effects on Akt-mediated endothelial signaling"

GSE11674-GPL81_series_matrix.txtimp_info.txt

vascular endothelial cadherin r is 6

Found inhibition in !Series_summary "In order to identify genes regulated by VE-cadherin expression, we compared a mouse VE-cadherin null cell line (VEC null) with the same line reconstituted with VE-cadherin wild type cDNA (VEC positive). The morphological and functional properties of these cell lines were described previously [Lampugnani,M.G. et al. Contact inhibition of VEGF-induced proliferation requires vascular endothelial cadherin, beta-catenin, and the phosphatase DEP-1/CD148. J. Cell Biol. 161, 793-804 (2003)]. By Affymetrix gene expression analysis we found several genes up-regulated by VE-cadherin, among which claudin-5 reached remarkably high levels. The up-regulation of these genes required not only VE-cadherin expression but also cell confluence suggesting that VE-cadherin clustering at junctions was needed."

GSE11674-GPL81_series_matrix.txtimp_info.txt

vascular endothelial cadherin r is 3

Found induced in !Series_summary "In order to identify genes regulated by VE-cadherin expression, we compared a mouse VE-cadherin null cell line (VEC null) with the same line reconstituted with VE-cadherin wild type cDNA (VEC positive). The morphological and functional properties of these cell lines were described previously [Lampugnani,M.G. et al. Contact inhibition of VEGF-induced proliferation requires vascular endothelial cadherin, beta-catenin, and the phosphatase DEP-1/CD148. J. Cell Biol. 161, 793-804 (2003)]. By Affymetrix gene expression analysis we found several genes up-regulated by

VE-cadherin, among which claudin-5 reached remarkably high levels. The up-regulation of these genes required not only VE-cadherin expression but also cell confluence suggesting that VE-cadherin clustering at junctions was needed."

GSE11674-GPL81_series_matrix.txtimp_info.txt

beta-catenin r is 7

Found induced in !Series_summary "In order to identify genes regulated by VE-cadherin expression, we compared a mouse VE-cadherin null cell line (VEC null) with the same line reconstituted with VE-cadherin wild type cDNA (VEC positive). The morphological and functional properties of these cell lines were described previously [Lampugnani,M.G. et al. Contact inhibition of VEGF-induced proliferation requires vascular endothelial cadherin, beta-catenin, and the phosphatase DEP-1/CD148. J. Cell Biol. 161, 793-804 (2003)]. By Affymetrix gene expression analysis we found several genes up-regulated by VE-cadherin, among which claudin-5 reached remarkably high levels. The up-regulation of these genes required not only VE-cadherin expression but also cell confluence suggesting that VE-cadherin clustering at junctions was needed."

GSE11674-GPL82_series_matrix.txtimp_info.txt

vascular endothelial cadherin r is 6

Found inhibition in !Series_summary "In order to identify genes regulated by VE-cadherin expression, we compared a mouse VE-cadherin null cell line (VEC null) with the same line reconstituted with VE-cadherin wild type cDNA (VEC positive). The morphological and functional properties of these cell lines were described previously [Lampugnani,M.G. et al. Contact inhibition of VEGF-induced proliferation requires vascular endothelial cadherin, beta-catenin, and the phosphatase DEP-1/CD148. J. Cell Biol. 161, 793-804 (2003)]. By Affymetrix gene expression analysis we found several genes up-regulated by VE-cadherin, among which claudin-5 reached remarkably high levels. The up-regulation of these genes required not only VE-cadherin expression but also cell confluence suggesting that VE-cadherin clustering at junctions was needed."

GSE11674-GPL82_series_matrix.txtimp_info.txt

vascular endothelial cadherin r is 3

Found induced in !Series_summary "In order to identify genes regulated by VE-cadherin expression, we compared a mouse VE-cadherin null cell line (VEC null) with the same line reconstituted with VE-cadherin wild type cDNA (VEC positive). The morphological and functional properties of these cell lines were described previously [Lampugnani,M.G. et al. Contact inhibition of VEGF-induced proliferation requires vascular endothelial cadherin, beta-catenin, and the phosphatase DEP-1/CD148. J. Cell Biol. 161, 793-804 (2003)]. By Affymetrix gene expression analysis we found several genes up-regulated by VE-cadherin, among which claudin-5 reached remarkably high levels. The up-regulation of these genes required not only VE-cadherin expression but also cell confluence suggesting that VE-cadherin clustering at junctions was needed."

GSE11674-GPL82_series_matrix.txtimp_info.txt

beta-catenin r is 7

Found induced in !Series_summary "In order to identify genes regulated by VE-cadherin expression, we compared a mouse VE-cadherin null cell line (VEC null) with the same line reconstituted with VE-cadherin wild type cDNA (VEC positive). The morphological and functional properties of these cell lines were described previously [Lampugnani,M.G. et al. Contact inhibition of VEGF-induced proliferation requires vascular endothelial cadherin, beta-catenin, and the phosphatase DEP-1/CD148. J. Cell Biol. 161, 793-804 (2003)]. By Affymetrix gene expression analysis we found several genes up-regulated by VE-cadherin, among which claudin-5 reached remarkably high levels. The up-regulation of these genes required not only VE-cadherin expression but also cell confluence suggesting that VE-cadherin clustering at junctions was needed."

GSE11684_series_matrix.txtimp_info.txt

GCN2 r is 2

Found activation in !Series_summary "We use array analysis to determine the global mRNA shift into polysomes following a stress response, and to compare the translational response following activation of GCN2 versus PERK, two of the four eIF2alpha kinases."

GSE11685_series_matrix.txtimp_info.txt

GCN2 r is 2

Found activation in !Series_title "Translational response following activation of GCN2 versus PERK"

GSE11685_series_matrix.txtimp_info.txt

GCN2 r is 2

Found activation in !Series_summary "We use array analysis to determine the global mRNA shift into polysomes following a stress response, and to compare the translational response following activation of GCN2 versus PERK, two of the four eIF2alpha kinases."

GSE11698_series_matrix.txtimp_info.txt

Trex1 r is 4

Found KO in !Series_title "Microarray of Trex1 WT and Trex1 KO hearts on RAG2KO background"

GSE11702_series_matrix.txtimp_info.txt

TGF-beta1 r is 2

Found treated in !Series_summary "Gene expression profiling of a total of 3,774 genes in primary osteoblastic cells treated with TGF-beta1"

GSE11702_series_matrix.txtimp_info.txt

beta1 r is 5

Found treated in !Series_overall_design "Primary osteoblasts cultured under serum-starved condition were treated or untreated with TGF-beta1 for 24 hr."

GSE11702_series_matrix.txtimp_info.txt

TGF-beta1 r is 4

Found treated in !Series_overall_design "Primary osteoblasts cultured under serum-starved condition were treated or untreated with TGF-beta1 for 24 hr."

GSE11766_series_matrix.txtimp_info.txt

p210 r is 2

Found expressing in !Series_title "Transcriptional repression of c-Myb and GATA-2 is involved in the effects of C/EBPα in p210 BCR/ABL-expressing cells"

GSE11770_series_matrix.txtimp_info.txt

DGK-zeta r is 2

Found induced in !Series_overall_design "single time point analysis of gene expression changes induced by DGK-zeta transfection in C2C12 cells. DGK-zeta-transfected cells are compared to untreated control cells and to EGFP-transfected cells."

GSE11775_series_matrix.txtimp_info.txt

Foxp3 r is 1

Found mutant in !Series_overall_design "To identify candidate genes that might be related to the suppressive activity, the Treg cells expressing functional and mutant Foxp3 transcription factor were compared"

GSE11775_series_matrix.txtimp_info.txt

Foxp3 r is 4

Found expressing in !Series_overall_design "To identify candidate genes that might be related to the suppressive activity, the Treg cells expressing functional and mutant Foxp3 transcription factor were compared"

GSE11788_series_matrix.txtimp_info.txt

norepinephrine transporter r is 2

Found deficient in !Series_summary "Results: We have determined by long serial analysis of gene expression (LongSAGE) the gene expression profiles of in vitro differentiating wild type and norepinephrine transporter-deficient (NETKO) neural crest derivatives. Comparison analyses with the wild type library (GSM 105765) have identified a number of important differentially expressed genes, including genes relevant to noradrenergic neuron differentiation and to the phenotype of NETKO mice. Furthermore we have identified novel differentially expressed genes."

GSE11788_series_matrix.txtimp_info.txt

norepinephrine transporter r is 4

Found deletion in !Series_summary "Background: The goal of this study was to determine the transcriptional consequences of norepinephrine transporter (NET) gene deletion in noradrenergic neuron differentiation. The norepinephrine transporter (NET) is the target of powerful mind-altering substances, such as tricyclic antidepressants and the drug of abuse, cocaine. NET function in adult noradrenergic neurons of the peripheral and central nervous systems is that of a scavenger that internalizes norepinephrine from the synaptic cleft. By contrast, norepinephrine (NE) transport has a different role in embryogenesis. It promotes differentiation of neural crest cells and locus ceruleus progenitors into noradrenergic neurons, whereas NET inhibitors, such as the tricyclic antidepressant desipramine and the drug of abuse, cocaine, inhibit noradrenergic differentiation. While NET structure und regulation of NET function is well described, little is known about downstream targets of NE transport."

GSE11809_series_matrix.txtimp_info.txt

Irf1 r is 5

Found -/- in !Series_overall_design "In two independent blocks of experiments, Ifng-/- and Irf1-/- mice on a C57Bl/6 background and their respective controls were infected with Mycobacterium avium via aerosol. After 14 weeks, mice were sacrificed and the lung RNA prepared using TriFast FL (Peqlab). Total RNA (1µg) was labeled and hybridized to Affymetrix mouse MOE430A 2.0 GeneChips according to the manufacturer's recommendations. For each condition three biological replicates (except Ifng-/-, no infection) were used. Total of 23 Samples."

GSE11821_series_matrix.txtimp_info.txt

Igf-1 r is 2

Found -/- in !Series_title "Expression data from Igf-1 -/- and Igf-1+/+ mouse cochleas"

GSE11821_series_matrix.txtimp_info.txt

Igf-1 r is 7

Found -/- in !Series_summary "We used microarrays to define the genetic signatures of Igf-1 +/+ and Igf-1-/- mouse cochea and identify the differentially expressed genes."

GSE11821_series_matrix.txtimp_info.txt

Igf-1 r is 8

Found -/- in !Series_overall_design "Cochleae from two E18.5 were isolated from both Igf-1+/+ wild type and Igf-1-/- null mice and pooled to obtain RNA. Heterozygous male and female with a genetic background C57BL/6J were mated to obtain embryos 18.5 days post coitus (E18.5). Three independent pools were used. Cochlear tissues included the otic capsule but not vestibular tissues."

GSE11844_series_matrix.txtimp_info.txt

Igf-1 r is 7

Found -/- in !Series_summary "We used microarrays to define the genetic signatures of Igf-1 +/+ and Igf-1-/- mouse cochea and identify the differentially expressed genes."

GSE11844_series_matrix.txtimp_info.txt

Igf-1 r is 8

Found -/- in !Series_overall_design "Cochleae from two E18.5 were isolated from both Igf-1+/+ wild type and Igf-1-/- null mice and pooled to obtain RNA. Heterozygous male and female with a genetic background C57BL/6J were mated to obtain embryos 18.5 days post coitus (E18.5). Three independent pools were used. Cochlear tissues included the otic capsule but not vestibular tissues."

GSE11844_series_matrix.txtimp_info.txt

Igf1 r is 1

Found null in !Series_title "Mouse Igf1 null publication analysis"

GSE11859_series_matrix.txtimp_info.txt

Olig2 r is 7

Found expressing in !Series_summary "Origins of the brain tumor, medulloblastoma, from stem cells or restricted pro-genitor cells are unclear. To investigate this, we activated oncogenic Hedgehog signaling in multipotent and lineage-restricted CNS progenitors. We observed that normal unipo-tent cerebellar granule neuron precursors (CGNP) derive from hGFAP+ and Olig2+ rhombic lip progenitors. Hedgehog activation in a spectrum of early and late stage CNS progenitors generated similar medulloblastomas, but not other brain cancers, indicating that acquisition of CGNP identity is essential for tumorigenesis. We show in human and mouse medulloblastoma that cells expressing the glia-associated markers Gfap and Olig2 are neoplastic and that they retain features of embryonic-type granule lineage progenitors. Thus, oncogenic Hedgehog signaling promotes medulloblastoma from lineage-restricted granule cell progenitors."

GSE11867_series_matrix.txtimp_info.txt

Dll1 r is 1

Found mutant in !Series_title "Expression Profiling of Dll1 mutant mouse lines on different genetic background"

GSE11867_series_matrix.txtimp_info.txt

Dll1 r is 1

Found mutant in !Series_summary "The two analysed Dll1 mutant mouse lines carry the same mutation but are on different genetic background: 1. Heterozygous F1-animals were backcrossed several times to the 129SV/J wild type; 2. Heterozygous F1-animals were outcrossed 11 generation to C3HeB/FeJ wild type"

GSE11867_series_matrix.txtimp_info.txt

Dll1 r is 1

Found mutant in !Series_overall_design "Four organs (liver, spleen, thymus, brain) of two Dll1 mutant mouse lines on different genetic background carrying the same mutation were analysed by cDNA microarray technology. Experiment include 4-5 biological replicates for reference (wildtype) and mutant animals. Up to 4 technical replicates for each mutant mouse were performed. As reference pooled RNA was used. 50% of the chip hybridisations are dye sway experiments."

GSE11897-GPL1261_series_matrix.txtimp_info.txt

Pou5f1 r is 7

Found loss of in !Series_summary "Lhx8 is a member of the LIM-homeobox transcription factor family and preferentially expressed in oocytes and germ cells within the mouse ovary. We discovered that Lhx8 knockout females lose oocytes within 7 days after birth. At the time of birth, histological examination shows that Lhx8 deficient (Lhx8(-/-)) ovaries are grossly similar to the newborn wild type ovaries. Lhx8(-/-) ovaries fail to maintain the primordial follicles and the transition from primordial to growing follicles does not occur. Lhx8(-/-) ovaries misexpress oocyte-specific genes such as Gdf9, Pou5f1, and Nobox. Very rapid loss of oocytes may partly be due to drastic the down-regulation of Kit and Kitl in Lhx8(-/-) ovaries. We compared Lhx8(-/-) and wild-type ovaries using Affymetrix 430 2.0 microarray platform. Eighty (44%) of 180 of the genes down-regulated more than 5-fold in Lhx8(-/-) ovaries were preferentially expressed in oocytes, whereas only 3 (2%) of 146 genes up-regulated more than 5-fold in the absence of Lhx8 were preferentially expressed in oocytes. In addition, the comparison of genes regulated in Lhx8(-/-) and Nobox(-/-) newborn ovaries discovered a common set of 34 genes whose expression level is affected in both Lhx8 and Nobox deficient mice. Our findings show that Lhx8 is a critical factor for maintenance and differentiation of the oocyte during early oogenesis and it acts in part by down-regulating the Nobox pathway."

GSE11897-GPL1261_series_matrix.txtimp_info.txt

Lhx8 r is 1

Found knockout in !Series_summary "Lhx8 is a member of the LIM-homeobox transcription factor family and preferentially expressed in oocytes and germ cells within the mouse ovary. We discovered that Lhx8 knockout females lose oocytes within 7 days after birth. At the time of birth, histological examination shows that Lhx8 deficient (Lhx8(-/-)) ovaries are grossly similar to the newborn wild type ovaries. Lhx8(-/-) ovaries fail to maintain the primordial follicles and the transition from primordial to growing follicles does not occur. Lhx8(-/-) ovaries misexpress oocyte-specific genes such as Gdf9, Pou5f1, and Nobox. Very rapid loss of oocytes may partly be due to drastic the down-regulation of Kit and Kitl in Lhx8(-/-) ovaries. We compared Lhx8(-/-) and wild-type ovaries using Affymetrix 430 2.0 microarray platform. Eighty (44%) of 180 of the genes down-regulated more than 5-fold in Lhx8(-/-) ovaries were preferentially expressed in oocytes, whereas only 3 (2%) of 146 genes up-regulated more than 5-fold in the absence of Lhx8 were preferentially expressed in oocytes. In addition, the comparison of genes regulated in Lhx8(-/-) and Nobox(-/-) newborn ovaries discovered a common set of 34 genes whose expression level is affected in both Lhx8 and Nobox deficient mice. Our findings show that Lhx8 is a critical factor for maintenance and differentiation of the oocyte during early oogenesis and it acts in part by down-regulating the Nobox pathway."

GSE11897-GPL1261_series_matrix.txtimp_info.txt

Lhx8 r is 4

Found -/- in !Series_summary "Lhx8 is a member of the LIM-homeobox transcription factor family and preferentially expressed in oocytes and germ cells within the mouse ovary. We discovered that Lhx8 knockout females lose oocytes within 7 days after birth. At the time of birth, histological examination shows that Lhx8 deficient (Lhx8(-/-)) ovaries are grossly similar to the newborn wild type ovaries. Lhx8(-/-) ovaries fail to maintain the primordial follicles and the transition from primordial to growing follicles does not occur. Lhx8(-/-) ovaries misexpress oocyte-specific genes such as Gdf9, Pou5f1, and Nobox. Very rapid loss of oocytes may partly be due to drastic the down-regulation of Kit and Kitl in Lhx8(-/-) ovaries. We compared Lhx8(-/-) and wild-type ovaries using Affymetrix 430 2.0 microarray platform. Eighty (44%) of 180 of the genes down-regulated more than 5-fold in Lhx8(-/-) ovaries were preferentially expressed in oocytes, whereas only 3 (2%) of 146 genes up-regulated more than 5-fold in the absence of Lhx8 were preferentially expressed in oocytes. In addition, the comparison of genes regulated in Lhx8(-/-) and Nobox(-/-) newborn ovaries discovered a common set of 34 genes whose expression level is affected in both Lhx8 and Nobox deficient mice. Our findings show that Lhx8 is a critical factor for maintenance and differentiation of the oocyte during early oogenesis and it acts in part by down-regulating the Nobox pathway."

GSE11897-GPL339_series_matrix.txtimp_info.txt

Pou5f1 r is 7

Found loss of in !Series_summary "Lhx8 is a member of the LIM-homeobox transcription factor family and preferentially expressed in oocytes and germ cells within the mouse ovary. We discovered

that Lhx8 knockout females lose oocytes within 7 days after birth. At the time of birth, histological examination shows that Lhx8 deficient (Lhx8(-/-)) ovaries are grossly similar to the newborn wild type ovaries. Lhx8(-/-) ovaries fail to maintain the primordial follicles and the transition from primordial to growing follicles does not occur. Lhx8(-/-) ovaries misexpress oocyte-specific genes such as Gdf9, Pou5f1, and Nobox. Very rapid loss of oocytes may partly be due to drastic the down-regulation of Kit and Kitl in Lhx8(-/-) ovaries. We compared Lhx8(-/-) and wild-type ovaries using Affymetrix 430 2.0 microarray platform. Eighty (44%) of 180 of the genes down-regulated more than 5-fold in Lhx8(-/-) ovaries were preferentially expressed in oocytes, whereas only 3 (2%) of 146 genes up-regulated more than 5-fold in the absence of Lhx8 were preferentially expressed in oocytes. In addition, the comparison of genes regulated in Lhx8(-/-) and Nobox(-/-) newborn ovaries discovered a common set of 34 genes whose expression level is affected in both Lhx8 and Nobox deficient mice. Our findings show that Lhx8 is a critical factor for maintenance and differentiation of the oocyte during early oogenesis and it acts in part by down-regulating the Nobox pathway."

GSE11897-GPL339_series_matrix.txtimp_info.txt

Lhx8 r is 1

Found knockout in !Series_summary "Lhx8 is a member of the LIM-homeobox transcription factor family and preferentially expressed in oocytes and germ cells within the mouse ovary. We discovered that Lhx8 knockout females lose oocytes within 7 days after birth. At the time of birth, histological examination shows that Lhx8 deficient (Lhx8(-/-)) ovaries are grossly similar to the newborn wild type ovaries. Lhx8(-/-) ovaries fail to maintain the primordial follicles and the transition from primordial to growing follicles does not occur. Lhx8(-/-) ovaries misexpress oocyte-specific genes such as Gdf9, Pou5f1, and Nobox. Very rapid loss of oocytes may partly be due to drastic the down-regulation of Kit and Kitl in Lhx8(-/-) ovaries. We compared Lhx8(-/-) and wild-type ovaries using Affymetrix 430 2.0 microarray platform. Eighty (44%) of 180 of the genes down-regulated more than 5-fold in Lhx8(-/-) ovaries were preferentially expressed in oocytes, whereas only 3 (2%) of 146 genes up-regulated more than 5-fold in the absence of Lhx8 were preferentially expressed in oocytes. In addition, the comparison of genes regulated in Lhx8(-/-) and Nobox(-/-) newborn ovaries discovered a common set of 34 genes whose expression level is affected in both Lhx8 and Nobox deficient mice. Our findings show that Lhx8 is a critical factor for maintenance and differentiation of the oocyte during early oogenesis and it acts in part by down-regulating the Nobox pathway."

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Lhx8 r is 4

Found -/- in !Series_summary "Lhx8 is a member of the LIM-homeobox transcription factor family and preferentially expressed in oocytes and germ cells within the mouse ovary. We discovered that Lhx8 knockout females lose oocytes within 7 days after birth. At the time of birth, histological examination shows that Lhx8 deficient (Lhx8(-/-)) ovaries are grossly similar to the newborn wild type ovaries. Lhx8(-/-) ovaries fail to maintain the primordial follicles and the transition from primordial to growing follicles does not occur. Lhx8(-/-) ovaries misexpress oocyte-specific genes such as Gdf9, Pou5f1, and Nobox.

Very rapid loss of oocytes may partly be due to drastic the down-regulation of Kit and Kitl in Lhx8(-/-) ovaries. We compared Lhx8(-/-) and wild-type ovaries using Affymetrix 430 2.0 microarray platform. Eighty (44%) of 180 of the genes down-regulated more than 5-fold in Lhx8(-/-) ovaries were preferentially expressed in oocytes, whereas only 3 (2%) of 146 genes up-regulated more than 5-fold in the absence of Lhx8 were preferentially expressed in oocytes. In addition, the comparison of genes regulated in Lhx8(-/-) and Nobox(-/-) newborn ovaries discovered a common set of 34 genes whose expression level is affected in both Lhx8 and Nobox deficient mice. Our findings show that Lhx8 is a critical factor for maintenance and differentiation of the oocyte during early oogenesis and it acts in part by down-regulating the Nobox pathway."

GSE11897-GPL340_series_matrix.txtimp_info.txt

Pou5f1 r is 7

Found loss of in !Series_summary "Lhx8 is a member of the LIM-homeobox transcription factor family and preferentially expressed in oocytes and germ cells within the mouse ovary. We discovered that Lhx8 knockout females lose oocytes within 7 days after birth. At the time of birth, histological examination shows that Lhx8 deficient (Lhx8(-/-)) ovaries are grossly similar to the newborn wild type ovaries. Lhx8(-/-) ovaries fail to maintain the primordial follicles and the transition from primordial to growing follicles does not occur. Lhx8(-/-) ovaries misexpress oocyte-specific genes such as Gdf9, Pou5f1, and Nobox. Very rapid loss of oocytes may partly be due to drastic the down-regulation of Kit and Kitl in Lhx8(-/-) ovaries. We compared Lhx8(-/-) and wild-type ovaries using Affymetrix 430 2.0 microarray platform. Eighty (44%) of 180 of the genes down-regulated more than 5-fold in Lhx8(-/-) ovaries were preferentially expressed in oocytes, whereas only 3 (2%) of 146 genes up-regulated more than 5-fold in the absence of Lhx8 were preferentially expressed in oocytes. In addition, the comparison of genes regulated in Lhx8(-/-) and Nobox(-/-) newborn ovaries discovered a common set of 34 genes whose expression level is affected in both Lhx8 and Nobox deficient mice. Our findings show that Lhx8 is a critical factor for maintenance and differentiation of the oocyte during early oogenesis and it acts in part by down-regulating the Nobox pathway."

GSE11897-GPL340_series_matrix.txtimp_info.txt

Lhx8 r is 1

Found knockout in !Series_summary "Lhx8 is a member of the LIM-homeobox transcription factor family and preferentially expressed in oocytes and germ cells within the mouse ovary. We discovered that Lhx8 knockout females lose oocytes within 7 days after birth. At the time of birth, histological examination shows that Lhx8 deficient (Lhx8(-/-)) ovaries are grossly similar to the newborn wild type ovaries. Lhx8(-/-) ovaries fail to maintain the primordial follicles and the transition from primordial to growing follicles does not occur. Lhx8(-/-) ovaries misexpress oocyte-specific genes such as Gdf9, Pou5f1, and Nobox. Very rapid loss of oocytes may partly be due to drastic the down-regulation of Kit and Kitl in Lhx8(-/-) ovaries. We compared Lhx8(-/-) and wild-type ovaries using Affymetrix 430 2.0 microarray platform. Eighty (44%) of 180 of the genes down-regulated more than 5-fold in Lhx8(-/-) ovaries were preferentially expressed in oocytes, whereas only 3 (2%) of 146 genes up-regulated more

than 5-fold in the absence of Lhx8 were preferentially expressed in oocytes. In addition, the comparison of genes regulated in Lhx8(-/-) and Nobox(-/-) newborn ovaries discovered a common set of 34 genes whose expression level is affected in both Lhx8 and Nobox deficient mice. Our findings show that Lhx8 is a critical factor for maintenance and differentiation of the oocyte during early oogenesis and it acts in part by down-regulating the Nobox pathway."

