

Supplementary Table 1: Guideline values for risk assessment of BaP from various subnational, national and international agencies.

Agency	Category	Value µg/L	Method	Basis for value
WHO (2003)	Health-based guideline	0.7	Oral carcinogenicity in mice using two-stage mutation model. Neal and Rigdon (1967) forestomach tumors	Oral cancer in mice
Cal/EPA (2010)	Public health goal	0.007	Time to tumor model to obtain a lower-bound estimate of dose associated with 10% increased incidence of tumors. Assumed a linear dose-response relationship at low doses. Increased potency factor by 1.7 to correct for early-life exposure. Exposure of 0.052 L/kg bw per day of water (an age-adjusted upper 95th percentile of drinking water consumption for age range for cancer potency correction). q* Cancer potency 1.7 (mg/kg bw per day)-1 [2.9 (mg/kg bw per day)-1 when corrected for early-life exposure]	Incidence of oral cavity or forestomach tumors in female mice (Kroese et al., 2001)
Cal/EPA (2010)	Health protective level	4	LOAEL of 5 mg/kg bw per day from subchronic study. Uncertainty factor of 3000 and RSC of 0.1 and drinking water ingestion of 0.044 L/kg bw per day.	Renal toxicity (Knuckles et al., 2001)
U.S. EPA (1994; 2007) Currently under review	Maximum contaminant level	0.2	Used Neal and Rigdon (1967) and Rabstein (1973); both incidence of mouse forestomach tumors from feeding study	Reproductive difficulties; increased risk of cancer (stomach)
NHMRC (2004)	Health-based guideline	0.01	Based on limit of detection, which is slightly less than value derived using a risk assessment from WHO (2003) using Neal and Rigdon (1967).	Mouse tumors
Health Canada (1988, reaffirmed in 2005)	Health-based guideline	0.01	Increased stomach tumors in Neal and Rigdon (1967) feeding study in mice. Used surface area correction and robust linear extrapolation model to estimate lifetime risk associated with 1 µg/l BaP in drinking water of 5×10^{-5}	Mouse stomach tumors

Supplementary Table 2: All epidemiology studies evaluated.

Study	Case-control/cohort	Exposure route	N	Endpoint	BaP concentration	Outcome	Duration of exposure	Association with BaP
Inhalation								
Arif et al. (2006)	Case-control	Cigarette smoking (comparison of human tissues from cancer patients)	50	Highly lipophilic DNA adducts	Unknown	DNA adducts are present, but are not related to PAHs or BaP ^a	Chronic	Negative
Armstrong et al. (1986)	Case-control	Inhalation among aluminum smelter workers	85 cases and 255 referents	Bladder cancer	1 mg/m ³	2.3% increase in bladder cancer	Chronic	Positive
Armstrong et al. (1994)	1) Case-cohort; 2) Subcohort	Inhalation in aluminum production plant	1) 338 2) 1138	Lung cancer mortality	1) cumulative < 10, 10–99, 100–199, 200–299, ≥ 300 µmol/m ³ -year 2) 342.2 µg/m ³ / 190.1 µg/m ³	1) Smoking-adjusted rate ratio ≥ 1 year between 1950 and 1979 2) probability of causation over 50% first achieved and according to the upper 95% confidence limit	Chronic	Positive
Armstrong and Thériault (1996)	Cohort	From compensation claims from lung cancer patients exposed to coal tar pitch volatiles (occupational exposure)	Not reported	Lung cancer	1) 100 µg m ⁻³ ·years 2) 342.2 µg/m ³ / 190.1 µg/m ³	1) probability of causation over 50% first achieved and according to the upper 95% confidence limit	Chronic	Positive
Armstrong and Gibbs (2009)	Cohort	Inhalation from aluminum smelters in Quebec	16 431	Lung cancer	100 µg/m ³ BaP-years of employment	Relative risk = 1.35 (95% CI 1.22–1.51)	at least 1 year of employment	Positive
Bartsch et al. (1998)	Case-control	Lung cancer patients vs. controls	N/A	BPDE-DNA adducts (also PAH DNA adduct)	Unknown	CYP1A1 mutation increases chronic	BPDE-DNA adducts	Positive
Burstin et al. (2005)	Cohort	Inhalation in asphalt workers across Europe	12 367 males	Ischemic heart disease mortality	Highest exposure group was 273+ ng/m ³ and 2013+ ng/m ³ for cumulative exposure	Significant trend with dose and cumulative exposure; 1.64 relative ratio for average exposure and 1.58 in the cumulative exposure	Minimum of one work season	Positive
Cross et al. (2010)	Cohort	Meat and meat component intake as well as meat cooking by-products	Not reported	Esophageal and gastric cancer	Increased risk estimated for each additional 10 ng/d BaP	No relation for BaP; increased cancer risk was observed for regular consumption	Chronic	Negative
De Flora et al. (1993)	Cohort	Smoking	39 for adducts and 31 for MN	BPDE-DNA adducts and micronuclei (MN not specific to BaP)	Not defined	Not significant for MN; however, BPDE-DNA adducts were significant according to number of cigarettes smoked per day	Not defined	Positive
De Stefani et al. (2009)	Case-control	Intake of meat and meat mutagens	846 cases and 846 controls	Lung cancer	1) less than 26.9 ng/g; 2) 27.0–42.1 ng/g; 3) 42.2–51.8 ng/g; 4) 51.9+ ng/g	BaP significantly associated with risk of lung cancer. 1) OD = 1.0, 2) OD = 1.41; 3) OD = 1.49; 4) OD = 2.08	Chronic	Positive
Engholm et al. (1996)	Cohort	Residents of Copenhagen in comparison with those living in rural areas	927 470 men and 486 130 women	Lung cancer	Up to 10 ng/m ³	Region had very small effect, and role of BaP is unclear	Chronic	Inconclusive
Friesen et al. (2007)	Cohort	Inhalation at aluminum smelter	6423 males	Bladder and lung cancer incidence, mortality due to myocardial infarction	7.63–122.0 µg/m ³ ·year	BaP (and benzene-soluble material) were strongly associated with bladder cancer and lung cancer, but modestly associated with myocardial infarction	More than 3 years of work experience	Positive
Friesen et al. (2009)	Cohort	Inhalation at aluminum smelter	4316 male smelter workers	Mortality and cancer incidence	Unknown	Marginal significance of trend for BaP (also inhalable dust and fluoride exposure)	Chronic	Inconclusive
Gu et al. (2008)	Case-control	Smoking	203 cases and 198 controls	BPDE-induced 9p21 aberrations in cultured peripheral blood lymphocytes (bladder cancer)	Not defined	In smokers individuals with cancer relative to control; BPDE 9p21 aberrations significantly associated with bladder cancer (OR = 5.29, 95% CI = 3.26–8.59)	Not defined	Positive
Gustavsson et al. (1995)	Cohort	Inhalation in Swedish graphite electrode plant	901	Mortality and cancer incidence	In the highest exposure group, cumulative exposure was 33 µg BaP/m ³ ·years, mean exposure time was 10.8 years and average exposure level	No excess risk of death from cancer	Average of 10.8 years	Negative
Haugen et al. (1986)	Cohort	Inhalation in coke oven workers	Not reported	BPDE-DNA adducts in lymphocytes	7.3 µg/m ³ on collected particulate matter	One third of the workers had detectable BPDE-DNA adducts	Chronic	Positive
He et al. (1991)	Case-control	Indoor smoky coal exposure	110 cases and 426 controls	Lung cancer mortality	35.60–248.50 µg/100 m ³	Strong association between BaP exposure and lung cancer mortality	Chronic	Positive
Hemminki et al. (1990)	Cohort	Inhalation in coke workers	91	Aromatic DNA adducts in white blood cells	0.25–90 µg/m ³	Significant in battery workers in comparison with other jobs; significant in nonbattery workers outside with current smoking controls	Current job considered	Positive
Izzotti et al. (1991)	Case-control	Cigarette smoking	39	BPDE-DNA adducts	Unknown	No adducts were detected in samples from nonsmokers or ex-smokers, whereas 84.6% of samples from current smokers exhibited typical fluorescence peaks	Chronic	Positive
Jeng et al. (2011)	Cross-sectional	Inhalation in coke workers	1000	IgA, IgE levels (only endpoint relevant to BaP); other endpoints for all PAHs included malondialdehyde (MDA) and 8-OHDG	Mean concentrations of 1603.06 ng/m ³ in top-oven workers (considered high-exposure group in this study) and 62.47 ng/m ³ in side-oven workers (considered low-exposure group)	IgA and IgE correlated strongly with BaP exposure; MDA was significantly increased, but not 8-OHDG	At least 1 year of employment	Positive
Junior et al. (1994)	Cohort	Males working in steel foundries	206	BPDE adducts to hemoglobin	Assessed by personal air sampling devices; precise air concentrations not reported	Air concentrations of BaP not associated with levels	Current job considered	Negative
Knox et al. (2005)	Cohort	Home address relative to hotspots for chemical exposure	22 258 cancer-related deaths before age of 16	Cancer mortality	Not defined	Excess risk of cancer-related death within 0.3 km of hotspots for BaP (and many other chemicals including PM ₁₀ , benzene and toluene, etc.)	Chronic	Positive
Lavoué et al. (2007); Gibbs Cohort and Sevigny (2007a); and Gibbs and Sevigny (2007b) (four-part study)	Cohort	Inhalation in workers from aluminum smelters in Québec	Part I) 28 910 jobs; Part II) 21 5977 workers; Part III) 9726 workers; Part IV) 5977 workers	All cancer incidence and mortality	0.01–68.08 µg/m ³	Part I) excess risk of exposure; Part II) excess of death before 1951 significant for bladder cancer, COPD, cancers of stomach, digestive system (unspecified), rectum and rectosigmoid, pancreas and larynx, Alzheimer's disease, cerebrovascular disease; Part 3) cause of death after 1951 significant for breast and respiratory cancer (also esophagus, rectum and rectosigmoid junction, pancreas, larynx, lung, non-Hodgkin's lymphoma, cerebrovascular disease and asthma; Part 4) overall cancer incidence significant lung and bladder cancer (BaP specific); laryngeal and buccal cavity cancer also increased with BaP exposure	Chronic	Positive
Mumford et al., 1993	Cohort	Coal or wood smoke during cooking or heating vs. women using natural gas in Beijing	9 Xuan Wei women with chimney; 9 Xuan Wei women without chimney; 9 Beijing controls	DNA adducts in peripheral blood and cord blood white blood cells and placental tissue	19.25 µg/m ³ for smoky coal and 3.24 µg/m ³ for wood	More DNA adducts were reported in exposed women but significance is not reported.	At home throughout lifetime	Positive
Mori, 2002	Cohort	Inhalation in graphite electrode factory; exposure to coal tar and coal tar pitch volatiles	332 male employees	Mortality, all causes	Unknown	Increased mortality due to lung cancer (SMR=2.62, compared to 2.35 in general population) lymphatic and hematopoietic cancer (SMR=3.46). Relative contribution of BaP unknown.	More than 5 years	Inconclusive
Nadon et al. (1995)	Case-control	Cancer patients vs. controls in Montreal area	3730 cancer patients and 533 controls	14 types of cancer (esophagus, stomach, colon, rectum, pancreas, lung (+ oat cell, squamous cell and adenocarcinoma), prostate, bladder, kidney, melanoma of skin and non-Hodgkin's lymphoma	Unknown (only estimated as low, medium and high according to questionnaire)	Increased cancer cases for stomach, pancreas and prostate; increased lung cancer risk observed in nonsmokers and light smokers only	Chronic	Positive

Niu et al. (2011)	Cohort	Inhalation in coke oven workers	176 coke oven workers and 48 warehouse controls.	Emotional and cognitive function; concentrations of monoamine and amino acid neurotransmitters; urinary levels of 1-hydroxyxypyrene for exposure assessment	Mean concentrations of BaP were 19.5, 185.9 and 1623.5 ng/m ³ at the bottom, side and top of the coke oven, respectively; concentration for controls was 10.2 ng/m ³	I-OH-Py levels increased; learning and memory decreased; concentrations of norepinephrine decreased; acetylcholine esterase activity decreased; overall, the study shows that exposure to BaP may reduce neurobehavioral function and neurotransmitter levels	Chronic	Positive
Övrebo et al. (1995)	Cohort	Inhalation in coke workers	13/- for control, 23/17 for low, 26/18 for medium and 18/13 for high exposure workers (January/June)	Anti-BPDE-DNA adducts and hydroxethylvaline hemoglobin adducts; urinary 1-hydroxyxypyrene as a measure of exposure	high=top side workers, medium=side workers, low=maintenance workers	Anti-BPDE-DNA adducts did not correlate urinary marker of exposure or cumulative exposure; nonsignificant increase was observed for hydroxethylvaline-hemoglobin adducts	Chronic	Negative
Pan et al. (1998)	Cohort	Inhalation in coke oven workers (top, middle and bottom workers, and controls)	25 males	Urinary 1-hydroxyxypyrene, leukocyte aromatic DNA adducts; serum p53, glutathione S-transferase M1 (all for PAHs)	Mean BaP level for each worksite of 3.16 µg/m ³ (top), 3.02 µg/m ³ (push side), 0.98 µg/m ³ (coker side), 0.10 µg/m ³ (bottom) and 0.01 µg/m ³ (control); BaP exposure information also available for	Serum p53 correlated with cumulative BaP exposure; no correlation was found for DNA adducts and PAH exposure	Chronic	Positive
Pastorelli et al. (1996)	Cohort	Inhalation of BaP from traffic exhaust	53	BaP diol epoxide adducts with hemoglobin	Estimate of 1-3 ng/m ³ in high traffic areas	Significant difference observed for high-traffic exposure in nonsmokers	Chronic	Positive
Perera et al. (1982)	Case-control	Lung cancer patients (normal tissue, tumor tissue and blood samples)	15	DNA adducts	Unknown	Inconclusive due to small sample size	Unknown	Inconclusive
Perera et al. (1988)	Cohort	Inhalation in Finnish foundry workers	35 + 10 controls	DNA adducts in white blood cells	Low (< 0.05 µg/m ³); medium (0.05-0.2 µg/m ³); high (> 0.2 µg/m ³)	Significant for each exposure group	Current job considered	Positive
Perera et al. (1993)	Cohort	Inhalation in Finnish foundry workers	48	HPRT and GPA mutation; DNA adducts	< 5-60 µg/m ³	Not significant for any endpoint	Current job considered	Negative
Phillips et al. (1988)	Cohort	Inhalation in Finnish iron foundry	41	Aromatic DNA adducts in white blood cells	> 0.2 (high), 0.05-0.2 (medium) and < 0.05 (low) µg/m ³	Adducts present in 3/4 (high), 8/10 (medium), 4/18 (low) and 1/20 (control)	Current job considered	Positive
Rojas et al. (1995)	Cohort	Inhalation in coke workers (smoking, nonsmoking and controls)	39 exposed, 39 controls	Anti-BPDE-DNA adducts in lymphocytes/monocytes	Ranging from ≤ 0.15 to ≥ 4 µg/m ³ in year of collection	8 times higher in occupationally exposed individuals	At least 4-6 months prior to blood collection	Positive
Santella et al. (1993)	Cohort	Inhalation in workers near or in a Finnish iron foundry	48	PAH-DNA adducts in white blood cells	2-60 ng/m ³	Not significant	Chronic	Negative
Szczeklik et al. (1994)	Cohort	Inhalation in Polish iron workers	274 (199 coke oven workers and 76 cold-rolling mill workers)	Humoral immunity (IgG, IgA, IgM and IgE concentrations in blood)	0.2-50 µg/m ³ in coke plant workers was 3-5 times magnitude than in cold-rolling mill employees	Decreased IgG and IgA in coke oven workers indicating immunosuppression with BaP exposure	Average of 15 years	Positive
Tas et al. (1994)	Cohort	Inhalation in two steel foundries and one graphite electrode producing plant	260 (133 controls and 127 exposed)	BPDE adducts on albumin	Not clearly defined	Significantly higher in relative to controls; significantly associated with air BaP levels	Current job considered	Positive
Winker et al. (1996)	Cohort	Inhalation exposure in coke oven workers (one newer/clean facility and another with high levels of PAHs)	24	Immunotoxic effects from blood samples	New facility = 651 ng/m ³ air, old facility = 5396 ng/m ³	Reduced mitogenic response of T cells to phytohemagglutinin; impairment of B cell activity; reduced oxidative burst in monocytes after stimulation with <i>E. coli</i> ; no effects on lymphocyte subpopulations and immunoglobulin levels in serum	6-30 years	Positive and Negative
Xu et al. (1996)	Nested case-control	Inhalation in Chinese iron steel complex	610 lung cancer cases, 292 stomach cancer cases and 959 controls	Lung cancer and stomach cancer cases	< 0.84, 0.85-1.96, 1.97-3.2, ≥ 3.2 µg/m ³	Odds ratio of 0.9, 1.7, 1.3, 1.7 for each dose category, respectively; significant trend	More than 15 years	Positive
Ingestion								
Anderson et al. (2005)	Case-control	Meat intake and preparation method	193 cases and 674 controls	Pancreatic cancer	Median 0.3-53.7 ng/day	OR = 2.2 (1.2-4.0)	Chronic	Positive
Butler et al. (2003)	Case-control	Meat intake and preparation method	701 African Americans and 957 Caucasians	Colon cancer	Mean BaP levels of 22.5 and 16.7 ng/day for African American cases and controls; 41.9 and 35.4 ng/day for white cases and controls	Significant association observed in African Americans only	Chronic	Positive
Cross et al. (2005)	Case-control	Meat intake	29 361	Prostate cancer	≥ 1031.5 ng/day	No association between prostate cancer and BaP intake	Chronic	Negative
Cross et al. (2006)	Case-control	Meat intake and preparation method	383 controls and 458 cases	Non-Hodgkin's lymphoma	Mean intake of 37.1 ng/day and median intake of 16.8 ng/day	No increased risk	Chronic	Negative
Ferrucci et al. (2012)	Case-control	Meat consumption	17 072	Distal colon and rectal adenoma	Unknown	Not associated with colon adenoma but significantly associated with rectal adenoma (OR = 1.52)	Chronic	Positive
Fu et al. (2011)	Case-control	Meat and meat-derived mutagen intake	2386 cases and 1703 controls	Breast cancer	Unknown	Intake of red meat may be associated with breast cancer; however, this correlation does not exist for BaP	Chronic	Negative
Gunter et al. (2005)	Case-control	Meat intake and preparation method	628 cases and 689 controls	Colorectal adenoma	> 0.29-515.2 ng/day	6% increase of large adenoma per 10 ng/day consumption of BaP	Monitored over the last year	Positive
Hakami et al. (2008)	Case-control	Intake through diet (staple foods including bread and rice) and water in northern Iran	40 cases, 40 controls from the same area and 40 from a low-risk region	Esophageal cancer	For bread + rice + water (data from previous study) total daily intake was 99.0, 91.4 and 70.6 ng/day for cases, controls of the same region and low-risk region, respectively	BaP was significantly higher in cases and controls of the high-risk area, in comparison with the low-risk area; BaP may be associated with esophageal cancer	Chronic	Inconclusive
Lam et al. (2009)	Case-control	Meat intake	2120 controls and 2101 cases	Lung cancer	Not well defined	Significant association for BaP, heterocyclic amines and red meat consumption	Chronic	Positive
Li et al. (2007)	Case-control	Meat intake and preparation method	626 cases and 530 controls	Pancreatic cancer	Mean of 69.9 in cases and 41.3 in controls. Median of 42.3 in cases and 37.3 in controls. These differences were not significant	BaP was a significant predictor of pancreatic cancer	Chronic	Positive
Sinha et al. (2005a)	Case-control	BaP intake from meat and other foods	146 cases and 228 controls	Colorectal adenoma	Median 5 ng/day from meat and 73 ng/day from food in controls; median 17 ng/day from meat and 76 ng/day from other foods in cases	Trend is significant for both meat and food sources; ORs from meat were 1.19, 1.71, 2.16 and 2.82 and from food were 2.61, 4.21, 4.25 and 5.6 for the second, third, fourth and fifth quintiles, respectively	Chronic	Positive
Sinha et al. (2005b)	Case-control	BaP intake from meat and processed meats	3696 cases and 34 817 controls	Colorectal adenoma (left-sided descending sigmoid colon and rectum)	0.8-168.1 ng/day	BaP intake associated with marginal elevation of colorectal adenoma risk	Chronic	Positive
Dermal exposure								
Scheepers et al. (2009)	Cohort	Nurses who apply ointments containing coal tar	35 nurses	Traces of BaP on hands with and without gloves and 1-hydroxyxypyrene urinary biomarker	33.0 and 16.4 ng/cm ² on hands	Use of gloves decreased absorption of BaP by 51.5%. Use of suggested protocol reduces absorption of BaP by more than 57%	Chronic	Positive
Human cells								
Rojas et al. (2004)		Smoking	22 normal bronchial epithelial cells obtained with lung parenchyma in smokers and nonsmokers with lung cancer	BaP BPDE-N2-dG (adducts)	Unknown	Adducts were significantly more elevated in smokers and in bronchial epithelia in comparison with parenchyma		Positive

