GSE10029-GPL6328_series_matrix.txtimp_info.txt

PBE

Found treated in !Series_overall_design "The experiments addressed the effect of TNF exposure. 8-12-week-old NOD.BDC2.5 transgenic mice were injected i.p. with PBS or 3 ug murine TNFalpha; on alternate days for three weeks. At day 24 of treatment, suspensions of LNs and/or splenocytes were stimulated with the indicated concentration of the mimotope (1047-7 peptide (YVAPVWVRME)) at a density of 7-8 x 106 cells/ml, in a final volume of 1 ml in 24-well plates. After prolonged in vitro culture, cells were harvested 12 hrs post transplantation. CD4+ T cells were purified by labeling of monoclonal Abs of surface markers, Vbeta4-FITC and CD4-PE and enriched by Magnetic beads selection followed by FACS sorting. The purity of CD4+ T cells ranged from 95% to 99% depending on individual CD4 T cells preparation. The data in column represents six independent repeated experiments with half of the slides being reverse replicates. We compared RNA from PBE treated group with TNF treated group without including reference RNA."

GSE10029-GPL6329_series_matrix.txtimp_info.txt

PBE

Found treated in !Series_overall_design "The experiments addressed the effect of TNF exposure. 8-12-week-old NOD.BDC2.5 transgenic mice were injected i.p. with PBS or 3 ug murine TNFalpha; on alternate days for three weeks. At day 24 of treatment, suspensions of LNs and/or splenocytes were stimulated with the indicated concentration of the mimotope (1047-7 peptide (YVAPVWVRME)) at a density of 7-8 x 106 cells/ml, in a final volume of 1 ml in 24-well plates. After prolonged in vitro culture, cells were harvested 12 hrs post transplantation. CD4+ T cells were purified by labeling of monoclonal Abs of surface markers, Vbeta4-FITC and CD4-PE and enriched by Magnetic beads selection followed by FACS sorting. The purity of CD4+ T cells ranged from 95% to 99% depending on individual CD4 T cells preparation. The data in column represents six independent repeated experiments with half of the slides being reverse replicates. We compared RNA from PBE treated group with TNF treated group without including reference RNA."

GSE10029-GPL6330_series_matrix.txtimp_info.txt

PBE

Found treated in !Series_overall_design "The experiments addressed the effect of TNF exposure. 8-12-week-old NOD.BDC2.5 transgenic mice were injected i.p. with PBS or 3 ug murine TNFalpha; on alternate days for three weeks. At day 24 of treatment, suspensions of LNs and/or splenocytes were stimulated with the indicated concentration of the mimotope (1047-7 peptide (YVAPVWVRME)) at a density of 7-8 x 106 cells/ml, in a final volume of 1 ml in 24-well plates. After prolonged in vitro culture, cells were harvested 12 hrs post transplantation. CD4+ T cells were purified by labeling of monoclonal Abs of surface markers, Vbeta4-FITC and CD4-PE and enriched by Magnetic beads selection followed by FACS sorting. The purity of CD4+ T cells ranged from 95% to 99% depending on individual CD4 T cells

preparation. The data in column represents six independent repeated experiments with half of the slides being reverse replicates. We compared RNA from PBE treated group with TNF treated group without including reference RNA."

GSE10067_series_matrix.txtimp_info.txt

lipase

Found knockout in !Series_title "Gene expression data from murine liver samples comparing hormone sensitive lipase (HSL) knockout mice vs. wildtype mice"

GSE10067_series_matrix.txtimp_info.txt

HSL

Found knockout in !Series_summary "HSL is a key enzyme in in the mobilization of fatty acids from the triglyceride stores of white adipose tissue. In addition, it is expressed in mice liver. In the present microarray study, changes in the transcript profile of murine liver samples due to global HSL knockout were investigated."

GSE10067_series_matrix.txtimp_info.txt

HSL

Found knockout in !Series_overall_design "Genetic modification to analyze the impact of a general knockout of the HSL gene on liver metabolism."

GSE10168_series_matrix.txtimp_info.txt

ET

Found activation in !Series_title "AhR activation by TCDD in the ET, cortical epithelial cell line"

GSE10200_series_matrix.txtimp_info.txt

MYC

Found inactivation in !Series_summary "Keywords: Dose response - MYC inactivation by doxycycline treatment"

GSE10218_series_matrix.txtimp_info.txt

Fos

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

GSE10235_series_matrix.txtimp_info.txt

NF

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

GSE10291_series_matrix.txtimp_info.txt

Rho

Found treated in !Series_summary "To gain insights into the molecular mechanisms of oligodendrocyte differentiation, we performed microarray expression profiling of the oligodendroglial cell line, Oli-neu, treated with the Rho kinase (ROCK) inhibitor, Y-27632 or with conditioned neuronal medium. This resulted in the identification of the transmembrane protein 10 (Tmem10/Opalin), a novel type I transmembrane protein enriched in differentiating oligodendrocytes. In primary cultures, Tmem10 was abundantly expressed in O4-positive oligodendrocytes, but not in oligodendroglial precursor cells, astrocytes, microglia or neurons. In mature oligodendrocytes Tmem10 was enriched in the rims and processes of the cells and was only found to a lesser extent in the membrane sheets."

GSE10312_series_matrix.txtimp_info.txt

Pb

Found exposure in !Series_summary "This study focuses on the impact of developmental Pb exposure on splenic gene expression. Pb exposure from gestational day 8 (gd8) to post-natal day 21 (pnd21) was used to obtain insight into how Pb induces anemia and negatively influences the immune response"

GSE10341_series_matrix.txtimp_info.txt

Th

Found deletion in !Series_summary "The catecholamine norepinephrine is required for fetal survival, but its essential function is unknown. When catecholamine-deficient [tyrosine hydroxylase (Th) null] mouse fetuses die at E13.5-E14.5, they resemble wild type fetuses exposed to hypoxia. They exhibit bradycardia (28% reduction in heart rate), thin ventricular myocardium (20% reduction in tissue), epicardial detachment, and death with vascular congestion, hemorrhage and edema. At E12.5, prior to the appearance of morphological deficits associated with Th deletion, catecholamine-deficient fetuses are preferentially killed by experimentally-induced hypoxia and have lower tissue pO2 than wild type siblings. Catecholamine-deficient fetuses also induced HIF-1 target genes to a greater extent than wild type siblings, supporting the notion that null fetuses experience greater hypoxia or have an enhanced response to hypoxia. Hypoxia induces a 13-fold increase in plasma norepinephrine levels, which would be expected to increase heart rate, thereby, improving oxygen delivery in wild type mice. Surprisingly, increasing maternal oxygen (FiO2 33% or 63%) prevents the effects of catecholamine-deficiency, restoring heart rate, myocardial mass and survival of Th null fetuses. We suggest that norepinephrine mediates fetal survival by maintaining oxygen homeostasis as vulnerability to constitutive hypoxia increases as fetal growth accelerates during normal development."

GSE10421_series_matrix.txtimp_info.txt

hepcidin

Found induced in !Series_summary "Background & Aims: Although hepcidin expression was shown to be induced by the BMP signaling pathway, it is not yet known how iron regulates hepcidin and which of the BMP molecules is the endogenous regulator of iron homeostasis in vivo. We therefore assessed liver transcription profiles of mice fed an iron-deficient or an iron-enriched diet and looked for genes that were regulated similarly to hepcidin in that context."

GSE10422_series_matrix.txtimp_info.txt

tg

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

GSE10478_series_matrix.txtimp_info.txt

AAT

Found treated in !Series_summary "In this study, we performed the gene expression analysis of the Normal, Diabetic and AAT treated NOD mice to elucidate the transcriptional changes induced by AAT. This will assist in identifying the biological processes / pathways involved in curative mechanism of AAT."

GSE10478_series_matrix.txtimp_info.txt

AAT

Found treated in !Series_overall_design "Duplicate samples of Normal, Diabetic and AAT treated NOD mice were analyzed."

GSE10498-GPL339_series_matrix.txtimp_info.txt

Bax

Found null in !Series_title "Comparison of SCG expression profiles from Bax null versus NGF-Bax double null mice"

GSE10498-GPL339_series_matrix.txtimp_info.txt

SCG

Found null in !Series_title "Comparison of SCG expression profiles from Bax null versus NGF-Bax double null mice"

GSE10498-GPL339_series_matrix.txtimp_info.txt

fro

Found null in !Series_overall_design "This experiment examine gene expression differences in superior cervical ganglia fro P0 bax null versus NGF-Bax double null animals. The Bax genotype was used in order to prevent the neuronal cell death normally observed in the NGF null animal."

GSE10498-GPL340_series_matrix.txtimp_info.txt

Bax

Found null in !Series_title "Comparison of SCG expression profiles from Bax null versus NGF-Bax double null mice"

GSE10498-GPL340_series_matrix.txtimp_info.txt

SCG

Found null in !Series_title "Comparison of SCG expression profiles from Bax null versus NGF-Bax double null mice"

GSE10498-GPL340_series_matrix.txtimp_info.txt

fro

Found null in !Series_overall_design "This experiment examine gene expression differences in superior cervical ganglia fro P0 bax null versus NGF-Bax double null animals. The Bax genotype was used in order to prevent the neuronal cell death normally observed in the NGF null animal."

