Center for Hormonforstyrrende Stoffer

Litteraturgennemgang for perioden 16/12 2011 - 31/3 2013

Indholdsfortegnelse Side

Humane studier ved Afd for Vækst og Reproduktion, Rigshospitalet

Søgning er udført på PubMed og dækker perioden 16/12/2011 - 31/3/2012

Følgende søgeprofil er benyttet:

Bisphenol A
Phthalat*
Paraben*
(fluor* OR polyfluor*)
Triclocarban
Triclosan
(Flame retardant)
tributyltin

kombineret med nedenstående tekst:

AND expos* AND (human OR men OR women OR child* OR adult* OR adolescen* OR infan*)

Limits: title/abstract, English language

Ved brug af den "oprindelige søgeprofil: (endocrine disrupt*) AND expos* AND (human OR men OR women OR child* OR adult* OR adolescen* OR infan*) fremkom 58 resultater, hvoraf en del overlappede med resultaterne af ovenstående profiler. Dog blev der ved denne søgning også fundet en del artikler om DDE, PCB, og organochlorins, der alle er irrelevante. Fremover vil denne søgeprofil derfor *ikke* blive brugt.

Overvejelser vedrørende kriterier for udvælgelse

Når enkelte artikler udvælges til kommentering, sker det ud fra følgende kriterier:

- Informationer skal (efter vores vurdering) være brugbare for Miljøstyrelsens arbejde
- Studierne skal være danske eller inkludere populationer der er sammenlignelige med den danske.
- Studierne skal beskæftige sig med stoffer, der stadig er i brug, og dermed relevante rent reguleringsmæssigt.
- De biologiske endpoints skal gerne være endokrint relevante. Dog inkluderer vi også nogle gange studier der kigger på relevante stoffer i forhold til for eksempel hjertekarsygdomme eller immunsygdomme.
- Studierne skal være pålidelige og have en ønskelig størrelse.

Bemærk: når vi først har gennemgået et studie med henblik på kommentering, så inkluderer vi vores kommentarer, også selvom studiet viser sig at være ubrugeligt i henhold til ovennævnte kriterier. Dette vil så naturligvis fremgå af vores kommentarer.

Hum Reprod. 2012 Mar;27(3):910-20. Epub 2012 Jan 2.

Occupational exposure to chemicals and fetal growth: the Generation R Study.

Snijder CA, Roeleveld N, Te Velde E, Steegers EA, Raat H, Hofman A, Jaddoe VW, Burdorf A.

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BACKGROUND Developmental diseases, such as birth defects, growth restriction and preterm delivery, account for >25% of infant mortality and morbidity. Several studies have shown that exposure to chemicals during pregnancy is associated with adverse birth outcomes. The aim of this study was to identify whether occupational exposure to various chemicals might adversely influence intrauterine growth patterns and placental weight.

METHODS Associations between maternal occupational exposure to various chemicals and fetal growth were studied in 4680 pregnant women participating in a population-based prospective cohort study from early pregnancy onwards in the Netherlands (2002-2006), the Generation R Study.

Mothers who filled out a questionnaire during mid-pregnancy (response: 77% of enrolment) were included if they conducted paid employment during pregnancy and had a spontaneously conceived singleton live born pregnancy (n = 4680). A job exposure matrix was used, linking job titles to expert judgement on exposure to chemicals in the workplace. Fetal growth characteristics were repeatedly measured by ultrasound and were used in combination with measurements at birth. Placental weight was obtained from medical records and hospital registries. Linear regression models for repeated measurements were used to study the associations between maternal occupational exposure to chemicals and intrauterine growth.

RESULTS We observed that maternal occupational exposure to polycyclic aromatic hydrocarbons, phthalates, alkylphenolic compounds and pesticides adversely influenced several domains of fetal growth (fetal weight, fetal head circumference and fetal length). We found a significant association between pesticide and phthalate exposure with a decreased placental weight.

CONCLUSIONS Our results suggest that maternal occupational exposure to several chemicals is associated with impaired fetal growth during pregnancy and a decreased placental weight. Further studies are needed to confirm these findings and to assess post-natal consequences.

Int J Androl. 2012 Feb 9. doi: 10.1111/j.1365-2605.2011.01240.x. [Epub ahead of print]

Cumulative risk assessment of phthalate exposure of Danish children and adolescents using the hazard index approach.

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Human risk assessment of chemicals is traditionally presented as the ratio between the actual level of exposure and an acceptable level of exposure, with the acceptable level of exposure most often being estimated by appropriate authorities. This approach is generally sound when assessing the risk of individual chemicals. However, several chemicals may concurrently target the same receptor, work through the same mechanism or in other ways induce the same effect(s) in the body. In these cases, cumulative risk assessment should be applied. The present study uses biomonitoring data from 129 Danish children and adolescents and resulting estimated daily intakes of four different phthalates. These daily intake estimates are used for a cumulative risk assessment with anti-androgenic effects as the endpoint using Tolerable Daily Intake (TDI) values determined by the European Food Safety Authorities (EFSA) or Reference Doses for Anti-Androgenicity (RfD AA) determined by Kortenkamp and Faust [Int J Androl 33 (2010) 463] as acceptable levels of exposure. United States Environmental Protection Agency Reference Doses (US EPA RfD) could not be used as none of them identifies anti-androgenic effects as the most sensitive endpoint for the phthalates included in this article. Using the EFSA TDI values, 12 children exceeded the hazard quotient for the sum of di-n-butyl phthalate and di-iso-butyl phthalate (∑DBP((i+n))) and one child exceeded the hazard quotient for di-(2-ethylhexyl)phthalate (DEHP). Nineteen children exceeded the cumulated hazard index for three phthalates. Using the RfD AA values, one child exceeded the hazard quotient for DEHP and the same child exceeded the cumulated hazard index for four phthalates. The EFSA TDI

approach thus is more restrictive and identifies ∑DBP((i+n)) as the compound(s) associated with the greatest risk, while DEHP is the compound associated with the greatest risk when using the RfD AA approach.

J Expo Sci Environ Epidemiol. 2012 Feb 22. doi: 10.1038/jes.2012.7. [Epub ahead of print]

Urinary phthalate metabolites and their biotransformation products: predictors and temporal variability among men and women.

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Most epidemiology studies investigating the potential adverse health effects in relation to phthalates measure the urinary concentration of the free plus glucuronidated species of phthalate metabolites (i.e., total concentration) to estimate exposure. However, the free species may represent the biologically relevant dose. In this study, we collected 943 urine samples from 112 men and 157 women and assessed the between- and within-person variability and predictors of (1) the free and total urinary concentrations of phthalate metabolites, and (2) the percentage of free phthalate metabolites (a potential phenotypic indicator of individual susceptibility). We also explored the proportion of urinary di-(2-ethylhexyl) phthalate (DEHP) metabolites contributed to by the bioactive mono-2-ethylhexyl phthalate (MEHP), considered a possible indicator of susceptibility to phthalate exposure. The percentage of phthalate metabolites present in the free form was less stable over time than the total metabolite concentration, and, therefore, it is not likely a useful indicator of metabolic susceptibility. Thus, the added costs and effort involved in the measurement of free in addition to total metabolite concentrations in large-scale studies may not be justified. Conversely, the proportion of DEHP metabolites contributed to by MEHP was more stable within individuals over time and may be a promising indicator of susceptibility if time of day of sample collection is carefully considered.