GSE11897-GPL340_series_matrix.txtimp_info.txt

Lhx8 r is 4

Found -/- in !Series_summary "Lhx8 is a member of the LIM-homeobox transcription factor family and preferentially expressed in oocytes and germ cells within the mouse ovary. We discovered that Lhx8 knockout females lose oocytes within 7 days after birth. At the time of birth, histological examination shows that Lhx8 deficient (Lhx8(-/-)) ovaries are grossly similar to the newborn wild type ovaries. Lhx8(-/-) ovaries fail to maintain the primordial follicles and the transition from primordial to growing follicles does not occur. Lhx8(-/-) ovaries misexpress oocyte-specific genes such as Gdf9, Pou5f1, and Nobox. Very rapid loss of oocytes may partly be due to drastic the down-regulation of Kit and Kitl in Lhx8(-/-) ovaries. We compared Lhx8(-/-) and wild-type ovaries using Affymetrix 430 2.0 microarray platform. Eighty (44%) of 180 of the genes down-regulated more than 5-fold in Lhx8(-/-) ovaries were preferentially expressed in oocytes, whereas only 3 (2%) of 146 genes up-regulated more than 5-fold in the absence of Lhx8 were preferentially expressed in oocytes. In addition, the comparison of genes regulated in Lhx8(-/-) and Nobox(-/-) newborn ovaries discovered a common set of 34 genes whose expression level is affected in both Lhx8 and Nobox deficient mice. Our findings show that Lhx8 is a critical factor for maintenance and differentiation of the oocyte during early oogenesis and it acts in part by down-regulating the Nobox pathway."

GSE11963_series_matrix.txtimp_info.txt

p50 r is 3

Found activation in !Series_summary "Background: Lymphotoxin signaling via the lymphotoxin-β receptor (LTβR) has been implicated in several biological processes, ranging from development of secondary lymphoid organs, maintenance of splenic tissue, host defense against pathogens, autoimmunity, and lipid homeostasis. The major transcription factor that is activated by LTβR crosslinking is NF-κB. Two signaling pathways have been described that result in the activation of classical p50-RelA and alternative p52-RelB NF-κB heterodimers."

GSE11963_series_matrix.txtimp_info.txt

p52 r is 7

Found activation in !Series_summary "Background: Lymphotoxin signaling via the lymphotoxin-β receptor (LTβR) has been implicated in several biological processes, ranging from development of secondary lymphoid organs, maintenance of splenic tissue, host defense against pathogens, autoimmunity, and lipid homeostasis. The major transcription factor that is activated by LTβR

crosslinking is NF-κB. Two signaling pathways have been described that result in the activation of classical p50-RelA and alternative p52-RelB NF-κB heterodimers."

GSE11973_series_matrix.txtimp_info.txt

miR-223 r is 2

Found null in !Series_title "Wild-type cultured neutrophils versus miR-223 null cultured neutrophils "

GSE11973_series_matrix.txtimp_info.txt

miR-223 r is 2

Found null in !Series_summary "This array analysis is to study the regulation of target messages' expression in in vitro cultured murine neutrophils versus miR-223 null neutrophils. Culture media was SILAC-IMDM for MS analysis."

GSE11973_series_matrix.txtimp_info.txt

miR-223 r is 2

Found null in !Series_overall_design "Wild-type cultured neutrophils versus miR-223 null cultured neutrophils "

GSE11982_series_matrix.txtimp_info.txt

E2A r is 1

Found KO in !Series_title "Expression data from WT and E2A-KO bone marrow stem and progenitor cell subfractions."

GSE11982_series_matrix.txtimp_info.txt

E2A r is 1

Found KO in !Series_summary "Expression profiling of WT and E2A-KO LSK FLT3- and LMPP protenitor cells."

GSE11982_series_matrix.txtimp_info.txt

E2A r is 1

Found KO in !Series_overall_design "LSK FLT3- and LMPP stem/progenitor cells from WT and E2A-KO mice were FACS sorted. Subsequently RNA was extracted, labelled and hybridized to Affymetrix microarrays. Goal of experiment was to investigate expression changes between WT and KO LMPP cells."

GSE11990_series_matrix.txtimp_info.txt

p53 r is 1

Found deficient in !Series_title "Gene expression profiling of mouse p53-deficient epidermal carcinoma defines molecular determinants of human cancer malignancy (training dataset)"

GSE12001_series_matrix.txtimp_info.txt

miR-223 r is 2

Found null in !Series_title "Wild-type neutrophils and miR-223 null neutrophils"

GSE12001_series_matrix.txtimp_info.txt

miR-223 r is 2

Found null in !Series_summary "This array analysis is to study the regulation of target messages' expression in murine neutrophils versus miR-223 null neutrophils. "

GSE12001_series_matrix.txtimp_info.txt

miR-223 r is 2

Found null in !Series_overall_design "Three arrays for wild-type neutrophils and miR-223 null neutrophils"

GSE12003_series_matrix.txtimp_info.txt

miR-223 r is 2

Found null in !Series_title "4 days cultured progenitors and 8 days cultured mature neutrophils from WT vs miR-223 null neutrophils"

GSE12003_series_matrix.txtimp_info.txt

miR-223 r is 2

Found null in !Series_summary "This array analysis is to study developmental time course of the regulation of target messages' expression during culture of murine neutrophils versus miR-223 null neutrophils. Culture media was SILAC-IMDM for MS analysis."

GSE12003_series_matrix.txtimp_info.txt

miR-223 r is 2

Found null in !Series_overall_design "Two arrays of each for 4 days cultured progenitors and 8 days cultured mature neutrophils from wild-type cultured neutrophils versus miR-223 null cultured neutrophils"

GSE12008-GPL4134_series_matrix.txtimp_info.txt

p65 r is 3

Found knock-out in !Series_summary "LMX1B is a LIM-homeodomain transcription factor essential for development. Putative LMX1B target genes have been identified through mouse gene targeting studies; however, in the absence of in vivo molecular characterization of their regulation, their identity as direct LMX1B targets remains hypothetical. We describe here the first molecular characterization of LMX1B target gene regulation. A tetracycline-inducible expression system and microarray analysis showed that a subset of NF-kappa B target genes, including IL-6 and IL-8 are upregulated in LMX1B-expressing HeLa cells. Chromatin immunoprecipitation assays revealed that LMX1B binds to the proximal promoter region of IL-6 and IL-8 in vivo, in the vicinity of the characterized kappa B site, and that LMX1B recruitment correlates with an increased NF-kappa B DNA association. Inhibition of NF-kappa B activity by short interfering RNA-mediated knock-down of p65 impairs LMX1B-dependent induction of NF-kappa B target genes, while activation of NF-kappa B activity by TNF-alpha results in a synergistic induction of these genes by LMX1B. IL-6 promoter-driven reporter assays showed that the kappa B site and an adjacent putative LMX1B binding motif are both involved in LMX1B-mediated transcription. Expression of a number of NF-kappa B target genes is affected in the kidney of Lmx1b-/- knock-out mice, thus supporting the biological relevance of the data obtained in the human cell line. Together, these data demonstrate for the first time that LMX1B directly regulates transcription of a subset of NF-kappa B target genes in cooperation with nuclear p50/p65 NF-kappa B."

GSE12008-GPL4134_series_matrix.txtimp_info.txt

p65 NF-kappa B r is 3

Found knock-out in !Series_summary "LMX1B is a LIM-homeodomain transcription factor essential for development. Putative LMX1B target genes have been identified through mouse gene targeting studies; however, in the absence of in vivo molecular characterization of their regulation, their identity as direct LMX1B targets remains hypothetical. We describe here the first molecular characterization of LMX1B target gene regulation. A tetracycline-inducible expression system and microarray analysis showed that a subset of NF-kappa B target genes, including IL-6 and IL-8 are upregulated in LMX1B-expressing HeLa cells. Chromatin immunoprecipitation assays revealed that LMX1B binds to the proximal promoter region of IL-6 and IL-8 in vivo, in the vicinity of the characterized kappa B site, and that LMX1B recruitment correlates with an increased NF-kappa B DNA association. Inhibition of NF-kappa B activity by short interfering RNA-mediated knock-down of p65 impairs LMX1B-dependent induction of NF-kappa B target genes, while activation of NF-kappa B activity by TNF-alpha results in a synergistic induction of these genes by LMX1B. IL-6 promoter-driven reporter assays showed that the kappa B site and an adjacent putative LMX1B binding motif are both involved in LMX1B-mediated transcription. Expression of a number of NF-kappa B target genes is affected in the kidney of Lmx1b-/- knock-out mice, thus

supporting the biological relevance of the data obtained in the human cell line. Together, these data demonstrate for the first time that LMX1B directly regulates transcription of a subset of NF-kappa B target genes in cooperation with nuclear p50/p65 NF-kappa B."

GSE12008-GPL4134_series_matrix.txtimp_info.txt

Lmx1b r is 3

Found knock-out in !Series_overall_design "Mouse subset (GSM304379-GSM304384): Three kidney samples from newborn wild-type mice and from newborn Lmx1b-/- knock-out mice were processed for gene expression array analyses using an Agilent platform."

GSE12008-GPL4134_series_matrix.txtimp_info.txt

Lmx1b r is 2

Found -/- in !Series_overall_design "Mouse subset (GSM304379-GSM304384): Three kidney samples from newborn wild-type mice and from newborn Lmx1b-/- knock-out mice were processed for gene expression array analyses using an Agilent platform."

GSE12008-GPL4134_series_matrix.txtimp_info.txt

TNF-a r is 7

Found activation in !Series_summary "LMX1B is a LIM-homeodomain transcription factor essential for development. Putative LMX1B target genes have been identified through mouse gene targeting studies; however, in the absence of in vivo molecular characterization of their regulation, their identity as direct LMX1B targets remains hypothetical. We describe here the first molecular characterization of LMX1B target gene regulation. A tetracycline-inducible expression system and microarray analysis showed that a subset of NF-kappa B target genes, including IL-6 and IL-8 are upregulated in LMX1B-expressing HeLa cells. Chromatin immunoprecipitation assays revealed that LMX1B binds to the proximal promoter region of IL-6 and IL-8 in vivo, in the vicinity of the characterized kappa B site, and that LMX1B recruitment correlates with an increased NF-kappa B DNA association. Inhibition of NF-kappa B activity by short interfering RNA-mediated knock-down of p65 impairs LMX1B-dependent induction of NF-kappa B target genes, while activation of NF-kappa B activity by TNF-alpha results in a synergistic induction of these genes by LMX1B. IL-6 promoter-driven reporter assays showed that the kappa B site and an adjacent putative LMX1B binding motif are both involved in LMX1B-mediated transcription. Expression of a number of NF-kappa B target genes is affected in the kidney of Lmx1b-/- knock-out mice, thus supporting the biological relevance of the data obtained in the human cell line. Together, these data demonstrate for the first time that LMX1B directly regulates transcription of a subset of NF-kappa B target genes in cooperation with nuclear p50/p65 NF-kappa B."

GSE12008-GPL4134_series_matrix.txtimp_info.txt

TNF-alpha r is 7

Found activation in !Series_summary "LMX1B is a LIM-homeodomain transcription factor essential for development. Putative LMX1B target genes have been identified through mouse gene targeting studies; however, in the absence of in vivo molecular characterization of their regulation, their identity as direct LMX1B targets remains hypothetical. We describe here the first molecular characterization of LMX1B target gene regulation. A tetracycline-inducible expression system and microarray analysis showed that a subset of NF-kappa B target genes, including IL-6 and IL-8 are upregulated in LMX1B-expressing HeLa cells. Chromatin immunoprecipitation assays revealed that LMX1B binds to the proximal promoter region of IL-6 and IL-8 in vivo, in the vicinity of the characterized kappa B site, and that LMX1B recruitment correlates with an increased NF-kappa B DNA association. Inhibition of NF-kappa B activity by short interfering RNA-mediated knock-down of p65 impairs LMX1B-dependent induction of NF-kappa B target genes, while activation of NF-kappa B activity by TNF-alpha results in a synergistic induction of these genes by LMX1B. IL-6 promoter-driven reporter assays showed that the kappa B site and an adjacent putative LMX1B binding motif are both involved in LMX1B-mediated transcription. Expression of a number of NF-kappa B target genes is affected in the kidney of Lmx1b-/- knock-out mice, thus supporting the biological relevance of the data obtained in the human cell line. Together, these data demonstrate for the first time that LMX1B directly regulates transcription of a subset of NF-kappa B target genes in cooperation with nuclear p50/p65 NF-kappa B."

GSE12025_series_matrix.txtimp_info.txt

beta c r is 3

Found induced in !Series_title "Comparison of endocrine enriched genes in islet beta cells versus induced beta cells"

GSE12025_series_matrix.txtimp_info.txt

M3 r is 4

Found induced in !Series_summary "Endocrine enriched genes in adult islet beta cells were identified and compared with that of induced beta cells (with M3 transcription factors) in adult. The control sample is non-beta pancreatic cells."

GSE12025_series_matrix.txtimp_info.txt

beta c r is 1

Found induced in !Series_overall_design "Gene expression profile comparison of 3 samples, 3 independent repeats for each sample indicate a high degree of similarity between endogenous and induced beta cells in adult mouse."

GSE12028_series_matrix.txtimp_info.txt

interleukin-10 r is 3

Found deficient in !Series_title "Modulation of colon inflammation in interleukin-10 gene-deficient mice via dietary polyunsaturated fatty acid"

GSE12028_series_matrix.txtimp_info.txt

interleukin-10 r is 3

Found deficient in !Series_summary "Dietary n-3 polyunsaturated fatty acids can reduce inflammation via a range of mechanisms. This study tested the effect of dietary eicosapentaenoic acid (EPA) on intestinal inflammation using interleukin-10 gene-deficient (Il10-/-) mice. Methods: At 35 days of age, 12 weaned Il10-/- and 12 C57 mice were randomly assigned to one of two modified AIN-76A diets, supplemented with 3.7% purified ethyl esters of either EPA (n-3) or oleic acid (OA, control). To identify genes relevant to colon inflammation, transcription profiling (microarrays and qRT-PCR) and bioinformatic analyses were used. Results: In this study, dietary EPA reversed the decrease in colon fatty acid β-oxidation gene expression observed in OA-fed Il10-/- compared to C57 mice. Il10-/- mice fed the OA diet showed decreased expression of antioxidant enzyme genes, as well as those involved in detoxification of xenobiotics, compared to C57 mice on the same diet. In contrast, dietary EPA increased the expression of these genes in Il10-/- mice. Conclusions: These data indicate that dietary EPA induced endogenous lipid oxidation which might have a potential anti-inflammatory effect on colon tissue. This is supported by the activation of the Ppara gene that regulates the expression of pro-inflammatory and immunomodulatory genes and proteins."

GSE12067_series_matrix.txtimp_info.txt

immediate early r is 7

Found exposure in !Series_summary "The growth factor interleukin-3 (IL-3) promotes the survival and growth of multipotent hematopoietic progenitors and stimulates myelopoiesis. It has also been reported to oppose terminal granulopoiesis and to support leukemic cell growth through autocrine or paracrine mechanisms. We used kinetic microarray, Northern Blotting and bioinformatics analysis of IL-3 dependent myeloblasts to determine whether IL-3 acts in part by regulating the rate of turnover of mRNA transcripts in specific functional pathways. Our results indicate that exposure of myeloblasts to IL-3 causes immediate early stabilization of hundreds of transcripts in pathways relevant to myeloblast function. Examples include transcripts associated with proliferation and leukemic transformation (pik3cd, myb, pim-1), hematopoietic development (cited2), differentiation control (cdkn1a) and RNA processing (BRF1, BRF2). A domain in the 3'-utr of IL-6 that mediates IL-3 responsiveness contains AU-rich elements that bind proteins known to modulate mRNA stability, however a known destabilizing protein (AUF1) is shown not to mediate degradation in the absence of IL-3. These findings support a model of IL-3 action through mRNA stability control and suggest that aberrant stabilization of this network of transcripts could contribute to growth patterns observed in leukemia."

GSE12134_series_matrix.txtimp_info.txt

Tbr2 r is 8

Found loss of in !Series_summary "mid-neurogenesis and gain-of-function experiments show that AP2γ directly regulates the expression of several genes characteristic for basal progenitors, such as Math3 and Tbr2. The misspecification of basal progenitors upon loss of AP2γ resulted in their increased death and"

GSE12135_series_matrix.txtimp_info.txt

Emx2 r is 3

Found knockout in !Series_title "Expression data from Emx2 wildtype and knockout olfactory epithelium"

GSE12135_series_matrix.txtimp_info.txt

Emx2 r is 1

Found knockout in !Series_summary "Emx2 is a homeobox transcription factor that plays a critical role in development. Olfactory sensory neuron axons from Emx2 knockout mice fail to innervate their target tissue, the olfactory bulb. Homeobox transcription factors may also play an important role in olfactory receptor expression."

GSE12135_series_matrix.txtimp_info.txt

Emx2 r is 3

Found knockout in !Series_summary "We used microarrays to analyze differences in mRNA abundance in the olfactory epithelium of Emx2 wildtype and knockout embryonic day 18.5 mice."

GSE12135_series_matrix.txtimp_info.txt

Emx2 r is 5

Found knockout in !Series_overall_design "Total RNA from 9 Emx2 wildtype and 9 Emx2 knockout mice was analyzed. Equal amounts of RNA from 3 animals was pooled to create one biological sample, 3 pools per genotype, 6 chips total. RNA was hybridized to the Affymetrix Mouse Exon 1.0 ST array."

GSE12183_series_matrix.txtimp_info.txt

Mdr2 r is 1

Found knockout in !Series_summary "We crossed the HCV-Tg mice which do not produce HCC with the Mdr2-knockout (Mdr2-KO) mice which develop inflammation-associated HCC, to generate Mdr2-KO/HCV-Tg mice. We studied the effect of the HCV transgene on tumor incidence, hepatocyte mitosis and apoptosis, and on gene expression in the liver of produced mice."

GSE12185-GPL6096_series_matrix.txtimp_info.txt

Mdr2 r is 1

Found knockout in !Series_summary "We crossed the HCV-Tg mice which do not produce HCC with the Mdr2-knockout (Mdr2-KO) mice which develop inflammation-associated HCC, to generate Mdr2-KO/HCV-Tg mice. We studied the effect of the HCV transgene on tumor incidence, hepatocyte mitosis and apoptosis, and on gene expression in the liver of produced mice."

GSE12209_series_matrix.txtimp_info.txt

Crtc1 r is 7

Found deficient in !Series_summary "The adipocyte-derived hormone leptin maintains energy balance by acting on hypothalamic leptin receptors (Leprs) that trigger the signal transducer and activator of transcription 3 (Stat3). Although disruption of Lepr-Stat3 signaling promotes obesity in mice, other features of Lepr function, such as fertility, seem normal, pointing to the involvement of additional regulators. Here we show that the cyclic AMP responsive element–binding protein-1 (Creb1)-regulated transcription coactivator-1 (Crtc1) is required for energy balance and reproduction—Crtc1-/- mice are hyperphagic, obese and infertile. Hypothalamic Crtc1 was phosphorylated and inactive in leptin-deficient ob/ob mice; leptin administration increased amounts of dephosphorylated nuclear Crtc1. Dephosphorylated Crtc1 stimulated expression of the Cartpt and Kiss1 genes, which encode hypothalamic neuropeptides that mediate leptin's effects on satiety and fertility. Crtc1 overexpression in hypothalamic cells increased Cartpt and Kiss1 gene expression, whereas Crtc1 depletion decreased it. Indeed, leptin enhanced Crtc1 activity over the Cartpt and Kiss1 promoters in cells overexpressing Lepr and these effects were disrupted by expression of a dominant-negative Creb1 polypeptide. As leptin administration increased recruitment of hypothalamic Crtc1 to Cartpt and Kiss1 promoters, our results indicate that the Creb1-Crtc1 pathway mediates the central effects of hormones and nutrients on energy balance and fertility."

GSE12209_series_matrix.txtimp_info.txt

Crtc1 r is 1

Found knockout in !Series_overall_design "Mice were fasted overnight for 18h and refed for 6h. Hypothalami were obtained from 3 wild-type and 3 Crtc1 knockout mice. Total RNA was isolated from each sample and equal amounts from each sample were pooled for the microarray."

GSE12209_series_matrix.txtimp_info.txt

Stat3 r is 5

Found -/- in !Series_summary "The adipocyte-derived hormone leptin maintains energy balance by acting on hypothalamic leptin receptors (Leprs) that trigger the signal transducer and activator of transcription 3 (Stat3). Although disruption of Lepr-Stat3 signaling promotes obesity in mice, other features of Lepr function, such as fertility, seem normal, pointing to the involvement of additional regulators. Here we show that the cyclic AMP responsive element–binding protein-1 (Creb1)-regulated transcription coactivator-1 (Crtc1) is required for energy balance and reproduction—Crtc1-/- mice are hyperphagic, obese and infertile. Hypothalamic Crtc1 was phosphorylated and inactive in leptin-

deficient ob/ob mice; leptin administration increased amounts of dephosphorylated nuclear Crtc1. Dephosphorylated Crtc1 stimulated expression of the Cartpt and Kiss1 genes, which encode hypothalamic neuropeptides that mediate leptin's effects on satiety and fertility. Crtc1 overexpression in hypothalamic cells increased Cartpt and Kiss1 gene expression, whereas Crtc1 depletion decreased it. Indeed, leptin enhanced Crtc1 activity over the Cartpt and Kiss1 promoters in cells overexpressing Lepr and these effects were disrupted by expression of a dominant-negative Creb1 polypeptide. As leptin administration increased recruitment of hypothalamic Crtc1 to Cartpt and Kiss1 promoters, our results indicate that the Creb1-Crtc1 pathway mediates the central effects of hormones and nutrients on energy balance and fertility."

GSE12209_series_matrix.txtimp_info.txt

signal transducer and activator of transcription 3 r is 8

Found -/- in !Series_summary "The adipocyte-derived hormone leptin maintains energy balance by acting on hypothalamic leptin receptors (Leprs) that trigger the signal transducer and activator of transcription 3 (Stat3). Although disruption of Lepr-Stat3 signaling promotes obesity in mice, other features of Lepr function, such as fertility, seem normal, pointing to the involvement of additional regulators. Here we show that the cyclic AMP responsive element–binding protein-1 (Creb1)-regulated transcription coactivator-1 (Crtc1) is required for energy balance and reproduction—Crtc1-/- mice are hyperphagic, obese and infertile. Hypothalamic Crtc1 was phosphorylated and inactive in leptin-deficient ob/ob mice; leptin administration increased amounts of dephosphorylated nuclear Crtc1. Dephosphorylated Crtc1 stimulated expression of the Cartpt and Kiss1 genes, which encode hypothalamic neuropeptides that mediate leptin's effects on satiety and fertility. Crtc1 overexpression in hypothalamic cells increased Cartpt and Kiss1 gene expression, whereas Crtc1 depletion decreased it. Indeed, leptin enhanced Crtc1 activity over the Cartpt and Kiss1 promoters in cells overexpressing Lepr and these effects were disrupted by expression of a dominant-negative Creb1 polypeptide. As leptin administration increased recruitment of hypothalamic Crtc1 to Cartpt and Kiss1 promoters, our results indicate that the Creb1-Crtc1 pathway mediates the central effects of hormones and nutrients on energy balance and fertility."

GSE12209_series_matrix.txtimp_info.txt

Kiss1 r is 6

Found stimulated in !Series_summary "The adipocyte-derived hormone leptin maintains energy balance by acting on hypothalamic leptin receptors (Leprs) that trigger the signal transducer and activator of transcription 3 (Stat3). Although disruption of Lepr-Stat3 signaling promotes obesity in mice, other features of Lepr function, such as fertility, seem normal, pointing to the involvement of additional regulators. Here we show that the cyclic AMP responsive element–binding protein-1 (Creb1)-regulated transcription coactivator-1 (Crtc1) is required for energy balance and reproduction—Crtc1-/- mice are hyperphagic, obese and infertile. Hypothalamic Crtc1 was phosphorylated and inactive in leptin-deficient ob/ob mice; leptin administration increased amounts of dephosphorylated nuclear Crtc1. Dephosphorylated Crtc1 stimulated expression of the Cartpt and Kiss1 genes, which encode

hypothalamic neuropeptides that mediate leptin's effects on satiety and fertility. Crtc1 overexpression in hypothalamic cells increased Cartpt and Kiss1 gene expression, whereas Crtc1 depletion decreased it. Indeed, leptin enhanced Crtc1 activity over the Cartpt and Kiss1 promoters in cells overexpressing Lepr and these effects were disrupted by expression of a dominant-negative Creb1 polypeptide. As leptin administration increased recruitment of hypothalamic Crtc1 to Cartpt and Kiss1 promoters, our results indicate that the Creb1-Crtc1 pathway mediates the central effects of hormones and nutrients on energy balance and fertility."

GSE12294_series_matrix.txtimp_info.txt

Tbr2 r is 7

Found induced in !Series_summary "Basal (intermediate) progenitors are the major source of neurons in the mammalian cerebral cortex. The molecular machinery governing basal progenitor biogenesis is unknown. Here we show that the zinc finger transcription factor Insm1 (insulinoma-associated 1) is expressed specifically in progenitors undergoing neurogenic divisions and has a key role in basal progenitor formation. Mouse embryos lacking Insm1 contained half the number of basal progenitors and showed a marked reduction in cortical plate radial thickness. Forced premature expression of Insm1 in neuroepithelial cells resulted in their mitosis occurring at the basal (rather than apical) side of the ventricular zone and induced expression of the basal progenitor marker Tbr2. Remarkably, these cells remained negative for Tis21, a marker of neurogenic progenitors, and did not generate neurons but underwent self-amplification. Our data imply that Insm1 is involved in the generation and expansion of basal progenitors, a hallmark of cerebral cortex evolution."