GSE10574_series_matrix.txtimp_info.txt

Eed

Found KO in !Series_overall_design "We generated constitutive Eed KO mouse ES cells and observed gene expression using Affymetrix MOE430.2 microarray."

GSE10587_series_matrix.txtimp_info.txt

pendrin

Found knockout in !Series_summary "Determination of differential expression of genes in the stria vascularis of pendrin (Slc26a4) heterozygous and knockout mice before the onset of hearing at postnatal day 10 (P10)."

GSE10587_series_matrix.txtimp_info.txt

pendrin

Found knockout in !Series_overall_design "A total of Six samples of stria vascularis RNA obtained from P10 mice were analyzed. Triplicates from pendrin (Slc26a4) heterozygous and knockout mice were run and analyzed."

GSE10589_series_matrix.txtimp_info.txt

pendrin

Found knockout in !Series_summary "Determination of differential expression of genes in the thyroid of pendrin (Slc26a4) heterozygous and knockout mice at a time point corresponding to maximal thyroid gland activity, postnatal day 15 (P15). "

GSE10589_series_matrix.txtimp_info.txt

pendrin

Found knockout in !Series_overall_design "A total of Six samples of thyroid RNA obtained from P15 mice were analyzed. Triplicates from pendrin (Slc26a4) heterozygous and knockout mice were run and analyzed."

GSE10659_series_matrix.txtimp_info.txt

cubilin

Found knockout in !Series_summary "Keywords: reduced folate carrier knockout, folate receptor, cubilin, megalin, embryos, gene expression, neural tube defect, chorioallantoic fusion"

GSE10659_series_matrix.txtimp_info.txt

RFC

Found KO in !Series_title "AFFYMETRIX ANALYSIS OF E9.5 RFC MOUSE KO EMBRYOS REVEALS ALTERED EXPR'N OF GENES IN THE CUBILIN-MEGALIN COMPLEX"

GSE10659_series_matrix.txtimp_info.txt

RFC

Found KO in !Series_summary "Comparison of RFC KO, wildtype normal embryos vs. RFC KO, nullizygous affected embryos"

GSE10659_series_matrix.txtimp_info.txt

RFC

Found KO in !Series_overall_design "6 samples RFC KO mouse embryos, E9.5, folic acid treated: 3 Control, Wildtype, normal; 3 Affected, Nullizygous, CR/chorioallantoic defect; as paired-littermates with

one normal and one affected embryo per set from each of three separate litters for RNA extraction and hybridization on Affymetrix microarrays."

GSE10684_series_matrix.txtimp_info.txt

PXR

Found activation in !Series_summary "Damage of the intestinal epithelial barrier by xenobiotics or reactive oxygen species and a dysregulated immune response are both factors involved in the pathogenesis of inflammatory bowel diseases (IBD). Curcumin and rutin are polyphenolic compounds known to have anti-oxidant and anti-inflammatory activities, but their mechanism(s) of action are yet to be fully elucidated. Mdr1a-/- mice spontaneously develop intestinal inflammation, predominantly in the colon, with pathology similar to IBD, so this mouse model is relevant for studying diet-gene interactions and potential effects of foods on remission or development of IBD. This study tested whether the addition of curcumin or rutin to the diet would alleviate colonic inflammation in mdr1a-/- mice. Using whole-genome microarrays, the effect of dietary curcumin on gene expression in colon tissue was also investigated. Twelve mice were randomly assigned to each of three diets; control (AIN-76A), control + 0.2% curcumin or control + 0.1% rutin and monitored from the age of 7 to 24 weeks. Curcumin, but not rutin, significantly reduced histological signs of colonic inflammation in mdr1a-/- mice. Microarray and pathway analyses suggested that the effect of dietary curcumin on colon inflammation could be via an up-regulation of xenobiotic metabolism and a down-regulation of pro-inflammatory pathways probably mediated by PXR and PPARalpha activation of RXR. These results reveal the potential of global gene expression and pathway analyses to study and better understand the effect of foods in colonic inflammation."

GSE10684_series_matrix.txtimp_info.txt

PPARalpha

Found activation in !Series_summary "Damage of the intestinal epithelial barrier by xenobiotics or reactive oxygen species and a dysregulated immune response are both factors involved in the pathogenesis of inflammatory bowel diseases (IBD). Curcumin and rutin are polyphenolic compounds known to have anti-oxidant and anti-inflammatory activities, but their mechanism(s) of action are yet to be fully elucidated. Mdr1a-/- mice spontaneously develop intestinal inflammation, predominantly in the colon, with pathology similar to IBD, so this mouse model is relevant for studying diet-gene interactions and potential effects of foods on remission or development of IBD. This study tested whether the addition of curcumin or rutin to the diet would alleviate colonic inflammation in mdr1a-/- mice. Using whole-genome microarrays, the effect of dietary curcumin on gene expression in colon tissue was also investigated. Twelve mice were randomly assigned to each of three diets; control (AIN-76A), control + 0.2% curcumin or control + 0.1% rutin and monitored from the age of 7 to 24 weeks. Curcumin, but not rutin, significantly reduced histological signs of colonic inflammation in mdr1a-/- mice. Microarray and pathway analyses suggested that the effect of dietary curcumin on colon inflammation could be via an up-regulation of xenobiotic metabolism and a down-regulation of pro-inflammatory pathways probably mediated by PXR and PPARalpha activation of RXR. These results reveal the potential of global gene

expression and pathway analyses to study and better understand the effect of foods in colonic inflammation."

GSE10765_series_matrix.txtimp_info.txt

IRAK

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

GSE10807_series_matrix.txtimp_info.txt

Atm

Found deficient in !Series_title "Expression profiling of Atm deficient murine thymic lymphoma"

GSE10807_series_matrix.txtimp_info.txt

Atm

Found deficient in !Series_summary "To find genes deregulated in the pathogenisis of T-cells in Atm deficient mice, we performed expression profiling of Atm deficient thymic lymphomas, wildtype thymi and Atm deficient thymi without macroscopic enlargement, representing an intermediate stage in the process of tumorigenisis."

GSE10807_series_matrix.txtimp_info.txt

Atm

Found deficient in !Series_overall_design "To evaluate differential gene expression of Atm deficient thymic lymphoma we performed microarray based expression profiling of 8 Atm deficient thymic lymphomas, 6 wildtype thymi, 1 thymus of an Atm heterozygous mouse and 18 thymi of Atm deficient mice without macroscopic enlargement."

GSE10817_series_matrix.txtimp_info.txt

Kras

Found induced in !Series_summary "MLL5 is a novel trithorax group gene and a candidate tumor suppressor gene located within a 2.5 Mb interval of chromosome band 7q22 that is frequently deleted in human myeloid malignancy. Here we show that Mll5 is required for normal hematopoietic stem cell (HSC) homeostasis. Inactivation of the Mll5 gene in mice results in reduced cellularity of the long-term HSC compartment, which correlates with functional impairment of long-term repopulation potential under competitive conditions. Bone marrow cells from Mll5-deficient mice were defective in spleen colony-forming assays, and the mutant mice showed enhanced susceptibility to 5-Fluorouracil-induced myelosuppression. Heterozygous and homozygous Mll5 mutant mice did not spontaneously develop hematologic cancers, and loss of Mll5 did not alter the phenotype of a fatal myeloprolferative disorder

induced by oncogenic Kras in vivo. Collectively, the data reveal an important role for Mll5 in HSC homeostasis, and provide a basis for further studies to explore its role in leukemogenesis."

GSE10869_series_matrix.txtimp_info.txt

CaMKIV

Found induced in !Series_title "Effects of CaMKIV loss on cocaine-induced gene expression in the striatum"

GSE10869_series_matrix.txtimp_info.txt

CaMKIV

Found induced in !Series_summary "Our goal was to analyze how loss of CaMKIV will affect acitivity-regulated transcription induced by strong stimulation, i.e. cocaine."

GSE10902_series_matrix.txtimp_info.txt

cyclin

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."

GSE10915_series_matrix.txtimp_info.txt

leptin

Found treated in !Series_title "Comparative analysis of gene expression in ob/ob leptin-treated and ob/ob saline-treated lungs."

GSE11098-GPL1261_series_matrix.txtimp_info.txt

Fah

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

Fah

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)."

GSE11139_series_matrix.txtimp_info.txt

huntingtin

Found null in !Series_title "Elucidating a normal function of huntingtin by analysis of huntingtin-null mouse embryonic fibroblasts"

GSE11141_series_matrix.txtimp_info.txt

NgR

Found inhibition in !Series_summary "The lack of axonal growth after injury in the adult central nervous system (CNS) is partly due to the presence of growth-inhibitory molecules associated with myelin and the intrinsic growth-state of the injured neurons. To date, three inhibitors have been identified in myelin: Myelin- Associated Glycoprotein (MAG), Nogo A, and Oligodendrocyte-Myelin glycoprotein (OMgp). These three proteins all appear to be located in the periaxonal surface of the myelin membrane placing them in an optimal location to mediate axon-glial interaction. In addition, the three proteins have been shown to bind the same neuronal receptor, known as the Nogo-66 receptor (NgR). It has been hypothesized that inhibition of NgR may be a strategy to increase regeneration, plasticity and functional recovery of the lesioned central nervous system. Strong NgR mRNA expression is observed in the hippocampal pyramidal cell layers (CA1-3) and the granular layer of the dentate gyrus. It has been shown that animals exposed to entorhinal lesions show a biphasic regulation of NgR in the hippocampus, suggesting a tightly regulated mechanism mediated by this receptor. We have access to a transgenic model to over-express NgR in forebrain hippocampal neurons. Preliminary results have shown a phenotypic response in behavior and some molecular markers, as result of NgR overexpression. Knowledge of what genes are reacting in this novel transgenic model may provide insights into what pathways are affected by NgR to control synaptic plasticity in normal animals and during injury."