Environ Health Perspect. 2012 Jan 19. [Epub ahead of print]

Variability of Urinary Phthalate Metabolite and Bisphenol A Concentrations before and during Pregnancy.

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Background: Gestational phthalate and bisphenol A (BPA) exposure may increase the risk of adverse maternal/child health outcomes, but there is little data on the variability of urinary biomarkers before and during pregnancy. Objective: We characterized the variability of urinary phthalate metabolite and BPA concentrations before and during pregnancy and the ability of a single spot-urine sample to classify average gestational exposure. Methods: We collected 1,001 urine samples before and during pregnancy from 137 women who were partners in couples attending a Boston fertility clinic and had a live birth. Women provided spot-urine samples before (n>2) and during (n>2) pregnancy. We measured urinary concentrations of monoethyl phthalate (MEP), mono-n-butyl phthalate (MBP), mono-iso-butyl phthalate (MiBP), monobenzyl phthalate (MBzP), four metabolites of di-(2-ethylhexyl) phthalate (DEHP) and BPA. After adjusting for specific-gravity, we characterized biomarker variability using intraclass correlation coefficients (ICC) and conducted several surrogate category analyses to determine whether a single spot-urine sample could adequately classify average gestational exposure. Results: Concentrations of most phthalate metabolites and BPA were higher during pregnancy than before pregnancy. Variability was higher during pregnancy than before pregnancy for BPA and MBzP, but similar during and before pregnancy for MBP, MEP, and ΣDEHP. During pregnancy, MEP (ICC:0.50) and MBP (ICC:0.45) were less variable than BPA (ICC:0.12), MBzP (ICC:0.25), and ΣDEHP metabolites (ICC:0.08). Surrogate analyses suggested that a single spot-urine sample may reasonably classify MEP and MBP concentrations during pregnancy, but >1 sample may be necessary for MBzP, DEHP, and BPA. Conclusions: Urinary phthalate metabolites and BPA concentrations were variable before and during pregnancy, but the magnitude of variability was biomarker specific. A single spot-urine sample adequately classified MBP and MEP concentrations during pregnancy. The present results may be related to unique features of the women studied and replication in other pregnancy cohorts is recommended.

Environ Health Perspect. 2012 Mar;120(3):458-63. Epub 2011 Nov 23.
Association between Pregnancy Loss and Urinary Phthalate Levels around the Time of Conception.
Toft G, Jönsson BA, Lindh CH, Jensen TK, Hjollund NH, Vested A, Bonde JP.
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Background: Animal studies indicate that some phthalate metabolites may harm female reproductive function.Objectives: We assessed the associations between exposure to phthalate metabolites and pregnancy loss.Methods: Using a previously established cohort of couples planning their first pregnancy, we analyzed four primary and two oxidized secondary phthalate metabolites in urine samples collected on day 10 after the first day of the last menstrual period before conception occurred (n = 128) and during the previous cycle (if any, n = 111). Subclinical embryonal loss was identified by repeated measurement of urinary human chorionic gonadotropin, and information on clinical spontaneous abortions was obtained by telephone interview with the mother.Results: Pregnancy loss (n = 48) was increased among women with urinary concentration of monoethylhexyl phthalate (MEHP) in the upper tertile in the conception sample compared with women in the lowest tertile [adjusted odds ratio (OR) = 2.9; 95% confidence interval (CI): 1.1, 7.6]. The corresponding OR for subclinical embryonal loss (n = 32) was 40.7 (95% CI: 4.5, 69.5).Conclusions: The phthalate metabolite MEHP was associated with higher occurrence of pregnancy loss. Because this is the first human study to show this association and the sample size is small, the findings need to be corroborated in independent studies.

Environ Health Perspect. 2012 Mar;120(3):464-70. Epub 2011 Sep 7.
Exposure to Phthalates and Phenols during Pregnancy and Offspring Size at Birth.
Philippat C, Mortamais M, Chevrier C, Petit C, Calafat AM, Ye X, Silva MJ, Brambilla C, Pin I, Charles MA, Cordier S, Slama R.
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Background: Data concerning the effects of prenatal exposures to phthalates and phenols on fetal growth are limited in humans. Previous findings suggest possible effects of some phenols on male birth weight.Objective: Our aim was to assess the relationships between prenatal exposures to phthalates and phenols and fetal growth among male newborns.Methods: We conducted a case-control study on male malformations of the genitalia nested in two French mother-child cohorts with recruitment between 2002 and 2006. We measured, in maternal urinary samples collected between 6 and 30 gestational weeks, the concentrations (micrograms per liter) of 9 phenol (n = 191 pregnant women) and 11 phthalate metabolites (n = 287). Weight, length, and head circumference at birth were collected from maternity records. Statistical analyses were corrected for the oversampling of malformation cases.Results: Adjusted birth weight decreased by 77 g [95% confidence interval (CI): -129, -25] and by 49 g (95% CI: -86, -13) in association with a 1-unit increase in ln-transformed 2,4-dichlorophenol (DCP) and 2,5-DCP urinary concentrations, respectively. Benzophenone-3 (BP3) ln-transformed concentrations were positively associated with weight (26 g; 95% CI: -2, 54) and head circumference at birth (0.1 cm; 95% CI: 0.0, 0.2). Head circumference increased by 0.3 cm (95% CI: 0.0, 0.7) in association with a 1-unit increase in ln-transformed BPA concentration. For phthalate metabolites there was no evidence of monotonic associations with birth weight.Conclusions: Consistent with findings of a previous study, we observed evidence of an inverse association of 2,5-DCP and a positive association of BP3 with male birth weight.

Int J Androl. 2012 Mar 19. doi: 10.1111/j.1365-2605.2012.01260.x. [Epub ahead of print]

High urinary phthalate concentration associated with delayed pubarche in girls.

