Casual Friday Series

# Functional Blood Chemistry Series
# Pt. 20: CBC Connections (I)

A Biogenetix Clinical Presentation

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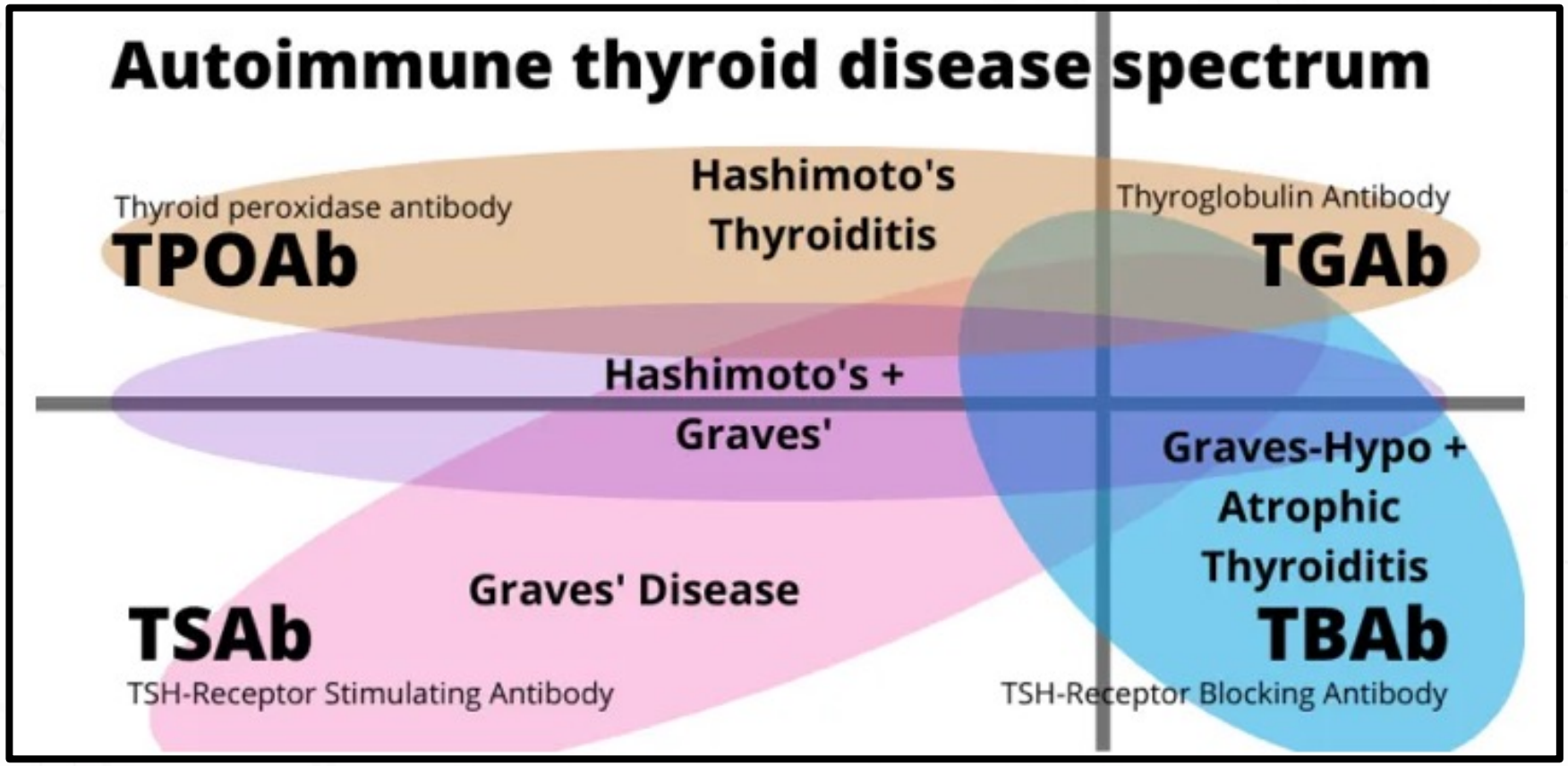
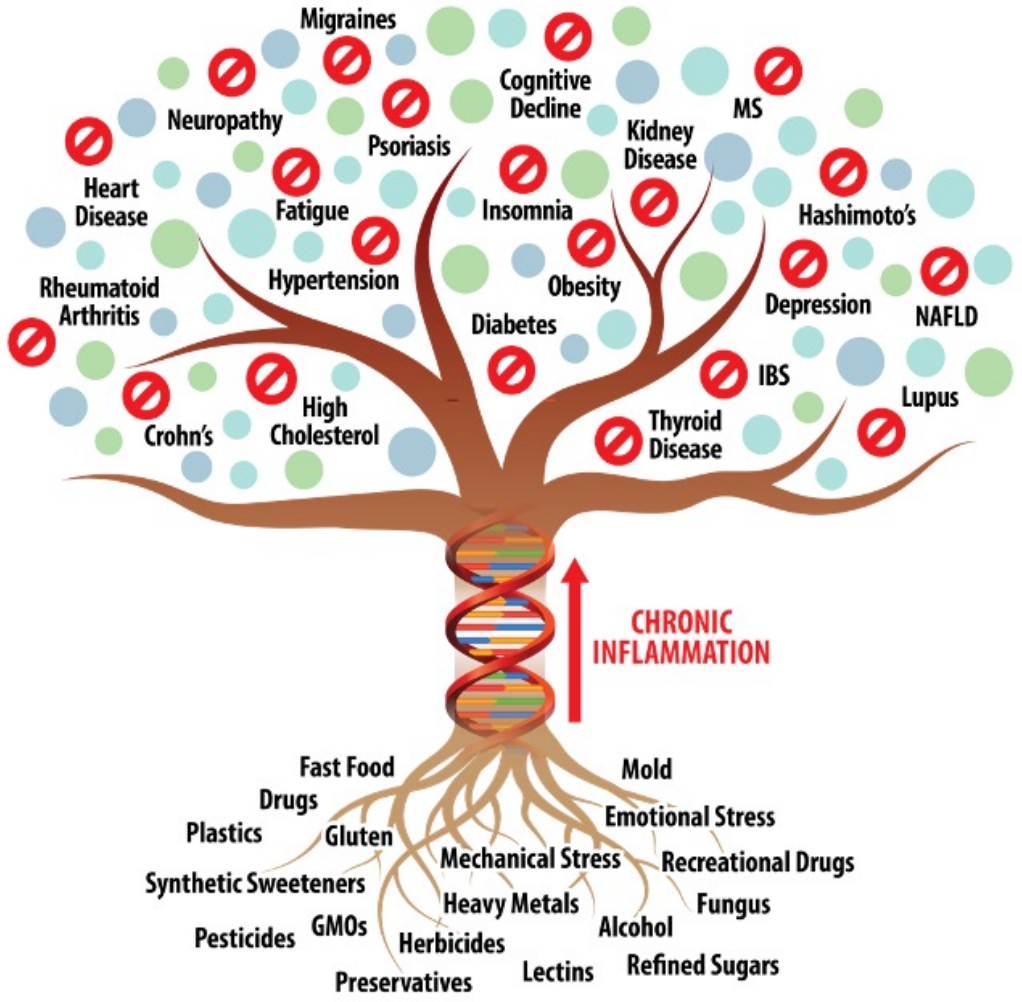
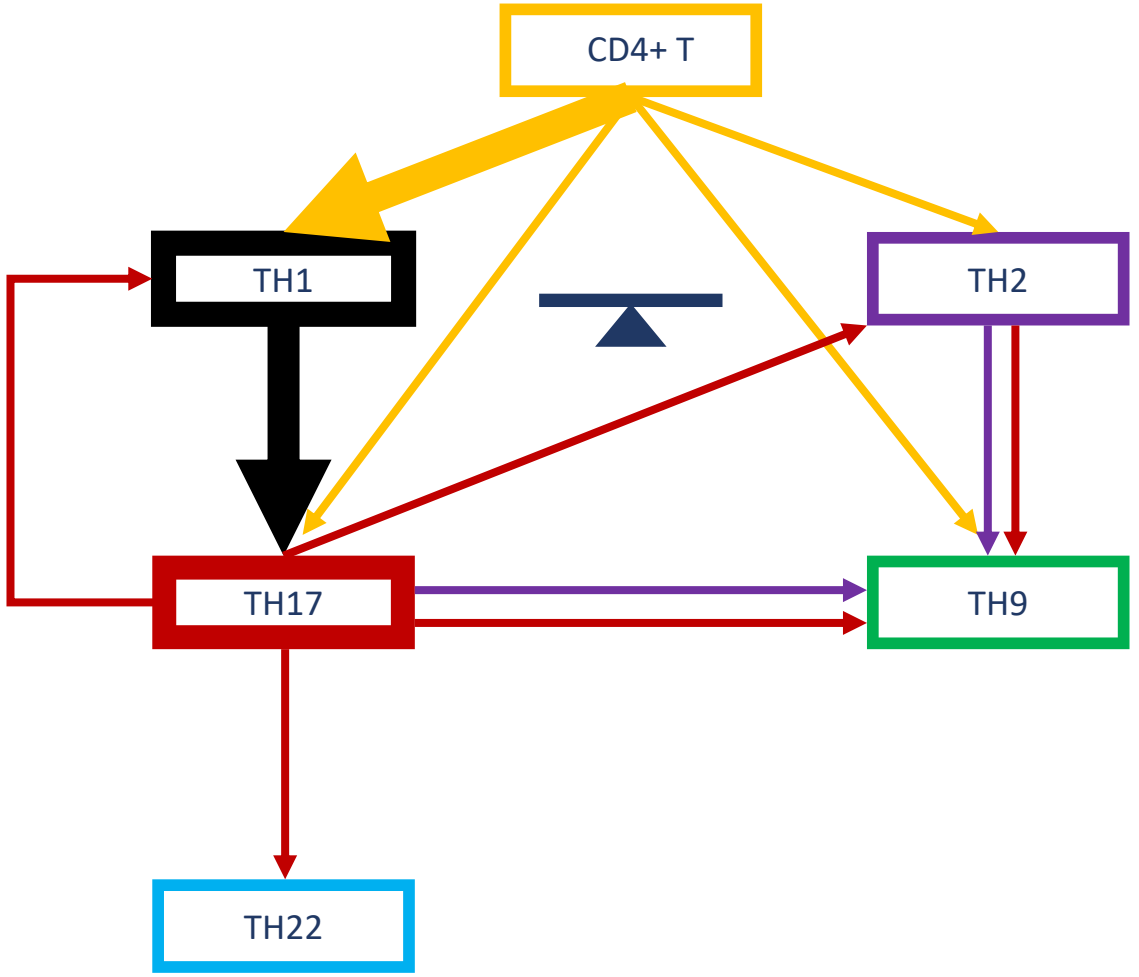
# Disclaimer