GSE11141_series_matrix.txtimp_info.txt

NgR

Found overexpression in !Series_title "Effects of NgR overexpression on the developing and mature forebrain (backm-affy-mouse-433094)"

GSE11141_series_matrix.txtimp_info.txt

NgR

Found overexpression in !Series_summary "- Effects of NgR overexpression in the mature forebrain -"

GSE11141_series_matrix.txtimp_info.txt

NgR

Found overexpression in !Series_summary "- Effects of NgR overexpression in the developing forebrain -"

GSE11141_series_matrix.txtimp_info.txt

NgR

Found overexpression in !Series_summary "We will also analyze the effects of NgR overexpression in bitransgenic animals raised on water. In these animals, the NgR transgene is expressed in the forebrain upon activation of the CamKII promoter. Since neuronal differentiation and neuronal pathways are being formed during development, we anticipate that NgR overexpression (de)effects in these animals will be markedly different to those observed in adult animals."

GSE11149_series_matrix.txtimp_info.txt

NCC

Found deficient in !Series_summary "FAK is a tyrosine kinase that transduces integrin and growth factor signaling pathways. Abnormal growth factor signaling leads to cardiovascular malformations caused by deficient cardiac NCC function. However, a direct role for FAK in NCC morphogenesis has not been demonstrated. We will test the hypothesis that FAK is a downstream target of essential growth factor signaling in cranial neural crest cells during development."

GSE11149_series_matrix.txtimp_info.txt

FAK

Found knockout in !Series_summary "1) Generate E11.5 mouse embryos that are either control (FAK+/flox) or NCC specific FAK knockout (Wnt1Cre;FAKflox/flox). 2) Determine genotype by PCR. 3) Dissect head and torax from the different genotypes. 4) Obtain NCC from dissected tissue by magnetic cell sorting using p75 antibody. 5) Prepare total RNA from speciments. We will pool the NCC from 3 different embryos of the same genotype, and send total RNA to TGEN for probe preparation, hybridization and array result analysis."

GSE11149_series_matrix.txtimp_info.txt

NCC

Found knockout in !Series_summary "1) Generate E11.5 mouse embryos that are either control (FAK+/flox) or NCC specific FAK knockout (Wnt1Cre;FAKflox/flox). 2) Determine genotype by PCR. 3) Dissect head and torax from the different genotypes. 4) Obtain NCC from dissected tissue by magnetic cell sorting using p75 antibody. 5) Prepare total RNA from speciments. We will pool the NCC from 3 different embryos of the same genotype, and send total RNA to TGEN for probe preparation, hybridization and array result analysis."

GSE11252_series_matrix.txtimp_info.txt

Aag

Found null in !Series_summary "We are investigating the transcriptional response of mice that are wt or null for Aag in response to alkylating agent MMS"

GSE11252_series_matrix.txtimp_info.txt

Aag

Found treated in !Series_overall_design "Mice (WT and Aag null) were treated with MMS and livers extracted at 6, 12, 24, and 48 hours"

GSE11253_series_matrix.txtimp_info.txt

Rb

Found deficient in !Series_summary "Keywords: unpaired WT versus Rb family deficient KLS"

GSE11253_series_matrix.txtimp_info.txt

Rb

Found induced in !Series_overall_design "We used mice that harbor floxed alleles for the Rb family and we induced the recombination by 6 weeks of age. KLS (c-Kit+ Lin - Sca-1+) cells were sorted 2 months after the recombination."

GSE11346_series_matrix.txtimp_info.txt

Apoe

Found knockout in !Series_overall_design "12-13 weeks old Apoe knockout mice (C57BL/6-Apoe tm1) mice were injected intraperitoneally with varying doses of SRM 2975. In a parallel experiment C57BL mice were exposed by inhalation for 90 minutes to four consecutive doses of 20mg/m3 of filtered air, NIST 2975 or Ptintex 90. Livers and aortic tissue were collected."

GSE11585_series_matrix.txtimp_info.txt

ERbeta

Found null in !Series_title "Granulosa cell gene expression in gonadotropin-treated ERbeta-het and ERbeta-null mice"

GSE11585_series_matrix.txtimp_info.txt

het

Found null in !Series_title "Granulosa cell gene expression in gonadotropin-treated ERbeta-het and ERbeta-null mice"

GSE11585_series_matrix.txtimp_info.txt

ERbeta

Found treated in !Series_title "Granulosa cell gene expression in gonadotropin-treated ERbeta-het and ERbeta-null mice"

GSE11585_series_matrix.txtimp_info.txt

het

Found treated in !Series_title "Granulosa cell gene expression in gonadotropin-treated ERbeta-het and ERbeta-null mice"

GSE11628_series_matrix.txtimp_info.txt

LIF

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)."

GSE11662_series_matrix.txtimp_info.txt

Akt

Found induced in !Series_title "GADD45a in ventilator-induced lung injury: role of Akt signaling"

GSE11684_series_matrix.txtimp_info.txt

Perk

Found knockout in !Series_title "Expression data from Perk wild-type and knockout mouse liver perfused without or with 2,5-di-tert-butylhydroquinone"

GSE11803_series_matrix.txtimp_info.txt

PPARdelta

Found treated in !Series_summary "We measured global skeletal muscle expression in sedentary and exercised mice treated with vehicle or PPARdelta ligand GW1516. PPARdelta is a transcriptional regulator of muscle oxidative metabolism and fatigue resistance."

GSE11804_series_matrix.txtimp_info.txt

PPARdelta

Found treated in !Series_summary "We have conducted skeletal muscle microarrays from mice treated with AMPK agoinst (AICAR), PPARdelta agonist (GW1516) or the combination of the two drugs to investigate the individual and interactive effects of the two on muscle genes."

GSE11836_series_matrix.txtimp_info.txt

Pten

Found mutant in !Series_summary "In our investigations of the molecular pathways of prostate tumorigenesis in Nkx3.1; Pten mutant mice using gene expression profiling, we now find that the AP-1 transcription factors, c-Jun and c-Fos, are significantly up-regulated during cancer progression. Forced expression of c-Fos and c-Jun in prostate cancer cells results in increased tumorigenicity, activation of Erk MAP kinase, and enhanced survival in the absence of androgens, which are hallmarks of disease progression. In humans, Jun and Fos proteins are significantly up-regulated during prostate cancer progression and significantly correlated with activation of Erk MAP kinase. Most notably, expression of Jun is associated with disease recurrence independent of other currently used prognostic indicators."

GSE11836_series_matrix.txtimp_info.txt

Pten

Found mutant in !Series_overall_design "Mouse prostate was collected from wild-type or the Nkx3.1; Pten compound mutant mice at the age of 8-16 months. One lobe of dosolateral prostate was snap-frozen in OCT and stored at -80ºC for laser capture microdissection (LCM). To obtain androgen-independent lesions, mice were castrated at 7 to 14 months of age. Mice were sacrificed for analysis at 8 to 16 months of age and one dosolateral prostatic lobe was snap-frozen in OCT and stored at -80ºC for LCM. Approximate 1000 Prostate epithelial cells were isolated from normal prostate, dysplasia, prostatic intraepithelial neoplasia (PIN) or cancer lesions using PixCell IIE LCM system (Arcturus), followed by RNA linear amplification and labeling using Small Sample Labeling Protocol VII (Affymetrix). Samples were labeled using a BioArray High Yield RNA transcript labeling kit (Enzo Life Scientific) and were hybridized to MOE430A GeneChips containing 22,690 well characterized mouse genes/ESTs (Affymetrix)."

GSE11836_series_matrix.txtimp_info.txt

Erk

Found activation in !Series_summary "In our investigations of the molecular pathways of prostate tumorigenesis in Nkx3.1; Pten mutant mice using gene expression profiling, we now find that the AP-1 transcription factors, c-Jun and c-Fos, are significantly up-regulated during cancer progression. Forced expression of c-Fos and c-Jun in prostate cancer cells results in increased tumorigenicity, activation of Erk MAP kinase, and enhanced survival in the absence of androgens, which are hallmarks of disease progression. In humans, Jun and Fos proteins are significantly up-regulated during prostate cancer progression and significantly correlated with activation of Erk MAP kinase. Most notably, expression of Jun is associated with disease recurrence independent of other currently used prognostic indicators."

GSE11859_series_matrix.txtimp_info.txt

Gfap

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."

GSE11884_series_matrix.txtimp_info.txt

furin

Found deletion in !Series_summary "Furin is a proprotein convertase induced in activated T cells, reported to processes the anti-inflammatory cytokine TGFb-1. Herein, we show that conditional deletion of furin in T cells allowed for normal T cell development but impaired the function of regulatory T cells and effector cells, which produced less TGFb-1. Furin-deficient Treg cells, were less protective in a T cell transfer colitis model and failed to induce Foxp3 in normal T cells. Furin-deficient effector cells were inherently overly active and were resistant to suppressive activity of wild-type Tregs. Thus, our results indicate that furin is indispensable in maintaining peripheral tolerance, which is due, at least in part, to its nonredundant, essential function in regulating TGFb-1 production. Targeting furin has emerged as a strategy in malignant and infectious disease. The current work suggests that inhibiting furin might activate immune responses, but may result in a breakdown in peripheral tolerance. "

GSE12028_series_matrix.txtimp_info.txt

Ppara

Found activation 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."

