ORAL COMMUNICATIONS

EFFECT OF DI-N-BUTYL PHTHALATE ON ADIPOGENESIS

B. Sgangarella Valvano[1], S. Boccia[2], A. Mileo[3], C. Pivonello[4], G. Guerra[1], A. De Luca[2], M. De Falco[5|6]

[1]Dept. of Medicine and Health Sciences "Vincenzo Tiberio", University of Molise, Campobasso, Italy; [2]Dept. of Mental and Physical Health and Preventive Medicine, University of Campania "Luigi Vanvitelli", Naples, Italy; [3]Dept. of Life Sciences, Health and Health Professions, Link Campus University, Rome, Italy; [4]Dept. of Public Health, University of Naples Federico II, Naples, Italy; [5]Dept. of Biology, University of Naples Federico II, Naples, Italy; [6]National Institute of Biostructures and Biosystems (INBB), Rome, Italy

Phthalates are ubiquitous endocrine disrupting chemicals (EDCs) associated with metabolic disorders and adipose tissue dysfunction. Among them, Di-n-butyl phthalate (DBP) has been proposed as a potential obesogen, although its role in adipogenesis remains unclear. This study investigated the effects of DBP on adipocyte differentiation in 3T3-L1 cells. Cells were induced to differentiate using a standard adipogenic protocol including insulin, 3-isobutyl-1-methylxanthine (IBMX), and dexamethasone (DEXA). DBP was tested at concentrations ranging from 10^{-6} M to 10^{-9} M under four experimental conditions to evaluate its ability to trigger or interfere with glucocorticoid signaling. Specifically, DBP was administered either as a substitute for DEXA or in combination with it (DBP+DEXA), during the induction phase (days 0–2) or throughout the entire differentiation period (days 0–8). MTT assay showed no cytotoxic effects at any tested concentration. Adipogenesis was evaluated by Oil Red O staining and Western blot analysis of the adipogenic markers C/EBPα and PPARγ. Oil Red O staining revealed altered lipid accumulation pattern in all DBP-treated cells compared with differentiated control, suggesting that DBP modulates adipogenesis independently of dose. In the absence of DEXA, DBP upregulated the expression of both adipogenic markers, with the strongest effect observed at 10^{-7} M in both treatment windows. Instead, in both time treatments, the mixture DBP+DEXA led to a downregulation of these markers, particularly at lower concentrations. Overall, DBP exerts a dual effect: alone, it induced a DEXA-like effect, stimulating the adipogenesis while in combination with DEXA, it inhibits its activity, displaying an antagonistic behavior and suggesting a context-dependent modulation of glucocorticoid signaling.