Supplementary Table 3: All rodent and *in vitro* studies evaluated

Reference	Health effect category (reproductive toxicity, developmental toxicity, carcinogenicity, mutagenicity, etc.)	Exposure (acute < 1 month, subchronic 1-3 months, chronic > 3 months)	Route of exposure (Inhalation, oral, intraperitoneal)	Test material (vehicle, control group, doses, test limit, duration of exposure, dose selection rationale [range-finding study], purity, stability)	Test animals (species, strain, sex, number, age, acclimation, weight, environmental conditions, diet, water)	Clinical observations, necropsy, histopathology, KEY ENDPOINT	Key events (known or speculated)	Findings (MOAs, LD ₅₀ , NOAEL/LOAEL, p-value, relative toxicity compounds)	Quality of study (strengths and weaknesses; use appropriate statistics?)	Notes	
Aboutabl et al. (2009)	Cardiovascular toxicity	Acute	i.p. injection	20 mg BaP/kg bw/day for 7 days vs. corn oil control	SD rats n=6 adults	inc. heart rate, bw, cardiac hypertrophic markers, atrial natriuretic peptide, brain neuropeptide. Inc. gene expression of cyp1a1,1b1,2e1,4f4, inc. DHETs, EETs, 20-HETE, total EETs	MOA AHRnecessity test with inhibitors	weak. Weakness in MOA	single dose		
Aboutabl et al. (2011)	Cardiovascular toxicity	Acute	i.p. injection	20 mg BaP/kg bw/day for 7 days vs. corn oil control. With & without soluble hydrolase enzyme inhibitor (14.5 DHET:14.5-4ET)	SD rat n=2 adults	inc. heart rate, bw, cardiac hypertrophic markers, atrial natriuretic peptide, brain neuropeptide. Inc. gene expression of cyp1a1,1b1,2e1,4f4, inc. DHETs, EETs, 20-HETE, total EETs(effects blocked by inhibitor)	MOA combined role of AhR & Cyp450sin	only 1 dose tested	single dose		
Carlson and White (1983)	Cardiovascular toxicity	Acute	i.p. injection	40 mg BaP/kg bw in corn oil 72 & 48 hours prior to phenobarbital, trichloroethylene or halothane exposure	male new zealand rabbits BaP n=7, control n= 20	BaP weak direct effect to increase number of arrhythmias. BaP some how facilitates sensitization of myocardium to epinephrine by trichloroethylene to cause arrhythmias	no	No	many	N	
Gentner and Weber (2011)	Cardiovascular toxicity	Acute	Intranasal	14 days before dosing, surgery to implant BP device and medicated with indomethacin, bupivacaine and isoflurane, desensitized with isoflurane	male SD rats n=6/group	14 days before dosing, surgery to implant BP device and medicated with indomethacin, bupivacaine and isoflurane, desensitized with isoflurane altered circadian pattern of blood pressure. Increased neutrophil recruitment in lungs. No effect on CV tissue histology, arterial stiffness, oxidative stress or fund/layer cyp1a activity	no	no	poor stats, lack details to key study		
Karvala et al. (2004)	Cardiovascular toxicity	Acute	In vitro	1 μm BaP for 45 hours	Thoracic vascular SMCs from congenic AHR+/+ & -/-	Identified TGFβ2 & IGF-1 as potential candidates for an AHR alternative pathway for cells to respond to BaP	postulated	no	not a good mechanism and exploratory	key study	
Matsuura et al. (2009)	Cardiovascular toxicity	Acute	In vitro & intranasal	1 μm BaP for 45 hours.	Human monocyte-macrophage	non stated. More mechanistic study.	BaP binds AHR to alter vitamin D3 catabolism by	no	not a good mechanism and exploratory	key study	
NDiaye et al. (2006)	Cardiovascular toxicity	Acute	In vitro & intranasal	In vitro: Intranasal instillation of 500 μg BaP in tricaprylin vehicle under anesthesia for 24 hours.	Human primary macrophages. Male adult	CCL1 (chemokine involved in CV diseases and inflammatory response) altered in vivo lung & in vitro by AHD mechanism. Increased early macrophage stress or fund/layer cyp1a activity	postulated	no	mechanistic and exploratory	useful as study details	
NDiaye et al. (2009)	Cardiovascular toxicity	Acute	In vitro	1 μm BaP for 1 hour	Human primary macrophages.	mechanism of CCL1 induction via AHR vs lipoprotein A	postulated	no	mechanistic not useful as mechanism	mechanistic	
Oesterling et al. (2008)	Cardiovascular toxicity	Acute	In vitro	AhR activation with B-NF (1 μm) for 16 hours then BaP for 24 hours	n = 3 Primary umbilical cells (HuVEC)	Pro-inflammatory MOA involving ICAM-1, Cxcl10 leading to increased vascular endothelial adhesiveness which may be a critical step in development of BaP induced atherosclerosis. AHR dependent metabolism of BaP	postulated	MOA but not for key study	mechanistic	mechanistic	
Deslaurier-Brown et al. (2009)	Cardiovascular toxicity	Acute	In vitro	AhR activation with B-NF (1 μm) for 16 hours then BaP for 24 hours	n = 3 Primary umbilical cells (HuVEC)	Flavonoids can protect against BaP induced ICAM-1 mechanism described in Oesterling et al 2008	postulated	MOA but not for key study	mechanistic	mechanistic	
Podechard et al. (2009)	Cardiovascular toxicity	Acute	In vitro & intranasal	In vitro: 1 μm BaP for 24 hours. Vehicle ?? In vivo: 500 μg BaP for 72 hours n = 3?	Primary human macrophages & C57BL/6 mice	BaP represses NAD(P)H oxidation via AhR in macrophages which may contribute to macrophage lipid accumulation involved in CV disease no effect on material or fetal survival rates. Decreased maternal BW, placental toxicity, fetal rupture and hemorrhage of blood vessels in skin, cranial and brain tissues.	postulated	MOA but not for key study	poor reporting of study details	not useful as mechanistic	
Sanyal and Li (2007)	Cardiovascular toxicity	Acute	i.p. injection	50, 100, 200 mg BaP/kg bw or corn oil vehicle PD 10, 12, 14 evaluated on PD 10	SD rats group 60-day old SD rats	hemorrhage seen even at lowest dose but no stats	no	no	no dose response	LOAEL 50 mg/kg	
Sauzeau et al. (2011)	Cardiovascular toxicity	Acute	i.p. injection	BaP 10 mg/kg bw for 24 hours.	4-month old AhR and Vav3 knock-out mice. N=5-7	Constitutive AhR regulation of Vav3 proto-oncogene to control cardiovascular & respiratory functions does not require AhR activation by BaP	postulated	no	no dose response		
Kerrey-Hamilton et al. (2012)	Cardiovascular toxicity	Chronic	Gavage & in food	Gavage: 1mg/kg bw/day for 10 weeks n = 5. Food: BaP 200 mg/kg bw in corn oil fed chow (BaP 800 mg/ml corn oil) estimated dose 10 mg/kg bw/week. Weekly injection from 4 weeks old to 24 weeks old of 40 mg BaP/kg bw or DMSO, n = 6	B6 strain (immature) & C57BL/6 D3 strain 90w old male white Leghorn chickens	B6 strain: altered growth of body and several organs and induced atherosclerosis and to a greater extent than in B6-D2. BaP had big impact on gene expression of the aorta (immune responses, muscle-specific genes) which support a MOA of inflammation leading to muscle	postulated	MOA but not for key study	no sufficient to be key	no sufficient to be key	
Pen and Snyder (1988)	Cardiovascular toxicity	Subchronic	i.p. (pectoral injection)	DMSO, n = 6		After 4 days: increased BPDE-DNA adducts in aorta. Decreased LDL & increased HDL. Direct and lipidperoxidation-induced DNA damage by BaP in aorta.	postulated	no	no	no	
Sorensen et al. (2003)	Cardiovascular toxicity			Hypothetical MOA linking PAH with biological effects. Help with MOA but not useful as key study for BaP assessment							
Knapen et al. (2007)	Cardiovascular toxicity	Acute	Gavage	5 mg/kg bw or tricaprylin vehicle, 2 treatments 1 week apart	APOE-/- male mice 17 weeks old n=5	increased mild level of atherosclerosis. MOA : vascular pro-inflammatory effects of BaP by AHR-mediated induction of MCP-1 proposed to lead to increased atherosclerosis.	postulated	MOA but not for key study	not sufficient to be key	not sufficient to be key	
Godschalx et al. (2003)	Cardiovascular toxicity	Acute	Gavage	High fat/high cholesterol diet for 25 days. Single oral dose of 5 mg BaP/kg bw or tricaprylin vehicle	n=5 streptom/diet. Male APOE-/- mice	After 4 days: increased BPDE-DNA adducts in aorta. Decreased LDL & increased HDL. Direct and lipidperoxidation-induced DNA damage by BaP in aorta.	postulated	no	not sufficient to be key	not sufficient to be key	
Penn (1990)	Cardiovascular toxicity			Review paper with MOA, dose range in chicken and mice 0.1–40 mg/kg bw							
Ferguson (2009)	Cardiovascular toxicity			Review paper with proposed MOA: mutation of SMC genes relevant to CVD leads to recruitment of macrophages, buildup of foam cells, fatty deposits in vessel wall and then to atherosclerosis							
Yang et al. (2009)	Cardiovascular toxicity	Chronic	Gavage	Olive oil vehicle, 2.5 mg BaP/kg bw/week for 24 weeks	male C57BL/6 mice. APOL-/- overexpress SOD	Increase mean lesion size in aortic tree and root 60% & 40% and increased oxidized lipids. BaP didn't alter lipid peroxidation or atherosclerotic lesions in aortas of any of the APOL-/- plus overexpress. Implicate role of ROS in BaP-induced atherosclerosis	postulated	MOA but not for key study	not sufficient to be key	not sufficient to be key	
Curfs et al. (2005)	Cardiovascular toxicity	Chronic	Gavage	5 mg/kg bw or tricaprylin control 1 time/week for 24 weeks	Male APOL-/- 5 weeks old n=20	Male APOL-/- 5 weeks old n=20. Increased greater than 2-fold T-lymphocytes in plaques and increase TGFβ local response	postulated	no	mechanistic	mechanistic	
Curfs et al. (2004)	Cardiovascular toxicity	Subchronic	Oral	5 mg/kg bw or tricaprylin control 1 time/week for 12 weeks (n = 31) or 24 weeks (n = 19)	Male APOL-/- 5 weeks old	Adducts formed. BaP didn't initiate atherosclerotic plaques but accelerates the progression via a local inflammatory response	postulated	no	mechanistic and exploratory	mechanistic and exploratory	
Rennels and Moorthy (2005)	Cardiovascular toxicity			Review provides MOA information but no dose-response data							
Nakamura et al. (2012)	Developmental/reproductive toxicology	Acute	Oral gavage	BaP 0.2, 10 mg/kg bw (dose selection based on previous study in CD-1 mice)	Male Gelm-/- Mice (Glutamate Cysteine Ligase) castrated seminiferous tubules at age. Gelm-/- mice had more pronounced effects than their Gelm +/- littermates. Increased Timed pregnant F344 rats	Prenatal exposure to BaP from GD7-16 was dose-dependently associated with sign. 1 testicular and epididymal weights, sperm counts & B6 strain index related to # of birth, BaP metabolic disposition (plasma, cortex), relative % of BaP metabolites in plasma and cortex over time, AhR and Cypl4a increased	Data demonstrate an important role of AhR in experiment	Experiment 1	Fundings relevant to length of experiment		
Wang et al. (2003)	Development	Acute	Noise-only exposure	11-21	Timed pregnant F344 rats	Birth index (related to # of birth), BaP metabolic disposition (plasma, cortex), relative % of BaP metabolites in plasma and cortex over time, AhR and Cypl4a increased	Prenatal BaP exposure --> Lower birth index at 75 days post coitus	Lower birth index at 75 days post coitus	Lengthy how much		
MacKenzie and Angvine (1981)	Development	Acute	Oral gavage	BaP from Aldrich; in 0.2 mL corn oil. GD 7-16	CD-1 mice, 10 weeks old, doses: 0.10, 40, 160 % pregnant, % viable offsprings, mean litter size, reproductive capacity of these animals (treated in utero) later in life.	Unknown. Lower birth weight at birth, lowered pup weight	Unknown. More abnormalities in C57BL/6 AhR-sensitive	Table 1 good for BMD	Can't do BMD on the		
Shum et al. (1979)	Development	Acute	i.p. injection	BaP from Sigma, dissolved in corn oil. GD 7 or 10. C57BL/6 responsive	Developmental abnormalities were studied (% stillborn, resorptions, malformations, etc.). AhR.						
Ball (1970)	Immune	Acute	subcutaneous injection	Acute (one time) subcutaneous injection of BaP (60 and 120 μg) right after birth induced thymic lymphoma formation (56% incidence vs. 1.5% in controls for 120 μg) while no immunosuppression was observed (hemolysis response of sheep erythrocytes), unlike DMBA (100% thymic lymphoma, about 50% immunosuppression).							
Daynes et al. (1979)	Immune	n.a.	n.a.	This paper looked at cotreatments of BaP and immunosuppressive agents, but not at the immunosuppressive properties of BaP							
Urso and Gengozian (1980)	Immune	Acute	i.p. injection	i.p. injection of BaP(100 and 150 mg/kg bw) at middle (11–13 days) or late (15–18) gestational days led to (about 50%) suppression of the anti-sheep erythrocyte plaque-forming response shortly after birth in the progeny and persisted later into life. Tumor frequency was 76 for mid-gestation and 64% for late gestation compared with controls.							
Urso and Gengozian (1982)	Immune	Acute	i.p. injection	Mice were exposed during gestational development, postnatally or as adults (150 mg BaP/kg bw, once). Animals exposed prenatally were more susceptible to immunosuppression & tumor formation compared with those treated postnatally. Supports the idea that immune deficiency (suppression) influences tumor formation.							
Urso and Gengozian (1984)	Immune	Acute	i.p. injection	Pregnant mice received 150 mg BaP/kg bw during GD 11–17 by i.p. injection. Progeny were assayed for humoral and cell-mediated immune response at various intervals after birth. Immature offspring (1–4 weeks) were severely suppressed in their ability to produce antibody-(plaque-) forming cells (PFC) against sheep red blood cells (SRBC).							
Bushee et al. (1984)	Immune			These data show that <i>in utero</i> exposure to the chemical carcinogen BaP alters development of components needed for establishing competent humoral and cell-mediated functions of the immune apparatus and leads to severe and sustained postnatal suppression of the defense mechanism. The immunodeficiency exhibited, particularly in the T-cell compartment, is likely due to BaP's effect on the thymus.							
Dean et al. (1983)	Immune	Acute	subcutaneous injection	A combination of <i>in vitro</i> and <i>in vivo</i> experiments on sheep and sheep lymphocytes. Showed that BaP is taken by lipoproteins. "We propose that lipophilic xenobiotic compounds interact with cells of the immune system via lymphatic lipoprotein transport of potentially mutagenic, carcinogenic, or immunosuppressive agents."	Adult B6C3F1 mice were exposed to 10 daily subcutaneous injections of BaP in corn oil for 14 days (oil, 50, 200 and 400 mg/kg bw). Exposure of mice to BaP resulted in a reduced number of IgM and IgG antibody plaque-forming cells						
White et al. (1984)	Immune	Acute	subcutaneous injection	Female B6C3F mice, subcutaneous injection of 0.5, 20 or 40 mg BaP/kg bw in corn oil. On day 11, all mice received sheep red blood cells (SRBC, i.p.). On day 15 animals were sacrificed, spleens dissociated into single-cell suspension and were assessed for the number of anti-SRBC antibody-forming cells producing IgM. Plaques were counted.							
Wojdani and Alfred (1984)	Immune	Acute	i.p. injection	C57, C3H and DBA mice, 8 weeks old, were dosed with phytohemagglutinin injection to activate splenic lymphocytes and 24, 96 or 216 hours later were treated with BaP (0.2, 1, 10, 50 mg/kg bw BaP single i.p. injection in oil). Isolated, T-cell-enriched mononuclear cell populations were assayed for AhR hydroxylase activity. % positive.							
Holladay (1994)	Immune	Acute	gavage	<i>In utero</i> exposure to BaP (0, 50, 100, 150 mg/kg bw/day on GD 13–17 by gavage), and offspring were examined at GD 18. Thymic atrophy, cellular depletion (e.g. CD4+8 ⁺ fetal thymocytes). So, BaP → thymic hypopcellularity, inhibits thymomaturation process, fetal liver hypopcellularity, including cells with hematopoietic subpopulations. Data							
Urso (2008)	Immune	Acute	i.p. injection	Pregnant dams of C3H/Ant mice received 150 µg/kg bw on the 12th day of pregnancy by i.p. injection. On day 18, dams were killed and fetuses were removed. Livers were removed and processed (adherent [macrophages, B cells and others] and non-adherent cells [T cells] were collected). Then, splenic responder (R) and stimulatory (S) cells were isolated and cultured. Splenocytes were stimulated with Concanavalin A (ConA) and anti-mouse AhR antibody. Cell proliferation was measured by [3H]thymidine incorporation. The proliferation of R cells was significantly inhibited by AhR antibody, while S cells proliferation was not affected.							
Allan and Sherr (2010)	Immune		<i>in vitro</i> cells	Peripheral blood mononuclear cells (PBMC) were prepared from individual blood donors, depleted of T cells and stained with FITC-labeled CD2-specific antibody and purified (B cells). Plasma cell generation: B cells were plated in irradiated CD40L-transfected L cells. After 4 days of coculture, B cells were harvested and secreted cytokines were measured.						REVIEW IN	
Fischer et al. (2011)	Immune	Subchronic	oral	Wistar rats, 9–13 weeks old, were fed daily with 150 mg/kg bw for 30 days. Bone marrow and spleen cell numbers were reduced in BaP-treated samples (~70% and 45% reduction, respectively). This is just one of the experiments. Also did some <i>in vitro</i> (cell culture) experiments.							
Smith (2010)	Immune	Subchronic	subcutaneous injection	Compared several models of delayed hypersensitivity using mouse foot pad swelling as endpoint. Female B6C3F1 mice 8–15 weeks of age were challenged with BaP in corn oil sc (subcutaneously) for 14 days at 5, 20 and 40 mg/kg bw. Then, three different antigens were used, and BaP-mediated effect was seen in all three						Not very	
Urso (2008)	Immune	Acute	i.p. injection	In this study, C3H mice were injected once with BaP (150 µg/kg bw) at day 12 of pregnancy and progeny lymphoid tissues were excised during gestation (day 18, GD 18) or at 1 or 6 weeks postpartum. The isolated lymphoid cells were analyzed by flow cytometry/immuno/fluorescence or assessed for function. In BaP-exposed fetuses, thymic T-cell proliferation was significantly reduced, while splenic B-cell proliferation was increased.						just	
Davila et al. (1996)	Immune		<i>in vitro</i> human cells	Examined the toxic effects of nine different PAHs on human peripheral blood T cell mitogenesis. Found that BaP was highly immunotoxic. Also showed that α-naphthoflavone (ANF), which functions as both an AhR antagonist and an inhibitor of cytochrome P450 activity, was able to block the suppressive effect of BaP. Mitogenesis = induction							
Allan et al. (2006)	Immune		<i>in vitro</i> human cells	The AhR is a ligand-activated transcription factor that mediates immunosuppression by environmental PAHs. Previous studies demonstrated that activation of mature human B cells upregulates AhR expression, suggesting that human B cells are direct PAH targets. To test this hypothesis and to determine the metabolic requirements for AhR activation, we used a panel of human B cell lines and primary B cells. Our results indicate that AhR activation is dependent on metabolic activation of BaP.						Very nice	
Galván et al. (2006)	Immune	Acute	i.p. injection	Four-week-old C57BL/6 mice were injected intraperitoneally with 50 mg BaP/kg bw. Mice were sacrificed 12, 24 and 48 hours after intraperitoneal injection with oil vehicle, BaP and bone marrow cells were purified. BaP treatment decreased the pre/pro B-lymphocytes and did not affect the immature B-lymphocytes or mature data.						NOT BMD	
Lee and Urso (2007)	Immune		<i>in vitro</i> studies with isolated spleen cells	Spleens of C3H/HeJ (source of responder, R cells) and CBV/D2 (source of stimulator cells, S cells) mice, 11–12 weeks of age were removed and made into cellular suspensions. The effect of BaP on the cellular proliferative response in response to allogeneic mixed lymphocyte response (MLR) on concanavalin (only R cells are BMD; this is						Nothing for BMD; this is	