GSE12073_series_matrix.txtimp_info.txt

Aire

Found expressing in !Series_title "Expression data from transgenic Aire expressing pancreatic islets"

GSE12209_series_matrix.txtimp_info.txt

Cartpt

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."

GSE12394_series_matrix.txtimp_info.txt

MYC

Found treated in !Series_summary "miRNA expression profiling of murine MYC-dependent lymphoma cell lines harboring the MYC-transgene in a Tet-off system comparing control untreated lymphoma cells (high MYC expression state) with 18hours Dox treated lymphoma cells (low MYC expression state)."

GSE12419-GPL2884_series_matrix.txtimp_info.txt

Arf

Found deficient in !Series_summary "The human CDKN2A locus encodes two distinct proteins, p16(INK4A) and p14(ARF) [mouse p19(Arf)], designated INK4A and ARF herein, that are translated from alternatively spliced mRNAs. Human ARF is implicated as a tumor suppressor gene, mainly in association with the simultaneous deletion of INK4A. However, questions remain as to whether loss of ARF alone is sufficient to drive tumorigenesis. Here we report that mice deficient for Arf are susceptible to accelerated asbestos-induced malignant mesothelioma (MM). MMs arising in Arf (+/-) mice consistently exhibit biallelic inactivation of Arf, but unexpectedly, do not acquire additional recurrent genetic alterations that we previously identified in asbestos-induced MMs arising in Nf2 (+/-) mice. Array CGH analysis was used to detect a recurrent deletion at chromosome 4C6. A candidate in this region, Faf1 (FAS-associated factor 1), was further explored because it encodes a protein implicated in tumor cell survival and in the pathogenesis of some human tumor types. We confirmed hemizygous loss of Faf1 and down regulation of Faf1 protein in a series of MMs from Arf (+/-) mice, and then showed that Faf1 regulates TNFalpha-mediated NFkappaB signaling, a mechanism previously implicated in asbestos-induced oncogenesis of human mesothelial cells. Collectively, these investigations divulge important information regarding the significance of Arf inactivation in MM pathogenesis, and implicate Faf1 as a key component in the TNFalpha/NFkappaB signaling node that has now been independently implicated in the asbestos-induced oncogenesis."

GSE12419-GPL4092_series_matrix.txtimp_info.txt

Arf

Found deficient in !Series_summary "The human CDKN2A locus encodes two distinct proteins, p16(INK4A) and p14(ARF) [mouse p19(Arf)], designated INK4A and ARF herein, that are translated from alternatively spliced mRNAs. Human ARF is implicated as a tumor suppressor gene, mainly in association with the simultaneous deletion of INK4A. However, questions remain as to whether loss of ARF alone is sufficient to drive tumorigenesis. Here we report that mice deficient for Arf are susceptible to accelerated asbestos-induced malignant mesothelioma (MM). MMs arising in Arf (+/-) mice consistently exhibit biallelic inactivation of Arf, but unexpectedly, do not acquire additional recurrent genetic alterations that we previously identified in asbestos-induced MMs arising in Nf2 (+/-) mice. Array CGH analysis was used to detect a recurrent deletion at chromosome 4C6. A candidate in this region, Faf1

(FAS-associated factor 1), was further explored because it encodes a protein implicated in tumor cell survival and in the pathogenesis of some human tumor types. We confirmed hemizygous loss of Faf1 and down regulation of Faf1 protein in a series of MMs from Arf (+/-) mice, and then showed that Faf1 regulates TNFalpha-mediated NFkappaB signaling, a mechanism previously implicated in asbestos-induced oncogenesis of human mesothelial cells. Collectively, these investigations divulge important information regarding the significance of Arf inactivation in MM pathogenesis, and implicate Faf1 as a key component in the TNFalpha/NFkappaB signaling node that has now been independently implicated in the asbestos-induced oncogenesis."

GSE12421_series_matrix.txtimp_info.txt

OBF

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

GSE12464_series_matrix.txtimp_info.txt

Itk

Found knockout in !Series_overall_design "CD3+ T-cells from pooled suspensions of spleen and lymph nodes of Wt and Itk knockout mice on C57BL/6 background were isolated after negative depletion. Unstimulated as well as stimulated T-cells were studied. Stimulations were done with anti-CD3 (1 mg/ml) with or without anti-CD28 (3 mg/ml) in the presence or absence of CsA (1 mg/ml) for 24 hrs. For each stimulus, at least duplicate samples were used."

GSE12465_series_matrix.txtimp_info.txt

Itk

Found knockout in !Series_overall_design "CD3+ CD4+ and CD8+ T-cells from pooled suspensions of spleen and lymph nodes of Wt and Itk knockout mice on C57BL/6 background were isolated after negative depletion. Unstimulated as well as stimulated T-cells were studied. Stimulations were done with anti-CD3 (1 mg/ml) for 24 hrs. For the CD4+ T-cells we collected triplicates from the Itk knockout mice and duplicates from the Wt group. For the CD8+ T-cells, we got duplicates from Itk knockout , while we obtained a single sample from Wt owing to the low cell yield for resting Wt CD8+ T-cells. After CD3-stimulation we got a single sample from the CD8+ subset of both Wt and Itk knockout, while for the CD4+ subsets we collected duplicates."

GSE12466_series_matrix.txtimp_info.txt

Itk

Found deficient in !Series_title "Transcriptional signatures of Itk-deficient CD3+, CD4+ and CD8+ T-cells"

GSE12467_series_matrix.txtimp_info.txt

LT

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

GSE12495_series_matrix.txtimp_info.txt

Trsp

Found knockout in !Series_title "Wild type mice (Trsp) vs Trsp-knockout mice (DTrsp) or A34 or G37L or G37H transgenic mice"

GSE12495_series_matrix.txtimp_info.txt

Trsp

Found mutant in !Series_summary "Sec (selenocysteine) is biosynthesized on its tRNA and incorporated into selenium-containing proteins (selenoproteins) as the 21st amino acid residue. Selenoprotein synthesis is dependent on Sec tRNA and the expression of this class of proteins can be modulated by altering Sec tRNA expression. The gene encoding Sec tRNA (Trsp) is a single-copy gene and its targeted removal in liver demonstrated that selenoproteins are essential for proper function wherein their absence leads to necrosis and hepatocellular degeneration. In the present study, we found that the complete loss of selenoproteins in liver was compensated for by an enhanced expression of several phase II response genes and their corresponding gene products. The replacement of selenoprotein synthesis in mice carrying mutant Trsp transgenes, wherein housekeeping, but not stress-related selenoproteins are expressed, led to normal expression of phase II response genes. Thus the present study provides evidence for a functional link between housekeeping selenoproteins and phase II enzymes."

GSE12495_series_matrix.txtimp_info.txt

selenoprotein

Found mutant in !Series_summary "Sec (selenocysteine) is biosynthesized on its tRNA and incorporated into selenium-containing proteins (selenoproteins) as the 21st amino acid residue. Selenoprotein synthesis is dependent on Sec tRNA and the expression of this class of proteins can be modulated by altering Sec tRNA expression. The gene encoding Sec tRNA (Trsp) is a single-copy gene and its targeted removal in liver demonstrated that selenoproteins are essential for proper function wherein their absence leads to necrosis and hepatocellular degeneration. In the present study, we found that the complete loss of selenoproteins in liver was compensated for by an enhanced expression of several phase II response genes and their corresponding gene products. The replacement of selenoprotein synthesis in mice carrying mutant Trsp transgenes, wherein housekeeping, but not stress-related selenoproteins are expressed, led to normal expression of phase II response genes. Thus the present study provides evidence for a functional link between housekeeping selenoproteins and phase II enzymes."

GSE12506_series_matrix.txtimp_info.txt

TR

Found induced in !Series_summary "The concept of immune regulation/suppression has been well-established. With thymus-derived CD4 CD25 regulatory T (TR) cells, it became clear that a variety of additional peripherally induced TR cells play vital roles in protection from many harmful immune responses including intestinal inflammation. In the present study, we have analyzed in vivo-induced Ag-specific CD4 TR cells with respect to their molecular and functional phenotype. By comparative genomics we could show that these Ag-specific TR cells induced by chronic Ag stimulation in vivo clearly differ in their genetic program from naturally occurring thymus-derived CD4 CD25 TR cells. This distinct population of induced TR cells express neither CD25 nor the TR-associated transcription factor Foxp3. Strikingly, CD25 is not even up-regulated upon stimulation. Despite the lack in Foxp3 expression, these in vivo-induced CD25 TR cells are able to interfere with an Ag-specific CD8 T cell-mediated intestinal inflammation without significant increase in CD25 and Foxp3 expression. Thus, our results demonstrate that in vivo-induced Ag-specific TR cells represent a distinct population of Foxp3 CD25 TR cells with regulatory capacity both in vitro and in vivo."