GSE12315_series_matrix.txtimp_info.txt

Gpx1 r is 5

Found knockout in !Series_summary "Using glutathione peroxidase, Gpx1 and Gpx2, double knockout (Gpx1/2-KO) mice as a model of inflammatory bowel disease predisposing to intestinal cancer, we analyzed genome-wide DNA methylation and chromatin changing's in the mouse ileum during chronic inflammation, aging and cancer. We found that inflammation leads to aberrant DNA methylation in Polycomb target genes, with 70% of the ~250 genes methylated in the inflamed tissue being PcG targets in embryonic stem cells and 58% of the methylated genes being marked by H3K27 trimethylation in the ileum of adult wildtype mice. Acquisition of DNA methylation at CpG islands in the ileum of Gpx-1/2-KO mice frequently correlated with loss of H3K27 trimethylation at the same loci. Inflammation-associated DNA methylation occurs preferentially in tissue-specific silent genes and, importantly, is much more frequently represented in tumors than is age-dependent DNA methylation. 60% of aberrant methylation found in tumors is also present in the inflamed tissue. In summary, inflammation creates a signature of aberrant DNA methylation, which is observed later in the malignant tissue and is directed by the PcG complex."

GSE12315_series_matrix.txtimp_info.txt

Gpx2 r is 3

Found knockout in !Series_summary "Using glutathione peroxidase, Gpx1 and Gpx2, double knockout (Gpx1/2-KO) mice as a model of inflammatory bowel disease predisposing to intestinal cancer, we analyzed genome-wide DNA methylation and chromatin changing's in the mouse ileum during chronic inflammation, aging and cancer. We found that inflammation leads to aberrant DNA methylation in Polycomb target genes, with 70% of the ~250 genes methylated in the inflamed tissue being PcG targets in embryonic stem cells and 58% of the methylated genes being marked by H3K27 trimethylation in the ileum of adult wildtype mice. Acquisition of DNA methylation at CpG islands in the ileum of Gpx-1/2-KO mice frequently correlated with loss of H3K27 trimethylation at the same loci. Inflammation-associated DNA methylation occurs preferentially in tissue-specific silent genes and, importantly, is much more frequently represented in tumors than is age-dependent DNA methylation. 60% of aberrant methylation found in tumors is also present in the inflamed tissue. In summary, inflammation creates a signature of aberrant DNA methylation, which is observed later in the malignant tissue and is directed by the PcG complex."

GSE12346_series_matrix.txtimp_info.txt

Th2 r is 5

Found -/- in !Series_summary "Th2 cells enable humoral immunity and host-defense to parasites. Whereas IL-4 drives Th2 differentiation and IL-2 is important later in this process by augmenting IL4 chromatin accessibility, we demonstrate that IL-2 serves an essential early role in regulating IL-4Rα expression by inducing binding of Stat5a and Stat5b to the IL4ra locus, with sustained binding during Th2 differentiation. Although IL-4 induces IL-4Rα expression, TCR-induced IL-4Rα expression was unexpectedly normal in ILr-/- but profoundly diminished in IL2-/- T cells. Remarkably, enforced IL-4Rα expression rescued Th2 differentiation in IL2-/- cells. These results reveal a novel function for IL-2, with IL-2 via Stat5 providing an early signal for IL4ra induction, thereby priming cells for Th2 differentiation and promoting/maintaining IL-4Rα expression in Th2-committed cells."

GSE12367_series_matrix.txtimp_info.txt

E1 r is 2

Found treated in !Series_overall_design "To compare the gene expression profiles of Deaf-1-transduced MECs relative to Deaf-1-deficient MECs, Affymetrix analysis was performed using the GeneChip® Mouse Expression Set 430 2.0 array which comprises 39,000 transcripts on a single array. Total RNA was harvested from two independent clones (C1 and E1) infected with either control or Deaf-1-expressing retrovirus making 4 samples in total. Clones C1 and E1 were treated as biological replicates in the subsequent analysis."

GSE12367_series_matrix.txtimp_info.txt

C1 r is 4

Found treated in !Series_overall_design "To compare the gene expression profiles of Deaf-1-transduced MECs relative to Deaf-1-deficient MECs, Affymetrix analysis was performed using the GeneChip® Mouse Expression Set 430 2.0 array which comprises 39,000 transcripts on a single array. Total RNA was harvested from two independent clones (C1 and E1) infected with either control or Deaf-1-expressing retrovirus making 4 samples in total. Clones C1 and E1 were treated as biological replicates in the subsequent analysis."

GSE12412_series_matrix.txtimp_info.txt

E15 r is 7

Found knockout in !Series_summary "The gene expression profiles of control vs AGTR2 knockout mouse whole brains at developmental stage E15 and postnatal day 1 were examined."

GSE12415_series_matrix.txtimp_info.txt

Hsf4 r is 3

Found lacking in !Series_summary "Microarray Analyses of Newborn Mouse lens lacking HSF4. Hsf4 is essential for lens development."

GSE12421_series_matrix.txtimp_info.txt

OBF-1 r is 2

Found overexpression in !Series_title "Analysis of OBF-1 overexpression in early B cells"

GSE12425_series_matrix.txtimp_info.txt

Notch4 r is 1

Found overexpression in !Series_title "Dox-regulated Notch4 overexpression in mouse ES cells redirects hemagioblasts to a cardiac fate."

GSE12425_series_matrix.txtimp_info.txt

Flk-1 r is 7

Found stimulation in !Series_summary "To investigate the role of Notch signalling in the establishment of cardiac lineages, we used a tet-inducible ES cell line (Ainv18) engineered to express an activated form of the Notch4 receptor following doxycycline treatment. This line also expresses a GFP cDNA from the Bry locus. Following 3.0-3.5 days of serum stimulation, three distinct populations based on Flk-1 and GFP expression are observed: Bry-GFP-/Flk-1-, Bry-GFP+/Flk-1- and Bry-GFP+/Flk-1+ cells. Previous studies have shown that the Bry-GFP+/Flk-1+ population contains hemangioblasts, whereas the Bry-GFP+/Flk-1- population displays cardiac potential."

GSE12430_series_matrix.txtimp_info.txt

Math1 r is 8

Found knockout in !Series_summary "Cells from 5 GFAP-Cre tumors, 5 Math1-Cre tumors, and 5 conventional patched-knockout tumors will be isolated using enzymatic digestion and Percoll gradient centrifugation. These procedures have been found to result in 85-95% pure tumor cell populations. In addition, 5 tumors will be FACS-sorted to isolate CD15+ and CD15- populations. RNA will be prepared from each of these samples using an RNeasy kit from Qiagen. Samples of 2 micrograms will be resuspended in 10 microliters of RNase-free water, and sent to the Consortium for labeling, hybridization and analysis. Cells from the three types of tumors, and CD15+ and CD15- cells, will be compared to one another."

GSE12441_series_matrix.txtimp_info.txt

Wnt3a r is 3

Found treated in !Series_overall_design "Cells derived from mouse embryonic stage 11.5 limb buds were cultured and treated with purified Wnt3a protein or vehicle controls. The transcriptional response was detected using spotted cDNA microarrays after 2 hrs or 4 hrs of treatment. 4 biological replicates were used per condition."

GSE12464_series_matrix.txtimp_info.txt

CD3 r is 1

Found stimulation in !Series_summary "The Tec-family kinase Itk plays an important role during T-cell activation and function, and controls also conventional versus innate-like T-cell development. We have characterized the transcriptome of Itk-deficient CD3+ T-cells, including CD4+ and CD8+ subsets, using Affymetrix microarrays. The largest difference between Itk-/- and Wt CD3+ T-cells was found in unstimulated cells, e.g. for killer cell lectin-like receptors. Compared to anti-CD3-stimulation, anti-CD3/CD28 significantly decreased the number of transcripts suggesting that the CD28 co-stimulatory pathway is mainly independent of Itk. The signatures of CD4+ and CD8+ T-cell subsets identified a greater differential expression than in total CD3+ cells. Cyclosporin (CsA)-treatment had a stronger effect on transcriptional regulation than Itk-deficiency, suggesting that only a fraction of TCR-mediated calcineurin/NFAT-activation is dependent on Itk. Bioinformatic analysis of NFAT-sites of the group of transcripts similarly regulated by Itk-deficiency and CsA-treatment, followed by chromatin-immunoprecipitation, revealed NFATc1-binding to the Bub1, IL7R, Ctla2a, Ctla2b, and Schlafen1 genes. Finally, to identify transcripts that are regulated by Tec-family kinases in general, we compared the expression profile of Itk-deficient T-cells with that of Btk-deficient B-cells and a common set of transcripts was found. Taken together, our study provides a general overview about the global transcriptional changes in the absence of Itk."

GSE12465_series_matrix.txtimp_info.txt

CD3 r is 1

Found stimulation in !Series_summary "The Tec-family kinase Itk plays an important role during T-cell activation and function, and controls also conventional versus innate-like T-cell development. We have characterized the transcriptome of Itk-deficient CD3+ T-cells, including CD4+ and CD8+ subsets, using Affymetrix microarrays. The largest difference between Itk-/- and Wt CD3+ T-cells was found in unstimulated cells, e.g. for killer cell lectin-like receptors. Compared to anti-CD3-stimulation, anti-CD3/CD28 significantly decreased the number of transcripts suggesting that the CD28 co-stimulatory pathway is mainly independent of Itk. The signatures of CD4+ and CD8+ T-cell subsets identified a greater differential expression than in total CD3+ cells. Cyclosporin (CsA)-treatment had a stronger effect on transcriptional regulation than Itk-deficiency, suggesting that only a fraction of TCR-mediated calcineurin/NFAT-activation is dependent on Itk. Bioinformatic analysis of NFAT-sites of the group of transcripts similarly regulated by Itk-deficiency and CsA-treatment, followed by chromatin-immunoprecipitation, revealed NFATc1-binding to the Bub1, IL7R, Ctla2a, Ctla2b, and Schlafen1 genes. Finally, to identify transcripts that are regulated by Tec-family kinases in general, we compared the expression profile of Itk-deficient T-cells with that of Btk-deficient B-cells and a common set of transcripts was found. Taken together, our study provides a general overview about the global transcriptional changes in the absence of Itk."

GSE12467_series_matrix.txtimp_info.txt

N-myc r is 2

Found deficient in !Series_title "Differentially regulated genes in control and c-myc N-myc deficient LT-HSCs"

GSE12467_series_matrix.txtimp_info.txt

N-myc r is 2

Found deficient in !Series_summary "Analysis of HSCs from control and c-myc N-myc deficient long-term hematopoietic stem cells. HSCs lacking both c-myc and N-myc display increased apoptosis rates. Data provide insight into the molecular changes occuring upon complete loss of Myc activity, clarifying the resulting apoptotic mechanism and the role of Myc family proteins in HSCs. "

GSE12489_series_matrix.txtimp_info.txt

V1 r is 6

Found treated in !Series_overall_design "Animals were injected i.p. 100mg/kg phenobarbital or vehicle (5% DMSO in corn oil). After 12h they were sacrificed and total RNA was isolated from the livers. Pools of untreated samples were mixed in each genetic variant group (wild type and CAR-/-, PXR-/- or CAR/PXR-/-) with the phenobarbital treated ones and hybridized to Sterolgene V1 arrays."

GSE12498_series_matrix.txtimp_info.txt

Tead2 r is 4

Found overexpression in !Series_summary "Regulation of organ size is important for development and tissue homeostasis. In Drosophila, Hippo signaling controls organ size by regulating the activity of a TEAD transcription factor, Scalloped, through modulation of its coactivator protein Yki. The role of mammalian Tead proteins in growth regulation, however, remains unknown. Here we examined the role of mouse Tead proteins in growth regulation. In NIH3T3 cells, cell density and Hippo signaling regulated the activity of Tead proteins by modulating nuclear localization of a Yki homologue, Yap, and the resulting change in Tead activity altered cell proliferation. Tead2-VP16 mimicked Yap overexpression, including increased cell proliferation, reduced cell death, promotion of EMT, lack of cell contact inhibition, and promotion of tumor formation. Growth promoting activities of various Yap mutants correlated with their Tead-coactivator activities. Tead2-VP16 and Yap regulated largely overlapping sets of genes. However, only a few of the Tead/Yapregulated genes in NIH3T3 cells were affected in Tead1-/-;Tead2-/- or Yap-/- embryos. Most of the previously identified Yap-regulated genes were not affected in NIH3T3 cells or mutant mice. In embryos, levels of nuclear Yap and Tead1 varied depending on cell types. Strong nuclear accumulation of Yap and Tead1 were seen in myocardium, correlating with requirements of Tead1 for proliferation. However, their distribution did not always correlate with proliferation. Taken together, mammalian Tead proteins regulate cell proliferation and contact inhibition as a transcriptional mediator of Hippo signaling, but the mechanisms by which Tead/Yap regulate cell proliferation differ depending on cell types, and Tead, Yap and Hippo signaling may play multiple roles in mouse embryos."

GSE12503_series_matrix.txtimp_info.txt

Agr2 r is 2

Found -/- in !Series_summary "Keywords: small intestine and colon gene expression profiles for Agr2-/- and littermate control mice"

GSE12506_series_matrix.txtimp_info.txt

CD25 r is 2

Found induced in !Series_overall_design "To define the molecular signature of Ag-specific in vivo-induced dtg CD25 TR cells in comparison to naturally occurring CD25 TR cells, we performed comparative gene expression profiling by Affymetrix microarray analysis. Sorted splenic wild-type (WT) TR cells, stg TR cells, dtg CD25- TR cells, dtg CD25+ TR cells, in vitro-stimulated stg 16h TA cells, stg 3d TA cells as well as stg TN cells, were included in the experiment and analyses were performed in triplicates."

GSE12509_series_matrix.txtimp_info.txt

V1 r is 6

Found treated in !Series_overall_design "Animals were injected i.p. 10mg/kg TCPOBOP or vehicle (5% DMSO in corn oil). After 12h they were sacrificed and total RNA was isolated from the livers. Pools of

untreated samples were mixed in each genetic variant group (wild type and CAR-/-, PXR-/- or CAR/PXR-/-) with the TCPOBOP treated ones and hybridized to Sterolgene V1 arrays."

GSE12536_series_matrix.txtimp_info.txt

N-myc r is 2

Found deficient in !Series_title "Differentially regulated genes in control and c-myc N-myc deficient progenitors"

GSE12536_series_matrix.txtimp_info.txt

N-myc r is 2

Found deficient in !Series_summary "Analysis of HSCs from control and c-myc N-myc deficient long-term hematopoietic stem cells. HSCs lacking both c-myc and N-myc display increased apoptosis rates. Data provide insight into the molecular changes occuring upon complete loss of Myc activity, clarifying the resulting apoptotic mechanism and the role of Myc family proteins in HSCs and commited progenitors."

GSE12538_series_matrix.txtimp_info.txt

N-myc r is 2

Found deficient in !Series_title "Differentially regulated genes in control and c-myc N-myc deficient LT-HSCs and progenitors"

GSE12541-GPL2872_series_matrix.txtimp_info.txt

Aoh2 r is 1

Found -/- in !Series_summary "The mouse aldehyde oxidase, Aoh2 (aldehyde oxidase homolog 2), is a molybdo-flavoenzyme. Harderian glands are the richest source of Aoh2, although the protein is detectable also in sebaceous glands, epidermis and other keratinized epithelia. The levels of Aoh2 in the Harderian gland and skin are controlled by genetic background, being maximal in CD1 or C57BL/6 and minimal in DBA/2, CBA or 129/Sv strains. Testosterone is a negative regulator of Aoh2 in Harderian glands. Homogenously purified Aoh2 oxidizes retinaldehyde into retinoic acid efficiently, while it is devoid of pyridoxal oxidizing activity, unlike other aldehyde oxidases. Aoh2 knockout mice are viable and fertile, although they have an absolute deficit of retinaldehyde oxidase activity in the Harderian gland, decreased retinoic acid levels, and down-regulation of retinoid-dependent genes. The Harderian gland's transcriptome of knockout mice is characterized by perturbations in pathways controlling lipid homeostasis and cellular secretion. This is particularly evident in sexually immature animals. Lower amounts of retinoic acid are also observed in the skin of Aoh2-/- animals. Knockout mice have thickening of the epidermis in basal conditions and after UV light exposure. This has correlates in the skin transcriptome, which shows enrichment and overall up-regulation of genes involved in hypertrophic responses."

GSE12541-GPL2872_series_matrix.txtimp_info.txt

aldehyde oxidase homolog 2 r is 2

Found -/- in !Series_summary "The mouse aldehyde oxidase, Aoh2 (aldehyde oxidase homolog 2), is a molybdo-flavoenzyme. Harderian glands are the richest source of Aoh2, although the protein is detectable also in sebaceous glands, epidermis and other keratinized epithelia. The levels of Aoh2 in the Harderian gland and skin are controlled by genetic background, being maximal in CD1 or C57BL/6 and minimal in DBA/2, CBA or 129/Sv strains. Testosterone is a negative regulator of Aoh2 in Harderian glands. Homogenously purified Aoh2 oxidizes retinaldehyde into retinoic acid efficiently, while it is devoid of pyridoxal oxidizing activity, unlike other aldehyde oxidases. Aoh2 knockout mice are viable and fertile, although they have an absolute deficit of retinaldehyde oxidase activity in the Harderian gland, decreased retinoic acid levels, and down-regulation of retinoid-dependent genes. The Harderian gland's transcriptome of knockout mice is characterized by perturbations in pathways controlling lipid homeostasis and cellular secretion. This is particularly evident in sexually immature animals. Lower amounts of retinoic acid are also observed in the skin of Aoh2-/- animals. Knockout mice have thickening of the epidermis in basal conditions and after UV light exposure. This has correlates in the skin transcriptome, which shows enrichment and overall up-regulation of genes involved in hypertrophic responses."

GSE12541-GPL7202_series_matrix.txtimp_info.txt

Aoh2 r is 1

Found -/- in !Series_summary "The mouse aldehyde oxidase, Aoh2 (aldehyde oxidase homolog 2), is a molybdo-flavoenzyme. Harderian glands are the richest source of Aoh2, although the protein is detectable also in sebaceous glands, epidermis and other keratinized epithelia. The levels of Aoh2 in the Harderian gland and skin are controlled by genetic background, being maximal in CD1 or C57BL/6 and minimal in DBA/2, CBA or 129/Sv strains. Testosterone is a negative regulator of Aoh2 in Harderian glands. Homogenously purified Aoh2 oxidizes retinaldehyde into retinoic acid efficiently, while it is devoid of pyridoxal oxidizing activity, unlike other aldehyde oxidases. Aoh2 knockout mice are viable and fertile, although they have an absolute deficit of retinaldehyde oxidase activity in the Harderian gland, decreased retinoic acid levels, and down-regulation of retinoid-dependent genes. The Harderian gland's transcriptome of knockout mice is characterized by perturbations in pathways controlling lipid homeostasis and cellular secretion. This is particularly evident in sexually immature animals. Lower amounts of retinoic acid are also observed in the skin of Aoh2-/- animals. Knockout mice have thickening of the epidermis in basal conditions and after UV light exposure. This has correlates in the skin transcriptome, which shows enrichment and overall up-regulation of genes involved in hypertrophic responses."

GSE12541-GPL7202_series_matrix.txtimp_info.txt

aldehyde oxidase homolog 2 r is 2

Found -/- in !Series_summary "The mouse aldehyde oxidase, Aoh2 (aldehyde oxidase homolog 2), is a molybdo-flavoenzyme. Harderian glands are the richest source of Aoh2, although the protein is detectable also in sebaceous glands, epidermis and other keratinized epithelia. The levels of Aoh2 in the Harderian gland and skin are controlled by genetic background, being maximal in CD1 or C57BL/6 and minimal in DBA/2, CBA or 129/Sv strains. Testosterone is a negative regulator of Aoh2 in Harderian glands. Homogenously purified Aoh2 oxidizes retinaldehyde into retinoic acid efficiently, while it is devoid of pyridoxal oxidizing activity, unlike other aldehyde oxidases. Aoh2 knockout mice are viable and fertile, although they have an absolute deficit of retinaldehyde oxidase activity in the Harderian gland, decreased retinoic acid levels, and down-regulation of retinoid-dependent genes. The Harderian gland's transcriptome of knockout mice is characterized by perturbations in pathways controlling lipid homeostasis and cellular secretion. This is particularly evident in sexually immature animals. Lower amounts of retinoic acid are also observed in the skin of Aoh2-/- animals. Knockout mice have thickening of the epidermis in basal conditions and after UV light exposure. This has correlates in the skin transcriptome, which shows enrichment and overall up-regulation of genes involved in hypertrophic responses."

GSE12545_series_matrix.txtimp_info.txt

Gfi1 r is 2

Found -/- in !Series_title "Global gene expression analysis between Gfi1+/+ and Gfi1-/- splenic B cells"

GSE12545_series_matrix.txtimp_info.txt

CD138 r is 1

Found -/- in !Series_overall_design "Splenic B220+CD19+ CD138- B cells of 4 week old Gfi1+/+ and Gfi1-/- mice were isolated and RNA was extracted from one sample per group and microarray analysis was performed."

GSE12581_series_matrix.txtimp_info.txt

murine leukemia retrovirus r is 4

Found induced in !Series_title "Gene expression profiles of the lymphoid and non-lymphoid leukemias induced by the Graffi murine leukemia retrovirus"

GSE12591_series_matrix.txtimp_info.txt

angiotensin I r is 3

Found -/- in !Series_summary "In this study we used microarrays to examine relative genes expression within the aorta of ApoE-/- infused with angiotensin II in relation to aneurysm formation. Infusion of angiotensin II induces aortic dilatation particularly of the suprarenal aorta in ApoE-/- mice. Based on studies carried out in our and other laboratories the response to angiotensin II is variable, with some

mice developing large aneurysms but other animals appearing resistant to aneurysm formation with aortic diameters similar to that of saline controls. We compared RNA expression from whole aortas of 17 week old male ApoE-/- mice exposed to angiotensin II (1.44 µg/kg/min) for 4 weeks where there was clear evidence of aortic aneurysm formation (n=5) with that of mice failing to develop aneurysms (n=7) and those exposed to saline infusion (n=6). AAA was defined as diameter of suprarenal aorta greated than 1.5mm measured on photographs of aortas at necroscopy."

GSE12609_series_matrix.txtimp_info.txt

factor A r is 2

Found null in !Series_title "Transcription factor Arx null brains (fulp-affy-mouse-364520)"

GSE12673_series_matrix.txtimp_info.txt

alpha-MHC r is 5

Found expressing in !Series_overall_design "Bi-allelic transgenic mice were created by crossing alpha-MHC promoter/tet transactivating protein expressing mice with tet responsive element promter/stable HIF-1alpha protein expressing mice. Mice were either, maintained on Doxycycline (inhibiting the expression of the HIF-1alpha transgene) or removed from doxycycline (inducing expression) for one or three days."

GSE12687_series_matrix.txtimp_info.txt

GAP-43 r is 6

Found lacking in !Series_summary "Mice lacking the growth associated protein, GAP-43, (KO) show multiple deficits in forebrain axon guidance and cortical cell differentiation (Donovan and McCasland, 2005). As a result, GAP-43 KO mice fail to form barrels in mouse somatosensory cortex (S1) (Maier et al., 1999). GAP-43 heterozygous (HZ) mice show abnormalities in axonal pathfinding and show larger than normal barrels in layer IV S1 due to widely branched thalamocortical afferents (TCAs). Regardless of abnormalities during early development, HZ barrels become indistinguishable from WT by postnatal day 26. One explanation for these findings is that compensatory mechanisms may be activated in GAP-43 HZ cortex. We have used mRNA microarray expression analysis to gain a more comprehensive view of genes involved in GAP-43 signaling during barrel map formation. Using laser microdissection , cortical cells of the barrel cortex were excised, RNA extracted and used in GeneChip analysis. Expression profiling and functional gene group analysis of RNA from WT, HZ, and KO cortex at postnatal day 5 was performed. We identified thousands of transcripts differentially expressed across the genotypes. Verification of selected changes in gene expression was accomplished using in situ hybridization. Our results suggest an adaptive modification in transcript expression of genes involved in cell-cell communication and synaptogenesis. These modifications appear important in forward and reverse signaling as well as maintaining synchrony between the cells. Compensatory up- and down regulation of synapse-associated genes may explain the reverse in HZ phenotype from P7 to P26. Moreover, these

findings provide new insight into the role GAP-43 plays in several pathways associated with synaptogenesis and trans-synaptic signaling."

GSE12694_series_matrix.txtimp_info.txt

p53 r is 1

Found null in !Series_summary "We used microarrays to detail the gene expression difference of the p53-null and p53/Pten-doubly null neural stem cell after differentiation ."

GSE12694_series_matrix.txtimp_info.txt

p53 r is 1

Found null in !Series_overall_design "transcriptome comparisons of 2 independent p53-null with 3 p53/Pten double-null murine NSCs at 1 day post exposure to the differentiation inducer."

GSE12697_series_matrix.txtimp_info.txt

TNF-a r is 2

Found induced in !Series_title "Trap-80-dependence of TNF-alpha-induced genes"

GSE12697_series_matrix.txtimp_info.txt

TNF-alpha r is 2

Found induced in !Series_title "Trap-80-dependence of TNF-alpha-induced genes"

GSE12697_series_matrix.txtimp_info.txt

TNF-a r is 7

Found stimulated in !Series_overall_design "RNA was extracted from three independent cultures of wild-type and Trap-80 knock-down fibroblasts, before and after stimulation for 1 hour with 5ng/ml TNF-alpha. The unstimulated and stimulated wild-type samples, and the stimulated Trap-80 knock-down samples, were used for microarray analysis."