De Jong (1999)	Immune	Subchronic	oral gavage	Followed OECD 407 study protocol. Used male Riv-Tox Wistar SPF rats, 6 weeks of age, treated with BaP (0, 3, 10, 30, 90 mg/kg bw/day) in soybean by gavage for 35 days (5 days/week, Monday to Friday). Reduced thymus weight was observed starting at 10 mg/kg bw. Thymus, the organ suggested for immunosuppression ex BEST data for modelling wide range.
Silkworth et al. (1995)	Immune	Acute	oral	C57BL/6 (AhR ^{+/+}) and B6 D2 (AhR ^{-/-}) mice were used to evaluate immunosuppressive potential of PAHs. Immunosuppression was quantified as the number of antibody-producing cells in spleen following sheep erythrocyte immunization. BaP was able to show immunosuppressive effect in AhR ⁺ animals, but concentrations dose range.
Rodriguez (1999)	Immune	Acute	i.p. injection	Compared frequency of T cells in the spleen of mice progeny treated with BaP <i>in utero</i> (150 mg BaP/kg bw by single i.p. injection in corn oil at day 11 of pregnancy. Found significant reduction in certain T cell types in 1-week-old progeny from BaP-treated C3H/HeB mice.
Rodriguez (2002)	Immune	Acute	i.p. injection	Similar study design as above. In addition to reduced number of T cells, saw BaP-DNA adducts in maternal, placental and progeny (immature T cells).
Wolisi et al. (2001)	Immune	Acute	i.p. injection	Thymectomized (without thymus) mice and their progeny were studied (C3H/HeB mice). Thymus was excised, and mice were mated, then injected with 150 mg BaP/kg bw once by i.p. injection at day 12 of pregnancy. Thymectomy+BaP reduced litter size by 40%. Increased thymic cell-mediated immunity in progeny of BaP + thymectomized mice.
Near (1999)	Immune	in vitro human cells		Bone marrow-derived preB cell line was cocultured with bone marrow-derived stromal cell line and with Hepa1c1c7 were treated with BaP. Saw apoptosis and blockage of that by aNF: AhR antagonist; therefore, apoptosis is dependent on AhR activity.
Krieger (1994)	Immune	in vitro human cells		The four references below are Ca ²⁺ -related.
Krieger (1995)	Immune	in vitro human cells		BaP produced sustained (4 hours after stimulus) increase in intracellular Ca ²⁺ in human T cell line → altered calcium homeostasis.
Romero (1997)	Immune	in vitro human cells		aNF, inhibitor of Cyp450 and AhR antagonist, reduced Ca ²⁺ elevation seen in human peripheral blood mononuclear cells; 1–10 μM BaP did not affect glutathione (GSH) concentration when exposed to BaP for 6, 48 or 72 hours, but reactive metabolites of BaP (diol epoxide and 4,5-epoxide) produced 20–30% GSH depletion after 6 hours. Conclusion: BaP may alter Ca ²⁺ in cells, T cells, monocytes and P450 metabolism may play a role in the immunotoxicity.
Mounou (1997)	Immune	in vitro human cells		BaP produced significant increase in intracellular Ca ²⁺ in certain T and B cells in human peripheral blood mononuclear cells. BaP metabolites were more effective than BaP. Conclusion: PAHs may alter Ca ²⁺ in cells, monocytes and P450 metabolism may play a role in the immunotoxicity.
Lyte (1986)	Immune	in vitro cells		A dose-dependent increase in lipopolysaccharide-stimulated IL-2 production, concomitant with decreased cell viability, was seen for macrophages treated with BaP. Peritoneal exudate macrophages from 8- to 12-week-old B6C3F1 mice were used. BaP was added to cell culture plates with LPS. Mice were also subcutaneously injected with LPS.
Ladies 1992a	Immune	in vitro cells		Murine splenic macrophages were incubated with radioactively labeled BaP and BPDE generation was measured and found to be Cyp450-dependent and independent pathways based on the ratio of anti/syn BPDE (450/peroxyl radical pathway).
Ladies 1992b	Immune	subcutaneous injection		B6C3F1 female mice received a daily s.c. injection of 0.01 ml/g bw for 4 days in the lower back region to yield a daily dose of 200 mg/kg bw produced equivalent immunosuppression [no PFC in response to sheep erythrocytes] as 14-day treatment with 40 mg/kg bw/day. BaP metabolites were seen in splenocytes, and the metabolite conce.
Van Crevelingen et al. (2003)	Immune	in vitro human cells		Peripheral blood mononuclear cells were from blood of human donors. BaP inhibited differentiation of human blood monocytes and formation of adherent macrophagic cells derived from monocytes in response to cytokines without altering cell viability or inducing apoptosis. The use of aNF counteracted inhibitory effects of BaP. Conclusion: BaP may alter Ca ²⁺ in cells, monocytes and P450 metabolism may play a role in the immunotoxicity.
Holladay (1995)	Immune	Acute	i.p. injection	B6C3F1 mice were treated with 0.50 or 100 mg/kg bw/day by i.p. injection in corn oil for 5 days. Thymuses and spleens were collected from mice, homogenized. Bone marrow hematopoietic cells were also collected. Thymic cellularity and splenic cellularity (no. of cells) were decreased by BaP treatment. Same in bone marrow. Conclusion: BaP may alter Ca ²⁺ in cells, monocytes and P450 metabolism may play a role in the immunotoxicity.
Blanton (1986)	Immune	Acute	subcutaneous injection	14-day exposure of C57BL/6 mice (0 and 40 mg/kg bw/day s.c.) were studied. BaP was then isolated, challenged with sheep erythrocytes and no. of PFCs were measured after 5 days. BaP alone completely suppressed immune response and LPS and PPD-induced no. of PFCs. Culturing spleen cells <i>in vitro</i> and subjecting them with BaP induce.
Knuckles et al. (2001)	kidney	subchronic	oral-diet	0, 5 or 100 mg/kg-day BaP for 90 days
Bouayed et al. (2012)	Neuro	Acute (daily for 17 days)	i.p. injection	BaP from Sigma Aldrich (St. Quentin, Fallavier, France) – purity not specified, dissolved in avocado oil (0.02–0.2–2–20 mg/kg bw).
Chen et al. (2012)	Neuro	Acute (daily for 6 days)	Oral gavage	BaP stock solution was prepared by mixing the compound with 0.9% NaCl. BaP (0.09M/1400V) stock solution was prepared by mixing the compound with 0.9% NaCl. Male Sprague-Dawley rats (4 weeks old); n=8 for each
Qiu et al. (2011)	Neuro	Subchronic (each day for 14 weeks)	i.p. injection	BaP at 99% purity (Sigma-Aldrich) was administered in peanut oil, 2 mg/kg bw/day for 14 weeks. Morris water maze performance was evaluated at day 14. BaP was then isolated, challenged with sheep erythrocytes and no. of PFCs were measured after 5 days. BaP alone completely suppressed immune response and LPS and PPD-induced no. of PFCs. Culturing spleen cells <i>in vitro</i> and subjecting them with BaP induce.
Chengzhi (2011)	Neuro	Subchronic	Gavage	BaP stock solution was dissolved in DMSO and subsequently diluted in corn oil. BaP stock solution was dissolved in DMSO and subsequently diluted in corn oil. BaP was randomly assigned to one of the following four treatment groups: BaP (100 mg/kg bw/day), BaP (10 mg/kg bw/day), BaP (1 mg/kg bw/day) and vehicle (0 mg/kg bw/day).
Xia et al. (2011)	Neuro	Subchronic (once daily for 13 weeks)	i.p. injection	Na ⁺ , ACh ⁺ and ChAT ⁺ (see → for abbreviations). Endogenous monoamine levels.
Sheng (2010)	Neuro	Acute	Oral gavage (to pregnant dames)	On embryonic day (ED) 14–17, Cpx/lox dams were exposed to BaP (150, 300 and 600 µg/kg bw/day) or oral gavage. The proper controls for the BaP-treated dams were vehicle-treated dams.
Dutta (2010)	Neuro	Acute	i.p. injection	BaP (0.02, 0.05, 0.1 and 0.2 mg/kg bw) were used for 4 consecutive days. BaP (0.02 and 0.2 mg/kg bw) dissolved in avocado oil (Cauvin, France) and avocodo oil (control mice). Doses are per day, for 21 or 28 days, n = 9–9.
Bouayed (2009a)	Neuro	Subchronic	"Oral route"	We used Swiss albino male mice (OFL) 9 weeks old. BaP was dissolved in olive oil. On day 21, the effects of BaP ¹⁷ on the copulatory behaviour were evaluated by using the resident-intruder test. On day 28, the effects of BaP ¹⁷ on the copulatory behaviour were evaluated by using the male sexual behaviour test.
Bouayed (2009b)	Neuro	Subchronic	Lactational	Nursing females received a daily oral administration of 0.05% BaP ¹⁷ or vehicle (0%) for 10 days. BaP ¹⁷ was dissolved in olive oil.
McCallister (2008)	Neuro	Acute	Oral gavage (to pregnant dames)	Number of pups, body weight, toxification and detoxification BaP metabolites (7,8-diol and 3-OH BaP, respectively), NR2B (glutamatergic NMDA receptor
Konstantin et al. (2007)	Neuro	Acute (10 days)	i.p. injection	BaP source not mentioned.
Groves et al. (2007)	Neurotoxic	Acute (10 days)	i.p. injection	BaP > 97% purity; Sigma
Neurotoxic	Neuro	Acute (4 days)	Gestational (oral gavage to mothers)	BaP source not specified.
Brown et al. (2007)	Neuro	Acute (14–17)	Gestational (oral gavage to mothers)	Timed-pregnant dams were exposed by oral gavage to BaP and BaP and metabolites in liver, hippocampus and cortex. Glutamate subunit NR2B and GlutR1 mRNA expression were measured.
Saunders et al. (2006)	Neuro	(acute)	Gavage	BaP (97% purity; from Aldrich Chemical Company, St. Louis, MO; BaP was dissolved in peanut oil).
Womley (2004b)	Neuro	(acute) (Acute (GD 11–21); in utero)	Nose-only exposure	Maternal effects (final total rat brain trichloroethane (TCE) metabolites, Anticoagulant Activities (SOD, CAT, GPx), Lipid peroxidation were measured. Pregnancy outcome (# births), electrophysiological parameters (evoked field potentials in response to entorhinal cortex stimulation, long-term potentiation using tetanus stimulation); NMDA receptor subunit 1 protein expression were measured.
Saunders et al. (2003)	Neuro	Acute		See Saunders 2006 above.
Tang (2003)	Neuro	in vitro human cells		BaP from Sigma Chemical Co. (St. Louis, MO). BaP was prepared as DMSO stock, 30 mM and used at 0, 0.03, 0.3, 3 and 30 µM final concentration in the
Wu et al. (2003)	Neuro	Acute (GD 11–21), in utero	Nose-only exposure	A. incorporation into proteins, total protein, triplar blue dye exclusion, chlorophenol cytotoxicity, AChE activity were assayed. B. BaP (97% pure) were purchased from Sigma Chemical Co. (St. Louis, MO) in peanut oil (research grade, Sigma Chemical Co., St. Louis, MO).
Saunders (2002)	Neuro	Acute (once)	Oral gavage	Timed-pregnant Sprague Dawley rats were exposed to BaP (100 mg/kg bw/day) for 1 week. BaP was dissolved in olive oil.
Saunders (2001)	Neuro	Acute (once)	Oral gavage	BaP (97% purity) as specified by the supplier was obtained from Sigma Chemical Company (St. Louis, MO).
Hood (2000)	Neuro	Acute (in utero and another group)	Nose-only exposure	Unspecified source.
Stepanova et al. (1998)	Neuro	Acute (single dose and twice per week)	i.p. injection	Adult male CD-1 mice (100 mg/kg BaP in olive oil
Jayasankar et al. (1992)	Neuro	Acute (twice per week, 3 weeks)	i.p. injection	Adult male CD-1 mice received 0, 5, 25, Maternal effects (final total rat brain trichloroethane (TCE) metabolites, Anticoagulant Activities (SOD, CAT, GPx), Lipid peroxidation were measured. Pregnancy outcome (# births), electrophysiological parameters (evoked field potentials in response to entorhinal cortex stimulation, long-term potentiation using tetanus stimulation); NMDA receptor subunit 1 protein expression were measured.
Arafat et al. (2009)	Reproductive	Acute	Oral	BaP (50 mg/kg bw/day; hepardin (HNDN, a citrus flavonoid); 200 mg/kg bw/day; vehicle = olive oil (0.2 mL/100 g/day); exposure = 10 days. Testicular toxicity - Acute (10 days)
Archibong et al. (2008)	Reproductive	Subchronic	Inhalation (nose-only)	BaP: 75 µg/m ³ ; Exposure: 4 hours/day for 60 days (one sperm cycle) Dose selection rationale: exposure concentration selected as it adversely affected reproductive outcomes in male and female rats in a previous study by these authors and is within a range present in environmental sources of BaP
Bui et al. (1986)	Reproductive	Acute	Subcutaneous	BaP and methanol both affected reproductive performance in pregnant rats (sign. ↑ # resorptions and fetal wastage, ↓ fetal weight, ↓ live fetuses); in pseudo-pregnant rats BaP ↑ uterine wet weight & cAMP & cGMP levels, methadone was without effect, no maternal deaths in either group
Chen et al. (2011)	Reproductive	Subchronic	Oral gavage	BaP: 50 mg/kg bw in corn oil (2 mL/kg bw); methadone: 5 mg/kg bw in saline (10 mL/kg bw); dose selection of BaP based on previous work in that laboratory, some species injected given days prior to gestation or days 6–11 in pregnant rats, in the treatment and pseudopregnant (PSP) rat; Acute (three injections for pregnant rats or six injections for PSP rats)
Craig et al. (2011)	Reproductive	Review		This review summarizes the effects of EDCs on ovarian function by describing how they interfere with hormone signaling via 2 mechanisms: (1) altering the availability of ovarian hormones (expression and/or activity of enzymes required for synthesis and/or catabolism of ovarian sex steroids); and (2) altering binding and activity of the hormone at the receptor level (alter the expression of hormone receptors and/or their ability to bind their endogenous ligands).