- *Information in this presentation is not intended to diagnose, treat, reverse, cure, or prevent any disease. While this presentation is based on medical literature, findings, and text, The following statements have not been evaluated by the FDA.*
- *The information provided in this presentation is for your consideration only as a practicing health care provider. Ultimately you are responsible for exercising professional judgment in the care of your own patients.*

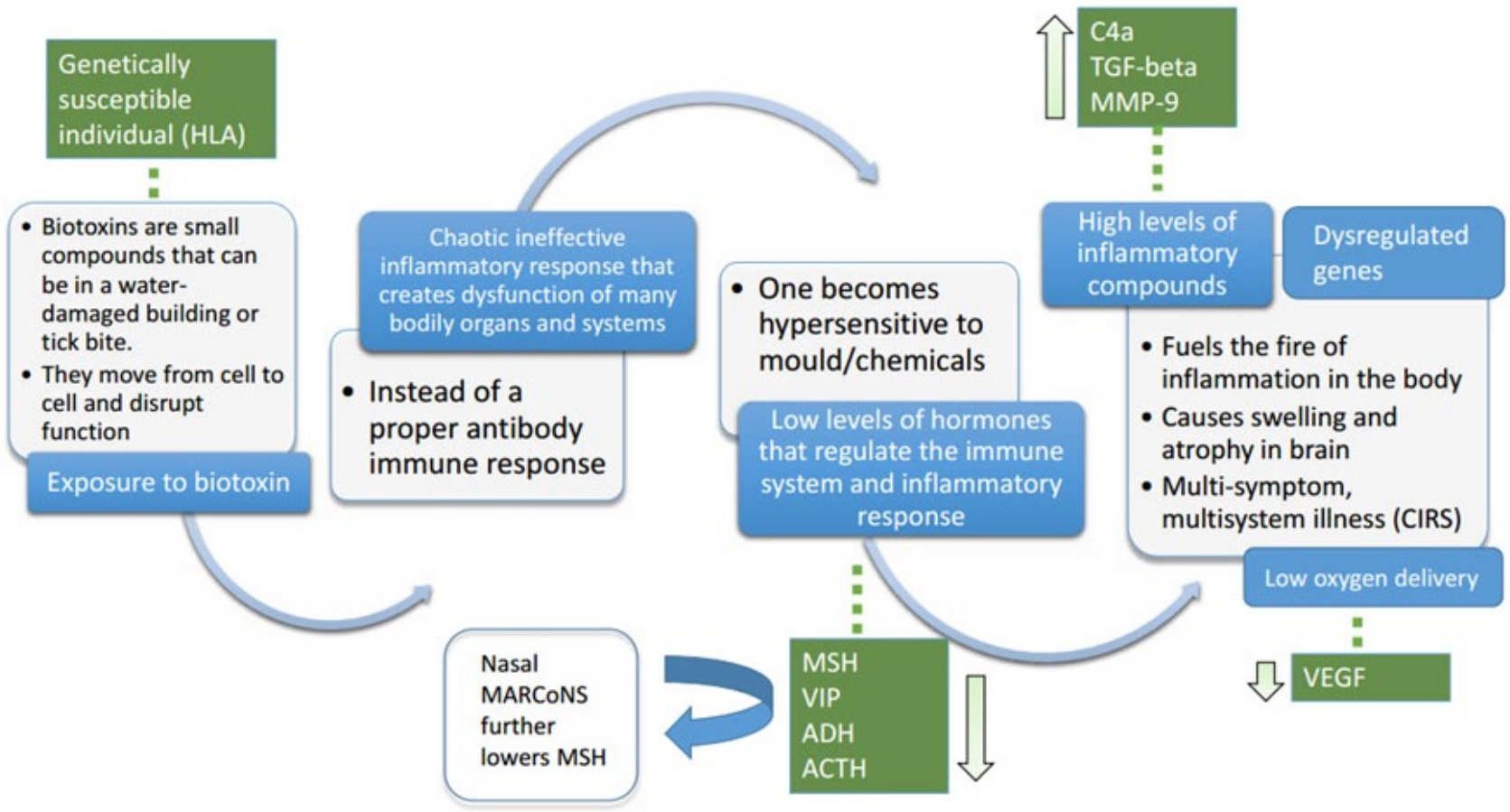