GSE12538_series_matrix.txtimp_info.txt

LT

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

GSE12576_series_matrix.txtimp_info.txt

PrP

Found expressing in !Series_overall_design "Wild type (WT), PrP-null (KO), and Tg(HQK) mice were fed food pellets either lacking or containing 6g doxycycline (Dox)/kg food to induce PrPC expression. Skeletal muscles from the quadriceps of hind legs were removed at day 0, 4, 7, 14, 30 and 60 days following administration of Dox. Total RNA was isolated from these tissues for use in subsequent microarray analysis. Mouse gene expression was analysed by two-colour microarray experiments using an inhouse manufactured 16K mouse cDNA microarray. Age matched reference mice (WT) and experimental (KO and HQK) Alexa Flour labeled aRNA were used in each competitive hybridization. Each sample was labeled individually with both Alexa Fluor 555 and 647 for subsequent dye-swapped hybridizations to account for intensity bias. 3 individual mice from each experimental group at each time point were individually processed for separate microarrays. We used the program EDGE to identify genes that were differentially expressed in mouse skeletal muscle in either transgenic HQK mice over expressing PrP, or PrP knock out (KO) mice after administration of Dox. We used a P value cut-off of 0.05 as the criteria of selection of significantly differentially expressed genes."

GSE12609_series_matrix.txtimp_info.txt

Arx

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

GSE12694_series_matrix.txtimp_info.txt

Pten

Found deletion in !Series_summary "Glioblastoma (GBM) is a highly lethal brain tumor presenting as one of two subtypes with distinct clinical histories and molecular profiles. The primary GBM subtype presents acutely as high-grade disease that typically harbors EGFR, PTEN and Ink4a/Arf mutations, and the secondary GBM subtype evolves from the slow progression of low-grade disease that classically possesses PDGF and p53 events1. Here, we show that concomitant CNS-specific deletion of p53 and Pten in the mouse CNS generates a penetrant acute-onset high-grade malignant glioma phenotype with striking clinical, pathological and molecular resemblance to primary GBM in humans. This genetic observation prompted p53 and PTEN mutational analysis in human primary GBM, demonstrating unexpectedly frequent inactivating mutations of p53 as well the expected PTEN mutations. Integrated transcriptomic profling, in silico promoter analysis and functional studies of murine neural stem cells (NSCs) established that dual, but not singular, inactivation of p53 and Pten promotes an undifferentiated state with high renewal potential and drives elevated c-Myc levels and its associated signature. Functional studies validated increased c-Myc activity as a potent contributor to the impaired differentiation and enhanced renewal of p53-Pten null NSCs as well as tumor neurospheres (TNSs) derived from this model. c-Myc also serves to maintain robust tumorigenic potential of p53-Pten null TNSs. These murine modeling studies, together with confirmatory transcriptomic/promoter studies in human primary GBM, validate a pathogenetic role of a common tumor suppressor mutation profile in human primary GBM and establish c-Myc as a key target for cooperative actions of p53 and Pten in the regulation of normal and malignant stem/progenitor cell differentiation, self-renewal and tumorigenic potential."

GSE12753_series_matrix.txtimp_info.txt

bp

Found null 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."

GSE12905_series_matrix.txtimp_info.txt

Kit

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

GSE12931_series_matrix.txtimp_info.txt

Kit

Found mutant in !Series_title "Murine model of gastrointestinal stromal tumors harboring a germline Kit K641E oncogenic mutant "

GSE12931_series_matrix.txtimp_info.txt

Kit

Found mutant in !Series_summary "Gastrointestinal stromal tumors (GIST) are thought to derive from the interstitial cells of Cajal (ICC) or an ICC precursor. Oncogenic mutations of the receptor tyrosine kinase KIT are present in most GIST. KIT K642E was originally identified in sporadic GIST and later found in the germ line of a familial GIST. A mouse model of harboring a germline Kit K641E mutant was created to model familial GIST. The expression profile was investigated in the gastric antrum in the knock-in Kit K641E murine GIST model by microarray."

GSE12982_series_matrix.txtimp_info.txt

Eed

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."

GSE13143_series_matrix.txtimp_info.txt

SMRT

Found mutant in !Series_title "Expression data from 3T3-MEFs derived from wild-type and SMRT RID mutant mice"

GSE13156_series_matrix.txtimp_info.txt

Crh

Found treated in !Series_summary "As the Crh-system and the HPA-axis are known to be crucially involved in the onset, development and maintainance of psychiatric disorders like anxiety and depression and regulate the behavioural and endocrine stress responses the further analysis of Crhr1-dependent signaling cascades is essential to understand the molecular mechanisms behind these psychiatric diseases. In this project, new candidate genes involved in Crhr1-dependent signaling cascades were dissected in the cell line model of AtT-20 cells by transcriptional profiling of mouse pituitary corticotroph cells comparing control untreated AtT-20 cells with AtT-20 cells treated with 100 nM Crh at 1, 3, 6, 12 and 24 hours."

GSE13156_series_matrix.txtimp_info.txt

Crh

Found treated in !Series_overall_design "Two condition experiment: untreated vs. 100 nM Crh treated AtT-20 cells with a time curve of 1, 3, 6, 12, 24 hours. Technical replicates: 6 for each time point, including dye-swap each with 3 replicates"

GSE13190_series_matrix.txtimp_info.txt

Esrrb

Found knockdown in !Series_title "Global gene-expression analyses of the Esrrb reprogrammed cells and Esrrb knockdown cells"

GSE13192_series_matrix.txtimp_info.txt

LXR

Found treated in !Series_overall_design "To study the importance of each of the receptor subtypes, myotube cultures derived from wild type (WT), LXRa and LXRb knockout (KO) mice were established. Sixteen samples were examined. Half the samples were treated with the unselective LXR agonist T0901317."

GSE13212_series_matrix.txtimp_info.txt

Esrrb

Found silencing in !Series_summary "Global gene expression effects of silencing the Esrrb gene. We used microarrays to detail the global programme of gene expression after silencing the Esrrb gene."

GSE13212_series_matrix.txtimp_info.txt

Esrrb

Found knockdown in !Series_title "Global gene-expression analyses of the Esrrb knockdown cells"

GSE13264_series_matrix.txtimp_info.txt

Sry

Found treated in !Series_summary "The purpose of this experiment was to determine the expression traits in Liver tissue from the Four Core Genotype treated group. Keywords: Sry transgene Four Core Genotype Mouse liver Tissue"

GSE13336_series_matrix.txtimp_info.txt

AngII

Found overexpressing in !Series_title "Gene expression profiling in cardiac AngII-overexpressing mice (TG1306/1R)"

GSE13380_series_matrix.txtimp_info.txt

ovalbumin

Found induced in !Series_overall_design "The analysis includes 9 samples of genomic DNA from isolated splenic CD11c+ dendritic cells (>95% pure) per group. The two groups are neonates born to mothers with induced allergy to ovalbumin, and normal control neonates. All neonates are genetically and environmentally identical, and allergen-naive."

GSE13408_series_matrix.txtimp_info.txt

Rb

Found knock-out in !Series_summary "The retinoblastoma cell cycle regulator pRb and the two related proteins p107 and p130 are thought to suppress cancer development both by inhibiting the G1/S transition of the cell cycle in response to growth-arrest signals and by promoting cellular differentiation. Here, we investigated the phenotype of Rb family triple knock-out (TKO) embryonic stem cells as they differentiate in vivo and in culture. Confirming the central role of the Rb gene family in cell cycle progression, TKO mouse embryos did not survive past mid-gestation and differentiating TKO cells displayed increased proliferation and cell death. However, patterning and cell fate determination were largely unaffected in these TKO embryos. Furthermore, a number of TKO cells, including in the neural lineage, were able to exit the cell cycle in G1 and terminally differentiate. This ability of Rb family TKO cells to undergo cell cycle arrest was associated with the repression of target genes for the E2F6 transcription factor, uncovering a pRb-independent control of the G1/S transition of the cell cycle. These results show that the Rb gene family is required for proper embryonic development but is not absolutely essential to induce G1 arrest and differentiation in certain lineages."

GSE13448_series_matrix.txtimp_info.txt

Mef

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

GSE13493_series_matrix.txtimp_info.txt

TCR

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."

GSE13582_series_matrix.txtimp_info.txt

KRAP

Found deficient in !Series_title "Expression data from BAT of the KRAP deficient mice"

GSE13583_series_matrix.txtimp_info.txt

KRAP

Found deficient in !Series_title "Expression data from liver of the KRAP deficient mice "

GSE13585_series_matrix.txtimp_info.txt

KRAP

Found deficient in !Series_title "Expression data from BAT and liver of the KRAP deficient mice"

GSE13643_series_matrix.txtimp_info.txt

ALS

Found mutant in !Series_summary "To better understand how the expression of a mutant gene that causes ALS can perturb the normal phenotype of astrocytes, and to identify genes that may have a role in their toxic effect on motor neurons, we used oligonucleotide arrays to compare the global gene expression profiles of glia overexpressing the mutant SOD1G93A protein with two different sets of controls: non-transgenic glia and glia overexpressing the wild type form of the human SOD1 protein ($P<0.001$) ."

GSE13707_series_matrix.txtimp_info.txt

myostatin

Found knockout in !Series_summary "More than 2,000 genes appear to be upregulated or downregulated in skeletal muscle of mice with constitutive knockout of myostatin (Steelman et al.,

FASEB J 20:580-2, 2006). This study was done to determine whether inhibition of myostatin activity in mature mice has similar effects on the pattern of gene expression."