Dent (2007)	Review: Strengths and limitations of using repeat-dose toxicity studies to predict effects on					Could rodent functional effects be predicted in a sub-chronic study? Yes/No (Reference) If yes, nature of effect: For BaP - Yes (Archibong et al., 2003) Testis: reduced weight; sperm analysis: reduced sperm motility and density (no testicular histology quoted). Also raised LH levels. The chromium compounds, some of the cadmium compounds (cadmium fluoride, cadmium chloride, and cadmium sulphate), benzol[ah]yrene, 1,2-dihydronaphthalene-3-chloropropene, 2-	The testicular effects that were predictive of functional effects could be the result of a number of toxic modes of action some of which may be shared with BaP.	Intelligent testing strategies therefore need to be developed which can take into account data from a wide range of toxic modes of action some of which may be shared with BaP.	Review focuses on a panel of test chemicals to determine priorities.		
Kristensen et al. (1995)	Reproductive	Acute	Oral	Four treatment groups (n = 9/group): (1) control; (2) lead (F ₀ given 1 g PbCl ₂ /L in drinking water until mating); (3) BaP (10 mg/kg bw daily by oral gavage on days 7-16 of F0 pregnancy); (4) combined lead and BaP. Dose selection rationale: lead dose was comparable to blood levels seen in occupational	Male and female F0 Female B6N:NNRj Mice were mated. At 9 weeks of age, F0 females were assigned to treatment	F1 groups exposed prenatally to BaP showed markedly reduced fertility with few ovarian follicles and corpora lutea, 1/ovarian weight; non-significant indication that the compounds in combination ↓ # offspring, # litters, and litter size; results suggest that lead and BaP have synergistic effects on the impairment of fertility			Authors state that the dose selection for lead and BaP were probably		
Mackenzie and Angevine (1981)	Reproductive (bit of developmental)	Acute	Oral gavage	BaP (0, 10, 40 or 160 mg/kg/day) by gavage on days 7-16 of gestation. Corn oil vehicle, controls given corn oil. Male breeding study: F1 males was placed with untreated females at 6 weeks of age. At 14 weeks of age, 19 females were sacrificed and 4 implants, fetuses and resorptions were recorded (F2 young were examined for gross abnormalities). Female breeding	Adult male and female CD-1 Mice; each test group with 10 mice pregnant dams	No maternal toxicity or embryolethality at any dose. Decrease pup weight all doses 160 mg/kg-day of BaP resulted in a reduced percentage of mice that were pregnant, reduced number of viable litters at parturition, and reduced mean pup weight at day 42 post-partum. Mean pup weight was significantly lower than control at 160 mg/kg-day. BaP Maternal offspring of dams treated with 10, 40 or 160 mg/kg-day and then bred with untreated females yielded decreased fertility (10 mg/kg or greater, with almost no pregnant females when the males were exposed to either 40 or 160 mg/kg-day in utero. Similarly, fertility was reduced in female	LOAEL: 10 mg/kg/day decreases fertility index in both males and females. Data demonstrate a sensitivity of fetal gonads	Multi-generational effects on the future of F0 pregnant females for 10			
Mukhopadhyay et al. (2010) Fertility and Sterility 94(2): 595-598.	Reproductive	Acute	In vitro (BaP was mixed with media)	In vitro (BaP was mixed with media)	Semen collected from 13 fertile, normozoospermic, non-smoking men; spermatozoa were washed and treated with BaP: 12.5, 25, 50, 100 µg/mL. Dose range selection was based on previously described studies and sperm survival studies done in their lab (1 hour exposure)	A statistically significant ↑ in sperm hyperactivation was observed at concentrations of BaP ≥ 50 µg/mL. Acrosome Halo Test: [BaP] ≥ 50 µg/mL significantly ↓ the percentage of halo formation, indicating an inappropriate (false) acrosome reaction	Conclusion: BaP significantly affected sperm functional competence in vitro, as evidenced by the increased	Acute in vitro study, but results seen were not statistically			
Tuttle et al. (2009)	Reproductive	In vivo subchronic (Inhalation (nose-only))	In vitro acute (24 hours)	In vivo: 2 cigarettes/day, exposed 5 days/week for a total of 8 weeks, including the 2-week lead-up period. In vitro BaP: 1-10000 ng/ml (24 h exposure)	Mice and isolated murine ovaries (in vivo/in vitro); female C57BL/6 mice (6-8 weeks old)	Ovaries were analysed for follicle loss and markers of apoptosis (TUNEL, Caspase 3, Caspase 8, Bax, Bel-2, Fas and FasL). Cigarette smoke exposure induced a significant ↓ in the number of primordial follicles, but not growing or antral follicles. Mainstream cigarette smoke exposure had no effect on any marker of apoptosis measured. Exposure of ovaries to BaP in vitro resulted in ↑ in the expression of the anti-apoptotic marker Bel-2, but no change in apoptosis. Significant reductions in the number of follicles in ovaries and ↓ in the number of antral follicles in isolated murine ovaries were also observed.	Data suggest that cigarette smoke-induced follicle loss is not mediated via BaP-induced apoptosis. Findings suggest that a decreased	Physiologically relevant exposure to cigarette smoke does not increase the rates of apoptosis in the ovary and hv.	Good study with in vivo and in vitro components to the research		
Nakanura et al. (2012)	Reproductive	Acute	Gavage	Dams were treated by oral gavage with 2 or 10 mg/kg BaP (Sigma-Aldrich Supelco, ≥99.8% purity) in sesame oil daily from GD7 to GD16. Control animals were gavaged with the same volume of sesame oil alone.	C57BL/6J genetic background (B6 129-Gelmintr1Tjk); hereafter referred to as Gelm(-)	We observed no changes in fertility, testicular weights, testicular sperm head counts, or testicular histology and subtle changes in cauda epididymal sperm counts, motility, and morphology in Gelm(-) males. Prenatal exposure to BaP from gestational day 7 to 16 was dose-dependently associated with significantly decreased testicular and epididymal weights, testicular and epididymal sperm counts, and with vacuolated seminiferous tubules at 10 weeks of age. Gelm(-) males exposed prenatally to BaP had greater decreases in testicular weights, testicular sperm head counts, epididymal sperm counts and epididymal sperm motility than Gelm(+) littermates. These results show no effects of BaP on testicular and epididymal follicle growth & development at concentrations of 5 mg/ml and higher, an effect attenuated by co-treatment with AhR antagonists. BaP caused a significant ↑ in oestriadiol and anti-Müllerian hormone (AMH) output, an effect attenuated by co-treatment with 1 of the 2 AhR antagonists tested.	Results suggest that the adverse effects of BaP on follicle growth, steroidogenesis and AMH output are mediated	In vitro model best represents in vivo scenario as concentrations			
Paltanaviciene et al. (2006)	Reproductive	Acute	Oral	Cadmium: 0.1, 0.5, 1.0, 1.5, 5.0 and 50.0 mg/kg; BaP: 0.00015, 0.0015, 33.3 (?) Toto polychlorinated biphenyls: 200, 400, 800, 1600, 3200, 6400, 12800, 200, 90.0 mg/kg; Exposure duration: 14, 28, 56, 90, 180 d. Combined exposures performed; oral administration at 1ml/100g BW/day. Vehicle = rapeseed oil; 40 groups, 6 control groups (3 water, 3 oil), n=7-10/group	Male Wistar rats (-320 total); age = 6-7 weeks; weight = 130 ± 40g;	Combined effects included: ↓ Spermatoids, ↓ viability, ↓ duration of motility, ↓ resistance in NaCl and HCl; relative weights of testicles, seminal vesicles & epididymis (data ambiguous due to combined dosing of 3 different substances)	Combined effects of these substances were similar to the effect seen by isolated substance based on test parameters	Poor quality of writing was poor (English was not the native language)	Looesley based on OECD test guidelines 407 and 408		
Ramesh et al. (2008)	Reproductive	Subchronic	Inhalation (nose-only)	Treatment group = 75 µg BaP/m ³ , 4 h daily for 60 days (1 sperm cycle). Control group = unexposed (UNC). Dose selection rationale: exposure concentration selected as it was adversely affected reproductive outcome in male and female rats in our previous study by these authors and is within a range of concentrations used in other studies.	Adult Male F-344 rats; age = 12-13 weeks; weight = 340-360g; n=10/group; BaP-DMSO ; BaP-vehicle ; BaP+carboxy-BaP ; BaP+baicalin	Blood samples were collected on day 60 (time 0), and subsequently at 24, 48 & 72 hr to assess plasma testosterone (T) and luteinizing hormone (LH). BaP exposure reduced testis weight and caused significant reductions in the components of the steroidogenic and spermatogenic compartment of the testes. Progressive motility and mean density of stored spermatozoa were reduced. Plasma [T] were decreased by two-fold and LH was increased by three-fold with increasing concentrations of BaP in BaP-exposed rats. [BaP]metabolites of tobacco	MOA: AhR and ARNT discussed	These data suggest that sub-chronic exposure to inhaled BaP causes reduced testicular and endometrial	Well written paper with many relevant references		
Sadeu and Foster (2011) Reproductive Toxicology 31: 402-408.	Reproductive	Acute	In vitro	BaP: DMSO & 0 ng/ml (BaP controls), 1.5, 5, 15 & 45 ng/ml. Dose Selection is representative of follicular fluid concentrations in women exposed to mainstream and/or sidestream cigarette smoke. sub-acute (13 days)	Follicles (100–130 µm) isolated from ovaries of F1 hybrid (C57BL/6J × CBA/Ca) mice (13 days post-puberty)	BaP treatment inhibited (p < 0.05) antral follicle development, decreased estradiol output and follicle survival had no effect on progesterone output or oocyte growth and nuclear maturation in surviving follicles.	Data suggest that BaP is an important toxic component of cigarette smoke that adversely affects regular follicles	The advantage and physiologically relevant aspect of the long-term			
Swartz and Mattison (1985)	Reproductive	Acute	Single i.p. injection, studied at weekly intervals from 1 to 4 weeks post-inj	BaP: 1, 5, 10, 50, 100 & 500 mg/kg. Vehicle = corn oil. 1 acute exposure study; 1, 2, 3 & 4 weeks post-exposure (designed to study the effect of BaP on ovarian function in intact mice not stimulated with exogenous gonadotropins).	Female C57BL/6N Mice; 6 weeks of age; water; standard chow ad libitum; 1 week acclimation before study	Dose and time dependent decrease in # of corpora lutea; General histological appearance of the ovary was assessed. # of corpora lutea counted; morphological variation was observed in treated mice; ovarian toxicity noted in highest dose group (complete absence of folliculogenesis); BaP had a 35% mortality rate at 500 mg/kg.	General histological appearance of the ovary was assessed. # of corpora lutea counted; morphological variation was observed in treated mice; ovarian toxicity noted in highest dose group (complete absence of folliculogenesis); BaP had a 35% mortality rate at 500 mg/kg.	These data suggest that external factors, like cigarette smoking, may be hazardous to the viability and function of follicles.			
Xu et al. (2010)	Reproductive	Subchronic	Oral	BaP: 5 & 10 mg/kg; di(2-ethylhexyl)phthalate (DEHP): 300 & 600 mg/kg or combination of BaP + DEHP, corn oil (control). Exposure: alternate days for 60 days; Dose Selection: dietary exposure to BaP and DEHP is likely as both are found in urban and rural water. Doses used are higher than the levels found in the general environment, but may be relevant in cases where the specific	Feminist Sprague-Dawley Rats; age = 5 weeks; weight = 80-100g; age = 5 weeks; access to purified water and standard chow	BaP and DEHP exerted cytotoxicity and suppression of sex hormone (17 β -estradiol), secretion and homeostasis, which is associated with prolonged estrous cycles, decreases in ovarian follicle populations and granulosa cell apoptosis involving a PPAR-mediated signaling pathway of action of the two chemicals. [ovarian weight & ovary:weight/BW ratios in treatment groups; P450 arom mRNA & protein expression. With ↑ in the dose of BaP + DEHP, there was a dose-response trend of prolonged duration in the EC and Nox/F phases of the estrus cycle	Granulosa cell apoptosis involving a PPAR-mediated signaling pathway of action of the two chemicals. Some AhR could interfere with BaP	Combined toxicological assessment of the combined toxicity; no interaction effects were observed following combined	Good study that looked at mRNA and protein levels		
Zenzenes (2000)	Reproductive	review paper	Inhalation (smoking)	BaP in cigarettes: 6-40/cigarette; 20 cigarettes in 8 h, could inhale 0.067-0.568 ug of BaP	Adult male Sprague-Dawley rats; age = 3 weeks	BaP's reactive metabolite binds covalently to DNA, forming adducts. Smoking-related adducts were detectable in ovarian granulosa-lutein cells, oocytes, spermatozoa & preimplantation embryos. Translocation of altered DNA from smoking by spermatozoa was demonstrated in preimplantation embryos and in association with increased risk of childhood cancer.	Discusses DNA damage with respect to adducts specific to BaP	Excellent review; discusses the role of BaP in carcinogenesis and of various forms of BaP; (2) smoking and clinical genotoxic outcomes in humans	Read this review paper		
Zenzenes et al. (1995)	Reproductive	Chronic	Inhalation (smoking)	Women included in study categorized based on smoking frequency (meiotic maturation of human oocytes) - chronic	Human oocytes - A total of 156 (54 smokers & 102 nonsmokers) women undergoing IVF, classified into four groups based on smoking status: non-smokers (n=102), light smokers (n=54), moderate smokers (n=30), and heavy smokers (n=10)	Found higher frequencies of diploid oocytes in smokers than in non-smokers, and a very significant dose effect (increased frequency with increased smoking). The observed increased proportion of analyzable oocytes in the smoker group suggests an earlier delay in oocyte maturation, compared with non-smokers.		Study shows that external factors, like cigarette smoking, may be hazardous to the viability and function of follicles.	Focus on human oocytes and smoking (not specifically BaP)		
Zhao et al. (2011)	Reproductive	Chronic	Oral - Nanjing city (China) tap water	Control = drinking water free of toxins; treatment = Nanjing tap water with contaminant levels determined by GC/MS. (90 days)	Male mice; age = 3 weeks; n=10/group; weight = 18±1g	In treated mice, flow cytometry analysis of testicular tissue indicated that the relative percentage of the elongated spermatid (IC) decreased significantly. Also slight increases in the relative percentage of round spermatids (IC) and primary spermatocytes (40) were noted. The ratios of 4C:2C (diploid germ cells) and 1C:2C increased, and testicular histopathology indicated an expansion of interstitial space and a decreased number and size of Leydig cells in treated mice.		Current study suggests that Nanjing tap water is toxic to the reproductive system of mice and additional study is needed to further elucidate the mechanism(s).	Drinking water study, including polycyclic aromatic hydrocarbons	Contaminant drinking water study, not specific to BaP	
Zheng et al. (2010)	Reproductive	Chronic	Oral gavage	(90 days) Rats were randomly divided into 7 groups; 2 groups received DBP + BaP (DBP+BaP; 50+1 or 250+5 mg/kg/day), 4 groups received DBP or BaP alone (DBP: 50 or 250 mg/kg/day; BaP: 1 or 5 mg/kg/day), and 1 group received vehicle alone (corn oil = control). Dose selection: little	Adult male Sprague-Dawley rats; age = 5 weeks old; BW = 70-100g; n=16/group	ED20 testicular macrophages (reactive with a differentiation-related antigen present on the resident macrophages) were activated and IL-1 β secretion was enhanced. DBP and BaP acted additively, as demonstrated by greater IL-1 β secretion relative to each compound alone. These observations suggest that exposure to DBP plus BaP induced greater suppression on testosteron production compared with each compound alone. DBP and BaP enhances the mRNA and protein expression of IL-1 β in testicular macrophages. Exposure to DBP and BaP alters testicular macrophage subset expression and enhances the ability and efficiency of resident macrophages to secrete IL-1 β and significantly suppresses testosteron	Hypothetical representation of the effects of chronic exposure to DBP and BaP on testicular macrophages. Both BaP and DBP had additive effects on increased IL-1 β secretion in rat testicular macrophages. BaP and DBP increased IL-1 β secretion in rat testicular macrophages, although this did not occur in a dose-dependent manner.				

Supplemental Table 4. BMD model fit parameters for apical endpoints in Table 3. Benchmark response: $BMD_{10}/BMDL_{10}$ for quantal data (tumor) and $BMD_{ISD}/BMDL_{ISD}$ continuous data (Neurotoxicity, developmental, immunotoxicity and mutations).

Reference	Effect	Best Model	AIC	P-value	BMD	BMDL
Chen et al. (2012)	Developmental toxicity	Hill	191.1	1.0	0.09 (0.05†)	0.05 (0.02†)
De Jong et al. (1999)	Immunotoxicity	Exponential	333.5	0.6	14.0 (7.6†)	8.9 (4.8†)
Lemieux et al. (2011)	Mutations: forestomach	Linear	148.3	0.6	0.49	0.3
Lemieux et al. (2011)	Mutations: lung	Linear	137.7	0.1	2.2	1.4
Lemieux et al. (2011)	Mutations: liver	Power	121.1	1.0	7.2	4.8
Culp et al. (1998)	Forestomach Tumor	LogLogistic	96.5	0.4	0.83	0.54
Wester et al. (2012)	Forestomach Tumor	Multistage	179.9	1.0	1.54 (0.83†)	0.75(0.41†)
Wester et al. (2012)	Liver Tumor	LogLogistic	127.8	0.5	3.27 (1.77†)	2.36 (1.28†)

† For comparison of rat and mouse BMDLs were scaled from rat to mouse by multiplying rat values by C based on the assumption that the physiological processes scale with body weight to the $\frac{3}{4}$ power (allometric scaling).

Supplementary Table 5: Custom gene list for PCR arrays, including housekeeping genes (denoted by one *) and controls (denoted by two **).