# Autoimmunity






(Dermatologic)




Adapted from The Biotoxin Pathway, © R.Shoemaker 2011

# Neutrophils

The most abundant and most motile of all white blood cells

Neutralizes an invader by phagocytosis

- Contains a number of enzymes and pyrogens that can also cause damage to host cells

Optimal range: 50-60%

# Lymphocytes

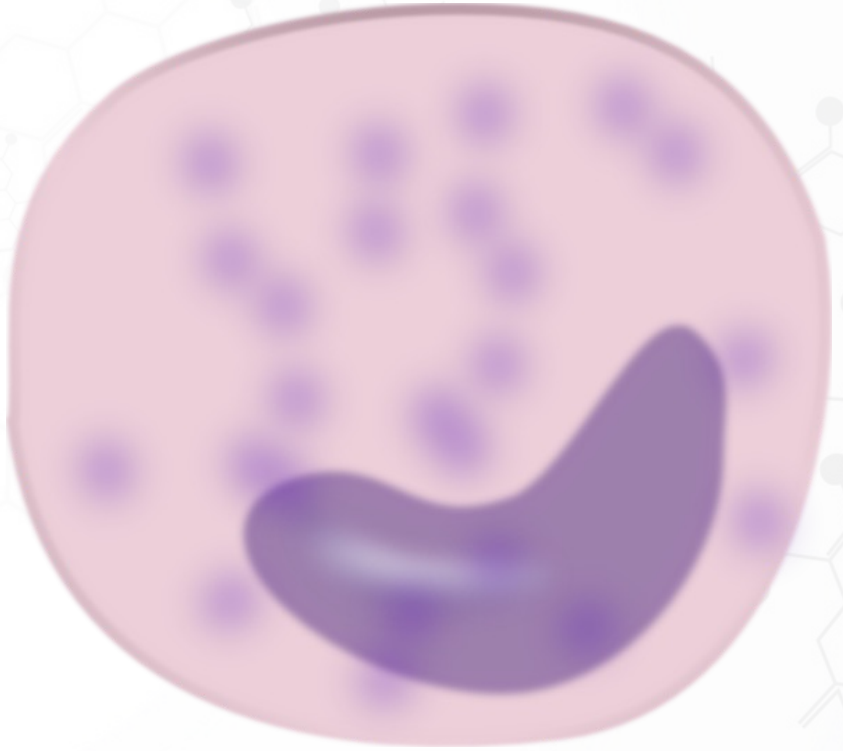
Lymphocytes contain a wide variety of white blood cells including B-cells, T-cells and Natural Killer Cells