GSE13753_series_matrix.txtimp_info.txt

Rb

Found knock-out in !Series_overall_design "In one experiment, embryonic Day 13.5 placentas from wild-type and Rb -/- knock-out mice were compared. In the second experiment, embronic day 11.5 placentas from wild-type and Rb -/-;E2f4 -/- double knock-out mice were compared."

GSE13753_series_matrix.txtimp_info.txt

Rb

Found -/- in !Series_overall_design "In one experiment, embryonic Day 13.5 placentas from wild-type and Rb -/- knock-out mice were compared. In the second experiment, embronic day 11.5 placentas from wild-type and Rb -/-;E2f4 -/- double knock-out mice were compared."

GSE13753_series_matrix.txtimp_info.txt

pRb

Found mutant in !Series_summary "Homozygous mutation of the murine retinoblastoma tumor suppressor gene, Rb, results in embryonic lethality between E13.5 and E15.5 with defects in cellular proliferation, differentiation and apoptosis. Many of these defects are suppressed by mutation of an activating E2F, E2f1 or E2f3, indicating that they are key downstream targets of the retinoblastoma protein, pRB. In this study, we assess how E2F4 contributes to the developmental consequences of pRb-loss. In stark contrast to the activating E2Fs, the homozygous mutation of E2f4 shortened the lifespan of Rb-/- embryos. This resulted from an exacerbation of the placental defect of the Rb-/- mice indicating that E2F4 and pRB cooperate in the development of this tissue. Further analyses indicated that this defect reflects an increase in trophectoderm-like cells. Under conditions where the placenta was wild-type but the embryo mutant for E2f4 and pRb embryos survived to birth and exhibited all of the defects that were observed in the E2f4 and Rb single mutant embryos. Thus, while pRB and E2F4 cooperate in placental development, they play largely non-overlapping roles the development of many embryonic tissues."

GSE13805_series_matrix.txtimp_info.txt

calreticulin

Found deficient in !Series_title "Expression data from wild type and calreticulin deficient murine embryonic stem cells"

GSE13805_series_matrix.txtimp_info.txt

calreticulin

Found knockout in !Series_overall_design "Stem cells cultured in triplicate (or more) were pooled to provide raw material per sample. Each sample represents material collected from three technical replicates or more. In this manner, two wild type samples, and five derived from calreticulin knockout samples, were obtained. Although sample content contains material from three or more technical replicates harvested contemporaneously, each sample is a distinct biological replicate. Total RNA was extracted from each of the samples and RNA pools were profiled on Affymetrix Mouse 430 2.0 Arrays to identify global gene expression changes invoked by genomic ablation of calreticulin."

GSE14089_series_matrix.txtimp_info.txt

ASP

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."

GSE14242_series_matrix.txtimp_info.txt

Hyp

Found activation in !Series_summary "We used gene array analysis of cortical bone to identify Phex-dependent gene transcripts regulating Fgf23 production and mineralization in Hyp mice. We discovered that activation of Fgf receptor- and Wnt-pathways contribute to increased Ffg23 gene transcription in Hyp bone. We found evidence in Hyp bone for increased expression of Fgf1, Fgf7, and Egr2 in the Fgf-signaling pathway and decrements in Sost and Cpz and increments in Sfrp1 and 4 in the Wnt-signaling pathway. Moreover, activation of Fgf and Wnt-signaling stimulated, whereas Tgf β inhibited Fgf23 promoter activity in osteoblasts. We also observed reductions in Bmp1, a metalloproteinase that metabolizes the Fgf23 regulatory extracellular matrix protein Dmp1. These findings suggest that elevation of Fgf23 expression in osteocytes is regulated by interactions between cell surface expression of Phex, extracellular matrix proteins and paracrine effects of Fgf and Wnt. Alterations were also found in enzymes regulating the posttranslational processing and stability of Fgf23, including decrements in the glycosyltransferase Galnt3 and the proprotein convertase Pcsk5. In addition, we found that the Pcsk5 and the glycosyltransferase Galnt3 were decreased in Hyp bone, suggesting that reduced post-translational processing of FGF23 may also contribute to increased Fgf23 levels in Hyp mice. With regards to mineralization, we identified additional candidates to explain the intrinsic mineralization defect in Hyp osteoblasts, including increases in the mineralization inhibitors Mgp and Thbs4, as well as increases in local pH altering factors, carbonic anhydrase 12 (Car12) and 3 (Car3) and the sodium-dependent citrate transporter (Slc13a5). These studies demonstrate the complexity of gene expression

alterations in bone that accompanies inactivating Phex mutations and identify novel pathways that may coordinate Fgf23 expression and mineralization of extracellular matrix in Hyp bone."

GSE14365_series_matrix.txtimp_info.txt

Aire

Found deficient in !Series_title "Expression data from WT and Aire deficient mTECs"

GSE14365_series_matrix.txtimp_info.txt

Aire

Found knock-out in !Series_overall_design "In this experiment there are 5 samples altogether which consist of two biological replicates of Aire knock-out mTECs and 3 biological replicates of wild type mTECs."

GSE14395_series_matrix.txtimp_info.txt

PPARalpha

Found knock-out in !Series_overall_design "Expression profile difference between male and female PPARalpha wild-type and knock-out mice in liver and heart (3 pools of 4 animals in each group). Wild-type (12 males and 12 females) and knock-out PPARalpha SV129 mice (12 males and 12 females) approximately 10 to 12 weeks of age were killed at ZT14 and their livers and hearts quickly removed and frozen."

GSE14416_series_matrix.txtimp_info.txt

ICSBP

Found expressing in !Series_overall_design "Total RNA was isolated from parental BaF3 cells as well as BaF3 cells expressing BCR-ABL, ICSBP, or both BCR-ABL and ICSBP. Using standard Affymetrix protocols the RNA samples were analyzed for gene expression using Affymetrix mouse 430_2 whole genome microarrays arrays. A threshold value of 50 was set for all genes and the list of genes filtered to include only those that had at least one Present flag (""P"" flag) in one of the 4 conditions. For each gene, the ratio of its expression in a particular condition and its expression in parental BaF3 cells was determined. Only genes that had at least a 3-fold up or down change in expression were considered, leaving a set of 1431 genes for further analysis. K-means clustering with Gene cluster 3.0 was used to group these 1431 genes into 15 clusters and JavaTree was used to visualize the results."

GSE14418_series_matrix.txtimp_info.txt

IgG

Found treated in !Series_overall_design "Cy3: B16F10 mouse melanoma cells, treated by unspecific goat IgG (control) for 2 hours."

GSE14438_series_matrix.txtimp_info.txt

IgG

Found treated in !Series_overall_design "Bone marrow cells were differentiated in RPMI + 10% FCS + 5 ng/ml mouse IL-3 + 40 ng/ml mouse SCF for >4 weeks. The purity of the cell cultures was >90% at this time point (FcERIa+/c-kit+ cells). These in vitro-differentiated immature mast cells were then treated by either control goat IgG or an agonist anti-mouse TIM-3 antibody (RnD Systems, 15 ug/ml for 2 or 16 hours). For the IgE/antigen-activated mouse mast cells, these in vitro-differentiated immature mast cells were sensitized by 5 ug/ml anti-DNP IgE (Sigma) for 1 hour and then treated with 100 ng/ml DNP-HSA (antigen, Sigma) and either control goat IgG or an agonist anti-mouse TIM-3 antibody (RnD Systems, 15 ug/ml) for 2 or 16 hours. The anti-TIM-3 samples were labeled by Cy5 and they were compared to the Cy3-labeled, goat IgG controls in a dual-color, paired experimental setup. The Agilent Whole Mouse Genome 4x44K expression microarray kit and Dual-Color Protocol version 5.5 were used in the experiments."

GSE14525_series_matrix.txtimp_info.txt

MLCK

Found knockout in !Series_summary "Acute lung injury (ALI), a major cause of acute respiratory failure with high morbidity and mortality, isare characterized by significant pulmonary inflammation and both alveolar and vascular barriers dysfunction. In Pprior studies have highlighted the role of nonmuscle myosin light chain kinase (nmMLCK) as an essential element of inflammatory response with MYLK polymorphisms associated withwhich alters ALI susceptibility. In the present study we sought to further define nmMLCK in acute inflammatory syndromes and examined We examined nmMLCK as a molecular target involved in increase of lung epithelial and endothelial barrier permeability. We utilized in two muirine models of inflammatory lung injury: intratracheal administration of endotoxin/lipopolysaccharide (LPS, 2.5 mg/kg) and VILI (ventilator-induced lung injury, tidal volume 40ml/kg). Two complementary strategies were used to reduce nmMLCK activity or expression. We found that membrane permeant oligopeptide, PIK, inhibited MLC kinase activity in vitro in aand displayed dose-dependent mannerinhibition of MLC kinase activity.. Intravenous delivery of PIK significantly attenuated LPS-induced lung inflammation reflected by decreasing accumulation of bronchoalveolar lavage (BAL) albumin (~ 50% reduction) as well as reduction in BAL cells, tissue MPO activity and tissue albumin in lung homogenates. A second regulatory approach explored targeting murine nmMLCK by administration of siRNA (5mg/kg) 3 days prior to LPS challenge. siRNA decreased of nmMLCK expression in lungs (~ 70% reduction) and resulted in significant attenuation LPS-induced lung inflammation (~ 40% reduction) as reflected by decreased BAL protein level and BAL cells. For targeting pulmonary vessels nmMLCK we used ACE antibody-conjugated liposomes with nmMLCK siRNA in murine ventilator-induced lung injury (VILI) model. Protein silencing of nmMLCK was evident by immunohistochemical analysis with a decrease in relative intensity of fluorescence in lung vessels compared with control animals. Furthermore, the inhibition of nmMLCK expression by siRNA in vessels significantly attenuated VILI lung injury as reflected by decreased BAL protein level (40% reduction).