GSE12697_series_matrix.txtimp_info.txt

TNF-alpha r is 7

Found stimulated in !Series_overall_design "RNA was extracted from three independent cultures of wild-type and Trap-80 knock-down fibroblasts, before and after stimulation for 1 hour with 5ng/ml TNF-alpha. The unstimulated and stimulated wild-type samples, and the stimulated Trap-80 knock-down samples, were used for microarray analysis."

GSE12697_series_matrix.txtimp_info.txt

NF-kappaB r is 2

Found stimulation in !Series_summary "In fibroblasts, p65-dependent genes can be sub-divided, depending on whether they are Trap-80-dependent or -independent. To examine the generality of this grouping, we performed a microarray analysis of wild-type and Trap-80 knock-down fibroblasts, before and after stimulation of NF-kappaB activity using TNF-alpha."

GSE12697_series_matrix.txtimp_info.txt

TNF-a r is 6

Found stimulation in !Series_summary "In fibroblasts, p65-dependent genes can be sub-divided, depending on whether they are Trap-80-dependent or -independent. To examine the generality of this grouping, we performed a microarray analysis of wild-type and Trap-80 knock-down fibroblasts, before and after stimulation of NF-kappaB activity using TNF-alpha."

GSE12697_series_matrix.txtimp_info.txt

TNF-alpha r is 6

Found stimulation in !Series_summary "In fibroblasts, p65-dependent genes can be sub-divided, depending on whether they are Trap-80-dependent or -independent. To examine the generality of this grouping, we performed a microarray analysis of wild-type and Trap-80 knock-down fibroblasts, before and after stimulation of NF-kappaB activity using TNF-alpha."

GSE12697_series_matrix.txtimp_info.txt

TNF-a r is 6

Found stimulation in !Series_overall_design "RNA was extracted from three independent cultures of wild-type and Trap-80 knock-down fibroblasts, before and after stimulation for 1 hour with 5ng/ml TNF-alpha. The unstimulated and stimulated wild-type samples, and the stimulated Trap-80 knock-down samples, were used for microarray analysis."

GSE12697_series_matrix.txtimp_info.txt

TNF-alpha r is 6

Found stimulation in !Series_overall_design "RNA was extracted from three independent cultures of wild-type and Trap-80 knock-down fibroblasts, before and after stimulation for 1 hour with 5ng/ml TNF-alpha. The unstimulated and stimulated wild-type samples, and the stimulated Trap-80 knock-down samples, were used for microarray analysis."

GSE12707_series_matrix.txtimp_info.txt

Atg16l1 r is 1

Found mutant in !Series_summary "The aim of this study is to survey global gene expression of total thymocytes from wild-type mice and Atg16l1 mutant (hypomorph) mice."

GSE12712_series_matrix.txtimp_info.txt

Rsl1 r is 6

Found null in !Series_overall_design "Total RNA was extracted from livers of adult male and female wild type, rsl-null and transgenic mice that overexpress Rsl1 or Rsl2, specifically in the liver. Equivalent amounts of RNA were pooled from five or more mice per sex and genotype. Two pools per sex and genotype were hybridized to the Affymetrix Mouse Genome 430 2.0 array."

GSE12712_series_matrix.txtimp_info.txt

Rsl2 r is 8

Found null in !Series_overall_design "Total RNA was extracted from livers of adult male and female wild type, rsl-null and transgenic mice that overexpress Rsl1 or Rsl2, specifically in the liver. Equivalent amounts of RNA were pooled from five or more mice per sex and genotype. Two pools per sex and genotype were hybridized to the Affymetrix Mouse Genome 430 2.0 array."

GSE12753_series_matrix.txtimp_info.txt

Rex1 r is 1

Found -/- in !Series_overall_design "We established ES cell lines with four different genotypes for Rex1; ES cells carrying the wild-type Rex1 alleles and the empty CAG-IZ vector (wt), the wild-type Rex1 alleles and the Rex1 transgene (wt-Tg), the Rex1-/- alleles and the empty vector(KO), and the Rex1-/- alleles and the Rex1 transgene (KO-Tg). The genetically-engineered ES cell lines were generated to analyze the function of Rex1 in the maintenance of pluripotency and to analyze its gain- and loss-of function. For loss-of-function analysis, we disrupted the endogenous Rex1 allele by conventional gene targeting via homologous recombination in ES cells. The knock-out (KO) allele should be a functionally null allele because the first 100 bp of the open reading frame in the exon 4 including the start codon was replaced by the pacEGFP chimeric gene cassette containing the puromycin-resistant gene (pac) and the green fluorescent protein (Egfp) cDNA. Interestingly, all of the puromycin resistant clones obtained by transfection of this KO vector carried the correctly targeted alleles. One of the Rex1+/- ES cell line (RKPG9) was cultured with high-dose puromycin to obtain the Rex1-/- ES cell lines generated via spontaneous gene conversion. Multiple Rex1-/- ES cell lines were established with extremely high efficiency (4 of 4 clones obtained after the selection were homozygous for Rex1 KO allele). Correct targeting events were confirmed by the loss of the polymorphic signature of the wild-type allele on the southern blot analysis of the genomic DNA, in which the 5.6 kb fragment corresponds to the Rex1 pseudogene on chromosome 15 reported previously as well as found in the mouse genome data. Northern blot revealed the loss of the transcript derived from the wild-type allele in Rex1-/- ES cells, which express the large transcripts composed by the truncated Rex1 and pacEGFP. Rex1-/- ES cells were

also established by introduction of the second knockout vector carrying the hygromycin-resistant gene as a selection marker."

GSE12753_series_matrix.txtimp_info.txt

Rex1 r is 6

Found -/- in !Series_overall_design "Rex1+/- ES cell line (RKPG9), Rex1-/- ES cells (HP3 and HP4), and one wild-type ES cells (EB5)"

GSE12836_series_matrix.txtimp_info.txt

PDGF-B r is 2

Found overexpression in !Series_summary "We analyzed the generation of mouse gliomas following the overexpression of PDGF-B in embryonic neural progenitors. Comparison of our microarray data, with published gene expression data sets for many different murine neural cell types, revealed a closest relationship between our tumor cells and oligodendrocyte progenitor cells, confirming definitively that PDGF-B-induced gliomas are pure oligodendrogliomas."

GSE12863_series_matrix.txtimp_info.txt

Ets2 r is 8

Found deficient in !Series_overall_design "Breast TAMs were isolated from early-stage PyMT-induced mammary tumors expressing Ets2 and also from the tumors with Ets2-deficient TAMs. Since macrophages have also been implicated in normal mammary gland remodeling, normal remeodeling macrophages were also purified from females expressing Ets2 and the ones where Ets2 is deleted in the macrophages. One RNA sample was extracted from each genetic group for gene-expression profiling."

GSE12863_series_matrix.txtimp_info.txt

Ets2 r is 4

Found induced in !Series_overall_design "Breast TAMs were isolated from early-stage PyMT-induced mammary tumors expressing Ets2 and also from the tumors with Ets2-deficient TAMs. Since macrophages have also been implicated in normal mammary gland remodeling, normal remeodeling macrophages were also purified from females expressing Ets2 and the ones where Ets2 is deleted in the macrophages. One RNA sample was extracted from each genetic group for gene-expression profiling."

GSE12863_series_matrix.txtimp_info.txt

Ets2 r is 1

Found expressing in !Series_overall_design "Breast TAMs were isolated from early-stage PyMT-induced mammary tumors expressing Ets2 and also from the tumors with Ets2-deficient TAMs. Since macrophages have also been implicated in normal mammary gland remodeling, normal remeodeling

macrophages were also purified from females expressing Ets2 and the ones where Ets2 is deleted in the macrophages. One RNA sample was extracted from each genetic group for gene-expression profiling."

GSE12881_series_matrix.txtimp_info.txt

Cav-3 r is 2

Found knockout in !Series_overall_design "All WT and Cav-3 knockout (KO) mice used in this study were in the FVB/N genetic background. 4-month old virgin female mice were utilized in a micro array study between 3 wildtype and 3 Caveolin-3 knock-out mammary glands."

GSE12905_series_matrix.txtimp_info.txt

Foxl2 r is 1

Found null in !Series_summary "Comparison of Foxl2-null ovaries to wildtype ovaries, ovaries lacking Wnt4 or Kit, or testes, throughout mouse development."

GSE12905_series_matrix.txtimp_info.txt

Wnt4 r is 8

Found null in !Series_summary "Comparison of Foxl2-null ovaries to wildtype ovaries, ovaries lacking Wnt4 or Kit, or testes, throughout mouse development."

GSE12905_series_matrix.txtimp_info.txt

Foxl2 r is 8

Found lacking in !Series_summary "Comparison of Foxl2-null ovaries to wildtype ovaries, ovaries lacking Wnt4 or Kit, or testes, throughout mouse development."

GSE12905_series_matrix.txtimp_info.txt

Wnt4 r is 1

Found lacking in !Series_summary "Comparison of Foxl2-null ovaries to wildtype ovaries, ovaries lacking Wnt4 or Kit, or testes, throughout mouse development."

GSE12950_series_matrix.txtimp_info.txt

normal AK r is 3

Found treated in !Series_summary "Cryptorchidism and scrotal heating result in abnormal spermatogenesis but the mechanism(s) proscribing this temperature sensitivity are unknown. It was previously reported that the AKR/N or MRL/MpJ-+/+ mouse testis is more heat resistant than the testis from the C57BL/6 strain. We have attempted to probe into the mechanism(s) involved in heat sensitivity by examining global gene expression profiles of normal and heat-treated testes from

C57BL/6, AKR/N and MRL/MpJ-+/+ mice by microarray analysis. In the normal C57BL/6 testis, 415 and 416 transcripts were differentially expressed (at least two-fold higher or lower) when compared to the normal AKR/N and MRL/MpJ-+/+ testis, respectively. The AKR/N and MRL/MpJ-+/+ strains revealed 268 differentially expressed transcripts between them. There were 231 transcripts differentially expressed between C57BL/6 and two purported heat-resistant strains, AKR/N and MRL/MpJ-+/+."

GSE12954_series_matrix.txtimp_info.txt

Dll1 r is 1

Found mutant in !Series_title "Expression Profiling of Dll1 mutant mouse line"

GSE12954_series_matrix.txtimp_info.txt

Dll1 r is 1

Found mutant in !Series_overall_design "Four organs (liver, spleen, thymus, brain) of the Dll1 mutant mouse line analysed by cDNA microarray technology. Experiments include four biological replicates for reference (wildtype) and mutant animals. Two technical replicates for each mutant mouse were performed. As reference pooled RNA of the same organ was used. 50% of the chip hybridisations are dye sway experiments."

GSE12957_series_matrix.txtimp_info.txt

90kDa r is 4

Found -/- in !Series_overall_design "The supplementary file show data generated by real-time quantitative PCR normalized to Hypoxanthine guanine phosphoribosyl transferase 1, Heat shock protein 90kDa alpha, and Glyceraldehyde-3-phosphate dehydrogenase, displayed as fold difference in the expression of each gene between R848- and Mock-treated samples. Two independent experiments are summarized for wild-type mice. TLR7- and IFNAR-/- animals were tested once."

GSE12965-GPL6193_series_matrix.txtimp_info.txt

Nova2 r is 1

Found KO in !Series_title "Wild type vs. Nova2 KO mouse P10 cortex RNA"

GSE12965-GPL8940_series_matrix.txtimp_info.txt

Nova2 r is 1

Found KO in !Series_title "Wild type vs. Nova2 KO mouse P10 cortex RNA"

GSE12982_series_matrix.txtimp_info.txt

Ezh2 r is 3

Found knockout in !Series_overall_design "To assay the global effects of the loss of polycomb proteins (Ezh2 or Eed) in embryonic stem (ES) cells , we compared the expression profiles of homologuous Ezh2 or Eed knockout ES cells to wild-type ES cells in undifferentiated or differentiated condition."

GSE13010_series_matrix.txtimp_info.txt

normal AK r is 3

Found treated in !Series_summary "Cryptorchidism and scrotal heating result in abnormal spermatogenesis but the mechanism(s) proscribing this temperature sensitivity are unknown. It was previously reported that the AKR/N or MRL/MpJ-+/+ mouse testis is more heat resistant than the testis from the C57BL/6 strain. We have attempted to probe into the mechanism(s) involved in heat sensitivity by examining global gene expression profiles of normal and heat-treated testes from C57BL/6, AKR/N and MRL/MpJ-+/+ mice by microarray analysis. In the normal C57BL/6 testis, 415 and 416 transcripts were differentially expressed (at least two-fold higher or lower) when compared to the normal AKR/N and MRL/MpJ-+/+ testis, respectively. The AKR/N and MRL/MpJ-+/+ strains revealed 268 differentially expressed transcripts between them. There were 231 transcripts differentially expressed between C57BL/6 and two purported heat-resistant strains, AKR/N and MRL/MpJ-+/+. Next, the testes of C57BL/6 and AKR/N mice were exposed to 43ºC for 15 min and harvested at different time points for TUNEL studies and microarrays. An increase of TUNEL-positive germ cell numbers was significant 8 hr after heat exposure in the C57BL/6 mouse. However, this increase was not observed in the AKR/N mouse until 10 hr after heat exposure. All tubules showed germ cell loss and disruption in C57BL/6 testis 24 hr after heat shock. In contrast, although a number of seminiferous tubules showed an abnormal morphology 24 hr post-heat shock in the AKR/N mouse, many tubules still retained a normal structure. Numerous transcripts exhibited differential regulation between the two strains within 24 hours after heat exposure. The differentially expressed transcripts in the testes 8 hr after heat exposure were targeted to identify the genes involved in the initial response rather than those due to germ cell loss. Twenty transcripts were significantly down-regulated and 19 genes were up-regulated by hyperthermia in C57BL/6 and did not show a parallel change in the AKR/N testis. Conversely, heat shock resulted in 30 up-regulated transcripts and 31 down-regulated transcripts in AKR/N that were not similarly regulated in C57BL/6. A number of genes shared similar differential expression patterns and differential regulation by hyperthermia in both strains of mice. Taken together, the present study indicates the diverse genetic backgrounds in the three strains lead to major differences in normal testis gene expression profiles while the differences in heat shock responses involves a significantly smaller number of genes. The data generated may provide insights regarding gene networks and pathways involved in heat stress and their relationship to spermatogenesis. "

GSE13062_series_matrix.txtimp_info.txt

Cry1 r is 4

Found KO in !Series_title "The effects of temporally restricted feeding on hepatic gene expression of Cry1, Cry2 double KO mice"

GSE13062_series_matrix.txtimp_info.txt

Cry2 r is 2

Found KO in !Series_title "The effects of temporally restricted feeding on hepatic gene expression of Cry1, Cry2 double KO mice"

GSE13062_series_matrix.txtimp_info.txt

Cry1 r is 4

Found KO in !Series_summary "Restricted feeding impacts the hepatic circadian clock of WT mice. Cry1, Cry2 double KO mice lack a circadian clock and are thus expected to show rhythmical gene expression in the liver. Imposing a temporally restricted feeding schedule on these mice shows how the hepatic circadian clock and rhythmic food intake regulate rhythmic transcription in parallel "

GSE13062_series_matrix.txtimp_info.txt

Cry2 r is 2

Found KO in !Series_summary "Restricted feeding impacts the hepatic circadian clock of WT mice. Cry1, Cry2 double KO mice lack a circadian clock and are thus expected to show rhythmical gene expression in the liver. Imposing a temporally restricted feeding schedule on these mice shows how the hepatic circadian clock and rhythmic food intake regulate rhythmic transcription in parallel "

GSE13062_series_matrix.txtimp_info.txt

Cry2 r is 2

Found KO in !Series_overall_design "Cry1, Cry2 double KO mice were entrained either to ad libitum or temporally restricted feeding (tRF) schedules. Food was made available to mice under the tRF regimen only between ZT(CT)1 and ZT(CT)9. Mice were then released into constant darkness while the respective feeding schedules were still maintained. Liver tissue was collected on the second day of constant darkness at the indicated timepoints. Total RNA was extracted and 5ug of RNA was used in the standard Affymetrix protocol for amplification, labeling and hybridization"

GSE13062_series_matrix.txtimp_info.txt

Cry1 r is 3

Found double KO in !Series_title "The effects of temporally restricted feeding on hepatic gene expression of Cry1, Cry2 double KO mice"

GSE13062_series_matrix.txtimp_info.txt

Cry2 r is 1

Found double KO in !Series_title "The effects of temporally restricted feeding on hepatic gene expression of Cry1, Cry2 double KO mice"

GSE13062_series_matrix.txtimp_info.txt

Cry1 r is 3

Found double KO in !Series_summary "Restricted feeding impacts the hepatic circadian clock of WT mice. Cry1, Cry2 double KO mice lack a circadian clock and are thus expected to show rhythmical gene expression in the liver. Imposing a temporally restricted feeding schedule on these mice shows how the hepatic circadian clock and rhythmic food intake regulate rhythmic transcription in parallel "

GSE13062_series_matrix.txtimp_info.txt

Cry2 r is 1

Found double KO in !Series_summary "Restricted feeding impacts the hepatic circadian clock of WT mice. Cry1, Cry2 double KO mice lack a circadian clock and are thus expected to show rhythmical gene expression in the liver. Imposing a temporally restricted feeding schedule on these mice shows how the hepatic circadian clock and rhythmic food intake regulate rhythmic transcription in parallel "

GSE13062_series_matrix.txtimp_info.txt

Cry2 r is 1

Found double KO in !Series_overall_design "Cry1, Cry2 double KO mice were entrained either to ad libitum or temporally restricted feeding (tRF) schedules. Food was made available to mice under the tRF regimen only between ZT(CT)1 and ZT(CT)9. Mice were then released into constant darkness while the respective feeding schedules were still maintained. Liver tissue was collected on the second day of constant darkness at the indicated timepoints. Total RNA was extracted and 5ug of RNA was used in the standard Affymetrix protocol for amplification, labeling and hybridization"

GSE13129_series_matrix.txtimp_info.txt

F10 r is 6

Found expressing in !Series_summary "We wished to examine the genes regulated by FoxD3 in pigment cells to gain understanding in how FoxD3 represses melanoblast specification in the neural crest. For technical reasons, we could not use neural crest cells, so we used melanoma cells, since they are derived from neural crest cells. To this end, we transfected B16-F10 mouse melanoma cells with constructs expressing FoxD3, or FoxD3-VP16, in which the C-terminal portion of FoxD3 (which contains the transcriptional repression domain) has been replaced by the VP16 transcriptional activation domain."

GSE13130_series_matrix.txtimp_info.txt

aryl hydrocarbon receptor r is 3

Found deficient in !Series_title "Langerhans cells from aryl hydrocarbon receptor deficient mice"

GSE13140_series_matrix.txtimp_info.txt

DAP12 r is 2

Found KO in !Series_title "Basal and IL-4 response in DAP12 (TYROBP) KO mice"

GSE13140_series_matrix.txtimp_info.txt

DAP12 r is 1

Found knockdown in !Series_overall_design "The current experiment was designed to evaluate the effect of DAP12 knockdown on macrophage gene expression, both in basal conditions and in the response to IL-4."

GSE13140_series_matrix.txtimp_info.txt

DAP12 r is 4

Found activation in !Series_summary "DAP12 is a transmembrane protein, expressed as a disulfide-bonded homodimer and bears an immunoreceptor tyrosine-based activation motif (ITAM). DAP12 is broadly expressed in hematopoietic cells and associates with a variety of cell surface receptors in lymphoid and myeloid cells. Macrophages express several DAP12-associated receptors including triggering receptors expressed by myeloid cells (TREM)-1,2 and 3, myeloid DAP12-associating lectin (MDL)-1, CD200R like proteins CD200R3/R4 and CD300C/D/E ."

GSE13147_series_matrix.txtimp_info.txt

Myd88 r is 4

Found -/- in !Series_summary "Microarray analysis of Myd88-/-Trif-/- and Myd88-/-Rip2-/- macrophage responses to WT or dotA mutant L. pneumophila."

GSE13147_series_matrix.txtimp_info.txt

Myd88 r is 4

Found -/- in !Series_overall_design "Bone marrow-derived macrophages from Myd88-/-Trif-/- and Myd88-/-Rip2-/- mice were infected with WT L. pneumophila (Lp02) or dotA mutant L. pneumophila (Lp03) for 4 hours. The RNA was extracted, processed, and hybridized onto Affymetrix 430 2.0 microarrays"

GSE13163_series_matrix.txtimp_info.txt

Deaf1 r is 1

Found knock-out in !Series_summary "A microarray study performed in the pancreatic lymph nodes of Deaf1 knock-out and BALB/c control mice to identify genes that are regulated by the transcriptional regulator Deaf1. These experiments constitute a portion of the study described below:"

GSE13190_series_matrix.txtimp_info.txt

Klf4 r is 7

Found induced in !Series_summary "In the murine system, Oct4, Sox2, c-Myc and Klf4 are sufficient to convert fibroblasts to induced pluripotent stem (iPS) cells that exhibit many characteristics of embryonic stem (ES) cells. Herein, we show that the orphan nuclear receptor Esrrb works in conjunction with Oct4 and Sox2 to mediate reprogramming of mouse embryonic fibroblasts (MEFs) to iPS cells. Esrrb reprogrammed cells share similar expression and epigenetic signatures as ES cells. These cells are also pluripotent and can differentiate in vitro and in vivo into the three major embryonic cell lineages. Furthermore, these cells contribute to mouse chimeras and are germline transmissible. In ES cells, Esrrb targets many genes involved in selfrenewal and pluripotency. This suggests that Esrrb may mediate reprogramming through the up-regulation of ES cell-specific genes. Our findings also indicate that it is possible to reprogram MEFs without exogenous Klf transcription factors and link a nuclear receptor to somatic cell reprogramming."

GSE13207_series_matrix.txtimp_info.txt

Sirt6 r is 1

Found knockout in !Series_summary "We completed genome-wide microarray analysis in Sirt6 knockout (ko) mouse embryonic fibroblasts (MEFs) and control wildtype (wt) MEFs. Prior to RNA extraction, MEFs were treated with TNF-alpha (10 ng/ml) for the indicated times. Total RNA was extracted with TRIzol (Invitrogen) and amplified with Ambion Amino Allyl MessageAmp II Amplification kit. Amplified mouse Universal Reference RNA (Stratagene) was used as reference RNA for analysis. Array hybridization of mouse MEEBO arrays is as described (Hendrickson et al., PLoS ONE, 2008)."

GSE13207_series_matrix.txtimp_info.txt

Sirt6 r is 2

Found -/- in !Series_title "Mouse Sirt6-/- TNF-alpha timecourse"

GSE13207_series_matrix.txtimp_info.txt

TNF-a r is 1

Found -/- in !Series_title "Mouse Sirt6-/- TNF-alpha timecourse"

GSE13207_series_matrix.txtimp_info.txt

TNF-alpha r is 1

Found -/- in !Series_title "Mouse Sirt6-/- TNF-alpha timecourse"

GSE13207_series_matrix.txtimp_info.txt

TNF-a r is 2

Found treated in !Series_summary "We completed genome-wide microarray analysis in Sirt6 knockout (ko) mouse embryonic fibroblasts (MEFs) and control wildtype (wt) MEFs. Prior to RNA extraction, MEFs were treated with TNF-alpha (10 ng/ml) for the indicated times. Total RNA was extracted with TRIzol (Invitrogen) and amplified with Ambion Amino Allyl MessageAmp II Amplification kit. Amplified mouse Universal Reference RNA (Stratagene) was used as reference RNA for analysis. Array hybridization of mouse MEEBO arrays is as described (Hendrickson et al., PLoS ONE, 2008)."

GSE13207_series_matrix.txtimp_info.txt

TNF-alpha r is 2

Found treated in !Series_summary "We completed genome-wide microarray analysis in Sirt6 knockout (ko) mouse embryonic fibroblasts (MEFs) and control wildtype (wt) MEFs. Prior to RNA extraction, MEFs were treated with TNF-alpha (10 ng/ml) for the indicated times. Total RNA was extracted with TRIzol (Invitrogen) and amplified with Ambion Amino Allyl MessageAmp II Amplification kit. Amplified mouse Universal Reference RNA (Stratagene) was used as reference RNA for analysis. Array hybridization of mouse MEEBO arrays is as described (Hendrickson et al., PLoS ONE, 2008)."

GSE13208_series_matrix.txtimp_info.txt

Sirt6 r is 2

Found -/- in !Series_title "Mouse Sirt6-/- tissues"

GSE13209_series_matrix.txtimp_info.txt

Sirt6 r is 2

Found -/- in !Series_title "Mouse Sirt6-/- RelA+/- tissues"

GSE13209_series_matrix.txtimp_info.txt

Sirt6 r is 2

Found -/- in !Series_summary "We harvested spleen tissue from Sirt6-/- or Sirt6-/- RelA+/- animals and then completed genome-wide microarray analysis. Total RNA was extracted with TRIzol (Invitrogen) and amplified with Amino Allyl MessageAmp II Amplification kit. Amplified age-matched wild-type tissues were used as reference RNA for analysis. Array hybridization of mouse MEEBO arrays is as described (Hendrickson et al., PLoS ONE, 2008)."