The lymphocyte count does not differentiate between these – more specific testing is required to identify the lymphocyte subsets

Optimal range: 30-35%

# Monocytes

- Monocytes leave circulation within 16-36 hours and become macrophages
- Macrophages remove foreign substances via phagocytosis
  - They also participate in the destruction of old red blood cells, denatured proteins, microorganisms, and dead cells
- Macrophages can become Antigen Presenting Cells (APCs) and deliver an antigen to lymphocytes
- Optimal range: <6%

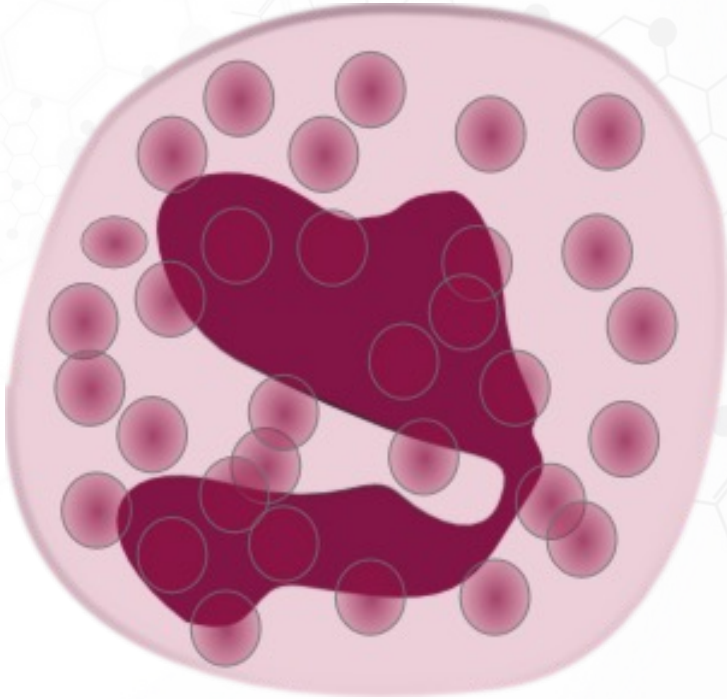


# Eosinophils

Eosinophils are also phagocytic immune cells and become active in the later stages of inflammation.

They are found in large numbers in the intestines and lungs.

Optimal range: <3%

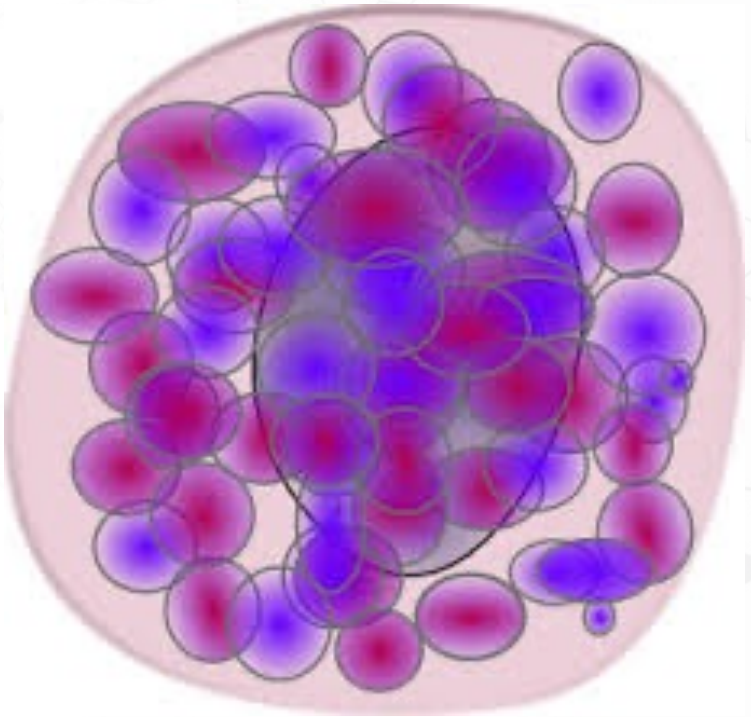


# Basophils

Basophils contain a large number of granules containing heparin, histamine, leukotriene, serotonin, etc.

They are associated with inflammation and hypersensitivities

Tissue basophils are called mast cells.



