

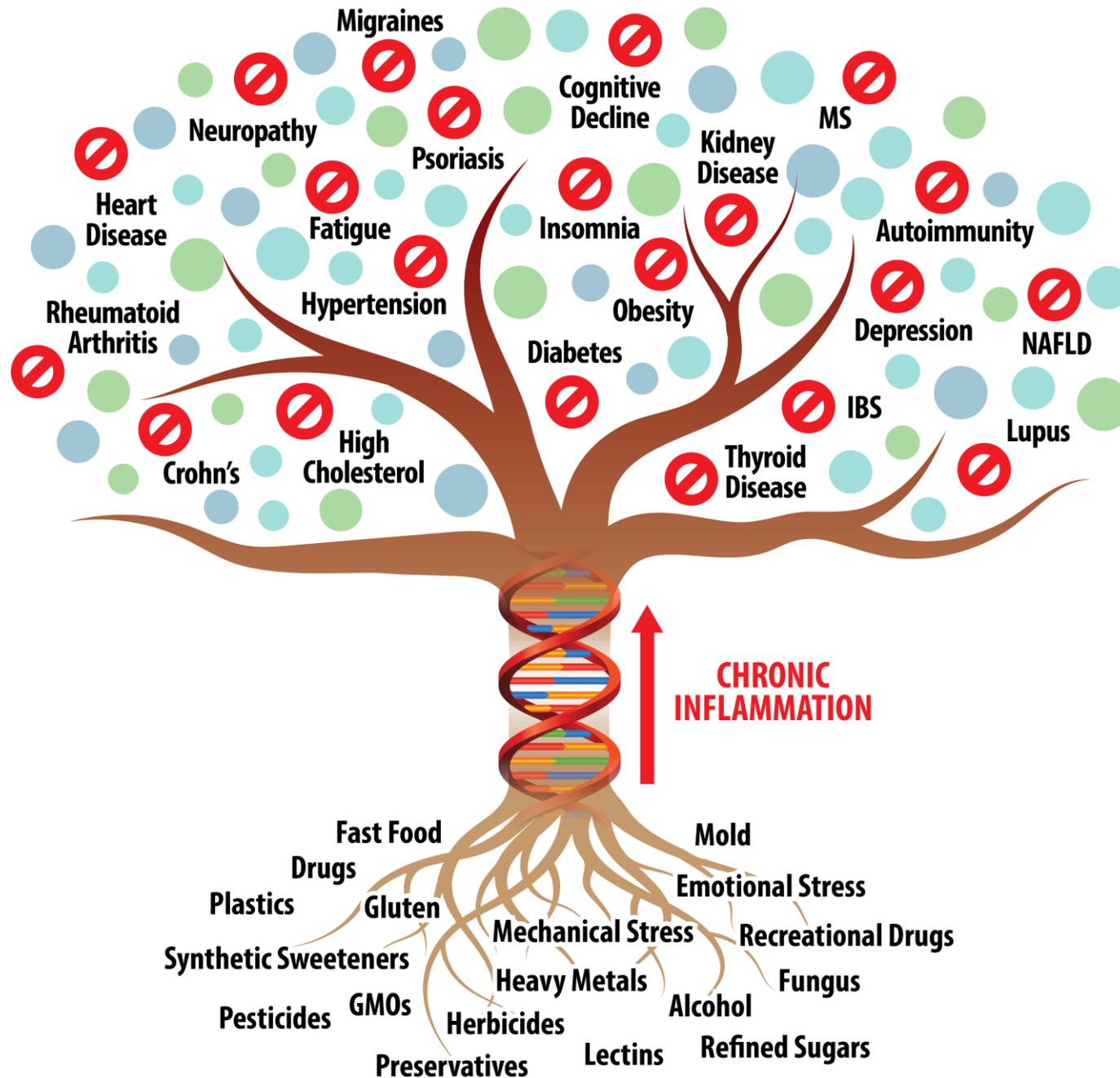
Casual Friday Presents

Prostate Focused FM

and Intervention Development

A BIOGENETIX CLINICAL PRESENTATION

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Three Most Common Prostate Conditions

1. Benign Prostatic Hyperplasia (BPH)

Enlarged prostate

Urinary frequency, weak stream, nocturia

Very common after 50

2. Prostatitis

Inflammatory (often non-bacterial)

Pelvic pain, urinary symptoms

Often linked to inflammation, stress, gut dysfunction

3. Prostate Cancer

Ranges from slow-growing to aggressive

Risk increases with age, metabolic dysfunction, and (obviously) inflammation.