Finally, MLCK knockout mice were significantly protected (reduced BAL protein and albumin) when exposed to a model of severe VILI (4h, 40ml/kg tidal volume). Conclusion: the MLCK gene KO and chemical biology results indicate that the targeting of nmMLCK in vivo attenuate the severity of LPS-induced or VILI acute lung injury."

GSE14549_series_matrix.txtimp_info.txt

Wrn

Found mutant in !Series_title "Gene expression profiling in liver of mice with a mutant Wrn protein treated with/without vitamin C compared to WT mice."

GSE14549_series_matrix.txtimp_info.txt

Wrn

Found mutant in !Series_summary "Werner syndrome (WS) is a rare disorder characterized by the premature onset of a number of age-related diseases. The gene responsible for WS is believed to be involved in different aspects of transcription, replication, and/or DNA repair. We generated a mouse model with a deletion in the helicase domain of the murine WRN homologue that recapitulates most of the WS phenotypes including an abnormal hyaluronic acid excretion, higher reactive oxygen species (ROS) levels, increased genomic instability and cancer incidence resulting in a 10-15% decreased life span expectancy. In addition, WS patients and Wrn mutant mice show hallmarks of a metabolic syndrome including premature visceral obesity, hypertriglyceridemia, insulin-resistant diabetes type 2 and associated cardiovascular diseases. In this study, we compared the expression profile of liver tissues from 9 months old Wrn mutant and wild type animals. Gene set enrichment analysis of the microarray data indicated that Wrn mutant mice exhibited down-regulation of genes normally decreased in several transgenic mouse models of hepatoma and during caloric restriction. Wrn mutant mice also altered the expression of genes involved in inflammation as well as in glutathione and xenobiotic metabolisms. These results indicate that Wrn mutant mice respond to the observed oxidative stress by altering the necessary pathways to survive. Vitamin C supplementation rescued the life span expectancy of Wrn mutant mice and reversed several age-related abnormalities in adipose, cardiac, and liver tissues, genomic integrity and inflammatory status. Finally, gene set enrichment analyses revealed that vitamin C decreased genes normally up regulated in human WS fibroblasts and cancers and it increased genes involved in tissue injury response and adipocyte dedifferentiation in obese mice."

GSE14549_series_matrix.txtimp_info.txt

Wrn

Found mutant in !Series_overall_design "Microarray analyses were performed on the liver tissues of 9 months old mice. Four independent biological replicates of this experiment (wild type vs Wrn mutant or wild type vs vitamin C treated mutant mice) were carried out with a dye swap on two replicates of each genotype."

GSE14549_series_matrix.txtimp_info.txt

Wrn

Found treated in !Series_title "Gene expression profiling in liver of mice with a mutant Wrn protein treated with/without vitamin C compared to WT mice."

GSE14550_series_matrix.txtimp_info.txt

podoplanin

Found overexpressing in !Series_title "Gene expression profiling of MCF7 breast cancer xenografts overexpressing podoplanin (human array)"

GSE14551_series_matrix.txtimp_info.txt

podoplanin

Found overexpressing in !Series_title "Gene expression profiling of tumor stroma of MCF7 breast cancer xenografts overexpressing podoplanin (mouse array)"

GSE14585_series_matrix.txtimp_info.txt

Xist

Found silencing in !Series_summary "The non-coding Xist RNA triggers silencing of one of the two female X chromosomes during X inactivation in mammals. Gene silencing by Xist is restricted to special developmental contexts found in cells of the early embryo and specific hematopoietic precursors. The absence of critical silencing factors might explain why Xist cannot silence outside these contexts. Here, we show that Xist can also initiate silencing in a lymphoma model. Using the tumor context we identify the special AT rich binding protein SATB1 as an essential silencing factor. We show that loss of SATB1 in tumor cells abrogates the silencing function of Xist. In normal female lymphocytes Xist localizes along SATB1 filaments and, importantly, forced Xist expression can relocalize SATB1 into the Xist cluster. This reciprocal influence on localization suggests a molecular interaction between Xist and SATB1. SATB1 and its close homologue SATB2 are expressed during the initiation window for X inactivation in embryonic stem cells and are recruited to surround the Xist cluster. Furthermore, ectopic expression SATB1 or SATB2 enables gene silencing by Xist in embryonic fibroblasts, which normally do not provide an initiation context. Thus, SATB1 functions as a crucial initiation factor and may act to organize genes for silencing by Xist during the initiation of X inactivation."

GSE14829_series_matrix.txtimp_info.txt

ras

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."

GSE14855_series_matrix.txtimp_info.txt

MnSOD

Found knockout in !Series_title "Effect of MnSOD knockout cells on gene profile expression"

GSE14870_series_matrix.txtimp_info.txt

Eda

Found mutant in !Series_summary "Sweat glands play a fundamental role in thermal regulation in man, but the molecular mechanism of their development remains unknown. To initiate analyses, we compared the model of Eda mutant Tabby mice, in which sweat glands were not formed, to wild-type mice. We inferred developmental stages and critical genes based on observations at 7 time points spanning embryonic, postnatal and adult life. In wild-type footpads, sweat gland germs were detected at E17.5. The coiling of secretory portions started at postnatal day 1 (P1), and sweat gland formation was essentially complete by P5. Consistent with a controlled morphological progression, expression profiling revealed stage-specific gene expression changes. Similar to the development of hair follicles the other major skin appendage controlled by EDA sweat gland induction and initial progression was accompanied by Eda-dependent up-regulation of the Shh pathway. During the further development of sweat gland secretory portions, Foxa1 and Foxi1, not at all expressed in hair follicles, were progressively up-regulated in wild-type but not in Tabby footpads. Upon completion of wild-type development, Shh declined to Tabby levels, but Fox family genes remained at elevated levels in mature sweat glands. The results provide a framework for the further analysis of phased downstream regulation of gene action, possibly by a signaling cascade, in response to Eda."

GSE14871_series_matrix.txtimp_info.txt

Eda

Found mutant in !Series_summary "Sweat glands play a fundamental role in thermal regulation in man, but the molecular mechanism of their development remains unknown. To initiate analyses, we compared the model of Eda mutant Tabby mice, in which sweat glands were not formed, to wild-type mice. We inferred developmental stages and critical genes based on observations at 7 time points spanning embryonic, postnatal and adult life. In wild-type footpads, sweat gland germs were detected at E17.5. The coiling of secretory portions started at postnatal day 1 (P1), and sweat gland formation was essentially complete by P5. Consistent with a controlled morphological progression, expression profiling revealed stage-specific gene expression changes. Similar to the development of hair follicles the other major skin appendage controlled by EDA sweat gland induction and initial progression was accompanied by Eda-dependent up-regulation of the Shh pathway. During the further development of sweat gland secretory portions, Foxa1 and Foxi1, not at all expressed in hair follicles, were progressively up-regulated in wild-type but not in Tabby footpads. Upon completion of wild-type development, Shh declined to Tabby levels, but Fox family genes remained at elevated levels in mature sweat glands. The results provide a framework for the further analysis of phased downstream regulation of gene action, possibly by a signaling cascade, in response to Eda."

GSE14872_series_matrix.txtimp_info.txt

Eda

Found mutant in !Series_summary "Sweat glands play a fundamental role in thermal regulation in man, but the molecular mechanism of their development remains unknown. To initiate analyses, we compared the model of Eda mutant Tabby mice, in which sweat glands were not formed, to wild-type mice. We inferred developmental stages and critical genes based on observations at 7 time points spanning embryonic, postnatal and adult life. In wild-type footpads, sweat gland germs were detected at E17.5. The coiling of secretory portions started at postnatal day 1 (P1), and sweat gland formation was essentially complete by P5. Consistent with a controlled morphological progression, expression profiling revealed stage-specific gene expression changes. Similar to the development of hair follicles the other major skin appendage controlled by EDA sweat gland induction and initial progression was accompanied by Eda-dependent up-regulation of the Shh pathway. During the further development of sweat gland secretory portions, Foxa1 and Foxi1, not at all expressed in hair follicles, were progressively up-regulated in wild-type but not in Tabby footpads. Upon completion of wild-type development, Shh declined to Tabby levels, but Fox family genes remained at elevated levels in mature sweat glands. The results provide a framework for the further analysis of phased downstream regulation of gene action, possibly by a signaling cascade, in response to Eda."