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Phthalates are a group of chemicals present in numerous consumer products. They have anti-androgenic properties in experimental studies and are suspected to be involved in human male reproductive health problems. A few studies have shown associations between phthalate exposure and changes in pubertal timing among girls, although controversies exist. We determined the concentration of 12 phthalate metabolites in first morning urine samples from 725 healthy Danish girls (aged 5.6-19.1 years) in relation to age, pubertal development (breast and pubic hair stage) and reproductive hormone levels (luteinizing hormone, oestradiol and testosterone). Furthermore, urinary phthalates were determined in 25 girls with precocious puberty (PP). In general, the youngest girls with less advanced pubertal development had the highest first morning urinary concentration of the monobutyl phthalate isoforms (∑MBP((i+n))), monobenzyl phthalate (MBzP), metabolites of di-(2-ethylhexyl) phthalate (∑DEHPm) and of di-iso-nonyl phthalate (∑DINPm). After stratification of the urinary phthalate excretion into quartiles, we found that the age at pubarche was increasing with increasing phthalate metabolite quartiles (except for MEP). This trend was statistically significant when all phthalate metabolites (except MEP) were summarized and expressed as quartiles. No association between phthalates and breast development was observed. In addition, there were no differences in urinary phthalate metabolite levels between girls with PP and controls. We demonstrated that delayed pubarche, but not thelarche, was associated with high phthalate excretion in urine samples from 725 healthy school girls, which may suggest anti-androgenic actions of phthalates in our study group of girls.

Circulation. 2012 Feb 21. [Epub ahead of print]

Urinary Bisphenol: A Concentration and Risk of Future Coronary Artery Disease in Apparently Healthy Men and Women.

Melzer D, Osborne NJ, Henley WE, Cipelli R, Young A, Money C, McCormack P, Luben R, Khaw KT, Wareham NJ, Galloway TS.

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BACKGROUND: The endocrine disrupting chemical Bisphenol A (BPA) is widely used in food and beverage packaging. Higher urinary BPA concentrations (uBPA) were cross-sectionally associated with heart disease in NHANES 2003/04 and NHANES 2005/6, independent of traditional risk factors.

METHODS AND RESULTS: We included 758 incident coronary artery disease (CAD) cases and 861 controls followed for 10.8 yrs from the European Prospective Investigation of Cancer - Norfolk UK. Respondents aged 40-74 yrs and free of CAD, stroke or diabetes provided baseline spot urine samples. uBPA concentrations (median value 1.3 ng/ml) were low. Per standard deviation (4.56ng/ml) increases in uBPA concentration were associated with incident CAD in age, sex and urinary creatinine adjusted models (n=1919, OR=1.13 95% CI 1.02 to 1.24, p=0.017). With CAD risk factor adjustment (including education, occupational social class, BMI category, systolic blood pressure, lipid concentrations and exercise) the estimate was similar but narrowly missed two-sided significance (n=1744 OR=1.11 95% CI: 1.00 to 1.23, p=0.058). Sensitivity analyses with the fully adjusted model, excluding early CAD (<3 year follow up); those with BMI>30; abnormal renal function; or adjusting additionally for vitamin C; C-reactive protein; or alcohol consumption, all produced similar estimates and all showed associations at p≤0.05.

CONCLUSIONS: Associations between higher BPA exposure (reflected in higher urinary concentrations) and incident CAD during over ten years of follow-up showed similar trends to previously reported cross-sectional findings in the more highly exposed NHANES respondents. Further work is needed to accurately estimate the prospective exposure response curve and to establish the underlying mechanisms.

J Clin Endocrinol Metab. 2011 Dec;96(12):3822-6. Epub 2011 Sep 28.
Relationship between urinary bisphenol A levels and diabetes mellitus.
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BACKGROUND: Bisphenol A (BPA) is a widely used chemical in the manufacture of polycarbonate plastics and epoxy resins. Recent animal studies have suggested that BPA exposure may have a role in the development of weight gain, insulin resistance, pancreatic endocrine dysfunction, thyroid hormone disruption, and several other mechanisms involved in the development of diabetes. However, few human studies have examined the association between markers of BPA exposure and diabetes mellitus. METHODS: We examined the association between urinary BPA levels and diabetes mellitus in the National Health and Nutritional Examination Survey (NHANES) 2003-2008. Urinary BPA levels were examined in quartiles. The main outcome of interest was diabetes mellitus defined according the latest American Diabetes Association guidelines. RESULTS: Overall, we observed a positive association between increasing levels of urinary BPA and diabetes mellitus, independent of confounding factors such as age, gender, race/ethnicity, body mass index, and serum cholesterol levels. Compared to quartile 1 (referent), the multivariate-adjusted odds ratio (95% confidence interval) of diabetes associated with quartile 4 was 1.68 (1.22-2.30) (p-trend = 0.002). The association was present among normal-weight as well as overweight and obese subjects. CONCLUSIONS: Urinary BPA levels are found to be associated with diabetes mellitus independent of traditional diabetes risk factors. Future prospective studies are needed to confirm or disprove this finding.

Environ Health Perspect. 2012 Jan 23. [Epub ahead of print]
Relationships of Perfluorooctanoate and Perfluorooctane Sulfonate Serum Concentrations Between Child-Mother Pairs in a Population with Perfluorooctanoate Exposure from Drinking Water.
Mondal D, Lopez-Espinosa MJ, Armstrong B, Stein CR, Fletcher T.
London School of Hygiene and Tropical Medicine.

Background: There are limited data on the associations between maternal, newborn and child exposure to perfluoroalkyl acids (PFAAs), including perfluorooctanoate (PFOA) and perfluorooctane sulfonate (PFOS). This study provides an opportunity to assess the association between PFAA concentrations in mother-child pairs in a population exposed to PFOA via drinking water. Objectives: To determine the relationship between child-mother PFAA serum concentrations, and examine how the child:mother ratio varies with child's age, child's gender, drinking water PFOA concentration, reported bottle water usage and mother's breastfeeding intention. Methods: We studied 4,943 child-mother pairs (child age: 1-19 years). The child:mother PFAA ratio was stratified by possible determinants. Results are summarized as geometric mean ratios and correlation coefficients between child-mother pairs, overall and within strata. Results: Child and mother PFOA and PFOS concentrations were correlated (r=0.82 and 0.26, respectively). Children had higher serum PFOA concentrations than their mothers up to about age 12 years. The highest child:mother PFOA ratio was found among children ≤5 years (44% higher than their mothers) which we attribute to in utero exposure and to exposure via breast milk and drinking water. Higher PFOS concentrations in children persisted until at least 19 years of age (42% higher than their mothers). Boys aged >5 years had significantly higher PFOA and PFOS child:mother ratios than girls. Conclusion: Concentrations of both PFOA and PFOS tended to be higher in children than their mothers. This difference persists until they are about 12 years for PFOA and at least until 19 years of age for PFOS.

Environ Health Perspect. 2012 Feb 3. [Epub ahead of print]
Prenatal Exposure to Perfluorooctanoate and Risk of Overweight at 20 Years of Age: A Prospective Cohort Study.
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University of Iceland.

