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Data Article

Data on environmentally relevant level of aflatoxin B₁-induced human dendritic cells' functional alteration

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ABSTRACT

We assessed the effects of naturally occurring levels of AFB₁ on the expression of key immune molecules and function of human monocyte-derived dendritic cells (MDDCs) by cell culture, RT-qPCR, and flow cytometry. Data here revealed that an environmentally relevant level of AFB₁ led to remarkably weakened key functional capacity of DCs, up-regulation of key transcripts and DCs apoptosis, down-regulation of key phagocytic element, CD64, and creation of pseudolicensing direction of DCs. Flow cytometry data confirmed a damage towards DCs, i.e., increased apoptosis. The detailed data and their mechanistic effects and the outcome are available in this research article (Mehrzad et al., 2018) [1]. The

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impaired phagocytosis capacity with triggered pseudolicensing direction of MDDCs caused by AFB₁ and dysregulation of the key functional genes could provide a mechanistic explanation for the observed *in vivo* immunotoxicity associated with this mycotoxin.

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Specifications table

Subject area	<i>Immunobiology, molecular biology and immunotoxicology</i>
More specific subject area	<i>Environmental-related pro-inflammation and immunotoxicity that potentially lead to immunodysregulation through dendritic cells (DCs)</i>
Type of data	<i>Very concise reader friendly graphs, figures, tables, text and file with 25 various immune genes and their designed sequences</i>
How data was acquired	<i>Cell culture, flow cytometry and RT-qPCR</i>
Data format	<i>Analyzed data with interpretive idea</i>
Experimental factors	<i>Naturally occurring levels of AFB₁ on mass generated DCs from pure monocytes of healthy individuals, the expression of key immune molecules and function of human MDDCs by cell culture, RT-qPCR, and flow cytometry.</i>
Experimental features	<i>in vitro experimental cell culture data and assays as detailed in the original article</i>
Data source location	<i>Laboratories of Immunology and Biotechnology, Department of Microbiology and Immunology and Department of Pathobiology, Faculty of Veterinary Medicine, and Institute of Biotechnology, University of Tehran and Ferdowsi University of Mashhad, Iran</i>
Data accessibility	<i>Data provided in this article is usefully accessible to the public</i>

Value of the data

- The novel experimental model is key for further *in vitro* tests in the area of Immunology, immunobiology, molecular diagnosis and immunotoxicology.
- Provision of primary pure MDDCs and their 25 more key genes' sequences can be a road for finding the etiology of various infectious/non-infectious diseases in human.
- The immunotoxic aspects of environmental mycotoxins especially AFB₁ and the key functional genes of MDDCs were designed as a model system for others to do further novel experiments in the area of mycotoxins-related infectious/non-infectious diseases, especially cancer.

1. Data

The analyses of the data on the expression of gene families in MDDCs indicated that the transcript levels of: 1) some key functional gene families, 2) some key TLR-related genes, 3) genes involved in the function of MDDCs and 4) some key cytokine transcripts were altered in post AFB₁-exposed MDDCs. Further, the flow cytometry-based phagocytosis and apoptosis assay revealed diminished phagocytic and survival capacity of MDDCs. The main finding of data on immune molecules at mRNA levels is briefed in Table 1.

Our data briefly illustrate (Fig. 1) the impaired phagocytosis capacity with triggered pseudolicensing [2] of MDDCs caused by AFB₁ and dysregulation of the key functional genes could provide a mechanistic explanation for the observed *in vivo* immunotoxicity associated with this mycotoxin [3–

Table 1

An overview of the effects of AFB₁ on the expression of various gene families and functional alterations in dendritic cells (DCs). The values (changes) are arbitrarily addressed according to the data in this article [1]. – = unchanged, ↑ = increased, ↑↑ = strongly increased, ↓ = decreased, ↓↓ = strongly decreased.