GSE14873_series_matrix.txtimp_info.txt

Eda

Found mutant in !Series_summary "Sweat glands play a fundamental role in thermal regulation in man, but the molecular mechanism of their development remains unknown. To initiate analyses, we compared the model of Eda mutant Tabby mice, in which sweat glands were not formed, to wild-type mice. We inferred developmental stages and critical genes based on observations at 7 time points

spanning embryonic, postnatal and adult life. In wild-type footpads, sweat gland germs were detected at E17.5. The coiling of secretory portions started at postnatal day 1 (P1), and sweat gland formation was essentially complete by P5. Consistent with a controlled morphological progression, expression profiling revealed stage-specific gene expression changes. Similar to the development of hair follicles the other major skin appendage controlled by EDA sweat gland induction and initial progression was accompanied by Eda-dependent up-regulation of the Shh pathway. During the further development of sweat gland secretory portions, Foxa1 and Foxi1, not at all expressed in hair follicles, were progressively up-regulated in wild-type but not in Tabby footpads. Upon completion of wild-type development, Shh declined to Tabby levels, but Fox family genes remained at elevated levels in mature sweat glands. The results provide a framework for the further analysis of phased downstream regulation of gene action, possibly by a signaling cascade, in response to Eda."

GSE14874_series_matrix.txtimp_info.txt

Eda

Found mutant in !Series_summary "Sweat glands play a fundamental role in thermal regulation in man, but the molecular mechanism of their development remains unknown. To initiate analyses, we compared the model of Eda mutant Tabby mice, in which sweat glands were not formed, to wild-type mice. We inferred developmental stages and critical genes based on observations at 7 time points spanning embryonic, postnatal and adult life. In wild-type footpads, sweat gland germs were detected at E17.5. The coiling of secretory portions started at postnatal day 1 (P1), and sweat gland formation was essentially complete by P5. Consistent with a controlled morphological progression, expression profiling revealed stage-specific gene expression changes. Similar to the development of hair follicles the other major skin appendage controlled by EDA sweat gland induction and initial progression was accompanied by Eda-dependent up-regulation of the Shh pathway. During the further development of sweat gland secretory portions, Foxa1 and Foxi1, not at all expressed in hair follicles, were progressively up-regulated in wild-type but not in Tabby footpads. Upon completion of wild-type development, Shh declined to Tabby levels, but Fox family genes remained at elevated levels in mature sweat glands. The results provide a framework for the further analysis of phased downstream regulation of gene action, possibly by a signaling cascade, in response to Eda."

GSE14876_series_matrix.txtimp_info.txt

Eda

Found mutant in !Series_summary "Sweat glands play a fundamental role in thermal regulation in man, but the molecular mechanism of their development remains unknown. To initiate analyses, we compared the model of Eda mutant Tabby mice, in which sweat glands were not formed, to wild-type mice. We inferred developmental stages and critical genes based on observations at 7 time points spanning embryonic, postnatal and adult life. In wild-type footpads, sweat gland germs were detected at E17.5. The coiling of secretory portions started at postnatal day 1 (P1), and sweat gland formation was essentially complete by P5. Consistent with a controlled morphological progression, expression profiling revealed stage-specific gene expression changes. Similar to the development of hair follicles the other

major skin appendage controlled by EDA sweat gland induction and initial progression was accompanied by Eda-dependent up-regulation of the Shh pathway. During the further development of sweat gland secretory portions, Foxa1 and Foxi1, not at all expressed in hair follicles, were progressively up-regulated in wild-type but not in Tabby footpads. Upon completion of wild-type development, Shh declined to Tabby levels, but Fox family genes remained at elevated levels in mature sweat glands. The results provide a framework for the further analysis of phased downstream regulation of gene action, possibly by a signaling cascade, in response to Eda."

GSE14877_series_matrix.txtimp_info.txt

Eda

Found mutant in !Series_summary "Sweat glands play a fundamental role in thermal regulation in man, but the molecular mechanism of their development remains unknown. To initiate analyses, we compared the model of Eda mutant Tabby mice, in which sweat glands were not formed, to wild-type mice. We inferred developmental stages and critical genes based on observations at 7 time points spanning embryonic, postnatal and adult life. In wild-type footpads, sweat gland germs were detected at E17.5. The coiling of secretory portions started at postnatal day 1 (P1), and sweat gland formation was essentially complete by P5. Consistent with a controlled morphological progression, expression profiling revealed stage-specific gene expression changes. Similar to the development of hair follicles the other major skin appendage controlled by EDA sweat gland induction and initial progression was accompanied by Eda-dependent up-regulation of the Shh pathway. During the further development of sweat gland secretory portions, Foxa1 and Foxi1, not at all expressed in hair follicles, were progressively up-regulated in wild-type but not in Tabby footpads. Upon completion of wild-type development, Shh declined to Tabby levels, but Fox family genes remained at elevated levels in mature sweat glands. The results provide a framework for the further analysis of phased downstream regulation of gene action, possibly by a signaling cascade, in response to Eda."

GSE14907_series_matrix.txtimp_info.txt

Eda

Found mutant in !Series_summary "Sweat glands play a fundamental role in thermal regulation in man, but the molecular mechanism of their development remains unknown. To initiate analyses, we compared the model of Eda mutant Tabby mice, in which sweat glands were not formed, to wild-type mice. We inferred developmental stages and critical genes based on observations at 7 time points spanning embryonic, postnatal and adult life. In wild-type footpads, sweat gland germs were detected at E17.5. The coiling of secretory portions started at postnatal day 1 (P1), and sweat gland formation was essentially complete by P5. Consistent with a controlled morphological progression, expression profiling revealed stage-specific gene expression changes. Similar to the development of hair follicles the other major skin appendage controlled by EDA sweat gland induction and initial progression was accompanied by Eda-dependent up-regulation of the Shh pathway. During the further development of sweat gland secretory portions, Foxa1 and Foxi1, not at all expressed in hair follicles, were progressively up-regulated in wild-type but not in Tabby footpads. Upon completion of wild-type development, Shh

declined to Tabby levels, but Fox family genes remained at elevated levels in mature sweat glands. The results provide a framework for the further analysis of phased downstream regulation of gene action, possibly by a signaling cascade, in response to Eda."

GSE14917_series_matrix.txtimp_info.txt

Cebpa

Found deletion in !Series_summary "We have previously demonstrated that deletion of the Cebpa gene in the developing fetal mouse lung caused death soon after birth from the failure of lung maturation. Many of the transcriptional pathways regulating morphogenesis of the fetal lung are induced postnatally and mediate repair of the injured lung. We hypothesized that C/EBPa plays a role in protection of the alveolar epithelium following hyperoxia injury of the mature lung. Transgenic CebpaΔ/Δ mice in which Cebpa was conditionally deleted from Clara cells (from early gestation) and type II cells (from near-term) were developed. CebpaΔ/Δ mice grow normally without any pulmonary abnormalities. CebpaΔ/Δ mice were highly susceptible to hyperoxia. CebpaΔ/Δ mice died within 4d after hyperoxia associated with severe lung inflammation and altered surfactant components at a time when all control mice survived. Microarrays were analyzed on isolated type II cells at an early stage (24h) of hyperoxia exposure to detect the primary genes influenced by deletion of Cebpa. The associated network analysis revealed the reduced expression of key genes related to surfactant lipid and protein homeostasis, such as Srebf, Scap, Lpcat1, Abca3, Sftpb, and Napsa. Genes for the cell signaling, immune response, and protective antioxidants, including GSH and Vnn-1,3, were decreased in the CebpaΔ/Δ mice lung. C/EBPa did not play a critical role in postnatal pulmonary function under normal conditions. In contrast, in the absence of C/EBPa, exposure to hyperoxia caused respiratory failure, supporting the concept that C/EBPa plays an important role in enhancing epithelial cell survival, surfactant lipid homeostasis, and maturation of SP-B from pro-SP-B. "

GSE14920_series_matrix.txtimp_info.txt

PPARalpha

Found activation in !Series_summary "Di-(2-ethylhexyl)-phthalate (DEHP), a widely used plasticizer, is detected in consumer’s body fluids. Contamination occurs through environmental and food chain sources. In mouse liver, DEHP activates the peroxisome proliferator-activated receptor alpha (PPARalpha) and regulates the expression of its target genes. Several in vitro investigations support the simultaneous recruitment of additional nuclear receptor pathways. We investigated, in vivo, the hepatic impact of low doses of DEHP on PPARalpha activation, and the putative activation of additional signalling pathways. Wild-type and PPARalpha-deficient mice were exposed to different doses of DEHP. Gene expression profiling delineated the role of PPARalpha and revealed a PPARalpha-independent regulation of several prototypic Constitutive Androstane Receptor (CAR) target genes. This finding demonstrates that CAR also represents a transcriptional regulator sensitive to phthalates. CAR-mediated effects of DEHP provide a new rationale for most endpoints of phthalates toxicity described previously, including endocrine disruption, hepatocarcinogenesis and the metabolic syndrome."

GSE14921_series_matrix.txtimp_info.txt

Lox

Found KO in !Series_overall_design "ix Lox control and six Liver-specific SIRT1 KO (SIRT1 LKO) mice in 95% C57BL/6 background were fed ad libitum with free access to water and food. They were then sacrificed at 4:00pm to analyze PPAR-alpha mediated phenotypes under physiologically relevant conditions. Livers from these mice were then dissected and RNA was isolated with a Qiagen RNA easy mini kit with on-column DNAseI treatment. RNA quality was validated with the Agilent 2100 Bioanalyzer in the microarray facility. RNA samples were then pooled with 2 animals/replicate and analyzed by Agilent Whole Mouse arrays. Data are generated by the Rosetta Resolver Error Model."