**[16] Systemic screening of Dioxin binding genomic regions using Chip-on-Chip analysis.** Seung Hun Shin[1,3], Ho Sik Kim[2], Yong Bok Jung[1,3], Yeun-Jun Chung[1,3]. [1]*Integrated Research Center for Genome Polymorphism,* [2]*Department of Microbiology,* [3]*Biochemistry. The Catholic University of Korea, College of Medicine. 505 Banpo-dong, Socho-gu, Seoul 137-701, Korea.*

The structure of the chromatin at the promoter of a gene is widely thought to influence directly or indirectly on the expression of the gene. Transcription initiation complex, comprised by transcription factor and promoter, is the key of gene expression regulation. Disruption of this orchestrated process by genotoxic chemicals may contribute or initiate disease. Chromatin immunoprecipitation (ChIP) basedDNA chip analysis (ChIP-on-chip) is a robust approach to screen the transcription factorbinding sites. This concept can be applied for the screening of the DNA binding sites of genotoxic chemicals. We have examined the cellular responses against Tetrachlorodibenzo-p-dioxin (TCDD). TCDD is an environmental contaminant, known to be a human carcinogen and exerts toxic effects on the skin, reproductive, immune and endocrine systems. Toxic and biological effects of TCDDare mediated through the aryl hydrocarbon receptor (AhR). After entering into cytoplasm, TCDD is transfers into nucleus mediated by AhR. In the nucleus, TCDD-AhR complex bind with Dioxin responsive element (DRE) mediated by ARNT. The most well-known gene activated by this TCDD-AhR pathway is CYP1A1, a xenobiotic metabolizing enzyme. TP53 is thought to be a downstream target of TCDD-AhR pathway, but the pathway is unclear yet. TCDD mediated anti-apoptosis and transformation cannot be explained by just activation of CYP1A1. Although some other TCDD-AhR sensitive genes such as $P21^{CYP1/WAF1}$ and CYP1A2 have been suggested, there might be more genes unidentified yet. In this study, we treated the target cell (HapG2) with different amount of TCDD and examined the TCDD-AhR binding sites by using whole-genome ChIP-on-chip analysis. Here we present the initial data.