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# Usefulness of Complete Blood Count (CBC) to Assess Cardiovascular and Metabolic Diseases in Clinical Settings: A Comprehensive Literature Review

In-Ho Seo and Yong-Jae Lee*

Alper Sonmez, Academic Editor

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Complete blood count (CBC) is one of the most common blood tests requested by clinicians and evaluates the total numbers and characteristics of cell components in the blood. Recently, many investigations have suggested that the risk of cancer, cardiovascular disease (CVD), arteriosclerosis, type 2 diabetes (T2DM), and metabolic syndrome can be predicted using CBC components. This review introduces that white blood cell (WBC), neutrophil-to-lymphocyte ratio (NLR), hemoglobin (Hb), mean corpuscular volume (MCV), red cell distribution width (RDW), platelet count, mean platelet volume (MPV), and platelet-to-lymphocyte ratio (PLR) are useful markers to predict CVD and metabolic diseases. Furthermore, we would like to support various uses of CBC by organizing pathophysiology that can explain the relationship between CBC components and diseases.

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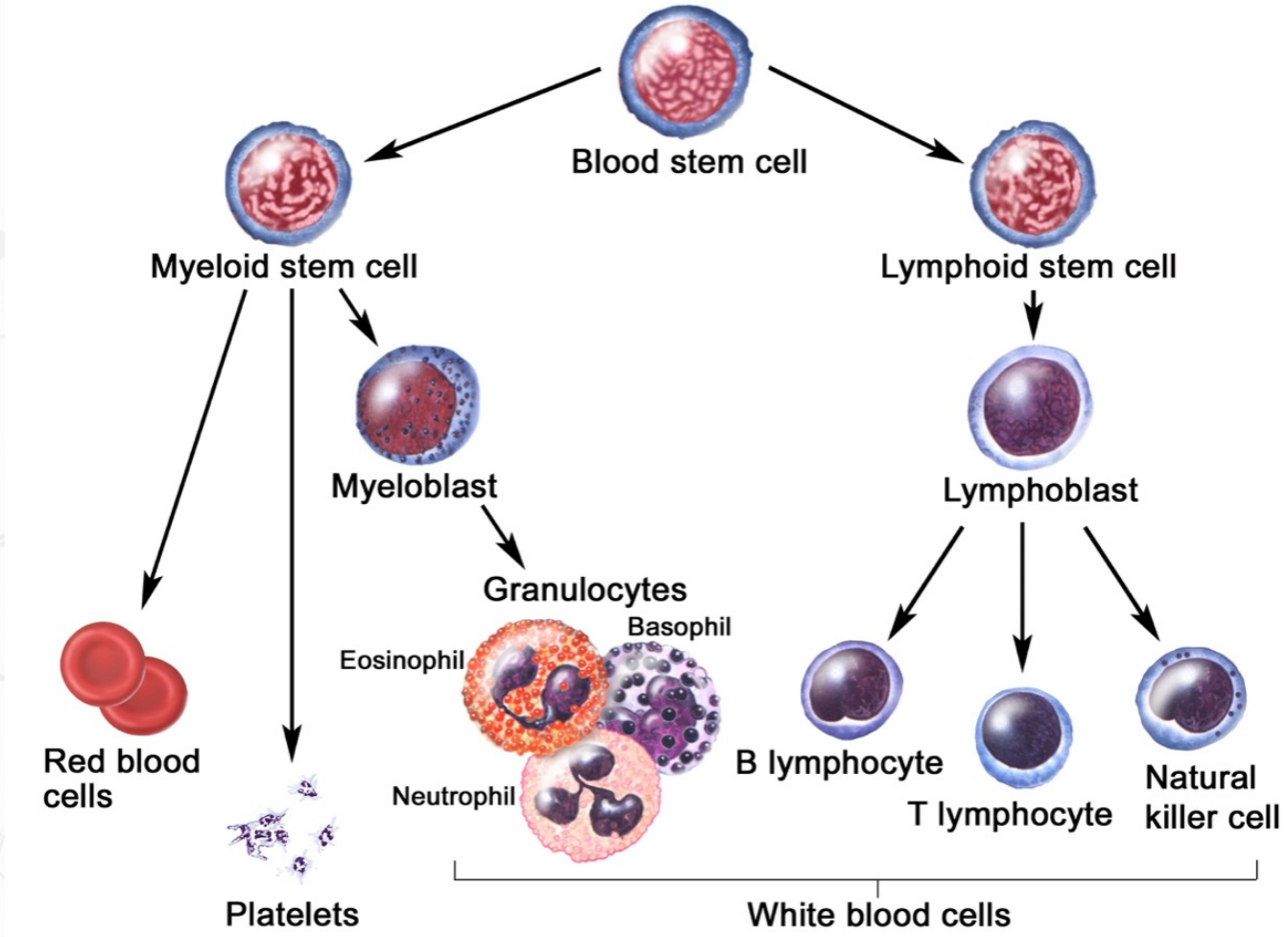
## 2.2. Neutrophil-to-Lymphocyte Ratio