A story of chronic inflammation and hormone imbalance...

Key hormones:

Testosterone

DHT (dihydrotestosterone)

Estradiol

SHBG

Insulin

*Insulin increases growth signaling in prostate tissue.

Key Inflammatory markers:

Non Specific:

CRP (C-reactive protein)

ESR (erythrocyte sedimentation rate)

Ferritin

Specific:

IL-1

IL-6

TNF-a

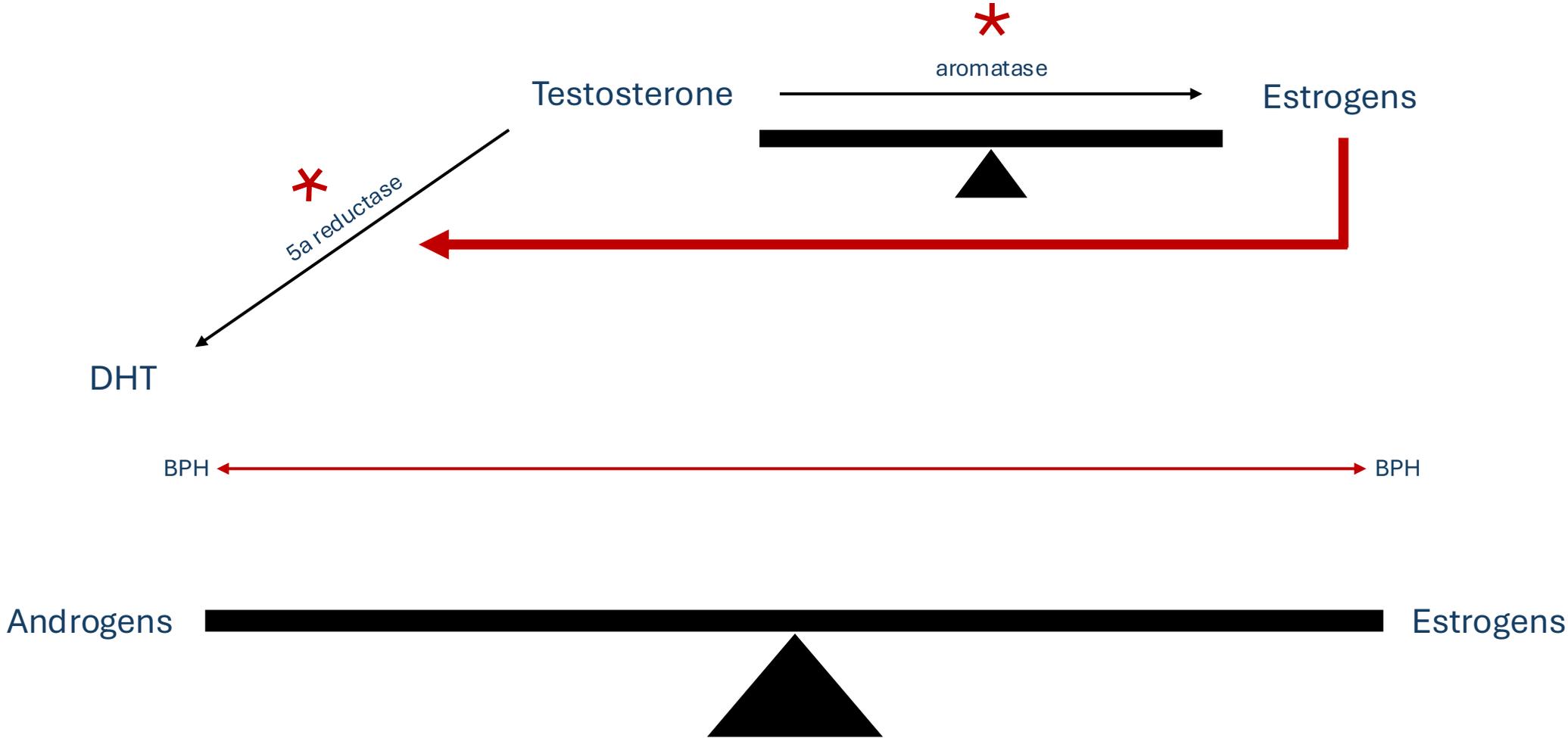
IL-8

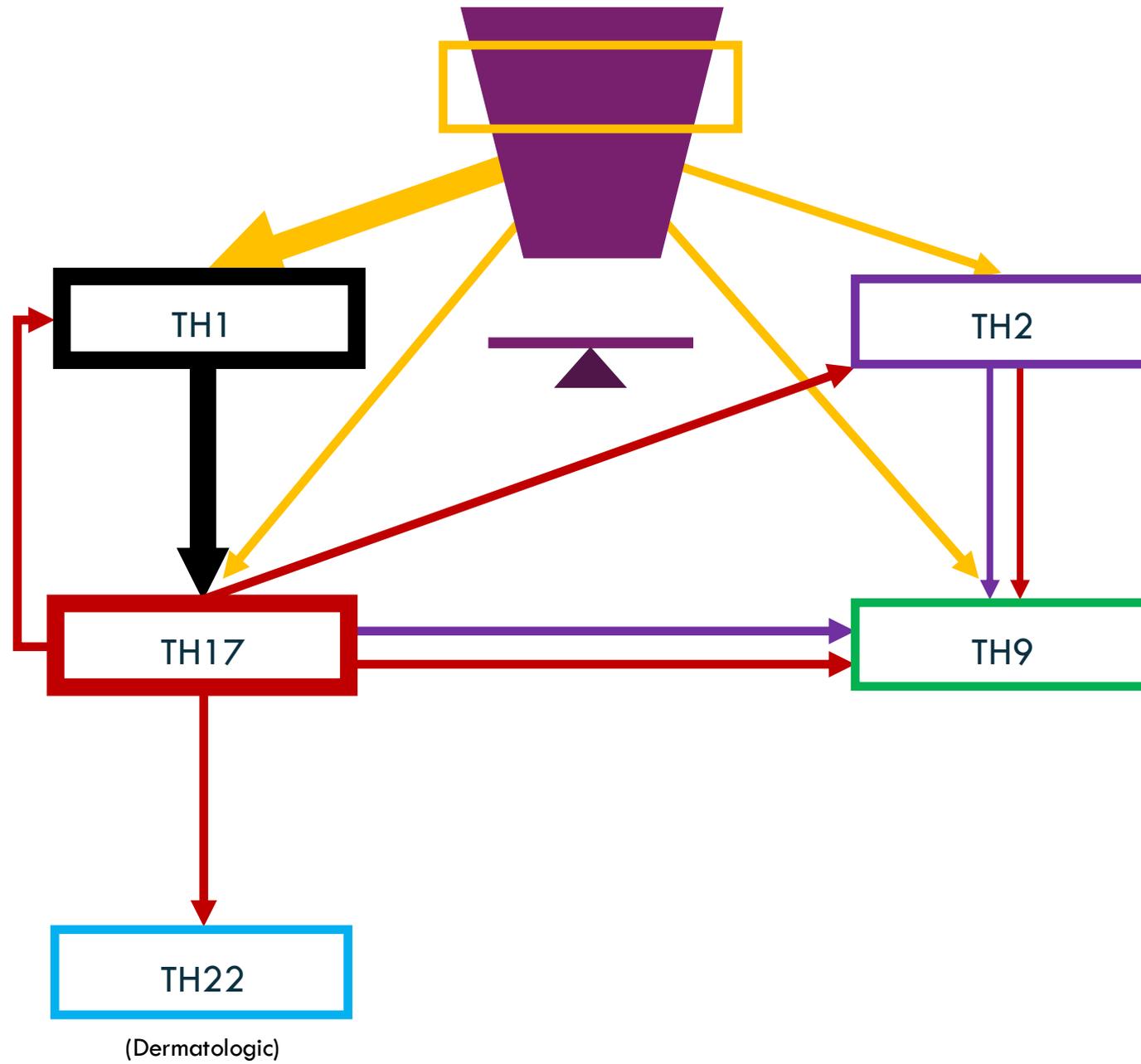
TGF-B1

VEGF



BPH: A hormone balancing act.





▶ Ther Adv Endocrinol Metab. 2021 Dec 8;12:20420188211066210. doi: [10.1177/20420188211066210](https://doi.org/10.1177/20420188211066210) [↗](#)

Metabolic syndrome is associated with prostate enlargement: a systematic review, meta-analysis, and meta-regression on patients with lower urinary tract symptom factors

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PMCID: PMC8664322 PMID: [34900218](https://pubmed.ncbi.nlm.nih.gov/34900218/)



Strong link between:

- Insulin resistance
- Abdominal obesity
- High triglycerides
- BPH & aggressive prostate cancer

▶ [BMC Urol. 2026 Feb 2;26:26. doi: 10.1186/s12894-025-02003-2](#) 

The gut-prostate axis in benign prostatic hyperplasia: systematic review of microbial dysbiosis and pathogenic mechanisms

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PMCID: PMC12866195 PMID: [41629891](#)



D. Gut-Prostate Axis

- Dysbiosis increases systemic inflammation.