Genes	Arbitrary changes
Aflatoxin B₁ metabolism genes	
AhR	↑
AKR7A2	–
CYP1A2	↓
CYP1B1	↑
CYP3A4	↓
GSTM1	–
TLRs-related genes	
TLR2	↑↑
TLR4	↑↑
MyD88	↑↑
COX2	↑
NF-κb	–
DC functional genes	
CD64	↓↓
LFA3	↑
HLA-DR	↑↑
CD209	↑
CD11c	↓↓
CD16	↑
C5aR	–
CCR7	↑
DC cytokines genes	
TNF-α	↑↑
IL-6	–
IL-8	–
IL-1β	–
TGF-β	↓
IL-10	–
DC phagocytic capacity	↓↓
DC apoptosis	↑

7]. Here, we assessed > 25 key molecules of AFB₁-exposed MDDCs at mRNA level with some DCs' key functional assays. Therefore, more in-depth molecular study at protein levels is needed.

2. Experimental design, materials and methods

Very briefly, experimental work plan were done on pure immature MDDCs with long membrane protrusions; accordingly, the MDDCs were treated with 0 or 10 ng of AFB₁/ml for 2 and 12 h (37 °C, 5% CO₂, 95% humidity) [3,6] and then used for cellular and molecular analyses [3–8]. Key functional gene families, TLR-related genes, genes involved in the function of MDDCs and some key cytokine transcripts were analyzed with qPCR assays [4–6,8]. Further the flow cytometry assays were used to quantify phagocytosis and survival capacity of MDDCs [3,6]. All data were as the mean ± SEM of 8 experiments.

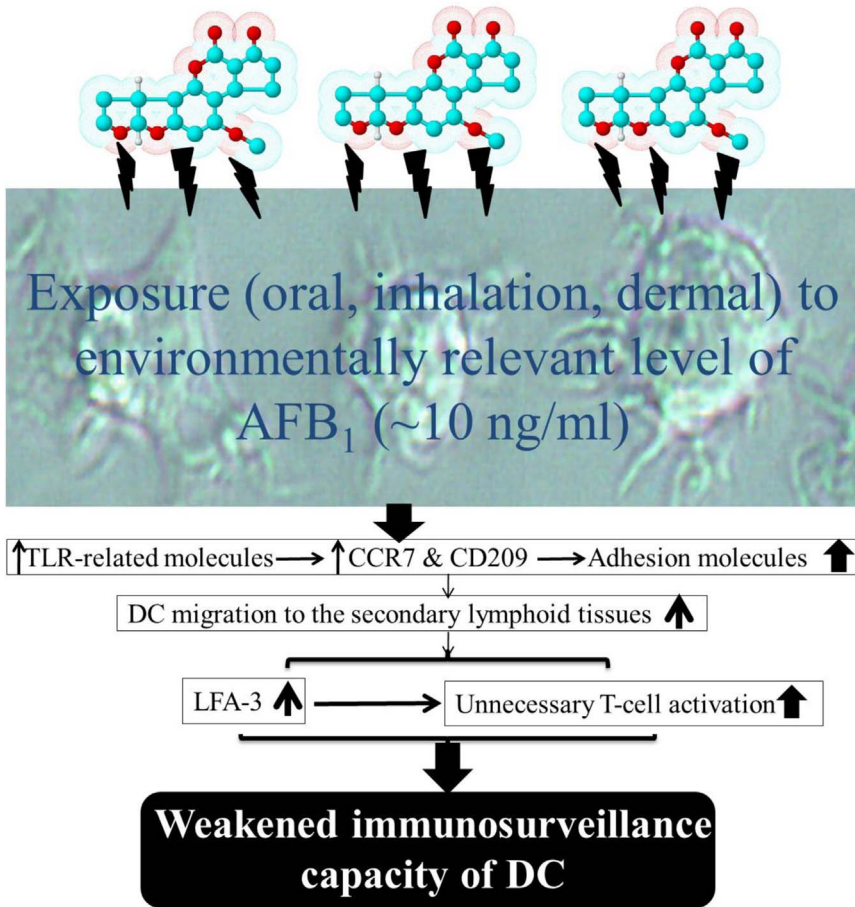


Fig. 1. Schematic representation from the conceptual interpretation of data of this paper. The scheme depicts how the proposed issue of pseudolicensing of AFB₁-exposed monocyte-derived dendritic cells (MDDCs) creates/leads to immunodisregulation *in vivo*. Though mRNA expression of CCR7, CD209 and LFA increase/change, nonetheless the protein levels of those molecules should be evaluated for future work.

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Transparency document. Supporting information

Supplementary data associated with this article can be found in the online version at <http://dx.doi.org/10.1016/j.dib.2018.04.104>.

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