Background: Perfluoroalkyl acids are persistent compounds used in various industrial applications. Of these compounds, perfluorooctanoate (PFOA) is currently detected in humans world-wide. A recent study on low dose developmental exposure to PFOA in mice reported increased weight and elevated biomarkers of adiposity in postpubertal female offspring. We examined whether those findings could be replicated in humans. Methods: A prospective cohort of 665 Danish pregnant women was recruited in 1988-1989 with offspring follow-up at 20 years. PFOA was measured in serum from gestational week 30. Offspring body mass index (BMI) and waist circumference was recorded at follow-up (n=665) and biomarkers of adiposity were quantified in a sub-set (n=422) of participants. Results: After adjustment for covariates, including maternal pre-pregnancy body mass index (BMI), smoking, education and birth weight, in utero exposure to PFOA was positively associated with anthropometry at 20 years in female but not male offspring. Adjusted relative risks comparing the highest to lowest quartile (median: 5.8 vs. 2.3ng/mL) of maternal PFOA concentration were 3.1 (95% confidence interval (CI): 1.4, 6.9) for overweight or obese (BM≥25 kg/m2) and 3.0 (95%CI: 1.3, 6.8) for waist circumference >88 cm among female offspring. This corresponded to estimated increases of 1.6 kg/m2 (95%CI: 0.6, 2.6) and 4.3 cm (95%CI: 1.4, 7.3) in average BMI and waist circumference, respectively. In addition, maternal PFOA concentrations were positively associated with serum insulin and leptin levels, and inversely associated with adiponectin levels in female offspring. Similar associations were observed for males, although point estimates were less precise due to fever number of observations. Maternal PFOS, PFOSA and PFNA concentrations were not independently associated with offspring anthropometry at 20 years. Conclusions: Our findings on low dose developmental exposures to PFOA are in line with experimental results suggesting obesogenic effects in female offspring at 20 years.

Hum Reprod. 2012 Mar;27(3):873-80. Epub 2012 Jan 13.

Association between perfluorinated compounds and time to pregnancy in a prospective cohort of Danish couples attempting to conceive.

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BACKGROUND: Perfluorinated chemicals (PFCs) have been widely used and have emerged as important food contaminants. A recent study on pregnant women suggested that PFC exposure was associated with a longer time to pregnancy (TTP). We examined the association between serum concentrations of PFCs in females and TTP in 222 Danish first-time pregnancy planners during the years 1992-1995. METHODS: The couples were enrolled in the study when discontinuing birth control and followed for six menstrual cycles or until a clinically recognized pregnancy occurred. Fecundability ratio (FR) was calculated using discrete-time survival models. In addition, odds ratio (OR) for TTP >6 cycles was calculated. RESULTS: OR for TTP >6 cycles for those with PFC concentrations above the median were 0.96 [95% confidence interval (CI): 0.54-1.64] for perfluorooctane sulfonic acid (PFOS), the major PFC, compared with those below the median. FRs for those with PFOS concentrations above the median were 1.05 (95% CI: 0.74-1.48) compared with those below the median. Other PFCs showed the same lack of association with TTP. The results were not affected by adjustment for covariates. PFOS and perfluorooctanoic acid concentrations were similar to those observed in a previous Danish study. CONCLUSIONS: These findings suggest that exposure to PFCs affects TTP only to a small extent, if at all.

Bruttoliste

Phthalater

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Flame retardants

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Tributyltin

1: Fiorini R, Pagliarani A, Nesci S, Pirini M, Tucci E, Ventrella V. Structural and functional changes in gill mitochondrial membranes from the Mediterranean mussel Mytilus galloprovincialis exposed to tri-n-butyltin. Environ Toxicol Chem. 2012 Apr;31(4):877-84. doi: 10.1002/etc.1764. Epub 2012 Feb 28. PubMed PMID: 22374617.
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In vitro studier ved DTU

***In vitro* studier ved DTU-FOOD**

Søgt i Pubmed med følgende kriterier:

" Endocrine disrupt* AND in vitro*" samt "Endocrine disrupt* AND expose* AND in vitro*" og "Paraben* AND in vitro*"

Limits activated: published in the last 180 days (January-March 2012)

Efter at have fjernet genganger, fra dem vi havde med på de forrige litteraturopdateringslister, gav litteratursøgningen, med de angivne søgekriterier, tilsammen en liste med i alt 22 artikler (Bruttolisten):

Udvalgte publikationer:

Tre artikler er blevet udvalgt til nærmere beskrivelse baseret på at de beskrevne resultater bidrager: 1) til ny eller mere viden omkring effekterne eller virkningsmekanismerne for den eller de beskrevet hormonforstyrrende stoffer, 2) at artiklen bidrager til ny viden omkring *in vitro* testningsmetoder og eller 3) at artiklen kommer med værdifuld information om hvordan vi generelt forbedrer måden, vi risikovurderer hormonforstyrrende stoffer.

Human testis steroidogenesis is inhibited by phthalates.

Desdoits-Lethimonier C, Albert O, Le Bizec B, Perdu E, Zalko D, Courant F, Lesné L, Guillé F, Dejucq-Rainsford N, Jégou B.

Abstract:

BACKGROUND: Phthalic acid esters are widely used in the manufacture of plastics. Numerous studies have shown that these phthalates impair testicular testosterone production in the rat. However, the scarce and contradictory data concerning humans have cast doubt over whether these compounds are also anti-androgenic in man. We therefore investigated the direct effects of di-(2-ethylhexyl) phthalate (DEHP) and mono-(2-ethylhexyl) phthalate (MEHP) on organo-cultured adult human testis and a human cell line.

METHODS: Adult human testis explants or NCI-H295R adrenocortical human cells were cultured with DEHP or MEHP. The effects of ketoconazole, used as a reference molecule, were also assessed.

RESULTS: In both models, DEHP and MEHP significantly inhibited testosterone production. The effects of both phthalates appeared to be specific for steroidogenesis, as INSL3 production by Leydig cells was not altered. Furthermore, the phthalates of interest had no effect on inhibin B production by Sertoli cells or on germ cell apoptosis. As only a small fraction of the phthalates added was found in the testis explants, and as these compounds were found to be metabolized, we estimate that the anti-androgenic effects observed occurred at concentrations of phthalates that are of the same order of magnitude as exposures reported in the literature for men.

CONCLUSIONS: We provide the first evidence that DEHP and MEHP can inhibit testosterone production in the adult human testis. This is consistent with recent epidemiological findings of an inverse correlation between exposure to MEHP and testosterone concentrations.

Bisphenol a and its **analogues** activate human pregnane x receptor.
Sui Y, Ai N, Park SH, Rios-Pilier J, Perkins JT, Welsh WJ, Zhou C.
Abstract:
Background: Bisphenol A (BPA) is a base chemical used extensively in many consumer products. BPA and its analogues are present in environmental and human samples. Many endocrine-disrupting chemicals, including BPA, have been shown to activate the pregnane X receptor (PXR), a nuclear receptor that functions as a master regulator of xenobiotic metabolism. However, the detailed mechanism by which these chemicals activate PXR remains unknown. Objective: We investigated the mechanism by which BPA interacts with and activates PXR and examined selected BPA analogues to determine whether they bind to and activate PXR. Methods: Cell-based reporter assays, in silico ligand-PXR docking studies, and site-directed mutagenesis were combined to study the interaction between BPA and PXR. We also investigated the influence of BPA and its analogues on the regulation of PXR target genes in human LS180 cells.
Results: We found that BPA and several of its analogues are potent agonists for human PXR (hPXR) but do not affect mouse PXR activity. We identified key residues within hPXR's ligand-binding pocket that constitute points of interaction with BPA. We also deduced the structural requirements of BPA analogues that activate hPXR. BPA and its analogues can also induce PXR target gene expression in human LS180 cells. Conclusions: The present study advances our understanding of the mechanism by which BPA interacts with and activates human PXR. Activation of PXR by BPA may explain some of the adverse effects of BPA in humans.