- LPS (endotoxin) increases prostate inflammatory signaling.
- B/F/Lactobacillus imbalance.
- Constipation worsens pelvic congestion.

The microbiome influences hormone metabolism.



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Review > Urol Oncol. 2012 Mar-Apr;30(2):216-9. doi: 10.1016/j.urolonc.2011.11.014.

Environmental exposures and prostate cancer

Jeffrey K Mullins¹, Stacy Loeb

Affiliations + expand

PMID: 22385992 DOI: 10.1016/j.urolonc.2011.11.014



E. Environmental Exposures
Endocrine disruptors (BPA, phthalates)
Glyphosate
Farming chemicals (phosphates)

***What about mycotoxin?**



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Key Inflammatory markers:

Non Specific:

CRP (C-reactive protein)

ESR (erythrocyte sedimentation rate)

Ferritin

Specific:

IL-1

IL-6

TNF- α

IL-8

TGF-B1

VEGF

What about Mycotoxin?

Deoxynivalenol-induced proinflammatory gene expression: mechanisms and pathological sequelae.

Pestka JJ¹ 

Author information

Toxins, 01 Jun 2010, 2(6):1300-1317

<https://doi.org/10.3390/toxins2061300> PMID: 22069639 PMCID: PMC3153246

Review



Free full text in Europe PMC

The trichothecene mycotoxin deoxynivalenol (DON) is commonly encountered in human cereal foods throughout the world as a result of infestation of grains in the field and in storage by the fungus *Fusarium*. Significant questions remain regarding the risks posed to humans from acute and chronic DON ingestion, and how to manage these risks without imperiling access to nutritionally important food commodities. Modulation of the innate immune system appears particularly critical to DON's toxic effects. Specifically, DON induces activation of mitogen-activated protein kinases (MAPKs) in macrophages and monocytes, which mediate robust induction of proinflammatory gene expression-effects that can be recapitulated in intact animals. The initiating mechanisms for DON-induced ribotoxic stress response appear to involve the (1) activation of constitutive protein kinases on the damaged ribosome and (2) autophagy of the chaperone GRP78 with consequent activation of the ER stress response. Pathological sequelae resulting from chronic low dose exposure include anorexia, impaired weight gain, growth hormone dysregulation and aberrant IgA production whereas acute high dose exposure evokes gastroenteritis, emesis and a shock-like syndrome. Taken together, the capacity of DON to evoke ribotoxic stress in mononuclear phagocytes contributes significantly to its acute and chronic toxic effects in vivo. It is anticipated that these investigations will enable the identification of robust biomarkers of effect that will be applicable to epidemiological studies of the human health effects of this common mycotoxin.