The most frequent type of WBC is neutrophils, accounting for 50–70% of the total WBC in blood circulation [12]. Neutrophils are the primary immune cells that respond to infection and are controlled under homeostatic conditions [12]. Lymphocytes are a critical population of WBCs and play an essential role in both innate and adaptive immunity [12]. They constitute 20–50% of total WBCs and consist of T cells, B cells, natural killer T cells, and innate lymphoid cells [12]. The ratio obtained by dividing neutrophils by lymphocytes is called the neutrophil–lymphocyte ratio (NLR) and is a useful biomarker that indicates the balance between systemic inflammation and immunity [13]. NLR has been investigated as a predictive and prognostic marker in several diseases, such as various cancers and CVD [14,15]. One advantage of using NLR is that, even when the WBC count is in the normal range, if NLR is 2.5 or higher, clinicians can identify chronic low-grade systemic inflammation [16]. In addition, NLR is useful in clinical practice because it can be calculated easily from CBC using the differential count result.




T- Cell Subtypes:

- Regulatory T Cells (suppress others)
- Helper T Cells (activate others)
- Cytotoxic T Cells (kill viruses, etc.)

B- Cells:

- Antibody creation for T cell activation

NK-Cell:

- Attack harmful cells but don't require activation like T Cell family.
- Largely TH1 response

https://nci-media.cancer.gov/pdq/media/images/526538.jpg

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The relationship between CAD and NLR has been studied extensively. NLR was associated with calcium score and risk factors for CAD, was associated independently with arterial stiffness, and was suggested to be a high predictive marker of arterial stiffness [21,22]. Because endothelial dysfunction is caused by neutrophil–endotherm interactions, an NLR increase could increase the risk of CAD. Interestingly, improvement in arterial stiffness in ST-elevation myocardial infarction (STEMI) patients who successfully underwent percutaneous coronary intervention (PCI) was associated with a decrease in NLR [23]. In addition, NLR has been used to predict the occurrence of major adverse cardiovascular events when patients with CAD undergo noncardiac surgery [24]. These findings indicate NLR as a clinical marker to confirm the improvement of CAD patients and an indicator to prevent and predict the occurrence of CAD.

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2.2.2. NLR and Metabolic Diseases Because T2DM is associated with chronic inflammation, we can presume that NLR is associated with T2DM. In a cross-sectional study, participants were distributed into four groups (normal, prediabetic, newly diagnosed diabetic, and previously diagnosed diabetic without complication groups) [25]. NLR was sequentially higher in the prediabetic, newly diagnosed, and previously diagnosed diabetic without complication groups than in the normal group. In addition, a cross-sectional study that investigated whether diabetic complications are associated with NLR in patients with T2DM showed that high NLR was associated with an increased prevalence of CVD and diabetic kidney disease (DKD) [26]. The relationship between NLR and diabetic retinopathy, one of the most important complications that can occur in T2DM patients, was investigated. Patients with diabetic retinopathy showed higher NLR than those with nondiabetic retinopathy [27]. In addition, NLR was an independent risk factor for diabetic retinopathy. Although more research is needed to confirm the findings of these cross-sectional studies, NLR has sufficient potential to predict T2DM and its related complications.

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Chronic inflammation is one explanation for the association of WBC count and NLR with CVD-related mortality, CVD incidence prediction, and disease severity. Arterial stiffness or atherosclerosis is closely related to CVD incidence, and an increase in WBC count and NLR is involved in the formation of arterial stiffness and atherosclerosis through interactions with the endothelium and platelets and overactivity of neutrophil extracellular traps [4,21,22]. In addition, a WBC increase causes a decrease in blood flow, especially in cardiac tissue, which can lead to coronary heart diseases and ischemic heart problems [70].

NLR is also thought to be useful as an excellent marker of CVD. An NLR increase indicates a reduction in lymphocytes induced by increased programmed cell death or infiltration of lymphocytes into cardiac tissue, which are both common in CVD [70].