Antiandrogenic activity of **phthalate mixtures**: Validity of concentration addition.
Christen V, Crettaz P, Oberli-Schrämmli A, Fent K.
Abstract:
Phthalates and bisphenol A have very widespread use leading to significant exposure of humans. They are suspected to interfere with the endocrine system, including the androgen, estrogen and the thyroid hormone system. Here we analyzed the antiandrogenic activity of six binary, and one ternary mixture of phthalates exhibiting complete antiandrogenic dose-response curves, and binary mixtures of phthalates and bisphenol A at equi-effective concentrations of EC(10), EC(25) and EC(50) in MDA-kb2 cells. Mixture activity followed the concentration addition (CA) model with a tendency to synergism at high and antagonism at low concentrations. Isoboles and the toxic unit approach (TUA) confirmed the additive to synergistic activity of the binary mixtures BBP+DBP, DBP+DEP and DEP+BPA at high concentrations. Both methods indicate a tendency to antagonism for the EC(10) mixtures BBP+DBP, BBP+DEP and DBP+DEP, and the EC(25) mixture of DBP+BPA. A ternary mixture revealed synergism at the EC(50), and weak antagonistic activity at the EC(25) level by the TUA. A mixture of five phthalates representing a human urine composition and reflecting exposure to corresponding parent compounds showed no antiandrogenic activity. Our study demonstrates that CA is an appropriate concept to account for mixture effects of antiandrogenic phthalates and bisphenol A. The interaction indicates a departure from additivity to antagonism at low concentrations, probably due to interaction with the androgen receptor and/or cofactors. This study emphasizes that a risk assessment of phthalates should account for mixture effects by applying the CA concept.

Bruttolisten

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Effects of a toxic cyanobacterial bloom (Planktothrix agardhii) on fish: Insights from histopathological and quantitative proteomic assessments following the oral exposure of medaka fish (Oryzias latipes).
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The novel endocrine disruptor tolylfluanid impairs insulin signaling in primary rodent and human adipocytes through a reduction in insulin receptor substrate-1 levels.
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Endocrine-disrupting chemicals in human follicular fluid impair in vitro oocyte developmental competence.
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Wang L, Zou W, Zhong Y, An J, Zhang X, Wu M, Yu Z.
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Binding of phthalate plasticizers to human serum albumin in vitro: a multispectroscopic approach and molecular modeling.
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Disruption of androgen receptor signaling in males by environmental chemicals.
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Minireview: PPARγ as the target of obesogens.
Janesick A, Blumberg B.
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Herudover er der yderligere 11 artikel, som ikke blev fanget af de valgte søgekriterier:

Human testis steroidogenesis is inhibited by phthalates.
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Billionnet C, Sherrill D, Annesi-Maesano I; GERIE study.
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Effects of bisphenol A exposure on the proliferation and senescence of normal human mammary epithelial cells.
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Bisphenol a and its analogues activate human pregnane x receptor.
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Predictive insight into the relationship between AhR binding property and toxicity of polybrominated diphenyl ethers by PLS-derived QSAR.
Gu C, Goodarzi M, Yang X, Bian Y, Sun C, Jiang X.
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In vitro characterization of the immunotoxic potential of several perfluorinated compounds (PFCs).
Corsini E, Sangiovanni E, Avogadro A, Galbiati V, Viviani B, Marinovich M, Galli CL, Dell'Agli M, Germolec DR.
Toxicol Appl Pharmacol. 2012 Jan 15;258(2):248-55. Epub 2011 Nov 18.

Antiandrogenic activity of phthalate mixtures: Validity of concentration addition.
Christen V, Crettaz P, Oberli-Schrämmli A, Fent K.
Toxicol Appl Pharmacol. 2012 Mar 1;259(2):169-76. Epub 2012 Jan 8.

In Vivo studier ved DTU - FOOD

Søgning er udført på PubMed og Scopus og dækker perioden 1/1 2012 – 28/3 2012
(Januar – marts 2012)
Følgende søgeprofil er benyttet: ”(endocrine disrupt*) AND (utero*) AND (rat OR mice OR mammal*)” samt ”(endocrine disrupt*) AND (rat OR mice OR mammal*)”. Derudover er der også søgt på ”(Paraben*) AND (in vivo*)”.
Efter at have fjernet genganger fra dem vi havde med på den forrige litteraturopdateringsliste, gav litteratursøgningen tilsammen en liste med i alt 31 artikler (Bruttolisten):

Tre artikler er blevet udvalgt til nærmere beskrivelse. Disse 3 er valgt fordi vi mener de bidrager til ny viden om phthalater (Driesche et al. og Mitchell et al.), relevans for mennesker (Mitchell et al., Christiansen et al.) og ny viden om samspilseffekter af en blanding af miljøkemikalier hos rotter (Christiansen et al.).

Ud fra bruttolisten (se længere nede i dokumentet) er udvalgt følgende 3 artikler til engelsk abstrakt og dansk resume og kommentarer:
Udvalgte publikationer:

Inter-relationship between testicular dysgenesis and Leydig cell function in the masculinization programming window in the rat.
Driesche S, Kolovos P, Platts S, Drake AJ, Sharpe RM.
PLoS One. 2012;7(1):e30111. Epub 2012 Jan 11.