Gene Symbol	Refseq #	Official Full Name
Bax	NM_007527	Bcl2-associated X protein
Bcl2	NM_009741	B-cell leukemia/lymphoma 2
Blk	NM_007549	B lymphoid kinase
Blnk	NM_008528	B-cell linker
Btk	NM_013482	Bruton agammaglobulinemia tyrosine kinase
Cd19	NM_009844	CD19 antigen
Ccnb2	NM_007630	Cyclin B2
Ccnd1	NM_007631	Cyclin D1
Ccng1	NM_009831	Cyclin G1
Cd19	NM_009844	CD19 antigen
Cd3g	NM_009850	CD3 antigen, gamma polypeptide
Cd40lg	NM_011616	CD40 ligand
Cd8a	NM_001081110	CD8 antigen, alpha chain
Cd8b1	NM_009858	CD8 antigen, beta chain 1
Cdkn1a	NM_007669	Cyclin-dependent kinase inhibitor 1A (P21)
Cxcr5	NM_007551	Chemokine (C-X-C motif) receptor 5
Cyp1a1	NM_009992	Cytochrome P450, family 1, subfamily a, polypeptide 1
Cyp1b1	NM_009994	Cytochrome P450, family 1, subfamily b, polypeptide 1
Cyr61	NM_010516	Cysteine rich protein 61
Dock2	NM_033374	Dedicator of cyto-kinesis 2
Gadd45a	NM_007836	Growth arrest and DNA-damage-inducible 45 alpha
Gadd45g	NM_011817	Growth arrest and DNA-damage-inducible 45 gamma
Gsta1	NM_008181	Glutathione S-transferase, alpha 1 (Ya)
Gsta2	NM_008182	Glutathione S-transferase, alpha 2 (Yc2)
Mdm2	NM_010786	Transformed mouse 3T3 cell double minute 2
Mgmt	NM_008598	O-6-methylguanine-DNA methyltransferase
Nqo1	NM_008706	NAD(P)H dehydrogenase, quinone 1
Pdgfa	NM_008808	Platelet derived growth factor, alpha
Pmaip1	NM_021451	Phorbol-12-myristate-13-acetate-induced protein 1
Polk	NM_012048	Polymerase (DNA directed), kappa
Sesn2	NM_144907	Sestrin 2
Srxn1	NM_029688	Sulfiredoxin 1 homolog (S. cerevisiae)
Tnfrsf10b	NM_020275	Tumor necrosis factor receptor superfamily, member 10b
Trp53inp1	NM_021897	Transformation related protein 53 inducible nuclear protein 1
Ugt1a9	NM_201644	UDP glucuronosyltransferase 1 family, polypeptide A9
Vegfa	NM_009505	Vascular endothelial growth factor A
Vegfc	NM_009506	Vascular endothelial growth factor C
Zmat3	NM_009517	Zinc finger matrin type 3
Gusb*	NM_010368	Glucuronidase, beta
Hprt*	NM_013556	Hypoxanthine guanine phosphoribosyl transferase
Gapdh*	NM_008084	Glyceraldehyde-3-phosphate dehydrogenase
MGDC**	SA_00106	Mouse Genomic DNA Contamination
RTC**	SA_00104	Reverse Transcription Control
PPC**	SA_00103	Positive PCR Control

Supplementary Table 6: Differentially expressed genes following exposure to BaP at 4 and 24 hours in human THK6 cells (FDR > 0.05% and FC > ±1.5^a.

Gene ID	Gene Name	Gene Symbol	Gene Type	Ranking	FC (log ₂)	p-value	Avg. 0.40 ug/mg, BaP vs Control	FDR (p-value)	Avg. 4 hr, log ₂ , BaP vs Control	FDR (p-value)	Avg. 4 hr, log ₂ , BaP vs Control	FDR (p-value)	Avg. 24 hr, log ₂ , BaP vs Control	FDR (p-value)	Avg. 24 hr, log ₂ , BaP vs Control	FDR (p-value)	Avg. 24 hr, log ₂ , BaP vs Control	FDR (p-value)			
A_23_P15232	NM_045044	GOF15	transcriptional factor T5	13.8	-0.40	0.10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00		
A_23_P362	NM_044861	TPMS3	tumor protein p53-induced protein 6	12.2	-0.00	0.24	1.0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
A_23_P32568	NM_045459	SCLC303	solute carrier family 30 (zinc transporter), member 3	9.6	0.00	0.00	-1.0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
A_23_P64721	NM_069118	HCR3	hydroxycarboxylic acid receptor 3	9.1	0.00	0.00	-0.10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
A_23_P11943	NM_001447	FUCA1	galactosidase, alpha-L-1, isoform 1	8.8	0.00	0.15	-1.0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
A_23_P26280	NM_21524	GPR56	G protein-coupled receptor 56	6.2	0.00	0.74	-1.0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
A_23_P36408	NM_001044	EPHA4	epithelial membrane protein 4	5.8	0.00	0.26	-1.0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00		
A_23_P79190	NM_002133	HMOX1	heme oxygenase (erythrocuprein) 1	5.4	0.00	0.47	-1.0	0.00	0.02	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00		
A_23_P354607	NM_02926	CCL4	chemokine (C-C motif) ligand 4	5.3	0.00	0.00	-0.10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
A_23_P23074	NM_006417	IFI44	interferon-induced protein 44	5.0	0.00	0.49	-1.0	0.00	0.74	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00		
A_23_P77608	NM_002625	PGL	placental growth factor	5.0	0.00	0.26	-1.0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00		
A_23_P46281	NM_13567	DQ4	DEAD box gene/ATPase 1	4.5	0.00	0.19	-1.0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00		
A_19_P005939	NM_00576	LGLSF2	leolin, galactose-binding, soluble 9	4.8	0.00	0.12	-1.0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
A_23_P14695	NM_01887	SLC2A9	solute carrier family 2 (facilitator), member 9	4.7	0.00	0.34	-1.0	0.00	0.31	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00		
A_23_P03369	NM_002369	GRIN2B	glutamate receptor, ionotropic, N-methyl-D-aspartate 2C	4.5	0.00	0.00	-0.10	0.00	0.25	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
A_19_P008051	NM_002524	MCAL2	MCAL-like 2	4.4	0.00	0.53	-1.0	0.00	0.11	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00		
A_23_P7278	NM_00104453	NURR1	nuuclear receptor, retinoid-X receptor, regulator, 1	4.2	0.00	0.00	-0.10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
A_23_P34353	NM_004111	NCAM1	cell adhesion molecule 1 (CAM-1)	4.2	0.00	0.00	-0.10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00		
A_23_P34354	NM_004467	NDPR1	NDPR-like 1	4.2	0.00	0.00	-0.10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00		
A_23_P37619	NM_0014137	RAGA1	interferon regulatory factor 7	4.1	0.00	0.00	-0.10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
A_19_P001859	NM_0014147	CCDC104	coiled-coil domain containing 104 (CCP1)	4.1	0.00	0.25	-1.0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
A_23_P22130	NM_00104437	CCDC33	coiled-coil domain containing 33 (CCP3-like)	4.0	0.00	0.00	-0.10	0.00	0.07	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
A_23_P00572	NM_002434	CD36	fatty acid binding protein, liver	3.9	0.00	0.00	-0.10	0.00	0.19	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
A_23_P26264	NM_0014111	CHAC1	CHAC1, cathepsin translocase homolog (E. coli)	3.8	0.00	0.03	-0.10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
A_33_P35881	NM_78351	LOC10192453	late confocal envelope 1C	3.8	0.00	0.07	-0.10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
A_23_P42262	NM_00102028	C4B5	complement component 4B, group B	3.7	0.00	0.00	-0.10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
A_23_P45878	NM_52133	C1orf56	chromosome 1 open reading frame 54	3.7	0.00	0.40	-1.0	0.00	0.31	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
A_19_P002815	NM_00104765	SLC6A4	monoamine transporter, family 6, member 4	3.7	0.00	0.29	-1.0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
A_23_P16288	NM_001017	DDX2	dead-specific DNA binding protein 2	3.6	0.00	0.00	-0.10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
A_23_P37016	NM_002791	SAT1	spontaneous DNA strand break-1	3.5	0.00	0.00	-0.10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
A_33_P29427	NM_00886	CYP450P450	cytochrome P450, family 450, subfamily P450, member 3	3.4	0.00	0.00	-0.10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
A_23_P37178	NM_002790	SAT1	spontaneous DNA strand break-1	3.4	0.00	0.00	-0.10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00		
A_23_P7199	NM_78111	GGTLC1	gamma-glutamyl transferase light chain	3.3	0.00	0.00	-0.10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
A_19_P0016344	NM_000174	NEAT1	nuclear paraspeckle assembly transcript 1	3.3	0.00	0.17	-1.0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
A_33_P343841	NM_16332	PRODH	proline dehydrogenase (oxoprolinase) 1	3.3	0.00	0.00	-0.10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
A_23_P26165	NM_001625	HMGBP1	poly(A)-dependent polymers of histone H3	3.3	0.00	0.51	-1.0	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
A_23_P26166	NM_00243	PTENP1	PTEN, phosphatase and tensin homolog	3.2	0.00	0.00	-0.10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
A_23_P26167	NM_000142	KRT17	krtin 17	3.2	0.00	0.00	-0.10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
A_23_P26168	NM_000142	PTENP1	PTEN, phosphatase and tensin homolog	3.2	0.00	0.00	-0.10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
A_23_P26169	NM_000142	PTENP2	PTEN, phosphatase and tensin homolog	3.2	0.00	0.00	-0.10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
A_23_P26170	NM_000142	PTENP3	PTEN, phosphatase and tensin homolog	3.2	0.00	0.00	-0.10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	
A_23_P26171	NM_000142	SANDB1	sterile alpha motif domain family member 1	3.2	0.00	0.00	-0.10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
A_23_P26172	NM_000142	SLC44A1	DEATH-domain-containing protein 1	3.2	0.00	0.00	-0.10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
A_23_P26173	NM_000142	SLC44A2	DEATH-domain-containing protein 2	3.2	0.00	0.00	-0.10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
A_23_P26174	NM_000142	SLC44A3	DEATH-domain-containing protein 3	3.2	0.00	0.00	-0.10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
A_23_P26175	NM_000142	SLC44A4	DEATH-domain-containing protein 4	3.2	0.00	0.00	-0.10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
A_23_P26176	NM_000142	SLC44A5	DEATH-domain-containing protein 5	3.2	0.00	0.00	-0.10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
A_23_P26177	NM_000142	SLC44A6	DEATH-domain-containing protein 6	3.2	0.00	0.00	-0.10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
A_23_P26178	NM_000142	SLC44A7	DEATH-domain-containing protein 7	3.2	0.00	0.00	-0.10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
A_23_P26179	NM_000142	SLC44A8	DEATH-domain-containing protein 8	3.2	0.00	0.00	-0.10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
A_23_P26180	NM_000142	SLC44A9	DEATH-domain-containing protein 9	3.2	0.00	0.00	-0.10	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
A_23_P26181	NM_000142	SLC44A10	DEATH-domain-containing protein 10</																		

A_24_P551842	NM_00127899	CYTB	cysteine-rich 9	2.0	0.00	0.61	-1.2	0.00	-2.0	0.82	-1.1	0.93	1.1	0.84	1.1	0.43	1.2	0.52	1.2	0.83	1.1	
A_23_P554027	NM_001020914	KCTD11	potassium channel tetramerization domain containing 11	2.0	0.00	0.48	1.1	0.05	1.2	0.00	1.4	0.81	-1.1	0.00	1.3	0.00	1.6	0.00	1.8	0.00	2.0	
A_23_P554865	NM_00128744	SORCS2	sortilin-related VHL domain containing receptor 2	2.0	0.00	0.43	1.1	0.05	1.3	0.00	1.5	0.71	0.88	-1.0	0.00	1.7	0.00	1.7	0.00	2.0	0.00	1.3
A_23_P559194	NM_003134	TTC1	tetralysine repeat domain 1	2.0	0.00	0.44	1.0	0.05	1.3	0.00	1.5	0.71	0.88	-1.0	0.00	1.7	0.00	1.7	0.00	2.0	0.00	1.3
A_23_P560208	NM_003134	TCOF1	coenzyme Q10 binding (CoQ10-binding)	2.0	0.00	0.50	1.0	0.05	1.2	0.00	1.3	0.73	0.91	-1.0	0.00	1.7	0.00	1.9	0.00	2.0	0.00	1.1
A_23_P560258	NM_00126258	APAF1	Apoptosis-associated factor 1	2.0	0.00	0.51	1.0	0.05	1.2	0.00	1.3	0.73	0.91	-1.0	0.00	1.7	0.00	1.9	0.00	2.0	0.00	1.4
A_23_P56088	NM_00124747	FGR	Gardiner-Rohlfing feline sarcoma viral (v-fgr) oncogene homolog	2.0	0.00	0.50	1.0	0.05	1.2	0.00	1.3	0.73	0.91	-1.0	0.00	1.7	0.00	1.9	0.00	2.0	0.00	1.6
A_23_P561332	NM_001020914	KCTD10	potassium channel tetramerization domain containing 10	2.0	0.00	0.47	1.1	0.05	1.3	0.00	1.5	0.71	0.88	-1.0	0.00	1.7	0.00	1.9	0.00	2.0	0.00	1.5
A_23_P561332	NM_0025950	PXT1	integrin alpha 1/beta 1 complex-specific	2.0	0.00	0.47	1.1	0.05	1.3	0.00	1.5	0.71	0.88	-1.0	0.00	1.7	0.00	1.9	0.00	2.0	0.00	1.4
A_23_P562028	NM_003879	TMC4	T-cell immunoglobulin and mucin domain-containing 4	2.0	0.00	0.62	-1.0	0.05	1.3	0.00	1.5	0.88	1.00	-1.0	0.00	1.7	0.00	1.9	0.00	2.0	0.00	1.6
A_23_P562028	NM_00127899	TMC5	T-cell immunoglobulin and mucin domain-containing 5	2.0	0.00	0.70	-1.0	0.05	1.2	0.00	1.3	0.96	1.00	-1.0	0.00	1.7	0.00	1.9	0.00	2.0	0.00	1.5
A_23_P562183	NM_00174742	LAMB3	lambin 3	2.0	0.00	0.75	-1.0	0.05	1.2	0.00	1.3	0.96	1.00	-1.0	0.00	1.7	0.00	1.9	0.00	2.0	0.00	1.6
A_23_P562183	NM_002968	COX1	cyclooxygenase 1	2.0	0.00	0.62	-1.0	0.05	1.2	0.00	1.3	0.96	1.00	-1.0	0.00	1.7	0.00	1.9	0.00	2.0	0.00	1.5
A_23_P562183	NM_0123225	COX2	cyclooxygenase 2	2.0	0.00	0.68	-1.0	0.05	1.2	0.00	1.3	0.96	1.00	-1.0	0.00	1.7	0.00	1.9	0.00	2.0	0.00	1.3
A_23_P562183	NM_0123225	COX3	cyclooxygenase 3	2.0	0.00	0.61	-1.0	0.05	1.2	0.00	1.3	0.96	1.00	-1.0	0.00	1.7	0.00	1.9	0.00	2.0	0.00	1.2
A_23_P562183	NM_0123225	COX4I1	cyclooxygenase 4	2.0	0.00	0.60	-1.0	0.05	1.2	0.00	1.3	0.96	1.00	-1.0	0.00	1.7	0.00	1.9	0.00	2.0	0.00	1.1
A_23_P562183	NM_0123225	COX4I2	cyclooxygenase 4	2.0	0.00	0.60	-1.0	0.05	1.2	0.00	1.3	0.96	1.00	-1.0	0.00	1.7	0.00	1.9	0.00	2.0	0.00	1.0
A_23_P562183	NM_0123225	COX4I3	cyclooxygenase 4	2.0	0.00	0.60	-1.0	0.05	1.2	0.00	1.3	0.96	1.00	-1.0	0.00	1.7	0.00	1.9	0.00	2.0	0.00	0.9
A_23_P562183	NM_0123225	COX4I4	cyclooxygenase 4	2.0	0.00	0.60	-1.0	0.05	1.2	0.00	1.3	0.96	1.00	-1.0	0.00	1.7	0.00	1.9	0.00	2.0	0.00	0.8
A_23_P562183	NM_0123225	COX4I5	cyclooxygenase 4	2.0	0.00	0.60	-1.0	0.05	1.2	0.00	1.3	0.96	1.00	-1.0	0.00	1.7	0.00	1.9	0.00	2.0	0.00	0.7
A_23_P562183	NM_0123225	COX4I6	cyclooxygenase 4	2.0	0.00	0.60	-1.0	0.05	1.2	0.00	1.3	0.96	1.00	-1.0	0.00	1.7	0.00	1.9	0.00	2.0	0.00	0.6
A_23_P562183	NM_0123225	COX4I7	cyclooxygenase 4	2.0	0.00	0.60	-1.0	0.05	1.2	0.00	1.3	0.96	1.00	-1.0	0.00	1.7	0.00	1.9	0.00	2.0	0.00	0.5
A_23_P562183	NM_0123225	COX4I8	cyclooxygenase 4	2.0	0.00	0.60	-1.0	0.05	1.2	0.00	1.3	0.96	1.00	-1.0	0.00	1.7	0.00	1.9	0.00	2.0	0.00	0.4
A_23_P562183	NM_0123225	COX4I9	cyclooxygenase 4	2.0	0.00	0.60	-1.0	0.05	1.2	0.00	1.3	0.96	1.00	-1.0	0.00	1.7	0.00	1.9	0.00	2.0	0.00	0.3
A_23_P562183	NM_0123225	COX4I10	cyclooxygenase 4	2.0	0.00	0.60	-1.0	0.05	1.2	0.00	1.3	0.96	1.00	-1.0	0.00	1.7	0.00	1.9	0.00	2.0	0.00	0.2
A_23_P562183	NM_0123225	COX4I11	cyclooxygenase 4	2.0	0.00	0.60	-1.0	0.05	1.2	0.00	1.3	0.96	1.00	-1.0	0.00	1.7	0.00	1.9	0.00	2.0	0.00	0.1
A_23_P562183	NM_0123225	COX4I12	cyclooxygenase 4	2.0	0.00	0.60	-1.0	0.05	1.2	0.00	1.3	0.96	1.00	-1.0	0.00	1.7	0.00	1.9	0.00	2.0	0.00	0.0
A_23_P562183	NM_0123225	COX4I13	cyclooxygenase 4	2.0	0.00	0.60	-1.0	0.05	1.2	0.00	1.3	0.96	1.00	-1.0	0.00	1.7	0.00	1.9	0.00	2.0	0.00	-1.0
A_23_P562183	NM_0123225	COX4I14	cyclooxygenase 4	2.0	0.00	0.60	-1.0	0.05	1.2	0.00	1.3	0.96	1.00	-1.0	0.00	1.7	0.00	1.9	0.00	2.0	0.00	-1.1
A_23_P562183	NM_0123225	COX4I15	cyclooxygenase 4	2.0	0.00	0.60	-1.0	0.05	1.2	0.00	1.3	0.96	1.00	-1.0	0.00	1.7	0.00	1.9	0.00	2.0	0.00	-1.2
A_23_P562183	NM_0123225	COX4I16	cyclooxygenase 4	2.0	0.00	0.60	-1.0	0.05	1.2	0.00	1.3	0.96	1.00	-1.0	0.00	1.7	0.00	1.9	0.00	2.0	0.00	-1.3
A_23_P562183	NM_0123225	COX4I17	cyclooxygenase 4	2.0	0.00	0.60	-1.0	0.05	1.2	0.00	1.3	0.96	1.00	-1.0	0.00	1.7	0.00	1.9	0.00	2.0	0.00	-1.4
A_23_P562183	NM_0123225	COX4I18	cyclooxygenase 4	2.0	0.00	0.60	-1.0	0.05	1.2	0.00	1.3	0.96	1.00	-1.0	0.00	1.7	0.00	1.9	0.00	2.0	0.00	-1.5
A_23_P562183	NM_0123225	COX4I19	cyclooxygenase 4	2.0	0.00	0.60	-1.0	0.05	1.2	0.00	1.3	0.96	1.00	-1.0	0.00	1.7	0.00	1.9	0.00	2.0	0.00	-1.6
A_23_P562183	NM_0123225	COX4I20	cyclooxygenase 4	2.0	0.00	0.60	-1.0	0.05	1.2	0.00	1.3	0.96	1.00	-1.0	0.00	1.7	0.00	1.9	0.00	2.0	0.00	-1.7
A_23_P562183	NM_0123225	COX4I21	cyclooxygenase 4	2.0	0.00	0.60	-1.0	0.05	1.2	0.00	1.3	0.96	1.00	-1.0	0.00	1.7	0.00	1.9	0.00	2.0	0.00	-1.8
A_23_P562183	NM_0123225	COX4I22	cyclooxygenase 4	2.0	0.00	0.60	-1.0	0.05	1.2	0.00	1.3	0.96	1.00	-1.0	0.00	1.7	0.00	1.9	0.00	2.0	0.00	-1.9
A_23_P562183	NM_0123225	COX4I23	cyclooxygenase 4	2.0	0.00	0.60	-1.0	0.05	1.2	0.00	1.3	0.96	1.00	-1.0	0.00	1.7	0.00	1.9	0.00	2.0	0.00	-1.8
A_23_P562183	NM_0123225	COX4I24	cyclooxygenase 4	2.0	0.00	0.60	-1.0	0.05	1.2	0.00	1.3	0.96	1.00	-1.0	0.00	1.7	0.00	1.9	0.00	2.0	0.00	-1.7
A_23_P562183	NM_0123225	COX4I25	cyclooxygenase 4	2.0	0.00	0.60	-1.0	0.05	1.2	0.00	1.3	0.96	1.00	-1.0	0.00	1.7	0.00	1.9	0.00	2.0	0.00	-1.6
A_23_P562183	NM_0123225	COX4I26	cyclooxygenase 4	2.0	0.00	0.60	-1.0	0.05	1.2	0.00	1.3	0.96	1.00	-1.0	0.00	1.7	0.00	1.9	0.00	2.0	0.00	-1.5
A_23_P562183	NM_0123225	COX4I27	cyclooxygenase 4	2.0	0.00	0.60	-1.0	0.05	1.2	0.00	1.3	0.96	1.00	-1.0	0.00	1.7	0.00	1.9	0.00	2.0	0.00	-1.4
A_23_P562183	NM_0123225	COX4I28	cyclooxygenase 4	2.0	0.00	0.60	-1.0	0.05	1.2	0.00	1.3	0.96	1.00	-1.0	0.00	1.7	0.00	1.9	0.00	2.0	0.00	-1.3
A_23_P562183	NM_0123225	COX4I29	cyclooxygenase 4	2.0	0.00	0.60	-1.0	0.05	1.2	0.00	1.3	0.96	1.00	-1.0	0.00	1.7	0.00	1.9	0.00	2.0	0.00	-1.2
A_23_P562183	NM_0123225	COX4I30	cyclooxygenase 4	2.0	0.00	0.60	-1.0	0.05	1.2	0.00	1.3	0.96	1.00	-1.0	0.00	1.7	0.00	1.9	0.00	2.0	0.00	-1.1
A_23_P562183	NM_0123225	COX4I31	cyclooxygenase 4	2.0	0.00	0.60	-1.0	0.05	1.2	0.00	1.3	0.96	1.00	-1.0	0.00	1.7	0.00	1.9	0.00	2.0	0.00	-1.0
A_23_P562183	NM_0123225	COX4I32	cyclooxygenase 4	2.0	0.00	0.60	-1.0	0.05	1.2	0.00	1.3	0.96	1.00	-1.0	0.00	1.7	0.00	1.9	0.00	2.0	0.00	-0.9
A_23_P562183	NM_0123225	COX4I33	cyclooxygenase 4	2.0	0.00	0.60	-1.0	0.05	1.2	0.00	1.3	0.96	1.00	-1.0	0.00	1.7	0.00	1.9	0.00	2.0	0.00	-0.8
A_23_P562183	NM_0123225	COX4I34	cyclooxygenase 4	2.0	0.00	0.60	-1.0	0.05	1.2	0.00	1.3	0.96	1.00	-1.0	0.00	1.7	0.00	1.9	0.00	2.0	0.00	-0.7
A_23_P562183	NM_0123225	COX4I35	cyclooxygenase 4	2.0	0.00	0.60	-1.0	0.05	1.2	0.00	1.3	0.96	1.00	-1.0	0.00	1.7	0.00	1.9	0.00	2.0	0.00	-0.6
A_23_P562183	NM_0123225	COX4I36	cyclooxygenase 4	2.0	0.00	0.60	-1.0	0.05	1.2	0.00	1.3	0.96	1.0									