GSE13211_series_matrix.txtimp_info.txt

Klf4 r is 7

Found induced in !Series_summary "In the murine system, Oct4, Sox2, c-Myc and Klf4 are sufficient to convert fibroblasts to induced pluripotent stem (iPS) cells that exhibit many characteristics of embryonic stem (ES) cells. Herein, we show that the orphan nuclear receptor Esrrb works in conjunction with Oct4 and Sox2 to mediate reprogramming of mouse embryonic fibroblasts (MEFs) to iPS cells. Esrrb reprogrammed cells share similar expression and epigenetic signatures as ES cells. These cells are also pluripotent and can differentiate in vitro and in vivo into the three major embryonic cell lineages. Furthermore, these cells contribute to mouse chimeras and are germline transmissible. In ES cells, Esrrb targets many genes involved in selfrenewal and pluripotency. This suggests that Esrrb may mediate reprogramming through the up-regulation of ES cell-specific genes. Our findings also indicate that it is possible to reprogram MEFs without exogenous Klf transcription factors and link a nuclear receptor to somatic cell reprogramming."

GSE13212_series_matrix.txtimp_info.txt

Klf4 r is 7

Found induced in !Series_summary "In the murine system, Oct4, Sox2, c-Myc and Klf4 are sufficient to convert fibroblasts to induced pluripotent stem (iPS) cells that exhibit many characteristics of embryonic stem (ES) cells. Herein, we show that the orphan nuclear receptor Esrrb works in conjunction with Oct4 and Sox2 to mediate reprogramming of mouse embryonic fibroblasts (MEFs) to iPS cells. Esrrb reprogrammed cells share similar expression and epigenetic signatures as ES cells. These cells are also pluripotent and can differentiate in vitro and in vivo into the three major embryonic cell lineages. Furthermore, these cells contribute to mouse chimeras and are germline transmissible. In ES cells, Esrrb targets many genes involved in selfrenewal and pluripotency. This suggests that Esrrb may mediate reprogramming through the up-regulation of ES cell-specific genes. Our findings also indicate that it is possible to reprogram MEFs without exogenous Klf transcription factors and link a nuclear receptor to somatic cell reprogramming."

GSE13298_series_matrix.txtimp_info.txt

Rb1 r is 1

Found deficient in !Series_overall_design "We have compared 3 cecal tumors with 3 duodenal tumors from Rb1 deficient Apc1638N mice."

GSE13306_series_matrix.txtimp_info.txt

beta s r is 6

Found activation in !Series_summary "CD4(+)Foxp3(+) regulatory T (Treg) cells originate primarily from thymic differentiation, but conversion of mature T lymphocytes to Foxp3 positivity can be elicited by several means, including in vitro activation in the presence of TGF-beta. Retinoic acid (RA) increases TGF-beta-induced expression of Foxp3, through unknown molecular mechanisms. We showed here that,

rather than enhancing TGF-beta signaling directly in naive CD4(+) T cells, RA negatively regulated an accompanying population of CD4(+) T cells with a CD44(hi) memory and effector phenotype. These memory cells actively inhibited the TGF-beta-induced conversion of naive CD4(+) T cells through the synthesis of a set of cytokines (IL-4, IL-21, IFN-gamma) whose expression was coordinately curtailed by RA. This indirect effect was evident in vivo and required the expression of the RA receptor alpha. Thus, cytokine-producing CD44(hi) cells actively restrain TGF-beta-mediated Foxp3 expression in naive T cells, and this balance can be shifted or fine-tuned by RA."

GSE13306_series_matrix.txtimp_info.txt

beta s r is 8

Found induced in !Series_summary "CD4(+)Foxp3(+) regulatory T (Treg) cells originate primarily from thymic differentiation, but conversion of mature T lymphocytes to Foxp3 positivity can be elicited by several means, including in vitro activation in the presence of TGF-beta. Retinoic acid (RA) increases TGF-beta-induced expression of Foxp3, through unknown molecular mechanisms. We showed here that, rather than enhancing TGF-beta signaling directly in naive CD4(+) T cells, RA negatively regulated an accompanying population of CD4(+) T cells with a CD44(hi) memory and effector phenotype. These memory cells actively inhibited the TGF-beta-induced conversion of naive CD4(+) T cells through the synthesis of a set of cytokines (IL-4, IL-21, IFN-gamma) whose expression was coordinately curtailed by RA. This indirect effect was evident in vivo and required the expression of the RA receptor alpha. Thus, cytokine-producing CD44(hi) cells actively restrain TGF-beta-mediated Foxp3 expression in naive T cells, and this balance can be shifted or fine-tuned by RA."

GSE13312_series_matrix.txtimp_info.txt

p53 r is 5

Found null in !Series_overall_design "Gene expression patterns were compared between p53 wt MEF and p53-null MEF cells."

GSE13364_series_matrix.txtimp_info.txt

BWF1 r is 8

Found treated in !Series_summary "Microarray analysis was performed on BWF1 mice spleenocyte cells in control and pCONS treated mice."

GSE13382_series_matrix.txtimp_info.txt

Foxa2 r is 6

Found -/- in !Series_summary "We analyzed the effects of these changes in FOXA transcription factor expression using Foxa2 transgenic mice and Foxa3-/- mice. We found that persistent expression of FOXA2 reduced mucus but the absence of FOXA3 had no effect on mucus production induced by allergen challenge."

GSE13382_series_matrix.txtimp_info.txt

Foxa3 r is 2

Found -/- in !Series_summary "We analyzed the effects of these changes in FOXA transcription factor expression using Foxa2 transgenic mice and Foxa3-/- mice. We found that persistent expression of FOXA2 reduced mucus but the absence of FOXA3 had no effect on mucus production induced by allergen challenge."

GSE13382_series_matrix.txtimp_info.txt

Foxa3 r is 2

Found -/- in !Series_summary "Keywords: gene expression comparison between Foxa3-/- and littermate control mice both challenged with OVA"

GSE13382_series_matrix.txtimp_info.txt

Foxa3 r is 1

Found KO in !Series_overall_design "DNA miocroarrays were used to analyze lung mRNA expression of Foxa3 KO and littermate control mice challenged with saline or OVA. The experiment incorporated a 1 color design and used Agilent arrays that contained roughly 44,000 60mer probes that provide complete coverage of the mouse genome. 11 arrays were hybridized and represent 3 lung samples for groups WT saline, WT OVA and KO OVA. There are 2 lung samples for the KO saline group."

GSE13432_series_matrix.txtimp_info.txt

VEGFR2 r is 5

Found activation in !Series_summary "The molecular mechanisms of angiogenesis in relation to adipose tissue metabolism remain poorly understood. Here we show that exposure of mice to cold led to conversion of white adipose tissue (WAT) to brown-like adipose tissue, accompanying the switch of an active angiogenic phenotype. Gene expression profile analysis showed VEGF was upregulated via most likely hypoxia-independent PGC-1 transcriptional activation. Intriguingly, VEGFR2 blockage abolished the cold-induced angiogenesis, significantly impaired nonshivering thermogenesis capacity, and markedly reduced adipose metabolism. Unexpectedly, VEGFR1 blockage resulted in opposite effects by increasing adipose vascularity and metabolism. These findings demonstrate that VEGFR2 and VEGFR1 mediate polarized activities in modulating adipose angiogenesis and metabolism. Taken together, our findings have conceptual implications in applying angiogenesis modulators for the treatment of obesity and metabolic disorders."

GSE13432_series_matrix.txtimp_info.txt

PGC-1 r is 3

Found activation in !Series_summary "The molecular mechanisms of angiogenesis in relation to adipose tissue metabolism remain poorly understood. Here we show that exposure of mice to cold led to conversion of white adipose tissue (WAT) to brown-like adipose tissue, accompanying the switch of an active angiogenic phenotype. Gene expression profile analysis showed VEGF was upregulated via most likely hypoxia-independent PGC-1 transcriptional activation. Intriguingly, VEGFR2 blockage abolished the cold-induced angiogenesis, significantly impaired nonshivering thermogenesis capacity, and markedly reduced adipose metabolism. Unexpectedly, VEGFR1 blockage resulted in opposite effects by increasing adipose vascularity and metabolism. These findings demonstrate that VEGFR2 and VEGFR1 mediate polarized activities in modulating adipose angiogenesis and metabolism. Taken together, our findings have conceptual implications in applying angiogenesis modulators for the treatment of obesity and metabolic disorders."

GSE13432_series_matrix.txtimp_info.txt

VEGFR2 r is 5

Found induced in !Series_summary "The molecular mechanisms of angiogenesis in relation to adipose tissue metabolism remain poorly understood. Here we show that exposure of mice to cold led to conversion of white adipose tissue (WAT) to brown-like adipose tissue, accompanying the switch of an active angiogenic phenotype. Gene expression profile analysis showed VEGF was upregulated via most likely hypoxia-independent PGC-1 transcriptional activation. Intriguingly, VEGFR2 blockage abolished the cold-induced angiogenesis, significantly impaired nonshivering thermogenesis capacity, and markedly reduced adipose metabolism. Unexpectedly, VEGFR1 blockage resulted in opposite effects by increasing adipose vascularity and metabolism. These findings demonstrate that VEGFR2 and VEGFR1 mediate polarized activities in modulating adipose angiogenesis and metabolism. Taken together, our findings have conceptual implications in applying angiogenesis modulators for the treatment of obesity and metabolic disorders."

GSE13448_series_matrix.txtimp_info.txt

p53 r is 1

Found -/- in !Series_overall_design "RNAs isolated from wild type, p53 -/- and p53 -/- Mef -/- LSK cells were used in oligonucleotide arrays (Affymetrix) "

GSE13448_series_matrix.txtimp_info.txt

Gfi-1 r is 3

Found null in !Series_summary "The importance of the p53 protein in the cellular response to DNA damage is well known, but its function during steady-state hematopoiesis has not been established. We have defined a critical role of p53 in regulating hematopoietic stem cell quiescence, especially in promoting the enhanced quiescence seen in HSCs that lack the MEF/ELF4 transcription factor. Transcription profiling of HSCs isolated from wild type and p53 null mice identified Gfi-1 and Necdin as p53 target genes and using lentiviral vectors to upregulate or knockdown the expression of these genes,

we show their importance in regulating HSC quiescence. Establishing the role of p53 (and its target genes) in controlling the cell cycle entry of HSCs may lead to therapeutic strategies capable of eliminating quiescent cancer (stem) cells."

GSE13467-GPL4128_series_matrix.txtimp_info.txt

Ezh1 r is 3

Found overexpressing in !Series_overall_design "Undifferentiated F9 cells were used for this experiment. Antibodies specificity were checked using a stable cell lines overexpressing a GAl4-Ezh1 or a Gal4-Ezh2 in an inducible manner followed by conventional ChIP at Gal4 responsive transgene. "

GSE13467-GPL4128_series_matrix.txtimp_info.txt

Ezh2 r is 7

Found overexpressing in !Series_overall_design "Undifferentiated F9 cells were used for this experiment. Antibodies specificity were checked using a stable cell lines overexpressing a GAl4-Ezh1 or a Gal4-Ezh2 in an inducible manner followed by conventional ChIP at Gal4 responsive transgene. "

GSE13467-GPL4129_series_matrix.txtimp_info.txt

Ezh1 r is 3

Found overexpressing in !Series_overall_design "Undifferentiated F9 cells were used for this experiment. Antibodies specificity were checked using a stable cell lines overexpressing a GAl4-Ezh1 or a Gal4-Ezh2 in an inducible manner followed by conventional ChIP at Gal4 responsive transgene. "

GSE13467-GPL4129_series_matrix.txtimp_info.txt

Ezh2 r is 7

Found overexpressing in !Series_overall_design "Undifferentiated F9 cells were used for this experiment. Antibodies specificity were checked using a stable cell lines overexpressing a GAl4-Ezh1 or a Gal4-Ezh2 in an inducible manner followed by conventional ChIP at Gal4 responsive transgene. "

GSE13493_series_matrix.txtimp_info.txt

Rag2 r is 1

Found deficient in !Series_title "Expression data from developing thymocytes of N15TCR transgenic Rag2 deficient mice"

GSE13493_series_matrix.txtimp_info.txt

Rag2 r is 1

Found deficient in !Series_summary "We used microarrays to identify the genes differentially expressed during CD8 single positive T cell development in N15 TCR transgenic Rag2 deficient mice."

GSE13522_series_matrix.txtimp_info.txt

IFN-g r is 2

Found deficient in !Series_summary "We used microarrays to detail the local host transcriptional response to intradermal T. cruzi infection in WT mice and mice depleted of NK cells, or deficient in IFN-gamma or type I IFN responses. Additionally we compared the local host-transcriptional response generated to infection with 3 different strains of Trypanosoma cruzi (Y, Brazil, and G)."

GSE13522_series_matrix.txtimp_info.txt

IFN-gamma r is 2

Found deficient in !Series_summary "We used microarrays to detail the local host transcriptional response to intradermal T. cruzi infection in WT mice and mice depleted of NK cells, or deficient in IFN-gamma or type I IFN responses. Additionally we compared the local host-transcriptional response generated to infection with 3 different strains of Trypanosoma cruzi (Y, Brazil, and G)."

GSE13522_series_matrix.txtimp_info.txt

IFN-g r is 2

Found KO in !Series_overall_design "Mice were infected by intradermal injection of 10^6 T. cruzi trypomastigotes in 100uL of saline split between 2 adjacent sites on the shaved side flank. Control mice were injected with an equal volume of saline. 24 hours post-injection approximately 75mm^2 of skin immediately surrounding the injection site was excised and RNA was isolated from the tissue. Balb/c mice were used for most experiments and IFN-gamma KO mice were on the Balb/c background. WT 129 mice were also used as IFNAR-/- mice were on the 129 background. In total 33 arrays were performed. 7 WT (Balb/c) control, 3 Y strain infected, 3 Brazil strain infected, 3 G strain infected, 2 IFN-gamma KO control, 2 IFN-gamma KO infected, 1 NK cell depleted control, 1 NK cell depleted infected, 3 WT (129) control, 3 WT (129) infected, 3 IFNAR KO control, 3 IFNAR KO infected"

GSE13522_series_matrix.txtimp_info.txt

IFN-gamma r is 2

Found KO in !Series_overall_design "Mice were infected by intradermal injection of 10^6 T. cruzi trypomastigotes in 100uL of saline split between 2 adjacent sites on the shaved side flank. Control mice were injected with an equal volume of saline. 24 hours post-injection approximately 75mm^2 of skin immediately surrounding the injection site was excised and RNA was isolated from the tissue. Balb/c mice were used for most experiments and IFN-gamma KO mice were on the Balb/c background. WT 129 mice were also used as IFNAR-/- mice were on the 129 background. In total 33 arrays were performed. 7 WT (Balb/c) control, 3 Y strain infected, 3 Brazil strain infected, 3 G strain infected, 2 IFN-gamma KO

control, 2 IFN-gamma KO infected, 1 NK cell depleted control, 1 NK cell depleted infected, 3 WT (129) control, 3 WT (129) infected, 3 IFNAR KO control, 3 IFNAR KO infected"

GSE13526_series_matrix.txtimp_info.txt

Nxf2 r is 1

Found KO in !Series_title "Transcript profiling of WT and Nxf2 KO post-natal day 21 testes"

GSE13526_series_matrix.txtimp_info.txt

Nxf2 r is 3

Found KO in !Series_summary "We used microarrays to check the expression profiles of the Nxf2 WT and KO 21d testes on C57BL/6 background."

GSE13526_series_matrix.txtimp_info.txt

Nxf2 r is 1

Found KO in !Series_overall_design "To examine the expression difference between WT and Nxf2 KO testes, we collected testes from juvenile mice of three ages (21d, 26d, 28d). Testis weight was similar between WT and KO mice at post-natal day 21. Three pairs of WT and KO 21d testes were chosen for microarray analysis."

GSE13526_series_matrix.txtimp_info.txt

Nxf2 r is 1

Found mutant in !Series_summary "In euakryotes, mRNAs must be exported from the nucleus to the cytsoplasm. NXF2 is highly expressed in the mouse male germ cells. We are interested in its function in spermatogenesis, espically in the nuclear RNA export in the testis. To this end, we made Nxf2 mutant mice by gene targeting. In an attempt to identify the mRNA substrates of NXF2, we perform the microarray experiments on testes."

GSE13549_series_matrix.txtimp_info.txt

PDGF-C r is 7

Found induced in !Series_summary "Tumor associated fibroblasts (TAF) from different tumors exhibit distinct angiogenic and tumorigenic properties. Unlike normal skin fibroblasts (NSF) or TAF (TAF-TIB6) from TIB6 tumors that are sensitive to anti-VEGF treatment, TAF (TAF-EL4) from resistant EL4 tumors can stimulate TIB6 tumor growth even when VEGF is inhibited. We show that platelet-derived growth factor (PDGF)-C is upregulated in TAFs from resistant tumors. PDGF-C neutralizing antibodies blocked the angiogenesis induced by such TAF in vivo and slowed the growth of EL4 and admixture (TAF-EL4 + TIB6) tumors and exhibited additive effects with anti-VEGF-A antibodies. Hence, our data reveal a

novel mechanism for TAF mediated tumorigenesis and suggest that some tumors may overcome inhibition of VEGF-mediated angiogenesis through upregulation of PDGF-C"

GSE13579_series_matrix.txtimp_info.txt

Bmal1 r is 1

Found mutant in !Series_summary "Hair follicles undergo recurrent cycling of controlled growth (anagen), regression (catagen), and relative quiescence (telogen) with a defined periodicity. Taking a genomics approach to study gene expression during synchronized mouse hair follicle cycling, we discovered that, in addition to circadian fluctuation, CLOCK-regulated genes are also modulated in phase with the hair growth cycle. During telogen and early anagen, circadian clock genes are prominently expressed in the secondary hair germ, which contains precursor cells for the growing follicle. Analysis of Clock and Bmal1 mutant mice reveals a delay in anagen progression, and the secondary hair germ cells show decreased levels of phosphorylated Rb and lack mitotic cells, suggesting that circadian clock genes regulate anagen progression via their effect on the cell cycle. Consistent with a block at the G1 phase of the cell cycle, we show a significant upregulation of p21 in Bmal1 mutant skin. While circadian clock mechanisms have been implicated in a variety of diurnal biological processes, our findings indicate that circadian clock genes may be utilized to modulate the progression of non-diurnal cyclic processes."

GSE13588_series_matrix.txtimp_info.txt

Foxp2 r is 4

Found ko in !Series_title "Gene expression in striatums of Foxp2-hum, Foxp2-ko and wild-type mice"

GSE13592_series_matrix.txtimp_info.txt

apolipoprotein E r is 2

Found deficient in !Series_summary "Dendritic cells (DCs) are essential for priming of immune responses. Although immune mechanisms are known to control the pathogenesis of atherosclerosis, the role of DCs remains elusive. Here we show that Ccl17 expressing mature, myeloid DCs accumulate within atherosclerotic lesions. Deletion of Ccl17 in apolipoprotein E-deficient (Apoe-/-) mice reduces the development and progression of atherosclerosis in several disease models. While Ccl17 expression by DCs dampened antigen-specific T cell proliferation, it is required for efficient polarization of T helper type 1 (Th1) and Th17 as reflected by a preponderance of Th2 cytokines in Ccl17-/- Apoe-/- mice. In line with these findings, only transfer of T cells from Apoe-/-, but not from Ccl17-/- Apoe-/- precipitated atherosclerosis in T cell depleted Apoe-/- recipients. These findings identify Ccl17+ DCs as central immune regulators in atherosclerosis and Ccl17 as a potential target in the treatment of this disease."

GSE13592_series_matrix.txtimp_info.txt

Ccl17 r is 1

Found expressing in !Series_summary "Dendritic cells (DCs) are essential for priming of immune responses. Although immune mechanisms are known to control the pathogenesis of atherosclerosis, the role of DCs remains elusive. Here we show that Ccl17 expressing mature, myeloid DCs accumulate within atherosclerotic lesions. Deletion of Ccl17 in apolipoprotein E-deficient (Apoe-/-) mice reduces the development and progression of atherosclerosis in several disease models. While Ccl17 expression by DCs dampened antigen-specific T cell proliferation, it is required for efficient polarization of T helper type 1 (Th1) and Th17 as reflected by a preponderance of Th2 cytokines in Ccl17-/- Apoe-/- mice. In line with these findings, only transfer of T cells from Apoe-/-, but not from Ccl17-/- Apoe-/- precipitated atherosclerosis in T cell depleted Apoe-/- recipients. These findings identify Ccl17+ DCs as central immune regulators in atherosclerosis and Ccl17 as a potential target in the treatment of this disease."

GSE13599_series_matrix.txtimp_info.txt

Mdr2 r is 1

Found knockout in !Series_summary "This study aims on the identification of the NF-kB dependent gene regulatory network during inflammation-associated liver carcinogenesis using the well-established Mdr2 knockout mouse model. We could identify 367 differentially expressed genes comparing expression profiles of tumor samples from Mdr2-KO mice to tumor samples derived from mice with an additional hepatocyte specific expression of an IkB-superrepressor. This IkB-superrepresser is undegradable upon ubiquitinylation initialized by Ikk dependent phosphorylation and therefore impedes NFkB activity."

GSE13599_series_matrix.txtimp_info.txt

Mdr2 r is 1

Found knockout in !Series_overall_design "In the experiments shown here, we compare gene expression profiles of HCCs from 4 idividual mice harboring a biallelic Mdr2 knockout to HCC specimen of 6 individual mice with Mdr2 knockout and a hepatocyte specific expression of the IkB-superrepressor. Each individual sample was hybridized aginst a universal reference mouse pool (Stratagene) on a two-color microarray. Furthermore was each sample/reference hybridization conducted as a dye swap experiment yielding two technical replicates with inverted dye channel orientation per sample. In addition a biological control using wildtype liver tissue from mice of the same genetic background was hybridized the same way aginst a universal reference. The latter was used to build a sample/control ratio for displays of indirect comparison."

GSE13599_series_matrix.txtimp_info.txt

Mdr2 r is 1

Found KO in !Series_title "NFkB dependent gene expression in a Mdr2-KO hepatocellular carcinoma (HCC) mouse model using a IkB-Superrepressor"

GSE13599_series_matrix.txtimp_info.txt

Mdr2 r is 1

Found KO in !Series_summary "Keywords: NFkB, IkB-superrepressor, inflammation-associated liver carcinogenesis, Mdr2-KO mouse model, hepatocellular carcinoma, gene expression, microarray"

GSE13626_series_matrix.txtimp_info.txt

VAP-1 r is 2

Found -/- in !Series_title "Comparison of gene expression in melanoma of wild-type and VAP-1 -/- mice"

GSE13626_series_matrix.txtimp_info.txt

semicarbazide-sensitive amine oxidase r is 7

Found -/- in !Series_summary "Vascular adhesion protein-1 (VAP-1) is an endothelial cell-surface protein. It is also an enzyme posessing semicarbazide-sensitive amine oxidase activity (EC.1.4.3.6). VAP-1 is involved in leukocyte traffic. To study the role of VAP-1 in tumor immunity, we compared gene expression profiles in melanomas growing in VAP-1 -/- mice and their wid-type littermates."

GSE13626_series_matrix.txtimp_info.txt

VAP-1 r is 2

Found -/- in !Series_overall_design "The B16-F10-Luc-G5 melanoma cells (Xenogen) were injected subcutaneously into abdominal area of 2 wild-type and 2 VAP-1 -/- mice. Tumors were grown until day 10 when mice were euthanized. Tumors were dissected and total RNA was isolated using the Qiagen RNA extraction kit."

GSE13635_series_matrix.txtimp_info.txt

cyclin D1 r is 2

Found -/- in !Series_title "Gene expression change in cyclin D1 -/- retinas in comparison to wildtype."

GSE13635_series_matrix.txtimp_info.txt

D1 r is 1

Found -/- in !Series_title "Gene expression change in cyclin D1 -/- retinas in comparison to wildtype."

GSE13642_series_matrix.txtimp_info.txt

apolipoprotein E r is 2

Found deficient in !Series_summary "Dendritic cells (DCs) are essential for priming of immune responses. Although immune mechanisms are known to control the pathogenesis of atherosclerosis, the role of DCs remains elusive. Here we show that Ccl17 expressing mature, myeloid DCs accumulate within atherosclerotic lesions. Deletion of Ccl17 in apolipoprotein E-deficient (Apoe-/-) mice reduces the development and progression of atherosclerosis in several disease models. While Ccl17 expression by DCs dampened antigen-specific T cell proliferation, it is required for efficient polarization of T helper type 1 (Th1) and Th17 as reflected by a preponderance of Th2 cytokines in Ccl17-/- Apoe-/- mice. In line with these findings, only transfer of T cells from Apoe-/-, but not from Ccl17-/- Apoe-/- precipitated atherosclerosis in T cell depleted Apoe-/- recipients. These findings identify Ccl17+ DCs as central immune regulators in atherosclerosis and Ccl17 as a potential target in the treatment of this disease."

GSE13642_series_matrix.txtimp_info.txt

Ccl17 r is 1

Found expressing in !Series_summary "Dendritic cells (DCs) are essential for priming of immune responses. Although immune mechanisms are known to control the pathogenesis of atherosclerosis, the role of DCs remains elusive. Here we show that Ccl17 expressing mature, myeloid DCs accumulate within atherosclerotic lesions. Deletion of Ccl17 in apolipoprotein E-deficient (Apoe-/-) mice reduces the development and progression of atherosclerosis in several disease models. While Ccl17 expression by DCs dampened antigen-specific T cell proliferation, it is required for efficient polarization of T helper type 1 (Th1) and Th17 as reflected by a preponderance of Th2 cytokines in Ccl17-/- Apoe-/- mice. In line with these findings, only transfer of T cells from Apoe-/-, but not from Ccl17-/- Apoe-/- precipitated atherosclerosis in T cell depleted Apoe-/- recipients. These findings identify Ccl17+ DCs as central immune regulators in atherosclerosis and Ccl17 as a potential target in the treatment of this disease."