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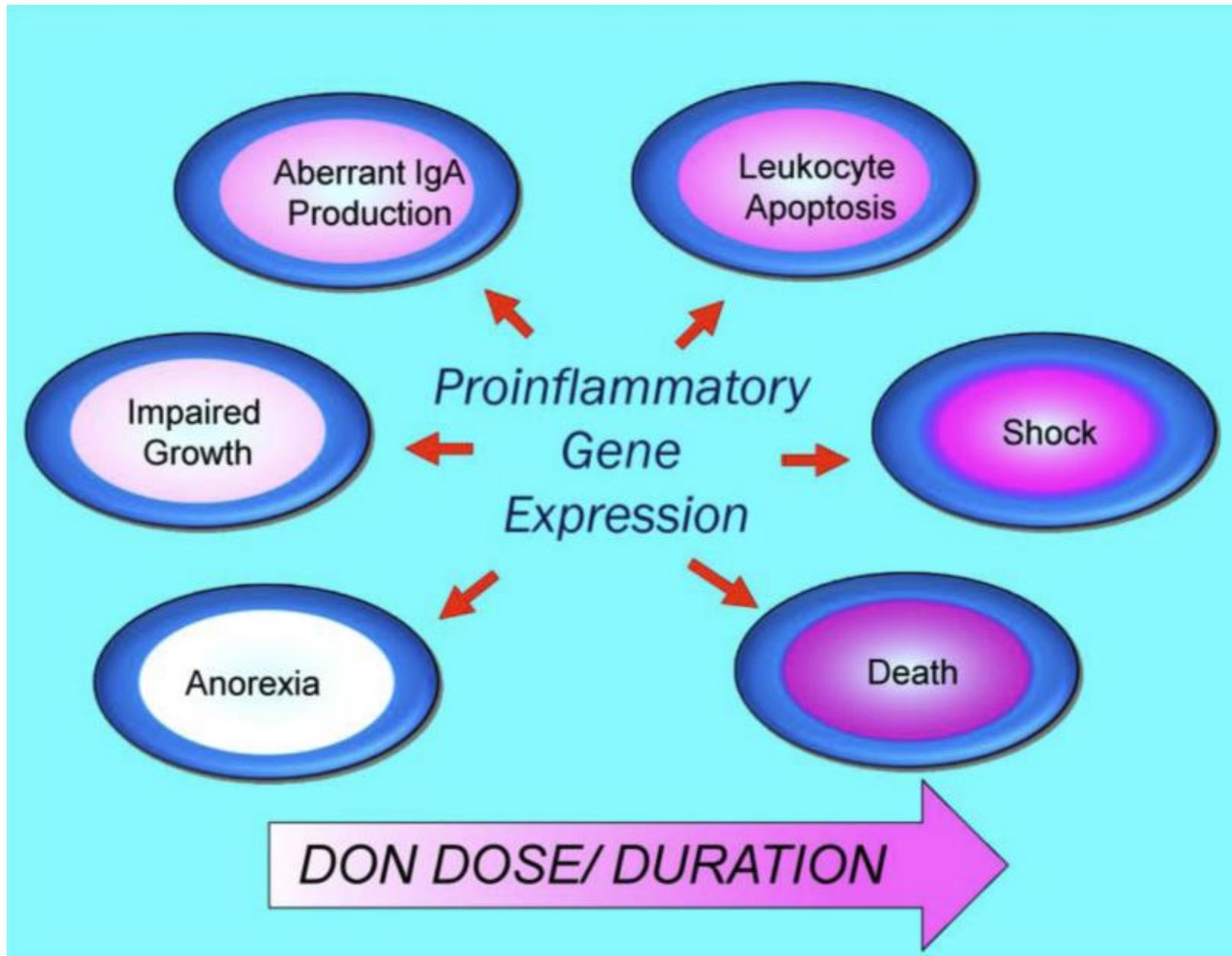


Table 1

Genes upregulated by deoxynivalenol in mice.