A_24_P224405	NM_012755	ANKRD11	ankyrin repeat domain 11	1.8	0.00	0.81	-1.0	0.00	-1.4	0.00	-1.8	0.85	1.1	0.88	-1.2	0.42	-1.1	0.41	-1.1	0.77	
A_24_P224406	NM_000973	FCN1	FCN1	1.8	0.00	0.97	-1.0	0.04	-1.1	0.00	-1.1	0.85	1.1	0.88	-1.0	0.42	-1.0	0.41	-1.0	0.77	
A_24_P224444	NM_005135	FHOD3	formin homology 2 domain containing 3	1.8	0.00	0.23	-1.0	0.00	-1.0	0.00	-1.8	1.00	1.0	1.00	0.20	1.1	0.07	1.2	0.38	1.1	0.00
A_24_P225057	NM_004207	NCORAI015	non-coding RNA 15B	1.8	0.00	0.22	-1.0	0.00	-1.0	0.00	-1.1	0.87	1.0	0.88	-1.0	0.40	-1.1	0.35	1.0	0.00	
A_24_P225060	NM_000978	NCORAI016	non-coding RNA 15C	1.8	0.00	0.33	-1.0	0.00	-1.0	0.00	-1.0	0.88	1.0	0.88	-1.0	0.40	-1.0	0.35	1.0	0.00	
A_24_P225076	NM_007387	FCRLA	FC receptor-like A	1.8	0.00	0.78	-1.0	0.00	-1.0	0.00	-2.8	1.1	0.95	-0.00	1.4	0.00	1.8	0.00	1.6	0.00	
A_24_P226002	NM_005448	LGLS2	lectin, galactose-binding, soluble, 2	1.8	0.00	0.67	-1.0	0.00	-1.0	0.00	-0.9	0.97	1.0	0.98	-0.9	0.40	-1.0	0.35	1.0	0.00	
A_24_P226003	NM_005448	LGLS3	lectin, galactose-binding, soluble, 3	1.8	0.00	0.67	-1.0	0.00	-1.0	0.00	-0.9	0.97	1.0	0.98	-0.9	0.40	-1.0	0.35	1.0	0.00	
A_24_P226005	NM_004873	TNP3	TNP493 interacting protein 3	1.8	0.00	0.68	-1.0	0.00	-1.0	0.00	-1.1	0.89	1.0	0.98	-0.00	1.3	0.00	1.8	0.00	1.4	0.00
A_24_P226006	NM_004873	TNP493	TNP493 interacting protein 3	1.8	0.00	0.67	-1.0	0.00	-1.0	0.00	-1.1	0.89	1.0	0.98	-0.00	1.3	0.00	1.8	0.00	1.4	0.00
A_24_P226008	NM_002201	ISCGD	interferon stimulated exonuclease gene 20kDa	1.8	0.00	0.45	-1.0	0.00	-1.0	0.00	-1.0	0.82	1.0	0.98	-1.0	0.40	-1.0	0.35	1.0	0.00	
A_24_P226009	NM_0103771	CBLN3	cereblon precursor	1.8	0.00	0.91	-1.0	0.00	-1.0	0.00	-1.2	0.92	-0.00	1.0	0.00	1.6	0.00	1.8	0.00	1.3	0.00
A_24_P226010	NM_0152452	TSC220	TSC22 domain family, member 3	1.8	0.00	0.43	-1.0	0.00	-1.0	0.00	-1.0	0.85	1.0	0.98	-1.0	0.40	-1.0	0.35	1.0	0.00	
A_24_P226011	NM_016446	TMEM88	transmembrane protein 88	1.8	0.00	0.58	-1.0	0.00	-1.0	0.00	-1.1	0.82	-0.00	1.0	0.00	1.8	0.00	1.5	0.00		
A_24_P226012	NM_004409	LGALS1	lectin, galactose-binding, soluble, 1	1.8	0.00	0.59	-1.0	0.00	-1.0	0.00	-1.0	0.87	1.0	0.98	-1.0	0.40	-1.0	0.35	1.0	0.00	
A_24_P226013	NM_0043	AURKA	aurora kinase A	1.8	0.00	0.27	-1.0	0.00	-1.0	0.00	-1.0	0.88	1.0	0.98	-1.0	0.40	-1.0	0.35	1.0	0.00	
A_24_P226014	NM_004304	ASPRY1	aspartyl-prolyl cyclase	1.8	0.00	0.76	-1.0	0.00	-1.0	0.00	-1.0	0.87	1.0	0.98	-1.0	0.40	-1.0	0.35	1.0	0.00	
A_24_P226015	NM_004304	CACNB2	calmodulin-binding, voltage-dependent, beta 2 subunit	1.8	0.00	0.64	-1.0	0.00	-1.0	0.00	-1.0	0.88	1.0	0.98	-1.0	0.40	-1.0	0.35	1.0	0.00	
A_24_P226016	NM_004304	CACNB3	calmodulin-binding, voltage-dependent, beta 3 subunit	1.8	0.00	0.59	-1.0	0.00	-1.0	0.00	-1.0	0.87	1.0	0.98	-1.0	0.40	-1.0	0.35	1.0	0.00	
A_24_P226017	NM_004304	CACNB6	calmodulin-binding, voltage-dependent, beta 6 subunit	1.8	0.00	0.59	-1.0	0.00	-1.0	0.00	-1.0	0.87	1.0	0.98	-1.0	0.40	-1.0	0.35	1.0	0.00	
A_24_P226018	NM_004304	CACNB7	calmodulin-binding, voltage-dependent, beta 7 subunit	1.8	0.00	0.59	-1.0	0.00	-1.0	0.00	-1.0	0.87	1.0	0.98	-1.0	0.40	-1.0	0.35	1.0	0.00	
A_24_P226019	NM_004304	CACNB8	calmodulin-binding, voltage-dependent, beta 8 subunit	1.8	0.00	0.59	-1.0	0.00	-1.0	0.00	-1.0	0.87	1.0	0.98	-1.0	0.40	-1.0	0.35	1.0	0.00	
A_24_P226020	NM_004304	CACNB9	calmodulin-binding, voltage-dependent, beta 9 subunit	1.8	0.00	0.59	-1.0	0.00	-1.0	0.00	-1.0	0.87	1.0	0.98	-1.0	0.40	-1.0	0.35	1.0	0.00	
A_24_P226021	NM_004304	CACNB10	calmodulin-binding, voltage-dependent, beta 10 subunit	1.8	0.00	0.59	-1.0	0.00	-1.0	0.00	-1.0	0.87	1.0	0.98	-1.0	0.40	-1.0	0.35	1.0	0.00	
A_24_P226022	NM_004304	CACNB11	calmodulin-binding, voltage-dependent, beta 11 subunit	1.8	0.00	0.59	-1.0	0.00	-1.0	0.00	-1.0	0.87	1.0	0.98	-1.0	0.40	-1.0	0.35	1.0	0.00	
A_24_P226023	NM_004304	CACNB12	calmodulin-binding, voltage-dependent, beta 12 subunit	1.8	0.00	0.59	-1.0	0.00	-1.0	0.00	-1.0	0.87	1.0	0.98	-1.0	0.40	-1.0	0.35	1.0	0.00	
A_24_P226024	NM_004304	CACNB13	calmodulin-binding, voltage-dependent, beta 13 subunit	1.8	0.00	0.59	-1.0	0.00	-1.0	0.00	-1.0	0.87	1.0	0.98	-1.0	0.40	-1.0	0.35	1.0	0.00	
A_24_P226025	NM_004304	CACNB14	calmodulin-binding, voltage-dependent, beta 14 subunit	1.8	0.00	0.59	-1.0	0.00	-1.0	0.00	-1.0	0.87	1.0	0.98	-1.0	0.40	-1.0	0.35	1.0	0.00	
A_24_P226026	NM_004304	CACNB15	calmodulin-binding, voltage-dependent, beta 15 subunit	1.8	0.00	0.59	-1.0	0.00	-1.0	0.00	-1.0	0.87	1.0	0.98	-1.0	0.40	-1.0	0.35	1.0	0.00	
A_24_P226027	NM_004304	CACNB16	calmodulin-binding, voltage-dependent, beta 16 subunit	1.8	0.00	0.59	-1.0	0.00	-1.0	0.00	-1.0	0.87	1.0	0.98	-1.0	0.40	-1.0	0.35	1.0	0.00	
A_24_P226028	NM_004304	CACNB17	calmodulin-binding, voltage-dependent, beta 17 subunit	1.8	0.00	0.59	-1.0	0.00	-1.0	0.00	-1.0	0.87	1.0	0.98	-1.0	0.40	-1.0	0.35	1.0	0.00	
A_24_P226029	NM_004304	CACNB18	calmodulin-binding, voltage-dependent, beta 18 subunit	1.8	0.00	0.59	-1.0	0.00	-1.0	0.00	-1.0	0.87	1.0	0.98	-1.0	0.40	-1.0	0.35	1.0	0.00	
A_24_P226030	NM_004304	CACNB19	calmodulin-binding, voltage-dependent, beta 19 subunit	1.8	0.00	0.59	-1.0	0.00	-1.0	0.00	-1.0	0.87	1.0	0.98	-1.0	0.40	-1.0	0.35	1.0	0.00	
A_24_P226031	NM_004304	CACNB20	calmodulin-binding, voltage-dependent, beta 20 subunit	1.8	0.00	0.59	-1.0	0.00	-1.0	0.00	-1.0	0.87	1.0	0.98	-1.0	0.40	-1.0	0.35	1.0	0.00	
A_24_P226032	NM_004304	CACNB21	calmodulin-binding, voltage-dependent, beta 21 subunit	1.8	0.00	0.59	-1.0	0.00	-1.0	0.00	-1.0	0.87	1.0	0.98	-1.0	0.40	-1.0	0.35	1.0	0.00	
A_24_P226033	NM_004304	CACNB22	calmodulin-binding, voltage-dependent, beta 22 subunit	1.8	0.00	0.59	-1.0	0.00	-1.0	0.00	-1.0	0.87	1.0	0.98	-1.0	0.40	-1.0	0.35	1.0	0.00	
A_24_P226034	NM_004304	CACNB23	calmodulin-binding, voltage-dependent, beta 23 subunit	1.8	0.00	0.59	-1.0	0.00	-1.0	0.00	-1.0	0.87	1.0	0.98	-1.0	0.40	-1.0	0.35	1.0	0.00	
A_24_P226035	NM_004304	CACNB24	calmodulin-binding, voltage-dependent, beta 24 subunit	1.8	0.00	0.59	-1.0	0.00	-1.0	0.00	-1.0	0.87	1.0	0.98	-1.0	0.40	-1.0	0.35	1.0	0.00	
A_24_P226036	NM_004304	CACNB25	calmodulin-binding, voltage-dependent, beta 25 subunit	1.8	0.00	0.59	-1.0	0.00	-1.0	0.00	-1.0	0.87	1.0	0.98	-1.0	0.40	-1.0	0.35	1.0	0.00	
A_24_P226037	NM_004304	CACNB26	calmodulin-binding, voltage-dependent, beta 26 subunit	1.8	0.00	0.59	-1.0	0.00	-1.0	0.00	-1.0	0.87	1.0	0.98	-1.0	0.40	-1.0	0.35	1.0	0.00	
A_24_P226038	NM_004304	CACNB27	calmodulin-binding, voltage-dependent, beta 27 subunit	1.8	0.00	0.59	-1.0	0.00	-1.0	0.00	-1.0	0.87	1.0	0.98	-1.0	0.40	-1.0	0.35	1.0	0.00	
A_24_P226039	NM_004304	CACNB28	calmodulin-binding, voltage-dependent, beta 28 subunit	1.8	0.00	0.59	-1.0	0.00	-1.0	0.00	-1.0	0.87	1.0	0.98	-1.0	0.40	-1.0	0.35	1.0	0.00	
A_24_P226040	NM_004304	CACNB29	calmodulin-binding, voltage-dependent, beta 29 subunit	1.8	0.00	0.59	-1.0	0.00	-1.0	0.00	-1.0	0.87	1.0	0.98	-1.0	0.40	-1.0	0.35	1.0	0.00	
A_24_P226041	NM_004304	CACNB30	calmodulin-binding, voltage-dependent, beta 30 subunit	1.8	0.00	0.59	-1.0	0.00	-1.0	0.00	-1.0	0.87	1.0	0.98	-1.0	0.40	-1.0	0.35	1.0	0.00	
A_24_P226042	NM_004304	CACNB31	calmodulin-binding, voltage-dependent, beta 31 subunit	1.8	0.00	0.59	-1.0	0.00	-1.0	0.00	-1.0	0.87	1.0	0.98	-1.0	0.40	-1.0	0.35	1.0	0.00	
A_24_P226043	NM_004304	CACNB32	calmodulin-binding, voltage-dependent, beta 32 subunit	1.8	0.00	0.59	-1.0	0.00	-1.0	0.00	-1.0	0.87	1.0	0.98	-1.0	0.40	-1.0	0.35	1.0	0.00	
A_24_P226044	NM_004304	CACNB33	calmodulin-binding, voltage-dependent, beta 33 subunit	1.8	0.00	0.59	-1.0	0.00	-1.0	0.00	-1.0	0.87	1.0	0.98	-1.0	0.40	-1.0	0.35	1.0	0.00	
A_24_P226045	NM_004304	CACNB34	calmodulin-binding, voltage-dependent, beta 34 subunit	1.8	0.00	0.59	-1.0	0.00	-1.0	0.00	-1.0	0.87	1.0	0.98	-1.0	0.40	-1.0	0.35	1.0	0.00	
A_24_P226046	NM_004304	CACNB35	calmodulin-binding, voltage-dependent, beta 35 subunit	1.8	0.00	0.59	-1.0	0.00	-1.0	0.00	-1.0	0.87	1.0	0.98	-1.0	0.40	-1.0	0.35	1.0	0.00	
A_24_P226047	NM_004304	CACNB36	calmodulin-binding, voltage-dependent, beta 36 subunit	1.8	0.00	0.59	-1.0	0.00	-1.0	0.00	-1.0	0.87	1.0	0.98	-1.0	0.40	-1.0	0.35	1.0	0.00	
A_24_P226048	NM_004304	CACNB37	calmodulin-binding, voltage-dependent, beta 37 subunit	1.8	0.00	0.59	-1.0	0.00	-1.0	0.00	-1.0	0.87	1.0	0.98	-1.0	0.40	-1.0	0.35	1.0	0.00	
A_24_P226049	NM_004304	CACNB38	calmodulin-binding, voltage-dependent, beta 38 subunit	1.8	0.00	0.59	-1.0	0.00	-1.0	0.00	-1.0	0.87	1.0	0.98	-1.0	0.40	-1.0	0.35	1.0	0.00	
A_24_P226050	NM_004304	CACNB39	calmodulin-binding, voltage-dependent, beta 39 subunit	1.8	0.00	0.59	-1.0	0.00	-1.0	0.00	-1.0	0.87	1.0	0.98	-1.0	0.40	-1.0	0.35	1.0	0.00	
A_24_P226051	NM_004304	CACNB40	calmodulin-binding, voltage-dependent, beta 40 subunit	1.8	0.00	0.59	-1.0	0.00	-1.0	0.00	-1.0	0.87	1.0	0.98	-1.0	0.40	-1.0	0.35	1.0	0	