GSE13645_series_matrix.txtimp_info.txt

p56Lck r is 6

Found null in !Series_summary "Signaling through the T cell antigen receptor is essential for the formation of regulatory T (Treg) cells in the thymus and for their involvement in antigen-directed suppression of immune responses. Using a conditional null allele of the gene encoding p56Lck we show here that T cell antigen receptor (TCR) signaling is also essential for sustaining the phenotype and homeostasis of Treg cells. Inactivation of p56Lck in Treg cells resulted in large-scale changes in their gene expression profile, blocked their capacity to suppress responses, inhibited their proliferation, and caused them to redistribute in the body. The results make clear multiple aspects of the Treg cell phenotype that are dependent on a sustained capacity to respond through their TCRs."

GSE13665_series_matrix.txtimp_info.txt

Crhr1 r is 4

Found knockout in !Series_title "Pituitaries of basal and stressed Crhr1 wild type and knockout mice"

GSE13665_series_matrix.txtimp_info.txt

Crhr1 r is 5

Found knockout in !Series_summary "Pituitaries of basal and stressed Crhr1-wildtype (wt) and -knockout (ko) mice (Timpl et al., 1998)"

GSE13686_series_matrix.txtimp_info.txt

Mef2c r is 1

Found deficient in !Series_overall_design "Expression profiles of multipotent progenitor cells (MPPs) from Mef2c deficient (Mx1-Cre Mef2cf/f) mice where compared to MPPs from control (Mef2cf/f) mice in triplicate."

GSE13718_series_matrix.txtimp_info.txt

Ebp1 r is 1

Found -/- in !Series_summary "Results: Ebp1 -/- mice were on average 30% smaller than wild type and heterozygous sex matched littermates. Growth retardation was apparent from Day 10 until Day 30. IGF-1 production and IGBP-3 and 4 protein levels were reduced in both embryo fibroblasts and adult knock-out mice. The proliferation of fibroblasts derived from Day 12.5 knock out embryos was also decreased as compared to that of wild type cells. Microarray expression analysis revealed changes in genes important in cell growth including members of the MAPK signal transduction pathway. In addition, the expression or activation of proliferation related genes such as AKT and the androgen receptor, previously demonstrated to be affected by Ebp1 expression in vitro, were altered in adult tissues."

GSE13718_series_matrix.txtimp_info.txt

Ebp1 r is 8

Found -/- in !Series_summary "Conclusions: These results indicate that Ebp1 can affect growth in an animal model, but that the expression of proliferation related genes is cell and context specific. The Ebp1-/- mouse line represents a new in vivo model to investigate Ebp1 function in the whole organism."

GSE13719_series_matrix.txtimp_info.txt

aryl hydrocarbon receptor r is 3

Found activation in !Series_summary "Effect of the over activation of the aryl hydrocarbon receptor on gene expression of spleen derived dendritic cells. "

GSE13740_series_matrix.txtimp_info.txt

NF-kappaB r is 2

Found activation in !Series_summary "Pneumonia is a serious problem worldwide. We recently demonstrated that innate defense mechanisms of the lung are highly inducible against pneumococcal pneumonia. To determine the breadth of protection conferred by stimulation of lung mucosal innate immunity, and to identify cells and signaling pathways activated by this treatment, mice were treated with an aerosolized bacterial lysate, then challenged with lethal doses of bacterial and fungal pathogens. Mice were highly protected against a broad array of Gram-positive, Gram-negative, and Class A bioterror bacterial pathogens, and Aspergillus fumigatus. Protection was associated with rapid pathogen killing within the lungs, and this effect was recapitulated in vitro using a respiratory epithelial cell line. Gene expression analysis of lung tissue showed marked activation of NF-kappaB, Type I and II interferon, and antifungal Card9-Bcl10-Malt1 pathways. Cytokines were the most strongly induced genes, but the inflammatory cytokines TNF and IL-6 were not required for protection. Lung-expressed antimicrobial peptides were also highly upregulated. Taken together, stimulated innate resistance (StIR) appears to occur through the activation of multiple host defense signaling pathways in lung epithelial cells, inducing rapid pathogen killing, and conferring broad protection against virulent bacterial and fungal pathogens. Augmentation of innate antimicrobial defenses of the lungs might have therapeutic value for protection of patients with neutropenia or impaired adaptive immunity against opportunistic pneumonia, and for defense of immunocompetent subjects against a bioterror threat or epidemic respiratory infection."

GSE13772_series_matrix.txtimp_info.txt

apoA-I r is 2

Found treated in !Series_overall_design "Three replicate experiments were analyzed. Each experiment consisted of a control sample and an apoA-I treated sample of RNA extracted from peritoneal macrophages isolated from both MyD88 +/+ and MyD88-/- mice, totaling four samples per experiment; 12 samples together."

GSE13772_series_matrix.txtimp_info.txt

apoA-I r is 2

Found induced in !Series_title "The apoA-I induced transcriptome and dependence on MyD88"

GSE13835_series_matrix.txtimp_info.txt

apolipoprotein E r is 2

Found deficient in !Series_summary "This study compared gene expression in smooth muscle cells (SMCs) in atherosclerosis-prone and atherosclerosis-resistant regions of the aorta of C57Bl/6 mice. In a parallel experiment, both regions were compared in young, plaque-free apolipoprotein E-deficient (apoE-/-) mice. Aortas of 3 male and 3 female C57Bl6 mice were isolated, perfused with triton X-100 to remove endothelial cells and divided in an atherosclerosis-prone region (AA: ascending aorta, aortic arch and proximal 2 mm of thoracic aorta) and an atherosclerosis-resistant region (TA: central thoracic aorta,i.e. 6 mm distal from the proximal 2mm of the thoracic aorta). Microarray analysis (VIB-MAF) of pooled total RNA showed differential expression (>2-fold difference) for 70 genes. Up- or

downregulation in the AA was observed for 33 and 37 genes respectively. Differential expression of 3 genes (ATPase, Na+/K+ transporting, beta 1 polypeptide, sarcolipin and homeo box B7) was confirmed using real-time PCR. Twenty five genes showed exclusively differential expression in C57BL6 mice. Only 7 could be linked to specific processes: development (4) and cell growth (3). The other 18 genes were all involved in different processes. Among the 45 genes showing differential expression in C57Bl/6 as well as apoE-/- mice, most were related to development (13), cell growth (8) and transcription (10). These results point to an altered transcriptome in SMCs of the C57Bl/6 aorta at an atherosclerosis-prone location. This is in agreement with findings in endothelial cells in atherosclerosis-prone regions. It could be due to biomechanical differences, for instance in wall tension or shear stress, or the different embryological origin of SMCs in AA and TA."

GSE13836_series_matrix.txtimp_info.txt

apolipoprotein E r is 2

Found deficient in !Series_summary "This study compared gene expression in smooth muscle cells (SMCs) in atherosclerosis-prone and atherosclerosis-resistant aorta segments in 4 months old apolipoprotein E-deficient (apoE-/-) mice before plaque development. In a parallel experiment, both regions were compared in young C57Bl/6 mice. Aortas of 3 male and 3 female ApoE-/- mice were isolated, perfused with triton X-100 to remove endothelial cells and divided in an atherosclerosis-prone region (AA: ascending aorta, aortic arch and proximal 2 mm of thoracic aorta) and a resistant region (TA: central thoracic aorta, i.e. 6 mm distal from the proximal 2 mm). Microarray analysis (VIB-MAF) of pooled total RNA showed differential expression (>2-fold difference) for 244 genes. Up- or downregulation in the AA was observed for 186 and 58 genes respectively. Differential expression of 6 genes was confirmed using real-time PCR. The 201 genes that showed exclusively differential expression in apoE-/- mice were related to processes involved in atherosclerosis, such as cell adhesion, proliferation, differentiation, motility and death, lipid metabolism and immune responses. Furthermore, the transcription profile of the AA was in accordance with a more synthetic SMC phenotype. These results point to an altered transcriptome in SMCs in the aorta of apoE-/- mice at the atherosclerosis-prone location before actual lesion development. This suggests that SMCs, in addition to endothelial cells, can facilitate plaque formation at predilection sites."

GSE13855-GPL7726_series_matrix.txtimp_info.txt

p75 r is 6

Found KO in !Series_title "p55 and p75 tumor necrosis factor receptor double KO and systemic lupus erythematosis"

GSE13855-GPL7726_series_matrix.txtimp_info.txt

tumor necrosis factor r is 5

Found KO in !Series_title "p55 and p75 tumor necrosis factor receptor double KO and systemic lupus erythematosis"

GSE13855-GPL7726_series_matrix.txtimp_info.txt

p75 r is 5

Found double KO in !Series_title "p55 and p75 tumor necrosis factor receptor double KO and systemic lupus erythematosis"

GSE13855-GPL7726_series_matrix.txtimp_info.txt

tumor necrosis factor r is 4

Found double KO in !Series_title "p55 and p75 tumor necrosis factor receptor double KO and systemic lupus erythematosis"

GSE13855-GPL7747_series_matrix.txtimp_info.txt

p75 r is 6

Found KO in !Series_title "p55 and p75 tumor necrosis factor receptor double KO and systemic lupus erythematosis"

GSE13855-GPL7747_series_matrix.txtimp_info.txt

tumor necrosis factor r is 5

Found KO in !Series_title "p55 and p75 tumor necrosis factor receptor double KO and systemic lupus erythematosis"

GSE13855-GPL7747_series_matrix.txtimp_info.txt

p75 r is 5

Found double KO in !Series_title "p55 and p75 tumor necrosis factor receptor double KO and systemic lupus erythematosis"

GSE13855-GPL7747_series_matrix.txtimp_info.txt

tumor necrosis factor r is 4

Found double KO in !Series_title "p55 and p75 tumor necrosis factor receptor double KO and systemic lupus erythematosis"

GSE13859_series_matrix.txtimp_info.txt

estrogen receptor r is 3

Found knockout in !Series_title "Global survey of miRNA microarray of whole embryo, wild type vs estrogen receptor alpha knockout mice"

GSE13869_series_matrix.txtimp_info.txt

Nxnl1 r is 2

Found -/- in !Series_title "Transcriptome of the Nxnl1-/- mouse retina"

GSE13935_series_matrix.txtimp_info.txt

Bapx1 r is 2

Found deficient in !Series_title "Gene expression in pylorus stomach tissue in Bapx1 (Nkx3.2) deficient mice"

GSE13935_series_matrix.txtimp_info.txt

Bapx1 r is 1

Found deficient in !Series_summary "Bapx1 (Nkx3.2) is an important factor for GI development. We document a morphologic change in the pylorus segment of the GI tract in Bapx1 deficient mice and we have used microarray analyses to assess gene exprofiles in the pylorus stomach region in knockout compared to wildtype littermates."

GSE13935_series_matrix.txtimp_info.txt

Bapx1 r is 2

Found deficient in !Series_summary "Keywords: Genetic modiufied mice deficient in Bapx1 expression"

GSE13935_series_matrix.txtimp_info.txt

Bapx1 r is 1

Found knockout in !Series_overall_design "RNA from 3 Bapx1 knockout animals and 3 control wildtype littermates were analyzed."

GSE13974_series_matrix.txtimp_info.txt

serine racemase r is 2

Found inactivation in !Series_title "Genetic inactivation of serine racemase produces behavioral phenotypes related to schizophrenia in mice"

GSE13981_series_matrix.txtimp_info.txt

Ccna2 r is 2

Found knockdown in !Series_title "Global gene expression profiles in Oct4-knockdown and Ccna2-knockdown mouse embryos."

GSE13981_series_matrix.txtimp_info.txt

Oct4 r is 1

Found knockdown in !Series_title "Global gene expression profiles in Oct4-knockdown and Ccna2-knockdown mouse embryos."

GSE13981_series_matrix.txtimp_info.txt

Ccna2 r is 1

Found knockdown in !Series_summary "Distinct subsets of genes are differentially expressed between Oct4 and Ccna2-knockdown embryos, and indicated differential functions. Further, a large panel of genes were confirmed to be differentially-expressed in Oct4-knockdown embryos by quantitative, real time RT-PCR."

GSE13981_series_matrix.txtimp_info.txt

Oct4 r is 3

Found knockdown in !Series_summary "Distinct subsets of genes are differentially expressed between Oct4 and Ccna2-knockdown embryos, and indicated differential functions. Further, a large panel of genes were confirmed to be differentially-expressed in Oct4-knockdown embryos by quantitative, real time RT-PCR."

GSE13992_series_matrix.txtimp_info.txt

c-Met r is 7

Found stimulated in !Series_overall_design "For gene array analysis c-MetDhepa and c-MetloxP/loxP controls were stimulated for 2 hours with 2µg recombinant mouse HGF.Three animals per group were treated in parallel, before and after i.p. injection of recombinant HGF or NaCl."

GSE14006_series_matrix.txtimp_info.txt

Bmal1 r is 1

Found mutant in !Series_summary "Hair follicles undergo recurrent cycling of controlled growth (anagen), regression (catagen), and relative quiescence (telogen) with a defined periodicity. Taking a genomics approach to study gene expression during synchronized mouse hair follicle cycling, we discovered that, in addition to circadian fluctuation, CLOCK-regulated genes are also modulated in phase with the hair growth cycle. During telogen and early anagen, circadian clock genes are prominently expressed in the secondary hair germ, which contains precursor cells for the growing follicle. Analysis of Clock and Bmal1 mutant mice reveals a delay in anagen progression, and the secondary hair germ cells show decreased levels of phosphorylated Rb and lack mitotic cells, suggesting that circadian clock genes regulate anagen progression via their effect on the cell cycle. Consistent with a block at the G1 phase of the cell cycle, we show a significant upregulation of p21 in Bmal1 mutant skin. While circadian clock

mechanisms have been implicated in a variety of diurnal biological processes, our findings indicate that circadian clock genes may be utilized to modulate the progression of non-diurnal cyclic processes."

GSE14021_series_matrix.txtimp_info.txt

Dyrk1a r is 1

Found overexpression in !Series_summary "The molecular mechanisms that lead to the cognitive defects characteristic of Down syndrome (DS), the most frequent cause of mental retardation, have remained elusive. Here we use a transgenic DS mouse model to show that DYRK1A gene dosage imbalance deregulates chromosomal clusters of genes located near neuron-restrictive silencer factor (REST/NRSF) binding sites. We found that DYRK1A binds the SWI/SNF-complex known to interact with REST/NRSF. Mutation of a REST/NRSF binding site in the promoter of the REST/NRSF target gene L1cam modifies the transcriptional effect of Dyrk1A-dosage imbalance on L1cam. DyrkA dosage imbalance perturbs Rest/Nrsf levels with decreased Rest/Nrsf expression in embryonic neurons and increased expression in adult neurons. We identified a coordinated deregulation of multiple genes that are responsible for the cellular phenotypic traits present in DS such as dendritic growth impairment and microcephaly during prenatal cortex development. Dyrk1a overexpression in primary mouse cortical neurons reduced the neuritic complexity. In the postnatal hippocampus, DYRK1A overexpression suppresses a form of synaptic plasticity that may be sufficient to cause DS cognitive defects. We propose that DYRK1A overexpression-related neuronal gene deregulation generates the brain phenotypic changes that characterize DS, with an accessory role for the gene dosage imbalance of other chromosome 21 genes."

GSE14030_series_matrix.txtimp_info.txt

Dyrk1a r is 1

Found overexpression in !Series_summary "The molecular mechanisms that lead to the cognitive defects characteristic of Down syndrome (DS), the most frequent cause of mental retardation, have remained elusive. Here we use a transgenic DS mouse model to show that DYRK1A gene dosage imbalance deregulates chromosomal clusters of genes located near neuron-restrictive silencer factor (REST/NRSF) binding sites. We found that DYRK1A binds the SWI/SNF-complex known to interact with REST/NRSF. Mutation of a REST/NRSF binding site in the promoter of the REST/NRSF target gene L1cam modifies the transcriptional effect of Dyrk1A-dosage imbalance on L1cam. DyrkA dosage imbalance perturbs Rest/Nrsf levels with decreased Rest/Nrsf expression in embryonic neurons and increased expression in adult neurons. We identified a coordinated deregulation of multiple genes that are responsible for the cellular phenotypic traits present in DS such as dendritic growth impairment and microcephaly during prenatal cortex development. Dyrk1a overexpression in primary mouse cortical neurons reduced the neuritic complexity. In the postnatal hippocampus, DYRK1A overexpression suppresses a form of synaptic plasticity that may be sufficient to cause DS cognitive defects. We propose that DYRK1A overexpression-related neuronal gene deregulation generates the brain phenotypic changes that characterize DS, with an accessory role for the gene dosage imbalance of other chromosome 21 genes."

GSE14031-GPL5105_series_matrix.txtimp_info.txt

estrogen receptor r is 3

Found knockout in !Series_title "Global survey of miRNA profiles in the uterus and an estrogen receptor alpha knockout embryo"

GSE14031-GPL5106_series_matrix.txtimp_info.txt

estrogen receptor r is 3

Found knockout in !Series_title "Global survey of miRNA profiles in the uterus and an estrogen receptor alpha knockout embryo"

GSE14072_series_matrix.txtimp_info.txt

Dyrk1a r is 1

Found overexpression in !Series_summary "REST/NRSF target gene L1cam modifies the transcriptional effect of Dyrk1Adosage imbalance on L1cam. DyrkA dosage imbalance perturbs Rest/Nrsf levels with decreased Rest/Nrsf expression in embryonic neurons and increased expression in adult neurons. We identified a coordinated deregulation of multiple genes that are responsible for the cellular phenotypic traits present in DS such as dendritic growth impairment and microcephaly during prenatal cortex development. Dyrk1a overexpression in primary mouse cortical neurons reduced the neuritic complexity. In the postnatal hippocampus, DYRK1A overexpression suppresses a form of synaptic plasticity that may be sufficient to cause DS cognitive defects. We propose that DYRK1A overexpression-related neuronal gene deregulation generates the"

GSE14088_series_matrix.txtimp_info.txt

Ube2m r is 7

Found knockdown in !Series_overall_design "Ube2m and Ube2f are E2 enzymes that direct protein modification by NEDD8. Here we explore the specific functions of Ube2f and Ube2m by comparing gene expression profiles following knockdown of their function in NIH 3T3 cells."

GSE14088_series_matrix.txtimp_info.txt

Ube2f r is 4

Found knockdown in !Series_overall_design "We used microarrays to detail the global programme of gene expression changes following knockdown of Ube2m and Ube2f in NIH 3T3 cells."

GSE14088_series_matrix.txtimp_info.txt

Ube2m r is 2

Found knockdown in !Series_overall_design "We used microarrays to detail the global programme of gene expression changes following knockdown of Ube2m and Ube2f in NIH 3T3 cells."

GSE14089_series_matrix.txtimp_info.txt

alpha-MSH r is 4

Found treated in !Series_summary "We determined the gene expression profiles of murine melan-a melanocytes treated with ASP or alpha-MSH over a 4 days time course using genome-wide oligonucleotide microarrays. As expected, the gene expression patterns emphasized the opposing effects of the 2 ligands, and there were significant reductions in expression of numerous melanogenic proteins elicited by ASP, which correlates with its inhibition of pigmentation. However, ASP also unexpectidly modulated the expression of genes involved in various other cellular pathways, including glutathione synthesis and redox metabolism. Many genes up-regulated by ASP are involved in morphogenesis, cell adhesion and ECM-receptor interactions."

GSE14101_series_matrix.txtimp_info.txt

Meis1 r is 3

Found expressing in !Series_overall_design "Murine Mll-AF9 leukemia (4166) cells were transduced with lentivirus expressing shRNA against Meis1 or control lentivirus (empty vector). Gene expression profiles were compared at 48 hours post transduction, using Puromycin as selection agent for transduced cells."

GSE14201_series_matrix.txtimp_info.txt

Lgr5 r is 6

Found expressing in !Series_overall_design "For the stem cell signature we used cell fractions of intestines from Lgr5-EGFP-ires-CreERT2 mice, expressing GFP under the control of the Lgr5 promoter. RNA was isolated from two FACS sorted cell populations, one expressing GFP at high levels (GFPhi) and the other expressing GFP at low levels (GFPlo). For the analysis of Ascl2 target genes RNA was isolated from intestinal epithelial cells of Ah-Cre/Ascl2floxed/floxed animals and Ah-Cre/Ascl2floxed/wt control animals 3 and 5 days post induction. Differentially labelled cRNA from GFPhi and GFPlo cells from two different sorts (each combining three different mice) were hybridised on 4X44K Agilent Whole Mouse Genome dual colour Microarrays (G4122F) in two dye swap experiments, resulting in four individual arrays. For the Ascl2 target gene analysis we analyzed the 3 and 5 days PI experiments in two dye swap experiments, resulting in four individual arrays."

GSE14216_series_matrix.txtimp_info.txt

Dnmt1 r is 4

Found deficient in !Series_overall_design "We compared gene expression patterns in Wildtype and DNA methylation deficient (Emx1-cre; Dnmt1 mutant) mouse dorsal cortex. We performed 3

replicates using different each individual mouse strain. The Sample GSM350992 table is the average log ratio for the 3 replicatesArrays were performed in triplicate."

GSE14216_series_matrix.txtimp_info.txt

Dnmt1 r is 1

Found mutant in !Series_summary "DNA methylation is a major epigenetic factor regulating genome reprogramming, cell differentiation, developmental gene expression. To understand the role DNA methylation in CNS neurons, we generated conditional Dnmt1 mutant mice that possess ~90% hypomethylated cortical and hippocampal cells in the dorsal forebrain from E13.5 on. The mutant mice were viable with a normal lifespan, but displayed severe neuronal cell death between E14.5 to 3-weeks postnatally. Accompanied with the striking cortical and hippocampal degeneration, adult mutant mice exhibited neurobehavioral defects in learning and memory in adulthood. Unexpectedly, a fraction of Dnmt1-/- cortical neurons survived through postnatal development, so that the residual cortex in mutant mice contained 20-30% of hypomethylated neurons throughout the life. Hypomethylated excitatory neurons exhibited multiple defects in postnatal maturation including abnormal dendritic arborization and impaired neuronal excitability. The mutant phenotypes are coupled with deregulation of those genes involved in neuronal layer-specification, cell death, and the function of ion channels. Our results suggest that DNA methylation, through its role in modulating neuronal gene expression, plays multiple roles in regulating cell survival, neuronal migration and maturation in the CNS."

GSE14219_series_matrix.txtimp_info.txt

Sall4 r is 1

Found null in !Series_title "Expression profile of Sall4-null ES cells and Sall4 heterozygous ES cells"

GSE14236_series_matrix.txtimp_info.txt

Flt3 r is 6

Found expressing in !Series_overall_design "We performed gene expression profiling: 32Dc vs. MLL-AF4 expressing 32Dc, 32Dc vs. Flt3 TKD+MLL-AF4 expressing 32Dc, and MLL-AF4 expressing 32Dc vs. Flt3 TKD+MLL-AF4 expressing 32Dc. A single sample for each expressing cells was analyzed."

GSE14336_series_matrix.txtimp_info.txt

p53 r is 1

Found mutant in !Series_title "Expression profiling of thymic lymphomas from p53 mutant (R270H) mice with varying HIF levels"

GSE14350_series_matrix.txtimp_info.txt

STAT5 r is 4

Found deficient in !Series_summary "Interleukin-2 receptor (IL-2R) signaling is essential for T regulatory (Treg) cell development and homeostasis. Here we show that expression of IL-2Rbeta chains that lack tyrosine residues important for the association of the adaptor Shc and the transcription factor STAT5 in IL-2Rbeta-deficient mice resulted in production of a normal proportion of natural Treg cells that suppressed severe autoimmunity related with deficiency in IL-2 or IL-2R. These mutant IL-2Rbeta chains supported suboptimal and transient STAT5 activation that upregulate the transcription factor Foxp3 to normal amounts in natural, but not induced, Treg cells. Using cells T cell obtained from normal C57BL/6 mice and mice harboring Treg cells with impaired IL-2R signaling, gene expression profiling revealed many targets in peripheral natural Treg cells that were IL-2-dependent and a substantial overlap between the Treg cell IL-2-dependent gene program and the Treg cell transcriptional signature. Collectively, these findings demonstrate that a critical, and perhaps minor, subset of IL-2-dependent targets in Treg cells is indexed to a low IL-2R signaling threshold and that a substantial proportion of the Treg cell gene program is regulated by IL-2. CD4 T effector cells also showed many IL-2R-dependent gene and these also overlapped in a distintive manner with the IL-2-dependent genes of Treg cells and the Treg gene signature."

GSE14350_series_matrix.txtimp_info.txt

Foxp3 r is 6

Found activation in !Series_summary "Interleukin-2 receptor (IL-2R) signaling is essential for T regulatory (Treg) cell development and homeostasis. Here we show that expression of IL-2Rbeta chains that lack tyrosine residues important for the association of the adaptor Shc and the transcription factor STAT5 in IL-2Rbeta-deficient mice resulted in production of a normal proportion of natural Treg cells that suppressed severe autoimmunity related with deficiency in IL-2 or IL-2R. These mutant IL-2Rbeta chains supported suboptimal and transient STAT5 activation that upregulate the transcription factor Foxp3 to normal amounts in natural, but not induced, Treg cells. Using cells T cell obtained from normal C57BL/6 mice and mice harboring Treg cells with impaired IL-2R signaling, gene expression profiling revealed many targets in peripheral natural Treg cells that were IL-2-dependent and a substantial overlap between the Treg cell IL-2-dependent gene program and the Treg cell transcriptional signature. Collectively, these findings demonstrate that a critical, and perhaps minor, subset of IL-2-dependent targets in Treg cells is indexed to a low IL-2R signaling threshold and that a substantial proportion of the Treg cell gene program is regulated by IL-2. CD4 T effector cells also showed many IL-2R-dependent gene and these also overlapped in a distintive manner with the IL-2-dependent genes of Treg cells and the Treg gene signature."