Gene Family	Gene
Proinflammatory Cytokines	<u>IL-1α, IL-1β, IL-6, IL-11, TNF-α, TGF-β</u>
T Cell Cytokines	IFN- γ , IL-2
Chemokines	MIP-2, MCP-1, Crg-2, CINC-1, MCP-3
Transcription Factors	cFos, cJun, Fra-2, Jun-B, NR4a1
Phosphatases	MKP1, CNAb, Ptpn8, Ptpnj
Suppressors of Cytokine Signaling (SOCS)	CIS1,SOCS1, SOCS2 ,SOCS3
Other	Cox-2, C3aR

See references [26,34,48,83,100,120].

Deoxynivalenol-induced proinflammatory gene expression: mechanisms and pathological sequelae.

Pestka JJ ¹ 

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Toxins, 01 Jun 2010, 2(6):1300-1317

What is the *in vivo* relevance of the DON-induced proinflammatory gene expression? DON is rapidly distributed throughout the body following oral exposure of mice [83,84] and thus would be present in many tissues containing mononuclear phagocytes. Consistent with *in vitro* studies, DON concomitantly induces a wide array of proinflammatory cytokines and chemokines that are detectable in spleen, liver, kidney and lung [37,49,84,85,86,87,88,89]. Ribotoxic stress in mononuclear phagocytes, with consequent induction of proinflammatory gene expression, is likely to be critical for the induction of acute and chronic sequelae associated with DON poisoning in experimental animals (Figure 3).

Aberrant elevation of inflammatory mediators, often referred to as a cytokine storm [90], mediate the shock-like effects of lipopolysaccharide (LPS) [91] and might likewise contribute to acute toxic effects of DON. Indeed, LPS and other toll-like receptor (TLR) agonists potentiate DON toxicity in mice [62,89,92,93,94,95]. Interestingly, DON has been reported to damage the integrity of intestinal cells and allow increased bacterial translocation [96,97,98,99]. Such collateral damage could greatly magnify DON toxicity.

Immunotoxicity of Three Environmental Mycotoxins and Their Risks of Increasing Pathogen Infections.

Sun Y¹ , Song Y¹, Long M¹ , Yang S¹

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Toxins, 02 Mar 2023, 15(3):187

<https://doi.org/10.3390/toxins15030187> PMID: 36977078 PMCID: PMC10054902

Aflatoxin B1 (AFB1), ochratoxin A (OTA), and deoxynivalenol (DON) are the three mycotoxins that have received the most scholarly attention and have been tested most routinely in clinics. These mycotoxins not only suppress immune responses but also induce inflammation and even increase susceptibility to pathogens. Here, we comprehensively reviewed the determining factors for the bidirectional immunotoxicity of the three mycotoxins, their effects on pathogens, and their action mechanisms. The determining factors include mycotoxin exposure doses and times, as well as species, sex, and some immunologic stimulants. Moreover, mycotoxin exposure can affect the infection severity of some pathogens, including bacteria, viruses, and parasites. Their specific action mechanisms include three aspects: (1) mycotoxin exposure directly promotes the proliferation of pathogenic microorganisms; (2) mycotoxins produce toxicity, destroy the integrity of the mucosal barrier, and promote inflammatory response, thereby improving the susceptibility of the host; (3) mycotoxins reduce the activity of some specific immune cells and induce immune suppression, resulting in reduced host resistance. The present review will provide a scientific basis for the control of these three mycotoxins and also provide a reference for research on the causes of increased subclinical infections.