A_23_P48217	NM_00817	APOLD1	apoptosis-related L domain containing 1	1.7	0.00	1.00	-1.0	0.31	1.1	0.01	1.2	0.81	-1.1	0.00	1.2	0.00	1.6	0.00	1.7	0.00	1.4	
A_23_P48218	NM_00818	APOLD2	apoptosis-related L domain containing 2	1.7	0.00	1.00	-1.0	0.30	1.1	0.01	1.3	0.81	-1.1	0.00	1.3	0.00	1.7	0.00	1.5	0.00	1.5	
A_23_P48219	NM_00819	MYLK2	myosin light chain kinase 2	1.7	0.00	0.81	-1.0	0.48	1.0	0.00	-1.3	0.81	1.1	0.01	1.2	0.00	1.5	0.00	1.7	0.00	1.7	
A_23_P48220	NM_00820	SFRP5	SLC24A5-like protein 5 (SFRP5-associated)	1.7	0.00	0.84	-1.0	0.30	0.97	-1.0	0.00	-1.1	0.89	-1.0	0.00	1.3	0.00	1.7	0.00	1.3	0.00	1.3
A_23_P48221	NM_00821	SH3BP5	SH3-binding protein 5 (SH3BP5-associated)	1.7	0.00	0.88	-1.0	0.86	1.0	0.00	-1.0	1.00	-0.9	0.00	1.3	0.00	1.7	0.00	1.3	0.00	1.3	
A_23_P48222	NM_00822	TBL1YL1	TRIM33-associated protein 1 (TBL1YL1-associated)	1.7	0.00	0.84	-1.0	0.86	1.0	0.00	-1.0	1.00	-0.9	0.00	1.3	0.00	1.7	0.00	1.3	0.00	1.3	
A_23_P48223	NM_00823	PTENP2	PTEN-induced putative kinase 2	1.7	0.00	0.88	-1.0	0.86	1.0	0.00	-1.0	1.00	-0.9	0.00	1.3	0.00	1.7	0.00	1.3	0.00	1.3	
A_23_P48224	NM_00824	CD40L	CD40 molecule, TNF receptor superfamily member 5	1.7	0.00	0.82	-1.0	0.00	-1.2	0.13	-1.1	0.94	-1.0	0.00	1.3	0.00	1.7	0.00	1.3	0.00	1.3	
A_23_P48225	NM_00825	MMP1	matrix metalloproteinase 1	1.7	0.00	0.89	-1.0	0.84	1.0	0.00	-1.0	1.00	-0.9	0.00	1.3	0.00	1.7	0.00	1.3	0.00	1.3	
A_23_P48226	NM_00826	DUSP2	dual specificity phosphatase 2	1.7	0.00	0.75	-1.0	0.00	-1.0	0.00	-1.0	0.95	1.0	0.01	1.3	0.00	1.7	0.00	1.3	0.00	1.3	
A_23_P48227	NM_00827	CER84	ceramide synthase 4	1.7	0.00	0.89	-1.0	0.91	1.0	0.00	-1.0	0.21	1.1	0.00	1.3	0.00	1.7	0.00	1.3	0.00	1.3	
A_23_P48228	NM_00828	WDR91	WD repeat domain 1	1.7	0.00	0.83	-1.0	0.94	1.0	0.00	-1.0	0.00	-1.0	0.00	1.3	0.00	1.7	0.00	1.3	0.00	1.3	
A_23_P48229	NM_00829	PNOC	prognostin	1.7	0.00	0.89	0.0	0.21	1.1	0.00	1.0	0.94	-1.0	0.00	1.3	0.00	1.7	0.00	1.3	0.00	1.3	
A_23_P48230	NM_00830	TCF10	chondroitin 14-sulfatase reading frame 182	1.7	0.00	0.85	0.0	0.86	1.0	0.00	-1.1	1.00	-0.9	0.00	1.3	0.00	1.7	0.00	1.3	0.00	1.3	
A_23_P48231	NM_00831	CTRC1	CREB-regulated transcript coactivator 1	1.7	0.00	1.00	0.0	0.98	1.0	0.00	0.95	1.0	0.00	1.3	0.00	1.7	0.00	1.3	0.00	1.3		
A_23_P48232	NM_00832	TCF10	chondroitin 14-sulfatase reading frame 182	1.7	0.00	0.85	0.0	0.86	1.0	0.00	-1.1	1.00	-0.9	0.00	1.3	0.00	1.7	0.00	1.3	0.00	1.3	
A_23_P48233	NM_00833	TCF10	chondroitin 14-sulfatase reading frame 182	1.7	0.00	0.85	0.0	0.86	1.0	0.00	-1.1	1.00	-0.9	0.00	1.3	0.00	1.7	0.00	1.3	0.00	1.3	
A_23_P48234	NM_00834	TCF10	chondroitin 14-sulfatase reading frame 182	1.7	0.00	0.85	0.0	0.86	1.0	0.00	-1.1	1.00	-0.9	0.00	1.3	0.00	1.7	0.00	1.3	0.00	1.3	
A_23_P48235	NM_00835	TCF10	chondroitin 14-sulfatase reading frame 182	1.7	0.00	0.85	0.0	0.86	1.0	0.00	-1.1	1.00	-0.9	0.00	1.3	0.00	1.7	0.00	1.3	0.00	1.3	
A_23_P48236	NM_00836	TCF10	chondroitin 14-sulfatase reading frame 182	1.7	0.00	0.85	0.0	0.86	1.0	0.00	-1.1	1.00	-0.9	0.00	1.3	0.00	1.7	0.00	1.3	0.00	1.3	
A_23_P48237	NM_00837	TCF10	chondroitin 14-sulfatase reading frame 182	1.7	0.00	0.85	0.0	0.86	1.0	0.00	-1.1	1.00	-0.9	0.00	1.3	0.00	1.7	0.00	1.3	0.00	1.3	
A_23_P48238	NM_00838	TCF10	chondroitin 14-sulfatase reading frame 182	1.7	0.00	0.85	0.0	0.86	1.0	0.00	-1.1	1.00	-0.9	0.00	1.3	0.00	1.7	0.00	1.3	0.00	1.3	
A_23_P48239	NM_00839	TCF10	chondroitin 14-sulfatase reading frame 182	1.7	0.00	0.85	0.0	0.86	1.0	0.00	-1.1	1.00	-0.9	0.00	1.3	0.00	1.7	0.00	1.3	0.00	1.3	
A_23_P48240	NM_00840	TCF10	chondroitin 14-sulfatase reading frame 182	1.7	0.00	0.85	0.0	0.86	1.0	0.00	-1.1	1.00	-0.9	0.00	1.3	0.00	1.7	0.00	1.3	0.00	1.3	
A_23_P48241	NM_00841	KIAA0013	TCF10-associated protein 1	1.7	0.00	0.85	0.0	0.86	1.0	0.00	-1.1	1.00	-0.9	0.00	1.3	0.00	1.7	0.00	1.3	0.00	1.3	
A_23_P48242	NM_00842	TCF10	chondroitin 14-sulfatase reading frame 182	1.7	0.00	0.85	0.0	0.86	1.0	0.00	-1.1	1.00	-0.9	0.00	1.3	0.00	1.7	0.00	1.3	0.00	1.3	
A_23_P48243	NM_00843	TCF10	chondroitin 14-sulfatase reading frame 182	1.7	0.00	0.85	0.0	0.86	1.0	0.00	-1.1	1.00	-0.9	0.00	1.3	0.00	1.7	0.00	1.3	0.00	1.3	
A_23_P48244	NM_00844	TCF10	chondroitin 14-sulfatase reading frame 182	1.7	0.00	0.85	0.0	0.86	1.0	0.00	-1.1	1.00	-0.9	0.00	1.3	0.00	1.7	0.00	1.3	0.00	1.3	
A_23_P48245	NM_00845	TCF10	chondroitin 14-sulfatase reading frame 182	1.7	0.00	0.85	0.0	0.86	1.0	0.00	-1.1	1.00	-0.9	0.00	1.3	0.00	1.7	0.00	1.3	0.00	1.3	
A_23_P48246	NM_00846	TCF10	chondroitin 14-sulfatase reading frame 182	1.7	0.00	0.85	0.0	0.86	1.0	0.00	-1.1	1.00	-0.9	0.00	1.3	0.00	1.7	0.00	1.3	0.00	1.3	
A_23_P48247	NM_00847	TCF10	chondroitin 14-sulfatase reading frame 182	1.7	0.00	0.85	0.0	0.86	1.0	0.00	-1.1	1.00	-0.9	0.00	1.3	0.00	1.7	0.00	1.3	0.00	1.3	
A_23_P48248	NM_00848	TCF10	chondroitin 14-sulfatase reading frame 182	1.7	0.00	0.85	0.0	0.86	1.0	0.00	-1.1	1.00	-0.9	0.00	1.3	0.00	1.7	0.00	1.3	0.00	1.3	
A_23_P48249	NM_00849	TCF10	chondroitin 14-sulfatase reading frame 182	1.7	0.00	0.85	0.0	0.86	1.0	0.00	-1.1	1.00	-0.9	0.00	1.3	0.00	1.7	0.00	1.3	0.00	1.3	
A_23_P48250	NM_00850	TCF10	chondroitin 14-sulfatase reading frame 182	1.7	0.00	0.85	0.0	0.86	1.0	0.00	-1.1	1.00	-0.9	0.00	1.3	0.00	1.7	0.00	1.3	0.00	1.3	
A_23_P48251	NM_00851	TCF10	chondroitin 14-sulfatase reading frame 182	1.7	0.00	0.85	0.0	0.86	1.0	0.00	-1.1	1.00	-0.9	0.00	1.3	0.00	1.7	0.00	1.3	0.00	1.3	
A_23_P48252	NM_00852	TCF10	chondroitin 14-sulfatase reading frame 182	1.7	0.00	0.85	0.0	0.86	1.0	0.00	-1.1	1.00	-0.9	0.00	1.3	0.00	1.7	0.00	1.3	0.00	1.3	
A_23_P48253	NM_00853	TCF10	chondroitin 14-sulfatase reading frame 182	1.7	0.00	0.85	0.0	0.86	1.0	0.00	-1.1	1.00	-0.9	0.00	1.3	0.00	1.7	0.00	1.3	0.00	1.3	
A_23_P48254	NM_00854	TCF10	chondroitin 14-sulfatase reading frame 182	1.7	0.00	0.85	0.0	0.86	1.0	0.00	-1.1	1.00	-0.9	0.00	1.3	0.00	1.7	0.00	1.3	0.00	1.3	
A_23_P48255	NM_00855	TCF10	chondroitin 14-sulfatase reading frame 182	1.7	0.00	0.85	0.0	0.86	1.0	0.00	-1.1	1.00	-0.9	0.00	1.3	0.00	1.7	0.00	1.3	0.00	1.3	
A_23_P48256	NM_00856	TCF10	chondroitin 14-sulfatase reading frame 182	1.7	0.00	0.85	0.0	0.86	1.0	0.00	-1.1	1.00	-0.9	0.00	1.3	0.00	1.7	0.00	1.3	0.00	1.3	
A_23_P48257	NM_00857	TCF10	chondroitin 14-sulfatase reading frame 182	1.7	0.00	0.85	0.0	0.86	1.0	0.00	-1.1	1.00	-0.9	0.00	1.3	0.00	1.7	0.00	1.3	0.00	1.3	
A_23_P48258	NM_00858	TCF10	chondroitin 14-sulfatase reading frame 182	1.7	0.00	0.85	0.0	0.86	1.0	0.00	-1.1	1.00	-0.9	0.00	1.3	0.00	1.7	0.00	1.3	0.00	1.3	
A_23_P48259	NM_00859	TCF10	chondroitin 14-sulfatase reading frame 182	1.7	0.00	0.85	0.0	0.86	1.0	0.00	-1.1	1.00	-0.9	0.00	1.3	0.00	1.7	0.00	1.3	0.00	1.3	
A_23_P48260	NM_00860	TCF10	chondroitin 14-sulfatase reading frame 182	1.7	0.00	0.85	0.0	0.86	1.0	0.00	-1.1	1.00	-0.9	0.00	1.3	0.00	1.7	0.00	1.3	0.00	1.3	
A_23_P48261	NM_00861	TCF10	chondroitin 14-sulfatase reading frame 182	1.7	0.00	0.85	0.0	0.86	1.0	0.00	-1.1	1.00	-0.9	0.00	1.3	0.00	1.7	0.00	1.3	0.00	1.3	
A_23_P48262	NM_00862	TCF10	chondroitin 14-sulfatase reading frame 182	1.7	0.00	0.85	0.0	0.86	1.0	0.00	-1.1	1.00	-0.9	0.00	1.3	0.00	1.7	0.00	1.3	0.00	1.3	
A_23_P48263	NM_00863	TCF10	chondroitin 14-sulfatase reading frame 182	1.7	0.00	0.85	0.0	0.86	1.0	0.00	-1.1	1.00	-0.9	0.00	1.3	0.00	1.7	0.00	1.3	0.00	1.3	
A_23_P48264	NM_00864	TCF10	chondroitin 14-sulfatase reading frame 182	1.7	0.00	0.85	0.0	0.86	1.0	0.00	-1.1	1.00	-0.9	0.00	1.3	0.00	1.7	0.00	1.3	0.00	1.3	
A_23_P48265	NM_00865	TCF10	chondroitin 14-sulfatase reading frame 182	1.7	0.00	0.85	0.0	0.86	1.0	0.00	-1.1	1.00	-0.9	0.00	1.3	0.00	1.7	0.00	1.3	0.00	1.3	
A_23_P48266	NM_00866	TCF10	chondroitin 14-sulfatase reading frame 182	1.7	0.00	0.85	0.0	0.86	1.0	0.00	-1.1	1.00	-0.9	0.00	1.3	0.00	1.7	0.00	1.3	0.00	1.3	
A_23_P48267	NM_00867	TCF10	chondroitin 14-sulfatase reading frame 182	1.7	0.00	0.85	0.0	0.86	1.0	0.00	-1.1	1.00	-0.9	0.00	1.3	0.00	1.7	0.00	1.3	0.00	1.3	
A_23_P48268	NM_00868	TCF10	chondroitin 14-sulfatase reading frame 182	1.7	0.00	0.85	0.0	0.86	1.0	0.00	-1.1	1.00	-0.9	0.00	1.3	0.00	1.7</					

