toma (SH-SY5Y) cell line. IC_{50} value of AKB48 was calculated as 160.91 μM by MTT assay. Oxidative damage potential was evaluated by determining reactive oxygen species (ROS) production by flowcytometer, and glutathione (GSH) levels by ELISA kit. Treatment with higher concentrations of AKB48 induced ROS generation (≥1.2-fold); however GSH levels did not changed. Additionally, the regulations in cannabinoid receptors and inflammation-related genes were determined on a qPCR platform. CB1 expression was increased approximately 15-fold at lower concentrations; whereas CB2 did not expressed. IL-6 and TNF-α were up-regulated with a dose-dependent manner, and the profiles were almost identical; however, MAPK8 and NF-κB were slightly regulated.

http://dx.doi.org/10.1016/j.toxlet.2017.07.660

P-07-07-04
Aryl hydrocarbon receptor is linked with novel food avoidance behaviour in Sprague-Dawley rats

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The aryl hydrocarbon receptor (AHR) mediates the toxicity of dioxins, but also plays important physiological roles. Previously, below-toxic doses of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin and shorter-acting AHR agonists, including β-naphthoflavone (BNF), were shown to induce strong avoidance of novel foods in rats. In contrast, 2,4,6-tryphenyldioxane-1,3, a phenobarbital-like inducer that activates constitutive androstane receptor instead of AHR, did not cause it. These results suggested dependence of the avoidance response specifically on AHR, a hypothesis tested here. We used littermate AHR-knockout, heterozygote and wild-type Sprague-Dawley rats. Young adult male rats were habituated to chocolate for ~20 h before exposure to a single dose of BNF (60 mg/kg ig) or the vehicle. Subsequently, chocolate consumption was monitored for another 24 h. The AHR-phenotype of each lineage was confirmed by quantifying hepatic *Cyp1a1* mRNA, a sensitive marker for AHR activation. *Cyp1a1* was not found to be expressed in AHR-knockouts or induced by BNF, contrary to the other two lineages. As hypothesised, BNF failed to influence chocolate intake in the knockouts; both groups consumed on average 6.3–6.6 g by 24 h ($p = 0.875$). In contrast, in both the heterozygote and wildtype lineages, BNF-treated rats exhibited strong chocolate avoidance, while the controls did not (respective 24-h consumptions: 0.35 vs 5.1 g, $p = 0.006$; and 1.3 vs 8.3 g, $p = 0.011$). These findings provide formal confirmation that AHR signalling is a prerequisite for BNF-induced novel food avoidance behaviour.

http://dx.doi.org/10.1016/j.toxlet.2017.07.661

P-07-07-05
***CYP1B1*2* and *CYP1B1*3* polymorphisms and clinical outcome in non-small cell lung cancer patients**

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The association between *CYP1B1*2* and *CYP1B1*3* polymorphisms and response to chemotherapy and survival of patients with non-small cell lung cancer (NSCLC) are limited and inconclusive. In this study, *CYP1B1*2* and *CYP1B1*3* polymorphisms and response to platinum-based chemotherapy and survival in 137 advanced stage NSCLC patients were investigated. Genetic polymorphism analyses were performed by conventional PCR and real time-PCR. The polymorphisms of *CYP1B1*2* and *CYP1B1*3* did not significantly influence the responses to chemotherapy and survival in NCSLC patients ($p > 0.05$). We also analysed these gene variants in combination with *CYP1A1*2C*, *CYP1B1*4*, *CYP2E1*5B*, *CYP2E1*6*, *CYP2E1*7B*, *GSTM1*, *GSTT1*, *GSTP1* exon 5, *GSTP1* exon 6, *GSTO1* (A140D), and *TP53* (Arg72Pro) polymorphic genes that we have previously genotyped in the same patients (Ada et al., *Neoplasma*, 57, 512–527, 2010; Karacaoglan et al., *Turk J Med Sci*, 47, 554–562, 2017). The multivariate analysis revealed that adjusted hazard ratio (HR) of death of the combined variant genotypes of *CYP1B1*2* and *CYP1A1*2C*, and *CYP1B1*2* and *CYP1B1*4* increased significantly as compared to wild-type genotypes (HR, 5.01; 95% CI, 1.52–16.19, $p = 0.008$, HR, 3.63; 95% CI, 1.12–11.78, $p = 0.032$, respectively). These results show that combined variant genotypes of *CYP1B1*2* and *CYP1A1*2C*, and *CYP1B1*2* and *CYP1B1*4* are associated with worsening of survival in NSCLC patients. (Supported by the grants from Research Funds of Ankara University, nos: 15L0237003 and 10A3336002.)

http://dx.doi.org/10.1016/j.toxlet.2017.07.662

P-07-07-06
Dysregulated lncRNA-UCA1 contributes to the progression of gastric cancer through regulation of the PI3K-Akt-mTOR signaling pathway

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The long non-coding RNA (lncRNA) urothelial carcinoma-associated 1 (UCA1) has been recently shown to be dysregulated during disease occurrence and to play an important role in the progression of several cancers. However, the biological role and potential regulation mechanism of UCA1 in the carcinogenesis of gastric cancer remain unclear. In the present study, we found that UCA1 was aberrantly upregulated in gastric cancer tissues and gastric cancer cell lines, and was associated with TNM stage and metastasis. UCA1 silencing significantly inhibited gastric cancer BGC-823 cell proliferation and increased its apoptosis. We also found that UCA1 played an important role in the migration and