GSE14361_series_matrix.txtimp_info.txt

Sca-1 r is 6

Found stimulation in !Series_summary "Maintenance of the blood system is dependent on dormant haematopoietic stem cells (HSCs) with long-term self-renewal capacity. Upon injury these cells are induced to proliferate in order to quickly re-establish homeostasis. The signalling molecules promoting the exit of HSCs out of the dormant stage remain largely unknown. Here we show that in response to

treatment of mice with interferon-alpha (IFNα), HSCs efficiently exit G0 and enter an active cell cycle. HSCs respond to IFNα treatment by increased phosphorylation of STAT1 and PKB/Akt, expression of IFNα target genes and up-regulation of stem cell antigen-1 (Sca-1). HSCs lacking either the interferon-α/β receptor (IFNAR), STAT1 or Sca-1 are insensitive to IFNα stimulation, demonstrating that STAT1 and Sca-1 mediate IFNα induced HSC proliferation. Although dormant HSCs are resistant to the anti-proliferative chemotherapeutic agent 5-FU1, HSCs pre-treated (primed) with IFNα and thus induced to proliferate are efficiently eliminated by 5-FU exposure in vivo. Conversely, HSCs chronically activated by IFNα are functionally compromised and are rapidly out competed by non-activatable IFNAR-/- cells in competitive repopulation assays. In summary, while chronic activation of the IFNα pathway in HSCs impairs their function, acute IFNα treatment promotes the proliferation of dormant HSCs in vivo. These data may help to clarify the so far unexplained clinical effects of IFNα on leukemic cells and raise the possibility for novel applications of type I interferons to target cancer stem cells. "

GSE14375_series_matrix.txtimp_info.txt

PECAM-1 r is 5

Found induced in !Series_summary "Tight homeostatic control of brain amino acids (AA) depends on transport via solute family carrier proteins expressed by the Blood-Brain Barrier (BBB) microvascular endothelial cells (BMEC). To characterize the mouse BMEC transcriptome and probe culture-induced changes microarray analyses of PECAM-1+ endothelial cells (ppMBMECs) were compared with primary MBMECs (pMBMEC) cultured in the presence or absence of glial cells, and with b.End5 endothelioma cell-line. Selected cell marker and AA transporter mRNA levels were further verified by real-time RT PCR."

GSE14395_series_matrix.txtimp_info.txt

PPAR-alpha r is 2

Found KO in !Series_title "Gender-specific gene repression of PPAR-alpha KO mice in liver and heart"

GSE14413_series_matrix.txtimp_info.txt

beta s r is 1

Found stimulated in !Series_title "Gene expression profiling of interferon-beta stimulated cells"

GSE14415_series_matrix.txtimp_info.txt

Foxp3 r is 8

Found induced in !Series_summary "We used gene expression microarrays to examine the transcriptional programs of natural and induced regulatory T cells and the function of Foxp3 in organizing the transcriptosomes of the respective cell type"

GSE14418_series_matrix.txtimp_info.txt

F10 r is 8

Found stimulation in !Series_title "The effect of TIM-3 stimulation on the gene expression profile of B16-F10 melanoma cells"

GSE14418_series_matrix.txtimp_info.txt

TIM-3 r is 2

Found stimulation in !Series_title "The effect of TIM-3 stimulation on the gene expression profile of B16-F10 melanoma cells"

GSE14418_series_matrix.txtimp_info.txt

TIM-3 r is 2

Found stimulation in !Series_summary "The effect of TIM-3 stimulation was studied on B16F10 mouse melanoma cells, in vitro."

GSE14430_series_matrix.txtimp_info.txt

beta c r is 2

Found loss of in !Series_summary "Glis3 mutant mice (Glis3zf/zf) die within the first week after birth due to overt diabetes, evidenced by hyperglycemia and hypoinsulinemia. Histopathological analysis showed that Glis3zf/zf mice develop a pancreatic phenotype with a dramatic loss of beta- (insulin) and delta- (somatostatin) cells contrasting a smaller relative loss of alpha- (glucagon), PP- (pancreatic polypeptide), and epsilon- (ghrelin) cells. Glis3zf/zf mice develop ductal cysts with decreased number of primary cilia, while the acini are not significantly affected. Gene expression profiling by microarray analysis demonstrated that the expression of terminal hormonal genes and several transcription factors important in endocrine development were significantly deregulated in Glis3zf/zf mice. During pancreatic development, Glis3 mRNA expression is induced during the secondary transition, a stage of cell lineage specification and extensive patterning. Changes in pancreatic development of Glis3zf/zf mice are noted during and after this stage; the number of cells staining positively for Ngn3, MafA, or Pdx-1 is greatly diminished. These observations indicate that Glis3 plays a key role in the development of mature beta cells."

GSE14430_series_matrix.txtimp_info.txt

pancreatic polypeptide r is 5

Found loss of in !Series_summary "Glis3 mutant mice (Glis3zf/zf) die within the first week after birth due to overt diabetes, evidenced by hyperglycemia and hypoinsulinemia. Histopathological analysis showed that Glis3zf/zf mice develop a pancreatic phenotype with a dramatic loss of beta- (insulin) and delta- (somatostatin) cells contrasting a smaller relative loss of alpha- (glucagon), PP-

(pancreatic polypeptide), and epsilon- (ghrelin) cells. Glis3zf/zf mice develop ductal cysts with decreased number of primary cilia, while the acini are not significantly affected. Gene expression profiling by microarray analysis demonstrated that the expression of terminal hormonal genes and several transcription factors important in endocrine development were significantly deregulated in Glis3zf/zf mice. During pancreatic development, Glis3 mRNA expression is induced during the secondary transition, a stage of cell lineage specification and extensive patterning. Changes in pancreatic development of Glis3zf/zf mice are noted during and after this stage; the number of cells staining positively for Ngn3, MafA, or Pdx-1 is greatly diminished. These observations indicate that Glis3 plays a key role in the development of mature beta cells."

GSE14430_series_matrix.txtimp_info.txt

Glis3 r is 4

Found induced in !Series_summary "Glis3 mutant mice (Glis3zf/zf) die within the first week after birth due to overt diabetes, evidenced by hyperglycemia and hypoinsulinemia. Histopathological analysis showed that Glis3zf/zf mice develop a pancreatic phenotype with a dramatic loss of beta- (insulin) and delta- (somatostatin) cells contrasting a smaller relative loss of alpha- (glucagon), PP- (pancreatic polypeptide), and epsilon- (ghrelin) cells. Glis3zf/zf mice develop ductal cysts with decreased number of primary cilia, while the acini are not significantly affected. Gene expression profiling by microarray analysis demonstrated that the expression of terminal hormonal genes and several transcription factors important in endocrine development were significantly deregulated in Glis3zf/zf mice. During pancreatic development, Glis3 mRNA expression is induced during the secondary transition, a stage of cell lineage specification and extensive patterning. Changes in pancreatic development of Glis3zf/zf mice are noted during and after this stage; the number of cells staining positively for Ngn3, MafA, or Pdx-1 is greatly diminished. These observations indicate that Glis3 plays a key role in the development of mature beta cells."

GSE14438_series_matrix.txtimp_info.txt

TIM-3 r is 2

Found stimulation in !Series_title "The effect of TIM-3 stimulation on the gene expression profile of IgE/antigen-activated mouse mast cells"

GSE14449_series_matrix.txtimp_info.txt

K-ras r is 2

Found mutant in !Series_title "Gene expression profiles of spontaneous metastasis in a K-ras/p53 mutant mouse model"

GSE14454_series_matrix.txtimp_info.txt

Eps8 r is 3

Found knockout in !Series_summary "In a variety of organisms, including mammals, caloric restriction improves metabolic status and lowers the incidence of chronic-degenerative diseases, ultimately leading to increased lifespan. Here we show that knockout mice for Eps8, a regulator of actin dynamics, display reduced bodyweight, partial resistance to age- or diet-induced obesity, and overall improved metabolic status. We present evidence that these phenotypes, which are associated to increased lifespan in the Eps8 null mice, are due to caloric restriction. This, in turn, is caused by reduced intestinal fat absorption, due to altered morphogenesis of microvilli in intestinal enterocytes. In the nematode, genetic removal of Eps8, causes a microvillar phenotype, indistinguishable from that observed in mice, which leads to early larval lethality. By exploiting the nematode model system, we demonstrate here that the actin bundling activity of Eps8 is indispensable for viability and proper intestinal morphogenesis. This result links a precise molecular function of Eps8 to proper microvillar morphogenesis, and therefore to the phenotype of Eps8-null mice. Our results implicate actin dynamics in individual variations in bodyweight, metabolic status and longevity."

GSE14457-GPL2881_series_matrix.txtimp_info.txt

p53 r is 1

Found mutant in !Series_summary "The retinoblastoma tumor suppressor, Rb, is implicated in luminal-B and basal-like breast carcinomas, yet its effect on mammary gland development and causal role in breast cancer subtypes remain undefined. Here we show that conditional deletion of Rb in mouse mammary epithelium led to expansion of the stem/progenitor cells and to focal acinar hyperplasia with squamous metaplasia. These uniform lesions progressed into histologically diverse, transplantable mammary adenocarcinomas and adenosquamous carcinomas with features of luminal-B or basal-like carcinomas. A subset of basal-like but none of the luminal-B tumors expressed mutant p53. These results demonstrate a causative role for Rb in the etiology of breast cancer subtypes and implicate p53 status as a determinant of tumor phenotype after Rb loss."

GSE14457-GPL2881_series_matrix.txtimp_info.txt

mutant p53 r is 0

Found mutant in !Series_summary "The retinoblastoma tumor suppressor, Rb, is implicated in luminal-B and basal-like breast carcinomas, yet its effect on mammary gland development and causal role in breast cancer subtypes remain undefined. Here we show that conditional deletion of Rb in mouse mammary epithelium led to expansion of the stem/progenitor cells and to focal acinar hyperplasia with squamous metaplasia. These uniform lesions progressed into histologically diverse, transplantable mammary adenocarcinomas and adenosquamous carcinomas with features of luminal-B or basal-like carcinomas. A subset of basal-like but none of the luminal-B tumors expressed mutant p53. These results demonstrate a causative role for Rb in the etiology of breast cancer subtypes and implicate p53 status as a determinant of tumor phenotype after Rb loss."

GSE14457-GPL4092_series_matrix.txtimp_info.txt

p53 r is 1

Found mutant in !Series_summary "The retinoblastoma tumor suppressor, Rb, is implicated in luminal-B and basal-like breast carcinomas, yet its effect on mammary gland development and causal role in breast cancer subtypes remain undefined. Here we show that conditional deletion of Rb in mouse mammary epithelium led to expansion of the stem/progenitor cells and to focal acinar hyperplasia with squamous metaplasia. These uniform lesions progressed into histologically diverse, transplantable mammary adenocarcinomas and adenosquamous carcinomas with features of luminal-B or basal-like carcinomas. A subset of basal-like but none of the luminal-B tumors expressed mutant p53. These results demonstrate a causative role for Rb in the etiology of breast cancer subtypes and implicate p53 status as a determinant of tumor phenotype after Rb loss."

GSE14457-GPL4092_series_matrix.txtimp_info.txt

mutant p53 r is 0

Found mutant in !Series_summary "The retinoblastoma tumor suppressor, Rb, is implicated in luminal-B and basal-like breast carcinomas, yet its effect on mammary gland development and causal role in breast cancer subtypes remain undefined. Here we show that conditional deletion of Rb in mouse mammary epithelium led to expansion of the stem/progenitor cells and to focal acinar hyperplasia with squamous metaplasia. These uniform lesions progressed into histologically diverse, transplantable mammary adenocarcinomas and adenosquamous carcinomas with features of luminal-B or basal-like carcinomas. A subset of basal-like but none of the luminal-B tumors expressed mutant p53. These results demonstrate a causative role for Rb in the etiology of breast cancer subtypes and implicate p53 status as a determinant of tumor phenotype after Rb loss."

GSE14457-GPL891_series_matrix.txtimp_info.txt

p53 r is 1

Found mutant in !Series_summary "The retinoblastoma tumor suppressor, Rb, is implicated in luminal-B and basal-like breast carcinomas, yet its effect on mammary gland development and causal role in breast cancer subtypes remain undefined. Here we show that conditional deletion of Rb in mouse mammary epithelium led to expansion of the stem/progenitor cells and to focal acinar hyperplasia with squamous metaplasia. These uniform lesions progressed into histologically diverse, transplantable mammary adenocarcinomas and adenosquamous carcinomas with features of luminal-B or basal-like carcinomas. A subset of basal-like but none of the luminal-B tumors expressed mutant p53. These results demonstrate a causative role for Rb in the etiology of breast cancer subtypes and implicate p53 status as a determinant of tumor phenotype after Rb loss."

GSE14457-GPL891_series_matrix.txtimp_info.txt

mutant p53 r is 0

Found mutant in !Series_summary "The retinoblastoma tumor suppressor, Rb, is implicated in luminal-B and basal-like breast carcinomas, yet its effect on mammary gland development and causal role in breast cancer subtypes remain undefined. Here we show that conditional deletion of Rb in mouse mammary epithelium led to expansion of the stem/progenitor cells and to focal acinar hyperplasia with squamous metaplasia. These uniform lesions progressed into histologically diverse, transplantable mammary adenocarcinomas and adenosquamous carcinomas with features of luminal-B or basal-like carcinomas. A subset of basal-like but none of the luminal-B tumors expressed mutant p53. These results demonstrate a causative role for Rb in the etiology of breast cancer subtypes and implicate p53 status as a determinant of tumor phenotype after Rb loss."

GSE14459-GPL1261_series_matrix.txtimp_info.txt

K-ras r is 2

Found mutant in !Series_title "NSCLC metastasis: K-ras/p53 mutant and syngeneic mouse models"

GSE14459-GPL339_series_matrix.txtimp_info.txt

K-ras r is 2

Found mutant in !Series_title "NSCLC metastasis: K-ras/p53 mutant and syngeneic mouse models"

GSE14512_series_matrix.txtimp_info.txt

MuRF1 r is 6

Found -/- in !Series_summary "Muscle ring finger (MuRF) proteins have been implicated in the transmission of mechanical forces to nuclear cell signaling pathways through their association with the sarcomere. We recently reported that MuRF1, but not MuRF2, regulated pathologic cardiac hypertrophy in vivo. This was surprising since MuRF1 and MuRF2 interact redundantly with sarcomeric proteins in yeast two hybrid studies, and form both homo- and hetero-dimers with each other. To determine if MuRF1 and MuRF2 were functionally redundant during development, we created mice lacking either 3 or 4 of the MuRF1 and MuRF2 alleles and compared them functionally. Surprisingly, only mice missing all four MuRF1 and MuRF2 alleles (MuRF1-/-//MuRF2-/-) developed a spontaneous hypertrophic cardiomyopathy - mice that were null for one of the genes, but heterozygous for the other (i.e. MuRF1-/-//MuRF2+/- or MuRF1+/-//MuRF2-/-) were phenotypically identical to wild type mice. Electron microscopy of the hearts of MuRF1-/-//MuRF2-/-(MuRF1/MuRF2 DN) mice identified altered Z disc and M line architecture, and a distinct swelling of mitochondria. MuRF1-/-//MuRF2-/- mouse hearts displayed increased expression of genes associated with fetal cardiac metabolism, including smooth muscle actin and b myosin heavy chain, suggesting that the cardiac hypertrophy seen in these mice was associated with a reversion to a fetal gene program. Despite our prediction that we would also see an increase in glucose compared to fatty acid oxidation (another trait of fetal cardiac metabolism) we saw that MuRF1-/-//MuRF2-/- heart homogenates oxidized significantly less glucose compared to controls, suggesting an important role for MuRF1 and MuRF2 in the regulation of glucose metabolism in vivo. This

study identifies a previously unreported redundancy in the function of MuRF proteins in normal cardiac development."

GSE14512_series_matrix.txtimp_info.txt

MuRF1 r is 1

Found -/- in !Series_overall_design "Four strain-matched groups of 12 week old mice were investigated: 1) MuRF1 -/- MuRF2 -/-; 2) MuRF1 +/+ MuRF2 +/+; 3) MuRF1 -/- MuRF2 +/-; 4) MuRF1 +/- MuRF2 -/-. Biological replicates: 4 WT, 4 MuRF1 -/- // MuRF2 -/-, 4 MuRF1 +/- //MuRF2 -/-, 4 MuRF1 -/- // MuRF2 +/-. Hearts harvested. One replicate per array."

GSE14536_series_matrix.txtimp_info.txt

AP-2 r is 2

Found mutant in !Series_overall_design "Biological triplicates of mouse lenses were analyzed (three wild-type lens samples and three Le-AP-2 mutant samples)"

GSE14539_series_matrix.txtimp_info.txt

Mdr2 r is 1

Found knockout in !Series_summary "Surgical resection is the preferred treatment for Hepatocellular carcinoma; however, it induces tumor recurrence. Our objective was to understand the molecular mechanisms linking liver regeneration under chronic-inflammation to tumorigenesis. Mdr2-knockout mice, a model of inflammation-associated cancer, underwent partial-hepatectomy which led to enhanced hepatocarcinogenesis. Yet, liver regeneration in these mice was severely attenuated. We demonstrate the activation of the DNA damage response machinery and altered genomic instability during early liver inflammatory stages resulting in hepatocyte apoptosis and cell-cycle arrest, and suggest their involvement in tumor recurrence subsequent to partial hepatectomy. We propose that under the regenerative proliferative stress induced by liver resection, the genomic unstable hepatocytes generated during chronic-inflammation, escape apoptosis and reenter the cell-cycle, triggering the enhanced tumorigenesis"

GSE14539_series_matrix.txtimp_info.txt

Mdr2 r is 2

Found -/- in !Series_overall_design "RNA was isolated from liver samples of 9-month-old Mdr2-/- and control mice obtained on days 0 (the removed lobe), 2 and 6 following PHx. Samples of d0 were obtained from the same mice that were sacrificed on later days. As we were concerned by the variability in the KO group 6 samples were obtained for d0. All other time points and groups contained 3 samples each."

GSE14561_series_matrix.txtimp_info.txt

Pig-a r is 2

Found knock-out in !Series_summary "Somatic mutation in the X-linked phosphatidylinositol glycan class A (PIG-A) gene causes glycosylphosphatidylinositol (GPI) anchor deficiency in humans with Paroxysmal Nocturnal Hemoglobinuria (PNH). Clinically, patients with PNH have intravascular hemolysis, venous thrombosis and bone marrow failure. We produced a conditional Pig-a knock-out mouse model specifically inactivating the Pig-a gene in hematopoietic cells to study the role of PIG-A deficiency in PNH pathophysiology. We used Affymetrix Mouse Genome 430 2.0 chips to investigate the gene expression pattern in the mouse model of targeted Pig-a deletion."

GSE14561_series_matrix.txtimp_info.txt

Pig-a r is 2

Found knock-out in !Series_overall_design "We performed microarray analysis on 3 pools of sorted GPI-deficient (GPI-) and GPI normal (GPI+) bone marrow cells derived from the same Pig-a knock-out animals, and identified 1275/669 genes of the 45,101 transcripts potentially available for screening using 2-fold change, <10% false discovery rate (FDR) and percentage of present calls as selection criteria. The major representative genes belong to the category of immune response. We tested whether the molecular differences between GPI- and GPI+ bone marrow cells could be preserved when GPI- cells were transplanted in lethally-irradiated wild type mice (C57BL/6). Microarray analysis was performed using sorted bone marrow cells from 4 animals transplanted with GPI-deficient bone marrow cells (T-GPI-) from Pig-a knock-out donors, 3 animals transplanted with GPI-normal bone marrow cells from C57BL/6 donors (T-GPI+), and GPI-normal bone marrow cells from 4 wild-type C57BL/6 mice (WT-GPI+). We found 296 probesets with 2-fold change cutoff, of which T-GPI- cells had 145 up-regulated genes in comparison to WT-GPI+ cells, and had 123 genes differentially expressed when compared to T-GPI+ cells. The gene expression of the GPI-deficient cells was very similar between the two sets of microarray experiments affirming the maintenance of the phenotype before and after bone marrow cell transplantation."

GSE14586_series_matrix.txtimp_info.txt

Cdx2 r is 1

Found overexpression in !Series_summary "(F) Potential CDX2-direct target genes based on ChIP-Seq and the alteration of expression by Cdx2-overexpression."

GSE14605_series_matrix.txtimp_info.txt

Ago2 r is 6

Found knockout in !Series_overall_design "gene expression profiling from two single wild-type oocytes, two single Dicer knockout oocyte, and one single Ago2 knockout oocyte"

GSE14654_series_matrix.txtimp_info.txt

Ctr9 r is 8

Found expressing in !Series_summary "To study the function of Paf1C in mouse ESCs, we generated an ES cell line stably expressing a location and affinity purification (LAP)-tagged Ctr9 fusiuon protein using the bacterial astificial chromosome (BAC)-based TransgeneOmics approach. To investigate whether pluripotency and lineage control genes differentially regulated upon Paf1C depletion are direct targets of the Paf1C, we analyzed the binding of the Ctr9-LAP fusion protein by ChIP-chip and identified 2175 promoter regions that were bound by Ctr9."

GSE14672_series_matrix.txtimp_info.txt

Stat5a r is 2

Found lacking in !Series_summary "GM-CSF controls the development of granulocytes but little is known about the contribution of the downstream mediating transcription factor STAT5A/B. To elucidate this pathway, we generated mice lacking the Stat5a and 5b genes in blood cells. Peripheral neutrophils were decreased and administration of 5-FU and GM-CSF failed to induce granulopoiesis in Stat5a/b-mutant mice. GMPs were isolated and cultured with GM-CSF. Both the number and size of STAT5A/B-null colonies were reduced and GM-CSF-induced survival of mature STAT5A/B-null neutrophils was impaired. Time-lapse cinematography and single cell tracking of GMPs revealed that STAT5A/B-null cells were characterized by a longer generation time and an increased cell death. Gene expression profiling experiments suggested that STAT5A/B directs GM-CSF signaling through the regulation of cell survival genes."

GSE14672_series_matrix.txtimp_info.txt

Stat5a r is 5

Found lacking in !Series_overall_design "Mice lacking or with the Stat5a and 5b genes in blood cells, which were treated w/o GMP"

GSE14691-GPL1261_series_matrix.txtimp_info.txt

Clcn1 r is 2

Found knockout in !Series_summary "Myotonic dystrophy type 1 (DM1) is an RNA dominant disease in which mutant transcripts containing an expanded CUG repeat (CUGexp) cause muscle dysfunction by interfering with biogenesis of other mRNAs. The toxic effects of mutant RNA are mediated partly through sequestration of splicing regulator Muscleblind-like 1 (Mbnl1), a protein that binds to CUGexp RNA. A gene that is prominently affected encodes chloride channel 1 (Clcn1), resulting in hyperexcitability of muscle (myotonia). To identify DM1-affected genes and study mechanisms for dysregulation, we performed global mRNA profiling in transgenic mice that express CUGexp RNA, as compared to Mbnl1 knockout and Clcn1 null mice. We found that the majority of changes induced by CUGexp RNA in skeletal muscle can be explained by reduced activity of Mbnl1, including many changes that are secondary to myotonia. The pathway most affected comprises genes involved in calcium

signaling and homeostasis. Some effects of CUGexp RNA on gene expression are caused by abnormal alternative splicing or downregulation of Mbnl1-interacting mRNAs. However, several of the most highly dysregulated genes showed altered transcription, as indicated by parallel changes of the corresponding premRNAs. These results support the idea that trans-dominant effects of CUGexp RNA on gene expression in this transgenic model may occur at the level of transcription, RNA processing, and mRNA decay, and are mediated mainly but not entirely through sequestration of Mbnl1."

GSE14691-GPL1261_series_matrix.txtimp_info.txt

Mbnl1 r is 1

Found knockout in !Series_summary "Myotonic dystrophy type 1 (DM1) is an RNA dominant disease in which mutant transcripts containing an expanded CUG repeat (CUGexp) cause muscle dysfunction by interfering with biogenesis of other mRNAs. The toxic effects of mutant RNA are mediated partly through sequestration of splicing regulator Muscleblind-like 1 (Mbnl1), a protein that binds to CUGexp RNA. A gene that is prominently affected encodes chloride channel 1 (Clcn1), resulting in hyperexcitability of muscle (myotonia). To identify DM1-affected genes and study mechanisms for dysregulation, we performed global mRNA profiling in transgenic mice that express CUGexp RNA, as compared to Mbnl1 knockout and Clcn1 null mice. We found that the majority of changes induced by CUGexp RNA in skeletal muscle can be explained by reduced activity of Mbnl1, including many changes that are secondary to myotonia. The pathway most affected comprises genes involved in calcium signaling and homeostasis. Some effects of CUGexp RNA on gene expression are caused by abnormal alternative splicing or downregulation of Mbnl1-interacting mRNAs. However, several of the most highly dysregulated genes showed altered transcription, as indicated by parallel changes of the corresponding premRNAs. These results support the idea that trans-dominant effects of CUGexp RNA on gene expression in this transgenic model may occur at the level of transcription, RNA processing, and mRNA decay, and are mediated mainly but not entirely through sequestration of Mbnl1."