Abstract
The testicular dysgenesis syndrome (TDS) hypothesis proposes that maldevelopment of the testis, irrespective of cause, leads to malfunction of the somatic (Leydig, Sertoli) cells and consequent downstream TDS disorders. Studies in rats exposed in utero to di(n-butyl) phthalate (DBP) have strongly supported the TDS concept, but so far no direct evidence has been produced that links dysgenesis per se to somatic cell dysfunction, in particular to androgen production/action during the ‘masculinization programming window’ (MPW; e15.5–e18.5). Normal reproductive tract development and anogenital distance (AGD) are programmed within the MPW, and TDS disorders arise because of deficiencies in this programming. However, DBP-induced focal testicular dysgenesis (Leydig cell aggregation, ectopic Sertoli cells, malformed seminiferous cords) is not evident until after the MPW. Therefore, we used AGD as a read-out of androgen exposure in the MPW, and investigated if this measure was related to objectively quantified dysgenesis (Leydig cell aggregation) at e21.5 in male fetuses exposed to vehicle, DBP (500 or 750 mg/kg/day) or the synthetic glucocorticoid dexamethasone (Dex; alone or plus DBP-500) from e15.5–e18.5 (MPW), e13.5–e20.5 or e19.5–e20.5 (late window). Dysgenesis was found only in animals exposed to DBP during the MPW, and was negatively correlated (R2 = 20.5) with AGD at e21.5 and at postnatal day 8, irrespective of treatment period. Dysgenesis was also negatively correlated (R2 = –0.5) with intratesticular testosterone (ITT) at e21.5, but only when treatments in short windows (MPW, late window) were excluded; the same was true for correlation between AGD and ITT. We conclude that AGD, reflecting Leydig cell function solely within the MPW, is strongly related to focal dysgenesis. Our results point to this occurring because of a common early mechanism, targeted by DBP that determines both dysgenesis and early (during the MPW) fetal Leydig cell dysfunction. The findings provide strong validation of the TDS hypothesis.

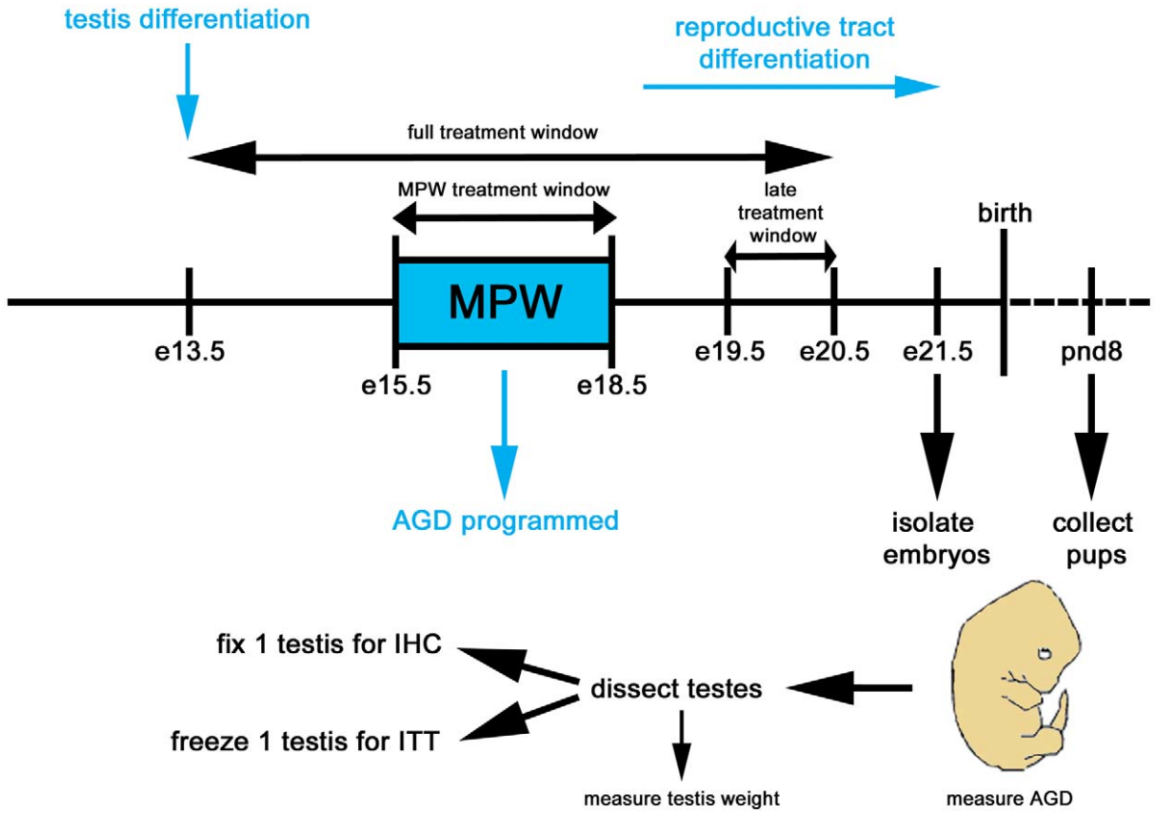

Figure 1. Schematic representation of the various treatment windows and experimental design. Also indicated in blue are testis differentiation in the rat (at , embryonic day (e)13.5), reproductive tract differentiation (from , e18.5 onwards) and the masculinization programming window (MPW, e15.5–e18.5), during which anogenital distance (AGD) is programmed. Three treatment windows were used in this study, namely 'full treatment window' (e13.5–e20.5), 'MPW treatment window' (e15.5–e18.5) and 'late treatment window' (e19.5–e20.5). At e21.5 embryos were isolated. For each embryo, AGD was measured, testes were dissected and weighed, before 1 testis was fixed for subsequent immunohistochemistry (IHC) and 1 testis was frozen on dry ice for subsequent intratesticular testosterone (ITT) measurement. In a separate experiment, pregnant rats were treated from e13.5–e21.5 and pups were collected on postnatal day (pnd) 8 and the same procedures as for the fetal samples were undertaken.
doi:10.1371/journal.pone.0030111.g001

Mitchell RT, Childs AJ, Anderson RA, van den Driesche S, Saunders PT, McKinnell C, Wallace WH, Kelnar CJ, Sharpe RM. Do Phthalates Affect Steroidogenesis by the Human Fetal Testis? Exposure of Human Fetal Testis Xenografts to Di-n-Butyl Phthalate. J Clin Endocrinol Metab, 11. januar 2012. http://www.ncbi.nlm.nih.gov/pubmed/22238399

Abstract

Context: Phthalates are ubiquitous environmental chemicals. Fetal exposure to certain phthalates [*e.g.* di-n-butyl phthalate (DBP)] causes masculinization disorders in rats, raising concern for similar effects in humans. We investigated whether DBP exposure impairs steroidogenesis by the human fetal testis.

Objective: The aim of the study was to determine effects of DBP exposure on testosterone production by normally growing human fetal testis xenografts.

Design: Human fetal testes (14–20wkgestation; n=12) were xenografted into castrate male nude mice that were treated for 4–21dwith vehicle, or 500mg/kg!dDBP, ormonobutylphthalate (active metabolite of DBP); all mice were treated with human chorionic gonadotropin to mimic normal human pregnancy. Rat fetal testis xenografts were exposed for 4 d to DBP as a positive control.