A_24_P84428	NM_044122	CACBYP	cyclin binding protein	1.6	0.00	0.85	-1.0	0.57	1.0	0.50	1.0	0.86	-1.0	0.00	-1.1	0.00	-1.4	0.00	-1.6	0.00	-1.2
A_24_P84429	NM_044620	CONG1	cyclin	1.6	0.00	0.44	-1.0	0.50	1.0	0.50	1.0	0.86	-1.0	0.00	-1.1	0.00	-1.4	0.00	-1.6	0.00	-1.3
A_19_P0086059	NM_070161	DUCHX	duchx domain containing 1C	1.6	0.00	0.98	-1.0	0.52	1.0	0.01	1.2	0.80	-1.0	0.00	-1.2	0.00	-1.4	0.00	-1.6	0.00	-1.3
A_23_P42217	NM_025286	USP9X	ubiquitin-specific peptidase 94	1.6	0.00	0.34	-1.0	0.00	1.0	0.00	1.0	0.86	-1.0	0.00	-1.1	0.00	-1.3	0.00	-1.5	0.00	-1.0
A_23_P81448	NM_044615	SESN3	sestolin-like	1.6	0.00	0.67	-1.0	0.98	1.0	0.00	1.0	0.97	-1.0	0.00	-1.1	0.00	-1.3	0.00	-1.5	0.00	-1.1
A_23_P81449	NM_044616	SESN3L	sestolin-like	1.6	0.00	0.69	-1.0	0.94	1.0	0.00	1.0	0.98	-1.0	0.00	-1.1	0.00	-1.3	0.00	-1.5	0.00	-1.1
A_23_P46725	NM_025029	EPIC1	enhancer of polycomb homolog 1 (Drosophila)	1.6	0.00	0.62	-1.0	0.84	1.0	0.28	-1.0	0.28	-1.0	0.98	-1.1	0.00	-1.1	0.00	-1.6	0.00	-1.1
A_23_P94722	NM_028033	BTBD9	BTB/POZ domain containing 9	1.6	0.00	0.19	-1.2	0.00	1.0	-0.06	-1.6	0.99	1.0	0.00	-1.0	0.39	1.1	0.00	1.0	0.00	1.1
A_24_P03469	NM_010761	TBL1	tbl1	1.6	0.00	0.44	-1.0	0.50	1.0	0.00	1.0	0.99	-1.0	0.00	-1.1	0.00	-1.3	0.00	-1.5	0.00	-1.1
A_24_P03469	NM_010761	TLST1	leukocyte specific transcript 1	1.6	0.00	0.51	-1.0	0.50	1.0	0.00	1.0	0.97	-1.0	0.00	-1.1	0.00	-1.3	0.00	-1.5	0.00	-1.1
A_24_P97369	NM_026219	MALAT1	metastasis associated lung adenocarcinoma transcript 1 (non-protein coding)	1.6	0.00	0.98	-1.0	1.00	1.0	0.00	1.0	0.99	-1.0	0.00	-1.1	0.00	-1.3	0.00	-1.5	0.00	-1.3
A_23_P87541	NM_025258	ICOG	zinc finger CCCH-type containing 1	1.6	0.00	0.49	-1.1	0.24	1.0	0.00	1.0	0.84	-1.0	0.00	-1.1	0.00	-1.3	0.00	-1.5	0.00	-1.2
A_23_P22444	NM_025454	HIST1H1A	histone cluster 1, H4	1.6	0.00	0.33	-1.0	0.00	1.0	0.00	1.0	0.98	-1.0	0.00	-1.1	0.00	-1.3	0.00	-1.5	0.00	-1.4
A_19_P0027719	NM_017156	PTBP1	PTBP1	1.6	0.00	0.48	-1.2	0.50	1.0	0.00	1.0	0.75	-1.0	0.00	-1.1	0.00	-1.3	0.00	-1.5	0.00	-1.1
A_23_P91776	NM_014147	MS4A8B	membrane-spanning 4-domains, subfamily A member 8B	1.6	0.00	0.57	-1.1	0.35	1.1	0.40	1.1	0.93	1.0	0.00	-1.2	0.00	1.3	0.00	1.6	0.00	1.1
A_23_P37875	NM_03116561	LOC105002970	putative protein	1.6	0.00	0.72	-1.0	0.00	1.0	0.00	1.0	0.85	-1.0	0.00	-1.2	0.00	-1.3	0.00	-1.5	0.00	-1.1
A_23_P91776	NM_010761	PTPB1	protein lymphocyte tyrosine 1VA, member 1	1.6	0.00	0.47	-1.1	0.00	1.0	0.00	1.0	0.86	-1.0	0.00	-1.1	0.00	-1.3	0.00	-1.5	0.00	-1.1
A_23_P91776	NM_010761	PTPB1	various isoforms	1.6	0.00	0.71	-1.0	0.54	1.0	0.00	1.0	0.77	-1.0	0.00	-1.1	0.00	-1.3	0.00	-1.5	0.00	-1.1
A_23_P91776	NM_010761	PTPB1	various isoforms	1.6	0.00	0.71	-1.0	0.54	1.0	0.00	1.0	0.77	-1.0	0.00	-1.1	0.00	-1.3	0.00	-1.5	0.00	-1.1
A_23_P91776	NM_010761	PTPB1	various isoforms	1.6	0.00	0.71	-1.0	0.54	1.0	0.00	1.0	0.77	-1.0	0.00	-1.1	0.00	-1.3	0.00	-1.5	0.00	-1.1
A_24_P03469	NM_011540	GOA2B	GOA2B	1.6	0.00	0.68	-1.1	0.73	1.0	0.00	1.0	0.89	-1.0	0.00	-1.1	0.00	-1.3	0.00	-1.5	0.00	-1.2
A_24_P03469	NM_011540	GOA2B	GOA2B	1.6	0.00	0.68	-1.1	0.73	1.0	0.00	1.0	0.89	-1.0	0.00	-1.1	0.00	-1.3	0.00	-1.5	0.00	-1.2
A_24_P17997	NM_010761	IRFD1	interferon-related developmental regulator 1	1.6	0.00	0.38	-1.2	0.00	1.0	-0.06	-1.3	0.87	1.1	0.00	-1.2	0.00	-1.5	0.00	-1.7	0.00	-1.1
A_23_P20460	NM_038165	MYEF2	myeloblastosis overexpressed (in a subset of 1114 positive multiple myeloma cell lines)	1.6	0.00	0.59	-1.0	0.60	1.0	0.00	1.0	0.86	-1.0	0.00	-1.2	0.00	-1.4	0.00	-1.6	0.00	-1.0
A_23_P20460	NM_038165	MYEF2	myeloblastosis overexpressed (in a subset of 1114 positive multiple myeloma cell lines)	1.6	0.00	0.59	-1.0	0.60	1.0	0.00	1.0	0.86	-1.0	0.00	-1.2	0.00	-1.4	0.00	-1.6	0.00	-1.0
A_23_P20460	NM_038165	MYEF2	myeloblastosis overexpressed (in a subset of 1114 positive multiple myeloma cell lines)	1.6	0.00	0.59	-1.0	0.60	1.0	0.00	1.0	0.86	-1.0	0.00	-1.2	0.00	-1.4	0.00	-1.6	0.00	-1.0
A_23_P20460	NM_038165	MYEF2	myeloblastosis overexpressed (in a subset of 1114 positive multiple myeloma cell lines)	1.6	0.00	0.59	-1.0	0.60	1.0	0.00	1.0	0.86	-1.0	0.00	-1.2	0.00	-1.4	0.00	-1.6	0.00	-1.0
A_23_P20460	NM_038165	MYEF2	myeloblastosis overexpressed (in a subset of 1114 positive multiple myeloma cell lines)	1.6	0.00	0.59	-1.0	0.60	1.0	0.00	1.0	0.86	-1.0	0.00	-1.2	0.00	-1.4	0.00	-1.6	0.00	-1.0
A_23_P20460	NM_038165	MYEF2	myeloblastosis overexpressed (in a subset of 1114 positive multiple myeloma cell lines)	1.6	0.00	0.59	-1.0	0.60	1.0	0.00	1.0	0.86	-1.0	0.00	-1.2	0.00	-1.4	0.00	-1.6	0.00	-1.0
A_23_P20460	NM_038165	MYEF2	myeloblastosis overexpressed (in a subset of 1114 positive multiple myeloma cell lines)	1.6	0.00	0.59	-1.0	0.60	1.0	0.00	1.0	0.86	-1.0	0.00	-1.2	0.00	-1.4	0.00	-1.6	0.00	-1.0
A_23_P20460	NM_038165	MYEF2	myeloblastosis overexpressed (in a subset of 1114 positive multiple myeloma cell lines)	1.6	0.00	0.59	-1.0	0.60	1.0	0.00	1.0	0.86	-1.0	0.00	-1.2	0.00	-1.4	0.00	-1.6	0.00	-1.0
A_23_P20460	NM_038165	MYEF2	myeloblastosis overexpressed (in a subset of 1114 positive multiple myeloma cell lines)	1.6	0.00	0.59	-1.0	0.60	1.0	0.00	1.0	0.86	-1.0	0.00	-1.2	0.00	-1.4	0.00	-1.6	0.00	-1.0
A_23_P20460	NM_038165	MYEF2	myeloblastosis overexpressed (in a subset of 1114 positive multiple myeloma cell lines)	1.6	0.00	0.59	-1.0	0.60	1.0	0.00	1.0	0.86	-1.0	0.00	-1.2	0.00	-1.4	0.00	-1.6	0.00	-1.0
A_23_P20460	NM_038165	MYEF2	myeloblastosis overexpressed (in a subset of 1114 positive multiple myeloma cell lines)	1.6	0.00	0.59	-1.0	0.60	1.0	0.00	1.0	0.86	-1.0	0.00	-1.2	0.00	-1.4	0.00	-1.6	0.00	-1.0
A_23_P20460	NM_038165	MYEF2	myeloblastosis overexpressed (in a subset of 1114 positive multiple myeloma cell lines)	1.6	0.00	0.59	-1.0	0.60	1.0	0.00	1.0	0.86	-1.0	0.00	-1.2	0.00	-1.4	0.00	-1.6	0.00	-1.0
A_23_P20460	NM_038165	MYEF2	myeloblastosis overexpressed (in a subset of 1114 positive multiple myeloma cell lines)	1.6	0.00	0.59	-1.0	0.60	1.0	0.00	1.0	0.86	-1.0	0.00	-1.2	0.00	-1.4	0.00	-1.6	0.00	-1.0
A_23_P20460	NM_038165	MYEF2	myeloblastosis overexpressed (in a subset of 1114 positive multiple myeloma cell lines)	1.6	0.00	0.59	-1.0	0.60	1.0	0.00	1.0	0.86	-1.0	0.00	-1.2	0.00	-1.4	0.00	-1.6	0.00	-1.0
A_23_P20460	NM_038165	MYEF2	myeloblastosis overexpressed (in a subset of 1114 positive multiple myeloma cell lines)	1.6	0.00	0.59	-1.0	0.60	1.0	0.00	1.0	0.86	-1.0	0.00	-1.2	0.00	-1.4	0.00	-1.6	0.00	-1.0
A_23_P20460	NM_038165	MYEF2	myeloblastosis overexpressed (in a subset of 1114 positive multiple myeloma cell lines)	1.6	0.00	0.59	-1.0	0.60	1.0	0.00	1.0	0.86	-1.0	0.00	-1.2	0.00	-1.4	0.00	-1.6	0.00	-1.0
A_23_P20460	NM_038165	MYEF2	myeloblastosis overexpressed (in a subset of 1114 positive multiple myeloma cell lines)	1.6	0.00	0.59	-1.0	0.60	1.0	0.00	1.0	0.86	-1.0	0.00	-1.2	0.00	-1.4	0.00	-1.6	0.00	-1.0
A_23_P20460	NM_038165	MYEF2	myeloblastosis overexpressed (in a subset of 1114 positive multiple myeloma cell lines)	1.6	0.00	0.59	-1.0	0.60	1.0	0.00	1.0	0.86	-1.0	0.00	-1.2	0.00	-1.4	0.00	-1.6	0.00	-1.0
A_23_P20460	NM_038165	MYEF2	myeloblastosis overexpressed (in a subset of 1114 positive multiple myeloma cell lines)	1.6	0.00	0.59	-1.0	0.60	1.0	0.00	1.0	0.86	-1.0	0.00	-1.2	0.00	-1.4	0.00	-1.6	0.00	-1.0
A_23_P20460	NM_038165	MYEF2	myeloblastosis overexpressed (in a subset of 1114 positive multiple myeloma cell lines)	1.6	0.00	0.59	-1.0	0.60	1.0	0.00	1.0	0.86	-1.0	0.00	-1.2	0.00	-1.4	0.00	-1.6	0.00	-1.0
A_23_P20460	NM_038165	MYEF2	myeloblastosis overexpressed (in a subset of 1114 positive multiple myeloma cell lines)	1.6	0.00	0.59	-1.0	0.60	1.0	0.00	1.0	0.86	-1.0	0.00	-1.2	0.00	-1.4	0.00	-1.6	0.00	-1.0
A_23_P20460	NM_038165	MYEF2	myeloblastosis overexpressed (in a subset of 1114 positive multiple myeloma cell lines)	1.6	0.00	0.59	-1.0	0.60	1.0	0.00	1.0	0.86	-1.0	0.00	-1.2	0.00	-1.4	0.00	-1.6	0.00	-1.0
A_23_P20460	NM_038165	MYEF2	myeloblastosis overexpressed (in a subset of 1114 positive multiple myeloma cell lines)	1.6	0.00	0.59	-1.0	0.60	1.0	0.00	1.0	0.86	-1.0	0.00	-1.2	0.00	-1.4	0.00	-1.6	0.00	-1.0
A_23_P20460	NM_038165	MYEF2	myeloblastosis overexpressed (in a subset of 1114 positive multiple myeloma cell lines)	1.6	0.00	0.59	-1.0	0.60	1.0	0.00	1.0	0.86	-1.0	0.00	-1.2	0.00	-1.4	0.00	-1.6	0.00	-1.0
A_23_P20460	NM_038165	MYEF2	myeloblastosis overexpressed (in a subset of 1114 positive multiple myeloma cell lines)	1.6	0.00	0.59	-1.0	0.60	1.0	0.00	1.0	0.86	-1.0	0.00	-1.2	0.00	-1.4	0.00	-1.6	0.00	-1.0
A_23_P20460	NM_038165	MYEF2	myeloblastosis overexpressed (in a subset of 1114 positive multiple myeloma cell lines)	1.6	0.00	0.59	-1.0	0.60	1.0	0.00	1.0	0.86	-1.0	0.00	-1.2	0.00	-1.4	0.00	-1.6	0.00	-1.0
A_23_P20460	NM_038165	MYEF2	myeloblastosis overexpressed (in a subset of 1114 positive multiple myeloma cell lines)	1.6	0.00	0.59	-1.0	0.60	1.0	0.00	1.0	0.86	-1.0	0.00	-1.2	0.00	-1.4	0.00	-1.6	0.00	-1.0
A_23_P20460	NM_038165	MYEF2	myeloblastosis overexpressed (in a subset of 1114 positive multiple myeloma cell lines)	1.6	0.00	0.59	-1.0	0.60	1.0	0.00	1.0	0.86	-1.0	0.00	-1.2	0.00	-1.4	0.00	-1.6	0.00	-1.0
A_23_P20460	NM_038165	MYEF2	my																		

* False Discovery Rate adjusted P value; ^ Fold Change

Supplementary Table 8. BMD modeling parameters for the lowest BMD₁₀ values at the 10th percentile of all genes in a pathway for different approaches for POD derivation.

Issue	Approach	Time point	IPS Pathway	P probes with S	BMD*	BMD(L)	BMD(L)z	BMD(L)z	P70%ile Lz	P70%ile(L)z	P70%ile(L)z	BMD(L)	
Liver	Toxicogenomics-key event preceding the committed step	3d+24h	Cell Cycle: G1/S Checkpoint Regulation	18	8.1	1.0	0.32397 ± 0.45522	-0.15 ± 0.19546 ± 0.147514 ± 0.43886 ± 0.410139 ± 0.3635	0.27790 ± 0.27730 ± 0.30546 ± 0.345610 ± 0.36957 ± 0.37	51.149702 ± 4.72656 ± 64.91563 ± -27.74493 ± -30.74493 ± 73.197176 ± -51.21917 ± -57.72944 ± -44.52287 ± -80.612486 ± -67.72057 ± -40.91771 ± 51.004579 ± -38.0291196 ± -61.082766			
Liver	Toxicogenomics-key event preceding the committed step	3d+24h	Notch Signaling	18	1.1	0.1	0.273902 ± 0.27239	-0.157526 ± 0.157526 ± 22.8394 ± 35.5465 ± 37.18948 ± 0.8795 ± 0.50	0.277902 ± 0.27730 ± 0.30546 ± 0.345610 ± 0.36957 ± 0.37	51.149702 ± 4.72656 ± 64.91563 ± -27.74493 ± -30.74493 ± 73.197176 ± -51.21917 ± -57.72944 ± -44.52287 ± -80.612486 ± -67.72057 ± -40.91771 ± 51.004579 ± -38.0291196 ± -61.082766			
Lung	Toxicogenomics-lowest pathway	3d+24h	Cell Cycle: G2/M DNA Damage Checkpoint Regulation	7	0.3	0.2	0.273902 ± 0.27239	-0.157526 ± 0.157526 ± 22.8394 ± 35.5465 ± 37.18948 ± 0.8795 ± 0.50	0.277902 ± 0.27730 ± 0.30546 ± 0.345610 ± 0.36957 ± 0.37	51.149702 ± 4.72656 ± 64.91563 ± -27.74493 ± -30.74493 ± 73.197176 ± -51.21917 ± -57.72944 ± -44.52287 ± -80.612486 ± -67.72057 ± -40.91771 ± 51.004579 ± -38.0291196 ± -61.082766			
Lung	Toxicogenomics-key event preceding the committed step	28d+3d	Cell Cycle: G2/M DNA Damage Checkpoint Regulation	5	1.3	0.7	0.273902 ± 0.27239	-0.157526 ± 0.157526 ± 22.8394 ± 35.5465 ± 37.18948 ± 0.8795 ± 0.50	0.277902 ± 0.27730 ± 0.30546 ± 0.345610 ± 0.36957 ± 0.37	51.149702 ± 4.72656 ± 64.91563 ± -27.74493 ± -30.74493 ± 73.197176 ± -51.21917 ± -57.72944 ± -44.52287 ± -80.612486 ± -67.72057 ± -40.91771 ± 51.004579 ± -38.0291196 ± -61.082766			
Lung	Toxicogenomics-lowest MOA-associated pathway	28d+3d	Cell Cycle: G2/M DNA Damage Checkpoint Regulation	5	14.8	3.7	0.273902 ± 0.27239	-0.157526 ± 0.157526 ± 22.8394 ± 35.5465 ± 37.18948 ± 0.8795 ± 0.50	0.277902 ± 0.27730 ± 0.30546 ± 0.345610 ± 0.36957 ± 0.37	51.149702 ± 4.72656 ± 64.91563 ± -27.74493 ± -30.74493 ± 73.197176 ± -51.21917 ± -57.72944 ± -44.52287 ± -80.612486 ± -67.72057 ± -40.91771 ± 51.004579 ± -38.0291196 ± -61.082766			
Fore stomach	Toxicogenomics-lowest pathway	28d+3d	Cellular Effects of Sildenafl (Viagra)	9	15.7	2.1	0.273902 ± 0.27239	-0.157526 ± 0.157526 ± 22.8394 ± 35.5465 ± 37.18948 ± 0.8795 ± 0.50	0.277902 ± 0.27730 ± 0.30546 ± 0.345610 ± 0.36957 ± 0.37	51.149702 ± 4.72656 ± 64.91563 ± -27.74493 ± -30.74493 ± 73.197176 ± -51.21917 ± -57.72944 ± -44.52287 ± -80.612486 ± -67.72057 ± -40.91771 ± 51.004579 ± -38.0291196 ± -61.082766			
Fore stomach	Toxicogenomics-key event preceding the committed step	28d+3d	Cellular Effects of Sildenafl (Viagra)	9	15.7	2.1	0.273902 ± 0.27239	-0.157526 ± 0.157526 ± 22.8394 ± 35.5465 ± 37.18948 ± 0.8795 ± 0.50	0.277902 ± 0.27730 ± 0.30546 ± 0.345610 ± 0.36957 ± 0.37	51.149702 ± 4.72656 ± 64.91563 ± -27.74493 ± -30.74493 ± 73.197176 ± -51.21917 ± -57.72944 ± -44.52287 ± -80.612486 ± -67.72057 ± -40.91771 ± 51.004579 ± -38.0291196 ± -61.082766			
Fore stomach	Toxicogenomics-lowest MOA-associated pathway	28d+3d	Notch Signaling	40	11.4	7.4	7.59405 ± 10.9697	-0.22075 ± 0.20676 ± 2.73824 ± 7.30227 ± 7.40956 ± 7.47967 ± 7.90	0.1434 ± 0.9481 ± 0.16	31.270717 ± 17.389053 ± 23.385343 ± 26.76416 ± 38.793948 ± 57.87791 ± 44.083418 ± 40.018496 ± 34.690842 ± 45.577534 ± 25.686571 ± 37.635487 ± 41.852796 ± 31.508078 ± 45.89395 ± 57.833543 ± 40.434633 ± 46.99345 ± 44.988943 ± 25.699202 ± 26.389448 ± 71.18845 ± 49.936767 ± 19.253156 ± 26.514993 ± 48			
Fore stomach	Toxicogenomics-lowest pathway	28d+3d	Chlorophyll Degradation IV (Mammalian, via Side Chain)	6	16.1	4.5	0.273902 ± 0.27239	-0.157526 ± 0.157526 ± 22.8394 ± 35.5465 ± 37.18948 ± 0.8795 ± 0.50	0.277902 ± 0.27730 ± 0.30546 ± 0.345610 ± 0.36957 ± 0.37	51.149702 ± 4.72656 ± 64.91563 ± -27.74493 ± -30.74493 ± 73.197176 ± -51.21917 ± -57.72944 ± -44.52287 ± -80.612486 ± -67.72057 ± -40.91771 ± 51.004579 ± -38.0291196 ± -61.082766			

*in mg BaP/kg bw day

†Similar to Thomas et al., (2011) but examined BMD (BMDx) at the 10th percentile rather than median.