GSE14691-GPL1261_series_matrix.txtimp_info.txt

Clcn1 r is 1

Found null in !Series_summary "Myotonic dystrophy type 1 (DM1) is an RNA dominant disease in which mutant transcripts containing an expanded CUG repeat (CUGexp) cause muscle dysfunction by interfering with biogenesis of other mRNAs. The toxic effects of mutant RNA are mediated partly through sequestration of splicing regulator Muscleblind-like 1 (Mbnl1), a protein that binds to CUGexp RNA. A gene that is prominently affected encodes chloride channel 1 (Clcn1), resulting in hyperexcitability of muscle (myotonia). To identify DM1-affected genes and study mechanisms for dysregulation, we performed global mRNA profiling in transgenic mice that express CUGexp RNA, as compared to Mbnl1 knockout and Clcn1 null mice. We found that the majority of changes induced by CUGexp RNA in skeletal muscle can be explained by reduced activity of Mbnl1, including many changes that are secondary to myotonia. The pathway most affected comprises genes involved in calcium signaling and homeostasis. Some effects of CUGexp RNA on gene expression are caused by abnormal alternative splicing or downregulation of Mbnl1-interacting mRNAs. However, several of the most highly

dysregulated genes showed altered transcription, as indicated by parallel changes of the corresponding premRNAs. These results support the idea that trans-dominant effects of CUGexp RNA on gene expression in this transgenic model may occur at the level of transcription, RNA processing, and mRNA decay, and are mediated mainly but not entirely through sequestration of Mbnl1."

GSE14691-GPL1261_series_matrix.txtimp_info.txt

Mbnl1 r is 4

Found null in !Series_summary "Myotonic dystrophy type 1 (DM1) is an RNA dominant disease in which mutant transcripts containing an expanded CUG repeat (CUGexp) cause muscle dysfunction by interfering with biogenesis of other mRNAs. The toxic effects of mutant RNA are mediated partly through sequestration of splicing regulator Muscleblind-like 1 (Mbnl1), a protein that binds to CUGexp RNA. A gene that is prominently affected encodes chloride channel 1 (Clcn1), resulting in hyperexcitability of muscle (myotonia). To identify DM1-affected genes and study mechanisms for dysregulation, we performed global mRNA profiling in transgenic mice that express CUGexp RNA, as compared to Mbnl1 knockout and Clcn1 null mice. We found that the majority of changes induced by CUGexp RNA in skeletal muscle can be explained by reduced activity of Mbnl1, including many changes that are secondary to myotonia. The pathway most affected comprises genes involved in calcium signaling and homeostasis. Some effects of CUGexp RNA on gene expression are caused by abnormal alternative splicing or downregulation of Mbnl1-interacting mRNAs. However, several of the most highly dysregulated genes showed altered transcription, as indicated by parallel changes of the corresponding premRNAs. These results support the idea that trans-dominant effects of CUGexp RNA on gene expression in this transgenic model may occur at the level of transcription, RNA processing, and mRNA decay, and are mediated mainly but not entirely through sequestration of Mbnl1."

GSE14691-GPL1261_series_matrix.txtimp_info.txt

Clcn1 r is 1

Found null in !Series_overall_design "All experiments involved generating expression profiles of quadriceps muscles taken from mice. Experiments 1 and 2: samples were hybridized to Moe430A and Moe430B arrays. Experiment 3: samples were hybridized to Mouse Genome 430 2.0 array. Experiment 1 compared expression profiles of wild-type mice (FVB strain) with two lines, designated 20b and 41, of CUGexp transgenic mice with FVB background. Experiment 2 compared expression profiles of Clcn1-null (myotonic) mice with the wild-type background strain (BALB). Experiment 3 compared expression profiles of Mbnl1-null mice with the wild-type background strain (FVB)."

GSE14691-GPL339_series_matrix.txtimp_info.txt

Clcn1 r is 2

Found knockout in !Series_summary "Myotonic dystrophy type 1 (DM1) is an RNA dominant disease in which mutant transcripts containing an expanded CUG repeat (CUGexp) cause muscle dysfunction by interfering with biogenesis of other mRNAs. The toxic effects of mutant RNA are mediated partly

through sequestration of splicing regulator Muscleblind-like 1 (Mbnl1), a protein that binds to CUGexp RNA. A gene that is prominently affected encodes chloride channel 1 (Clcn1), resulting in hyperexcitability of muscle (myotonia). To identify DM1-affected genes and study mechanisms for dysregulation, we performed global mRNA profiling in transgenic mice that express CUGexp RNA, as compared to Mbnl1 knockout and Clcn1 null mice. We found that the majority of changes induced by CUGexp RNA in skeletal muscle can be explained by reduced activity of Mbnl1, including many changes that are secondary to myotonia. The pathway most affected comprises genes involved in calcium signaling and homeostasis. Some effects of CUGexp RNA on gene expression are caused by abnormal alternative splicing or downregulation of Mbnl1-interacting mRNAs. However, several of the most highly dysregulated genes showed altered transcription, as indicated by parallel changes of the corresponding premRNAs. These results support the idea that trans-dominant effects of CUGexp RNA on gene expression in this transgenic model may occur at the level of transcription, RNA processing, and mRNA decay, and are mediated mainly but not entirely through sequestration of Mbnl1."

GSE14691-GPL339_series_matrix.txtimp_info.txt

Mbnl1 r is 1

Found knockout in !Series_summary "Myotonic dystrophy type 1 (DM1) is an RNA dominant disease in which mutant transcripts containing an expanded CUG repeat (CUGexp) cause muscle dysfunction by interfering with biogenesis of other mRNAs. The toxic effects of mutant RNA are mediated partly through sequestration of splicing regulator Muscleblind-like 1 (Mbnl1), a protein that binds to CUGexp RNA. A gene that is prominently affected encodes chloride channel 1 (Clcn1), resulting in hyperexcitability of muscle (myotonia). To identify DM1-affected genes and study mechanisms for dysregulation, we performed global mRNA profiling in transgenic mice that express CUGexp RNA, as compared to Mbnl1 knockout and Clcn1 null mice. We found that the majority of changes induced by CUGexp RNA in skeletal muscle can be explained by reduced activity of Mbnl1, including many changes that are secondary to myotonia. The pathway most affected comprises genes involved in calcium signaling and homeostasis. Some effects of CUGexp RNA on gene expression are caused by abnormal alternative splicing or downregulation of Mbnl1-interacting mRNAs. However, several of the most highly dysregulated genes showed altered transcription, as indicated by parallel changes of the corresponding premRNAs. These results support the idea that trans-dominant effects of CUGexp RNA on gene expression in this transgenic model may occur at the level of transcription, RNA processing, and mRNA decay, and are mediated mainly but not entirely through sequestration of Mbnl1."

GSE14691-GPL339_series_matrix.txtimp_info.txt

Clcn1 r is 1

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GSE14691-GPL339_series_matrix.txtimp_info.txt

Mbnl1 r is 4

Found null in !Series_summary "Myotonic dystrophy type 1 (DM1) is an RNA dominant disease in which mutant transcripts containing an expanded CUG repeat (CUGexp) cause muscle dysfunction by interfering with biogenesis of other mRNAs. The toxic effects of mutant RNA are mediated partly through sequestration of splicing regulator Muscleblind-like 1 (Mbnl1), a protein that binds to CUGexp RNA. A gene that is prominently affected encodes chloride channel 1 (Clcn1), resulting in hyperexcitability of muscle (myotonia). To identify DM1-affected genes and study mechanisms for dysregulation, we performed global mRNA profiling in transgenic mice that express CUGexp RNA, as compared to Mbnl1 knockout and Clcn1 null mice. We found that the majority of changes induced by CUGexp RNA in skeletal muscle can be explained by reduced activity of Mbnl1, including many changes that are secondary to myotonia. The pathway most affected comprises genes involved in calcium signaling and homeostasis. Some effects of CUGexp RNA on gene expression are caused by abnormal alternative splicing or downregulation of Mbnl1-interacting mRNAs. However, several of the most highly dysregulated genes showed altered transcription, as indicated by parallel changes of the corresponding premRNAs. These results support the idea that trans-dominant effects of CUGexp RNA on gene expression in this transgenic model may occur at the level of transcription, RNA processing, and mRNA decay, and are mediated mainly but not entirely through sequestration of Mbnl1."

GSE14691-GPL339_series_matrix.txtimp_info.txt

Clcn1 r is 1

Found null in !Series_overall_design "All experiments involved generating expression profiles of quadriceps muscles taken from mice. Experiments 1 and 2: samples were hybridized to Moe430A and Moe430B arrays. Experiment 3: samples were hybridized to Mouse Genome 430 2.0 array. Experiment 1 compared expression profiles of wild-type mice (FVB strain) with two lines, designated 20b and 41, of CUGexp transgenic mice with FVB background. Experiment 2 compared expression profiles of Clcn1-null (myotonic) mice with the wild-type background strain (BALB). Experiment 3 compared expression profiles of Mbnl1-null mice with the wild-type background strain (FVB)."

GSE14691-GPL340_series_matrix.txtimp_info.txt

Clcn1 r is 2

Found knockout in !Series_summary "Myotonic dystrophy type 1 (DM1) is an RNA dominant disease in which mutant transcripts containing an expanded CUG repeat (CUGexp) cause muscle dysfunction by interfering with biogenesis of other mRNAs. The toxic effects of mutant RNA are mediated partly through sequestration of splicing regulator Muscleblind-like 1 (Mbnl1), a protein that binds to CUGexp RNA. A gene that is prominently affected encodes chloride channel 1 (Clcn1), resulting in hyperexcitability of muscle (myotonia). To identify DM1-affected genes and study mechanisms for dysregulation, we performed global mRNA profiling in transgenic mice that express CUGexp RNA, as compared to Mbnl1 knockout and Clcn1 null mice. We found that the majority of changes induced by CUGexp RNA in skeletal muscle can be explained by reduced activity of Mbnl1, including many changes that are secondary to myotonia. The pathway most affected comprises genes involved in calcium signaling and homeostasis. Some effects of CUGexp RNA on gene expression are caused by abnormal alternative splicing or downregulation of Mbnl1-interacting mRNAs. However, several of the most highly dysregulated genes showed altered transcription, as indicated by parallel changes of the corresponding premRNAs. These results support the idea that trans-dominant effects of CUGexp RNA on gene expression in this transgenic model may occur at the level of transcription, RNA processing, and mRNA decay, and are mediated mainly but not entirely through sequestration of Mbnl1."

GSE14691-GPL340_series_matrix.txtimp_info.txt

Mbnl1 r is 1

Found knockout in !Series_summary "Myotonic dystrophy type 1 (DM1) is an RNA dominant disease in which mutant transcripts containing an expanded CUG repeat (CUGexp) cause muscle dysfunction by interfering with biogenesis of other mRNAs. The toxic effects of mutant RNA are mediated partly through sequestration of splicing regulator Muscleblind-like 1 (Mbnl1), a protein that binds to CUGexp RNA. A gene that is prominently affected encodes chloride channel 1 (Clcn1), resulting in hyperexcitability of muscle (myotonia). To identify DM1-affected genes and study mechanisms for dysregulation, we performed global mRNA profiling in transgenic mice that express CUGexp RNA, as compared to Mbnl1 knockout and Clcn1 null mice. We found that the majority of changes induced by CUGexp RNA in skeletal muscle can be explained by reduced activity of Mbnl1, including many changes that are secondary to myotonia. The pathway most affected comprises genes involved in calcium signaling and homeostasis. Some effects of CUGexp RNA on gene expression are caused by abnormal alternative splicing or downregulation of Mbnl1-interacting mRNAs. However, several of the most highly dysregulated genes showed altered transcription, as indicated by parallel changes of the corresponding premRNAs. These results support the idea that trans-dominant effects of CUGexp RNA on gene expression in this transgenic model may occur at the level of transcription, RNA processing, and mRNA decay, and are mediated mainly but not entirely through sequestration of Mbnl1."

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GSE14691-GPL340_series_matrix.txtimp_info.txt

Mbnl1 r is 4

Found null in !Series_summary "Myotonic dystrophy type 1 (DM1) is an RNA dominant disease in which mutant transcripts containing an expanded CUG repeat (CUGexp) cause muscle dysfunction by interfering with biogenesis of other mRNAs. The toxic effects of mutant RNA are mediated partly through sequestration of splicing regulator Muscleblind-like 1 (Mbnl1), a protein that binds to CUGexp RNA. A gene that is prominently affected encodes chloride channel 1 (Clcn1), resulting in hyperexcitability of muscle (myotonia). To identify DM1-affected genes and study mechanisms for dysregulation, we performed global mRNA profiling in transgenic mice that express CUGexp RNA, as compared to Mbnl1 knockout and Clcn1 null mice. We found that the majority of changes induced by CUGexp RNA in skeletal muscle can be explained by reduced activity of Mbnl1, including many changes that are secondary to myotonia. The pathway most affected comprises genes involved in calcium signaling and homeostasis. Some effects of CUGexp RNA on gene expression are caused by abnormal alternative splicing or downregulation of Mbnl1-interacting mRNAs. However, several of the most highly dysregulated genes showed altered transcription, as indicated by parallel changes of the corresponding premRNAs. These results support the idea that trans-dominant effects of CUGexp RNA on gene expression in this transgenic model may occur at the level of transcription, RNA processing, and mRNA decay, and are mediated mainly but not entirely through sequestration of Mbnl1."

GSE14691-GPL340_series_matrix.txtimp_info.txt

Clcn1 r is 1

Found null in !Series_overall_design "All experiments involved generating expression profiles of quadriceps muscles taken from mice. Experiments 1 and 2: samples were hybridized to Moe430A and Moe430B arrays. Experiment 3: samples were hybridized to Mouse Genome 430 2.0 array. Experiment 1 compared expression profiles of wild-type mice (FVB strain) with two lines, designated 20b and 41, of CUGexp transgenic mice with FVB background. Experiment 2 compared expression profiles of Clcn1-null (myotonic) mice with the wild-type background strain (BALB). Experiment 3 compared expression profiles of Mbnl1-null mice with the wild-type background strain (FVB)."

GSE14698_series_matrix.txtimp_info.txt

Stat5a r is 5

Found lacking in !Series_title "Microarray Experiments for mice lacking or with the Stat5a and 5b genes in blood cells that were treated w/o CMP"

GSE14698_series_matrix.txtimp_info.txt

Stat5a r is 2

Found lacking in !Series_summary "GM-CSF controls the development of granulocytes but little is known about the contribution of the downstream mediating transcription factor STAT5A/B. To elucidate this pathway, we generated mice lacking the Stat5a and 5b genes in blood cells. Peripheral neutrophils were decreased and administration of 5-FU and GM-CSF failed to induce granulopoiesis in Stat5a/b-mutant mice. CMPs were isolated and cultured with GM-CSF. "

GSE14698_series_matrix.txtimp_info.txt

Stat5a r is 5

Found lacking in !Series_overall_design "Microarray Experiments for mice lacking or with the Stat5a and 5b genes in blood cells that were treated w/o CMP"

GSE14699_series_matrix.txtimp_info.txt

BH3-only r is 7

Found deletion in !Series_summary "Consistent with defective cytolysis, these cells exhibited deficiencies in granzyme up-regulation. Furthermore, they showed antigen-driven Bcl-2 down-regulation and early up-regulation of the pro-apoptotic protein Bim, consistent with the requirement of this BH3-only protein for peripheral T cell deletion. Bim up-regulation was paralleled by defective IL-7Ra chain re-expression, suggesting that Bim-dependent death may be triggered by loss of IL-7/IL-7R signaling. Finally, we observed parallels in molecular signatures between deletion and anergy suggesting that these tolerance pathways may not be as molecularly distinct as previously surmised."

GSE14800_series_matrix.txtimp_info.txt

Lasp1 r is 2

Found loss of in !Series_summary "Chronic loss of Lasp1 alters the expression of other genes associated with cell motility/attachment, and/or other cellular functions. Results provide new information showing that loss of Lasp1 leads to up- and down-regulation of genes involved in cell motility/attachment/growth."

GSE14800_series_matrix.txtimp_info.txt

Lasp1 r is 1

Found -/- in !Series_overall_design "Total RNA isolated from Lasp1-/- MEFs compared to Lasp1+/+ MEFs."

GSE14813_series_matrix.txtimp_info.txt

Lasp1 r is 1

Found null in !Series_summary "Comparative analysis of gene expression in murine gastric fundic mucosa in wild-type and Lasp1-null littermates. The data provide a comprehensive overview of genes expressed in the mouse gastric mucosa and show that the expression of several known and unidentified genes is modified by disruption of the Lasp1 gene."

GSE14829_series_matrix.txtimp_info.txt

H-ras r is 6

Found knockout in !Series_summary "The similarity of transcription profiles among serum-starved fibroblasts of all different WT and ras knockout genotypes tested, indicated that H-Ras and N-Ras do not play significant roles in control of transcriptional responses to serum deprivation stress. In contrast, genomic disruption of H-ras or N-ras, individually or in combination, determined highly specific, differential gene expression profiles in response to post-starvation stimulation with serum for 1 hour (G0/G1 transition) or for 8 hours (mid-G1 progression). The absence of N-Ras caused significantly higher changes than the absence of H-Ras on the wave of transcriptional activation linked to G0/G1 transition. In contrast, the absence of H-Ras affected more potently the profile of the transcriptional wave detected during mid-G1 progression. Functional analysis demonstrated a predominant functional association of H-Ras with growth and proliferation, whereas N-Ras exhibited a closer functional link to development or cell cycle regulation as well as immunomodulation and apoptosis. Mechanistic analysis indicated that ERK-dependent activation of Stat1 mediates the regulatory effect of N-Ras on defense and immunity, whereas the pro-apoptotic effects of N-Ras are mediated through ERK and p38 signaling. Our observations support previous reports of an absolute requirement for different peaks of Ras activity during the initial stages of the cell cycle and confirm the notion of functional specificity for the H-Ras and N-Ras isoforms."

GSE14891_series_matrix.txtimp_info.txt

Zc3h12a r is 2

Found -/- in !Series_title "Expression data of LPS-stimulated macrophages from wild-type and Zc3h12a-/- mice."

GSE14891_series_matrix.txtimp_info.txt

Zc3h12a r is 2

Found -/- in !Series_overall_design "Peritoneal macrophages from wild-type and Zc3h12a-/- mice were stimulated with LPS for 0, 1, 2 and 4 hours, followed by RNA extraction. Then hybridization on affymetrix microarrays was performed."

GSE14891_series_matrix.txtimp_info.txt

Zc3h12a r is 6

Found stimulated in !Series_title "Expression data of LPS-stimulated macrophages from wild-type and Zc3h12a-/- mice."

GSE14891_series_matrix.txtimp_info.txt

Zc3h12a r is 5

Found stimulated in !Series_overall_design "Peritoneal macrophages from wild-type and Zc3h12a-/- mice were stimulated with LPS for 0, 1, 2 and 4 hours, followed by RNA extraction. Then hybridization on affymetrix microarrays was performed."

GSE14898_series_matrix.txtimp_info.txt

Tie2 r is 4

Found expressing in !Series_summary "Methods: Beginning at 8 weeks of age, male Tie2-GFP mice (transgenically expressing green fluorescent protein exclusively within the endothelia) were fed a 60% fat calorie diet (Bio-Serv #S3282); age-matched mice were fed normal chow. After 4, 6, and 8 weeks on the diet, aortae and skeletal muscles (gastrocnemius, biceps femoris, and plantaris) were excised, minced, and collagenolytically digested. Each tissue digest was then subjected to FACS in order to obtain 10,000 endothelial cells. Transcriptomic analyses were performed with microarrays containing the Operon Murine V4 oligo set, and highly dysregulated genes were confirmed by real-time PCR. Results: By 4 weeks, Tie2-GFP mice receiving a high fat diet exhibited a fasting glucose of 215+17 mg/dL vs. 134+23 mg/dL in controls; by 6 weeks, a high fat diet resulted in lower glucose tolerance vs. control diet. Following 4, 6, and 8 weeks of high-fat regimen, aortic endothelial transcripts up-regulated by greater than 2-fold in biologically replicate experiments included macrophage inflammatory protein 2 (Mip2), chemokine (C-C motif) ligand 9 (CCL9), galectin-3 (Gal-3), and 5-lipoxygenase-activating protein (FLAP). Endothelial transcripts up-regulated in skeletal muscle included Mip2, CCL8 and 9, FLAP, gal-1 and 3, and ferritin light chain 1 (FTL1); transcripts down-regulated in muscle included endothelin-1 (ET-1) and insulin-like growth factor II (IGF II). Discussion: Gal-3 and FTL-1 are known to increase in response to advanced glycation end-products and oxidized LDL, respectively. However, the down-regulation of ET-1

and IGFII was surprising, as the transcription of these genes has previously been thought to exacerbate atherosclerosis. In conclusion, a comprehensive analysis of the endothelial transcript-level response to a dietary model of Type II diabetes has revealed novel regulation of transcripts with roles in inflammation, insulin sensitivity, oxidative stress, and atherosclerosis. Understanding the mechanism of diabetes-associated endothelial dysfunction may lead to improved therapies that lower the risk of cardiovascular complications in diabetic patients. "

GSE14906_series_matrix.txtimp_info.txt

FOG2 r is 1

Found null in !Series_overall_design "We have analyzed 6 RNA samples total (3 from control hearts, 2 from FOG2 null hearts and 1 from GATA4ki hearts)"

GSE14921_series_matrix.txtimp_info.txt

PPAR-alpha r is 5

Found deletion in !Series_summary "Hepatic metabolic derangements are key components in the development of fatty liver, insulin resistance, and atherosclerosis. SIRT1, a NAD+-dependent protein deacetylase, is an important regulator of energy homeostasis in response to nutrient availability. Here we demonstrate that hepatic SIRT1 regulates fatty acid metabolism by positively regulating PPAR-alpha. Hepatocyte-specific deletion of SIRT1 impairs PPAR-alpha signaling and decreased fatty acid beta-oxidation in the liver. When challenged with a high-fat diet, liver-specific SIRT1 knockout mice develop hepatic steatosis, hepatic inflammation, and endoplasmic reticulum stress. Taken together, our data indicate that SIRT1 plays a vital role in the regulation of hepatic lipid homeostasis."

GSE14969_series_matrix.txtimp_info.txt

TNF-a r is 1

Found induced in !Series_summary "Although natural antibodies (NAbs) are present from birth, little is known about what drives their selection, and whether they have housekeeping functions. We now show that the prototypic T15-NAb, first identified because of its protective role in infection, is representative of a previously unknown type of NAb response that specifically recognizes and forms complexes with apoptotic cells, and which promotes cell-corpse engulfment by phagocytes. This T15-NAb-mediated process is dependent on the recruitment of C1q and mannose-binding lectin (MBL), which have known immune modulatory activities that also provide “eat me” signals for phagocytic clearance. Further investigation revealed that, the addition of T15-NAb significantly suppressed in vitro macrophage LPS-induced TNF-alpha and IL-6 secretion, as well as in vitro Toll-like receptor (TLR)-induced dendritic cell maturation and secretion of pro-inflammatory cytokines and chemokines. Significantly, high doses of this B-1 cell produced NAb also inhibited in vivo TLR–induced pro-inflammatory responses, and could suppress autoimmune inflammatory arthritis. These studies identify and characterize a previously unknown regulatory circuit by which a NAb product of innate-like B cells aids homeostasis by control of fundamental inflammatory pathways."

GSE14969_series_matrix.txtimp_info.txt

TNF-alpha r is 1

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GSE14971_series_matrix.txtimp_info.txt

Cux2 r is 1

Found knock-out in !Series_title "Comparative gene expression profile of Cux2 knock-out cortex"

GSE14971_series_matrix.txtimp_info.txt

Cux2 r is 1

Found ko in !Series_summary "Gene expression profiles of Cux2 ko cortex was compared to the profile of WT cortex."

GSE14971_series_matrix.txtimp_info.txt

Cux2 r is 1

Found ko in !Series_overall_design "Cerebral cortex of WT and Cux2 ko animals were dissected and total RNA was obtained. Gene expression profiles were obtained for each sample and compared."

GSE14980_series_matrix.txtimp_info.txt

Gata1 r is 8

Found deficient in !Series_summary "G1ME cells are GATA1-deficient murine bipotential megakaryocyte/erythrocyte progenitor cells derived from Gata1-negative murine ES cells. In order to assess the impact of GATA1 on gene regulation and cell differentiation, an expression construct was used to transiently produce high levels of GATA1. Cells transduced with this construct or a vector

control were harvested at 18 and 42 hours, and gene expression was analyzed using Affymetrix MOE430 version 2 arrays."

GSE14984_series_matrix.txtimp_info.txt

P5 r is 2

Found deletion in !Series_overall_design "Comparison 1: wild type vs. PWS deletion in P5 non-starved mice;"

GSE14984_series_matrix.txtimp_info.txt

P5 r is 2

Found deletion in !Series_overall_design "Comparison 2: wild type vs. PWS deletion in P5 fasting mice;"

GSE14984_series_matrix.txtimp_info.txt

P5 r is 2

Found deletion in !Series_overall_design "Comparison 4: non-starved vs. fasting in P5 PWS deletion mice;"

GSE10421_series_matrix.txtimp_info.txt

Smad4 r is 3

Found deficient in !Series_summary "Results: Among 1419 transcripts significantly modulated by the dietary iron content, four were regulated similarly to the hepcidin genes Hamp1 and Hamp2. They are coding for Bmp6, the regulator of Bmp/Smad signal transduction Smad7, the negative regulator of basic helix-loop-helix (bHLH) proteins Id1, and a protein with a bHLH domain, Atoh8. The iron overload developed by Smad4 and Hamp1-deficient mice also increased Bmp6 transcription. Body iron stores influence Smad1/5/8 phosphorylation and, as shown by analysis of mice with liver-specific disruption of Smad4, the binding partner for the receptor-activated Smads is necessary for activation of Smad7, Id1, and Atoh8 transcription by iron."