# Article information:

Bisphenol A and Di(2-Ethylhexyl) Phthalate promote pulmonary carcinoma in female rats via estrogen receptor beta: In vivo and in silico analysis - ScienceDirect  
<https://www.sciencedirect.com/science/article/pii/S0147651322013367?via%3Dihub>

# Article summary:

1. Combined exposure to BPA and DEHP can promote pulmonary carcinoma in female rats.

2. BPA and DEHP can directly bind and activate ESR2 in vivo and in silico.

3. BPA is the main effector in the combined exposure to BPA and DEHP, with ESR2 being related to multiple lung cancer pathways in female lung cancer.

# Article rating:

May be slightly imbalanced: The article presents the information in a generally reliable way, but there are minor points of consideration that could be explored further or claims that are not fully backed by appropriate evidence. Some perspectives may also be omitted, and you are encouraged to use the research topics section to explore the topic further.

# Article analysis:

The article “Bisphenol A and Di(2-Ethylhexyl) Phthalate promote pulmonary carcinoma in female rats via estrogen receptor beta: In vivo and in silico analysis” is a well-researched piece of work that provides an insight into the potential risks of endocrine disrupting chemicals (EDCs) on female lung cancer. The authors have used a rat pulmonary tumor model, combining bioinformatics analysis based on The Comparative Toxicogenomics Database (CTD) and The Cancer Genome Atlas (TCGA) databases, to explore the cancer-promoting effect of two representative substances of EDCs namely Bisphenol A (BPA) and Di(2-Ethylhexyl) Phthalate (DEHP). The article presents evidence that suggests that BPA and DEHP can enhance the promotion of pulmonary tumor in female rats, either alone or in combination, by binding to ESR2 protein, phosphorylating CREB protein, activating HDAC6 transcriptionally, inducing c-MYC production, etc.

The article is reliable as it provides evidence from both experimental studies as well as from databases such as CTD and TCGA which are trusted sources for scientific research. Furthermore, the authors have provided detailed explanations for their findings which makes it easier for readers to understand their conclusions. Additionally, they have also discussed potential counterarguments which adds credibility to their claims.

However, there are some points that could be improved upon such as providing more information about other EDCs that may affect hormone levels or exploring other pathways through which EDCs may affect lung cancer development. Additionally, while the authors have discussed potential counterarguments they could provide more detail about them so readers can better understand them. Finally, while the authors have discussed possible risks associated with EDCs exposure they could provide more information about how these risks can be minimized or avoided altogether.

# Topics for further research:

* Endocrine disrupting chemicals and lung cancer
* Estrogen receptor beta and cancer promotion
* Phosphorylation of CREB protein and cancer
* HDAC6 transcriptional regulation and cancer
* c-MYC production and cancer
* Minimizing risks of EDCs exposure

# Report location:

<https://www.fullpicture.app/item/05c626bbcda7d4b47e9ddb68d717c82c>