Main Outcome Measures: Testosterone production was assessed by measuring host serum testosterone and seminal vesicle (SV) weights at termination, plus testis gene expression (rats).
Results: Human fetal testis xenografts showed similar survival ("80%) and total graft weight (8.6 *vs*. 10.1 mg) in vehicle and DBP-exposed hosts, respectively. Serum testosterone (0.56 *vs*. 0.64 ng/ml; *P* # 0.05) and SV weight (67.2 *vs*. 81.9 mg; *P* # 0.05) also did not differ. Exposure to monobutyl phthalate gave similar results. In contrast, exposure of rat fetal xenografts to DBP significantly reduced SV weight and testis *Cyp11a1/StAR* mRNA expression and lowered testosterone levels, confirming that DBP exposure can inhibit steroidogenesis in xenografts, further validating the negative findings on testosterone production in the human.
Conclusions: Exposure of human fetal testes to DBP is unlikely to impair testosterone production as it does in rats. This has important safety and regulatory implications. **(*J Clin Endocrinol Metab* 97: 0000–0000, 2012).**

Mixtures of endocrine disrupting contaminants modelled on human high end exposures: an exploratory study in rats.Christiansen S, Kortenkamp A, Axelstad M, Boberg J, Scholze M, Jacobsen PR, Faust M, Lichtensteiger W, Schlumpf M, Burdorf A, Hass U.Int J Androl. 2012 Feb 28. doi: 10.1111/j.1365-2605.2011.01242.x. [Epub ahead of print]

Abstract
By diminishing the action of androgens during gestation, certain chemicals can induce irreversible demasculinization and malformations of sex organs in the male rat after gestational exposure. Studies with mixtures of such anti-andro- gens have shown that substantial combined effects occur even though each individual chemical is present at low, ineffective doses, but the effects of mixtures modelled based on human intakes have not previously been investigated. To address this issue for the first time, we selected 13 chemicals for a develop- mental mixture toxicity study in rats where data about in vivo endocrine disrupting effects and information about human exposures was available, including phthalates, pesticides, UV-filters, bisphenol A, parabens and the drug paracetamol. The mixture ratio was chosen to reflect high end human intakes. To make decisions about the dose levels for studies in the rat, we employed the point of departure index (PODI) approach, which sums up ratios between estimated exposure levels and no-observed-adverse-effect-level (NOAEL) values of individual substances. For high end human exposures to the 13 selected chemicals, we calculated a PODI of 0.016. As only a PODI exceeding 1 is expected to lead to effects in the rat, a total dose more than 62 times higher than human exposures should lead to responses. Considering the high uncer- tainty of this estimate, experience on lowest-observed-adverse-effect-level (LOAEL)/NOAEL ratios and statistical power of rat studies, we expected that combined doses 150 times higher than high end human intake estimates should give no, or only borderline effects, whereas doses 450 times higher should pro- duce significant responses. Experiments indeed showed clear developmental toxicity of the 450-fold dose in terms of increased nipple retention (NR) and reduced ventral prostate weight. The 150-fold dose group exhibited significantly increased NR. These observations suggest that highly exposed population groups, especially women of reproductive age, may not be protected sufficiently against the combined effects of chemicals that affect the hormonal milieu required for normal male sexual differentiation.

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Wildlife studier ved Biologisk Institut, Syddansk Universitet (SDU)

Søgningen er udført på Web of Science og dækker perioden 9/12 2011 - 26/3 2012.

Søgeprofilen kombinerer:	Endocrine disrupt*	og	Fish* Amphibia* Bird* OR Avia* Invertebrat* Mollus* Gastropod* Insect* Crustacea* Echinoderm* Ursus Reptil* OR Alligator Whal* OR seal OR dolphin

Fra bruttolisten (længere nede i dokumentet) er udvalgt fire artikler til medtagelse af abstract og yderligere kommentarer.
Kriterierne for udvælgelsen af publikationer til kommentering er, at de repræsenterer vigtig viden, som vurderes at have særlig interesse for Miljøstyrelsen bl.a. i forbindelse med styrelsen fokus på udvikling af testmetoder. Desuden kommenteres artikler, der omhandler 'nye' stoffer og miljøfaktorer, der har vist sig hormonforstyrrende; specielt hvis disse har relevans for danske forhold. Endelig medtages, efter Miljøstyrelsens ønske, artikler omhandlende parabener.

Artikel 1:

Holbech,H., Kinnberg,K.L., Brande-Lavridsen,N., Bjerregaard,P., Petersen,G.I., Norrgren,L., Orn,S., Braunbeck,T., Baumann,L., Bomke,C., Dorgerloh,M., Bruns,E., Ruehl-Fehlert,C., Green,J.W., Springer,T.A., and Gourmelon,A., 2012. Comparison of zebrafish (Danio rerio) and fathead minnow (Pimephales promelas) as test species in the Fish Sexual Development Test (FSDT). Comparative Biochemistry and Physiology C-Toxicology & Pharmacology 155, 407-415.

Abstract: Results are presented from a validation (with 5 laboratories) of the Fish Sexual Development Test (FSDT) developed to detect endocrine disrupters (EDs) and included in the OECD (Organisation for Economic Co-operation and Development) working program. The aromatase-inhibiting fungicide prochloraz was tested in zebrafish (*Danio rerio*) and fathead minnow (*Pimephales promelas*). The fish were exposed during sexual differentiation and development from 0 to 60 days post hatch (dph). After exposure, the vitellogenin (VTG) concentrations were quantified in head/tail homogenate and the sex ratio was determined (defined as female, male, intersex or undifferentiated). NOEC/LOEC and EC_x designs were compared to optimize the test approach. Results show that both species are highly sensitive to prochloraz during sexual development. They respond by skewing of the sex ratio towards male phenotype and by a VTG decline in females. The NOEC/LOEC approach is preferred because sex ratio is difficult to analyze with a regression model. The mean NOEC/LOEC for prochloraz on the sex ratio was 43.3/134 µg/L and 101/293 µg/L for zebrafish and fathead minnow, respectively. The mean NOEC/LOEC on the decline in female VTG concentration was 65/110 µg/L and ~ 30/68 µg/L respectively. In conclusion, zebrafish and fathead minnow are suitable species in the FSDT and their sexual differentiation is equally labile to EDs.

Artikel 2:

Stepankova,T., Ambrozova,L., Blaha,L., Giesy,J.P., and Hilscherova,K., 2011. In vitro modulation of intracellular receptor signaling and cytotoxicity induced by extracts of cyanobacteria, complex water blooms and their fractions. Aquatic Toxicology 105, 497-507.