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Toxins 02 Mar 2023, 15(3):187

2.1. AFB1

Aflatoxin, a low molecular secondary metabolite, is produced by *A. flavus* and *A. parasitica*. There are eighteen different varieties of aflatoxins, such as AFB1, AFB2, AFG1, AFG2, AFM1, and AFM2, of which AFB1 is the most important aflatoxin due to its toxicity and carcinogenic effects. AFB1 is widely distributed in moldy agricultural products, especially peanut meal and soybean meal (raw materials of animal feed), which seriously harm the health of livestock and poultry [13]. Many previous studies reported that AFB1 exposure can cause DNA damage, oxidative stress, and apoptosis and induce severe hepatotoxicity and nephrotoxicity [14,15,16].

2.2. OTA

After aflatoxin was discovered, ochratoxin ensured that people recognized mycotoxins again. Isocoumarin produces more than 20 chemicals, including ochratoxin, by cross-linking L-phenylalanine. OTA has attracted the attention of researchers due to its wide dissemination (moldy food and feed), serious toxicity, and significant impact on animals and agricultural products [13,17]. OTA is naturally produced by fungi such as *A. ocher*, *anthrax A.*, *A. niger*, and *Penicillium verrucosa*. OTA is toxic to livestock, and its primary target organ is the kidney. OTA can cause immunotoxicity, hepatotoxicity, apoptosis, decreased cell viability, and even affect oocyte maturation and embryonic development.



Table 1

Determining factors of the bidirectional immunotoxicity of the three mycotoxins.

Determining Factors	AFB1	OTA	DON
Exposure dose	(Low-dose exposure) inflammation [19]	(Low-dose exposure) inflammation [20]	(Low-dose exposure) inflammation [5]
	(High-dose exposure) immunosuppression [21]	(High-dose exposure) immunosuppression [22]	(High-dose exposure) immunosuppression [5]
Exposure time	(Short-term exposure) inflammation [8]	(Short-term exposure) inflammation [6]	-
	(Long-term exposure) immunosuppression [8]	(Long-term exposure) immunosuppression [6]	
Species	Pigs > ducklings > rats > sheep [23]	Pigs > rats > mice [24]	Pigs > mice > poultry > ruminants [25]
Sex	-	-	Female > male [26]
Immunologic stimulants	(With PHA) Immunosuppression [7,27]	-	(With LPS) Immunosuppression [28]
	(Without PHA) Inflammation [7]		(Without LPS) Inflammation [7]

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AFB1: Aflatoxin B1; OTA: ochratoxin A; DON: deoxynivalenol; Immunologic stimulants, including lipopolysaccharide (LPS), phytohemagglutinin (PHA), concanavalin A (CoA), and pokeweed mitogen, are determining factor affecting the bidirectional immunotoxicity of mycotoxins.

Immunotoxicity of Three Environmental Mycotoxins and Their Risks of Increasing Pathogen Infections.

Sun Y ¹ , Song Y ¹, Long M ¹ , Yang S ¹

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In conclusion, the mycotoxins AFB1, OTA, and DON have bidirectional immunotoxicity: low-dose or short-term mycotoxin exposure induces inflammation, while high-dose or long-term mycotoxin exposure results in immunosuppression; when immunologic stimulants are present, the three mycotoxins mainly manifest anti-inflammatory effects. The bidirectional immunotoxicity mechanism of mycotoxins is involved in the oxidative stress, apoptosis, and autophagy of some immune cells and some immunity-related signals. Moreover, exposure to these mycotoxins can affect the infection severity of bacteria, viruses, and parasites. Their action mechanisms include three aspects: (1) mycotoxin exposure directly promotes the proliferation of pathogenic microorganisms; (2) mycotoxins produce toxicity, destroy the integrity of the mucosal barrier, and promote inflammatory response, thereby increasing the susceptibility of the host; (3) mycotoxins reduce the activity of some specific immune cells and induce immunosuppression, resulting in reduced host resistance.



Sample FM Workup

1. Biogenetix General Screen
 - + insulin
 - + c-peptide
 - + Testosterone
 - + DHT (dihydrotestosterone)
 - + Estradiol
 - + SHBG
2. Stool test with Zonulin family peptide.
3. Total tox burden from Vibrant Wellness Laboratory (or similar).
4. Environmental Exposure Assessment
 - + The DUST test.

