# Plasmatic concentration of organochlorine lindane acts as metabolic disruptors in HepG2 liver cell line by inducing mitochondrial disorder.



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| Résumé en anglais | Lindane (LD) is a persistent environmental pollutant that has been the subject of several toxicological studies. However, concentrations used in most of the reported studies were relatively higher than those found in the blood of the contaminated area residents and effects of low concentrations remain poorly investigated. Moreover, effects on cell metabolism and mitochondrial function of exposure to LD have received little attention. This study was designed to explore the effects of low concentrations of LD on cellular metabolism and mitochondrial function, using the hepatocarcinoma cell line HepG2. Cells were exposed to LD for 24, 48 and 72 h and different parameters linked with mitochondrial regulation and energy metabolism were analyzed. Despite having any impact on cellular viability, exposure to LD at plasmatic concentrations led to an increase of maximal respiratory capacity, complex I activity, intracellular ATP and NO release but decreased uncoupled respiration to ATP synthesis and medium lactate levels. In addition, LD exposure resulted in the upregulation of mitochondrial biogenesis genes. We suggest that, at plasmatic concentrations, LD acts as a metabolic disruptor through impaired mitochondrial function and regulation with an impact on cellular energetic metabolism. In addition, we propose that a cellular assay based on the analysis of mitochondria function, such as described here for LD, may be applicable for larger studies on the effects of low concentrations of xenobiotics, because of the exquisite sensitivity of this organelle. |

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