Abstract: The biological activity of cyanobacteria and their chemical components have been widely studied due to their blooms in eutrophic waters worldwide. The primary goal of this study was to determine if individual cyanobacterial species and mixtures of cyanobacteria collected from the environment contain compounds with the potential for interaction with signaling pathways of the aryl hydrocarbon receptor (AhR), androgen receptor (AR), estrogen receptor (ER), glucocorticoid receptor (GR) and retinoid acid receptor (RAR). Cytotoxicity and specific toxic potencies of products of freshwater cyanobacteria were determined by use of in vitro reporter gene trans-activation assays. The testing included samples prepared from five selected single cyanobacterial species cultivated in laboratory and five complex cyanobacterial biomasses collected from blooms in surface waters in the Czech Republic. The results demonstrate estrogenic potencies of extracts of cyanobacterial biomasses. Among the laboratory single species, the extract of Planktothrix agardhii (intracellular metabolites) had a potency of estrogenic equivalents (EEQ) of 3.8 ng 17β-estradiol/g dw. The estimates of EEQs of samples prepared from complex cyanobacterial biomasses collected from freshwaters in the Czech Republic ranged from 19 to 2200 ng 17β-estradiol/g dw. Several samples prepared from the environmental cyanobacterial biomasses potentiated the androgenic potency of dihydrotestosterone. There was no dioxin-like, glucocorticoid or anti/retinoic activity observed for any of the extracts studied. Extracts of natural complex cyanobacterial biomasses exhibited greater and more frequent presence of compounds with specific modes of action, mainly estrogenic, and also greater cytotoxicity than extracts of single cyanobacterial species. The demonstrated estrogenic potency of the compounds present in complex cyanobacterial biomasses is of environmental relevance, and could potentially contribute to endocrine disruptive effects in aquatic ecosystems in case of great bloom densities.

Artikel 3:

Sychrova,E., Stepankova,T., Novakova,K., Blaha,L., Giesy,J.P., and Hilscherova,K., 2012. Estrogenic activity in extracts and exudates of cyanobacteria and green algae. Environment International 39, 134-140.

Abstract: Here is presented some of the first information on interactions of compounds produced by cyanobacteria and green algae with estrogen receptor signaling. Estrogenic potency of aqueous extracts and exudates (culture spent media with extracellular products) of seven species of cyanobacteria (10 different laboratory strains) and two algal species were assessed by use of in vitro trans-activation assays. Compounds produced by cyanobacteria and algae, and in particular those excreted from the cells, were estrogenic. Most exudates were estrogenic with potencies expressed at 50% of the maximum response under control of the estrogen receptor ranging from 0.2 to 7.2 ng 17β-estradiol (E2) equivalents (EEQ)/L. The greatest estrogenic potency was observed for exudates of Microcystis aerigunosa, a common species that forms water blooms. Aqueous extracts of both green algae, but only one species of cyanobacteria (Aphanizomenon gracile) elicited significant estrogenicity with EEQ ranging from 15 to 280 ng 17β-estradiol (E2)/g dry weight. Scenedesmus quadricauda exudates and extracts of Aphanizomenon flos-aquae were antagonistic to the ER when coexposed to E2. The EEQ potency was not correlated with concentrations of cyanotoxins, such as microcystin and cylindrospermopsin, which suggests that the EEQ was comprised of other compounds. The study demonstrates some differencesbetween the estrogenic potency of aqueous extracts prepared from the same species, but of different origin, while the effects of exudates were comparable within species. The observed estrogenic potencies are important namely in relation to the possible mass expansion of cyanobacteria and release of the active compounds into surrounding water.

Artikel 4:

Yamamoto,H., Tamura,I., Hirata,Y., Kato,J., Kagota,K., Katsuki,S., Yamamoto,A., Kagami,Y., and Tatarazako,N., 2011. Aquatic toxicity and ecological risk assessment of seven parabens: Individual and additive approach. Science of the Total Environment 410, 102-111.

Abstract: In the present study, aquatic concentrations of seven parabens were determined in urban streams highly affected by treated or untreated domestic sewage in Tokushima and Osaka, Japan. The detected highest concentrationswere 670, 207, and 163 ng l^{-1} for methylparaben, n-propylparaben, and n-butylparaben, respectively in sampling sites with watershed area of no sewer system in Tokushima. Conventional acute/chronic toxicity tests were conducted using medaka (Oryzias latipes), Daphnia magna, and Psuedokirchneriella subcapitata for four parabens, which was consistent with our previous study on three parabens, n-butylparaben, ibutylparaben, and benzylparaben. The aquatic toxicity on fish, daphnia, and algae was weaker for the parabens with a shorter alkyl chain than those with a longer alkyl chain as predicted by their hydrophobicity. Medaka vitellogenin assays and DNA microarray analysis were carried out for methylparaben and found induction of significant vitellogenin in male medaka at 630 μg l^{-1} of methylparaben, while the expression levels of genes encoding proteins such as choriogenin and vitellogenin increased for concentrations at 10 μg l^{-1} of methylparaben. Measured environmental concentrations (MECs) of seven parabens in Tokushima and Osaka were divided by predicted no effect concentrations (PNECs) and hazard quotient (MEC/PNEC) was determined for individual parabens. The MEC/PNEC was highest for n-propylparaben and was 0.010 followed by n-butylparaben (max. of 0.0086) and methylparaben (max. of 0.0042). The sum of the MEC/PNEC for the seven parabens was 0.0049. Equivalence factors were assigned for each paraben on the basis of the toxicity of n-butylparaben for each species, and n-butylparaben equivalence was calculated for the measured environmental concentrations. The MEC/PNEC approach was also conducted for the n-butylparaben-based equivalence values. The maximum MEC/PNEC was 0.018, which is lower than the trigger level for further detailed study such as large-scale monitoring for chronic toxicity tests including full-life cycle tests for fish.

Table 4
Results of acute toxicity tests for seven parabens using medaka, *Daphnia magna*, and green alagae.

	Algae (72 h-EC_{50})		Daphnia (48 h-EC_{50})		Fish (96 h-LC_{50})	
	Our results	Literature	Our results	Literature	Our results	Literature
Methyl-paraben	80,000 (ND[b])	91,000[a]	34,000 (30,000–39,000)	11,200[a] 41,100[c] 62,000[e] 24,600[d]	63,000 (50,000–93,000)	<160,000[d]
Ethyl-paraben	52,000 (ND[b])	18,000[a]	7400 (6200–8900)	20,000–50,000[a] 32,000[e] 18,700[d]	14,000 (10,000–19,000)	34,300[d]
n-propyl-paraben	36,000 (ND[b])	15,000[a]	2000 (770–2900)	15,400[a] 23,000[e] 12,300[d]	4900 (3600–6700)	9700[d]
i-propyl-paraben	48,000 (33,000–69,000)		3500 (3100–4200)	30,000[e] 8500[d]	4500 (3100–6800)	17,500[d]
n-butyl-paraben	9500[f]		1900[b] (1700–2600)	9200[e] 5300[d]	3100[b] (2500–8200)	4200[d]
i-butyl-paraben	4000[f]		3300[f]	9800[e] 7600[d]	4600[f]	6900[d]
Benzyl-paraben	1200[f]		2100[f]	6600[e] 4000[d]	730[f]	3300[d]

Unit: μg l^{-1}
Range of 95% confidence level was determined and presented within the parentheses for the acute data.
[a] From Madsen et al. (2001).
[b] Not determined due to the high jump in inhibition ratio.
[c] From Kamaya et al. (2005).
[d] From Dobbins et al. (2009).
[e] From Terasaki et al. (2008).
[f] From Yamamoto et al. (2007a).

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