The relationships of T2DM with WBC count and NLR can be explained by the pathophysiology of chronic inflammation. Various metabolic stimuli lead to an increase in monocytes in the WBC subpopulation of peripheral blood. These monocytes differentiate into macrophages and create an inflammatory state [71].

The possible mechanisms by which an inflammatory state lead to T2DM are a disturbance in insulin signaling in the liver by inflammatory molecules such as IL-6, a proinflammatory effect on insulin, or insulin resistance [71].

Published: 09 October 2017

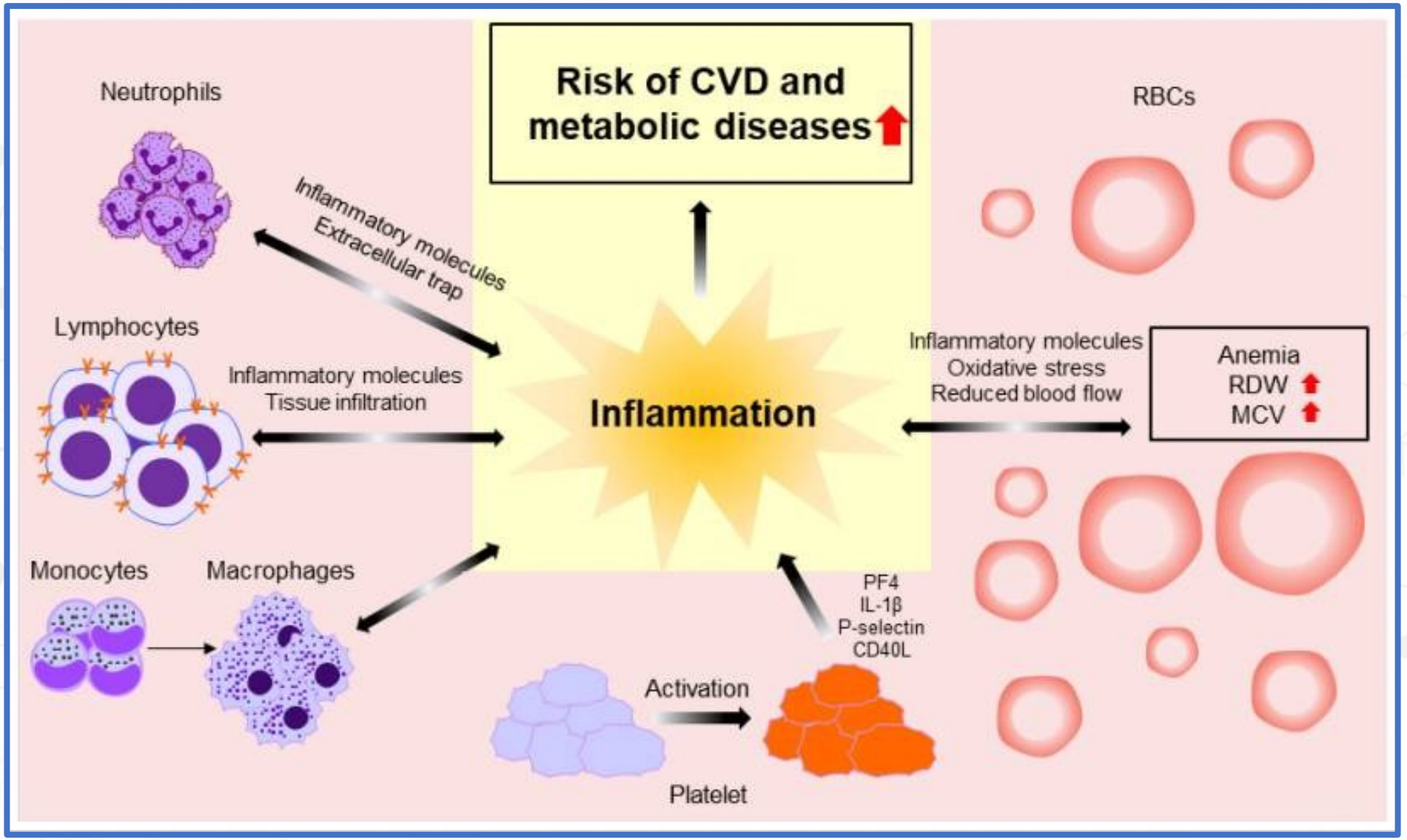
# Neutrophil extracellular traps in immunity and disease

Venizelos Papayannopoulos

*Nature Revie*

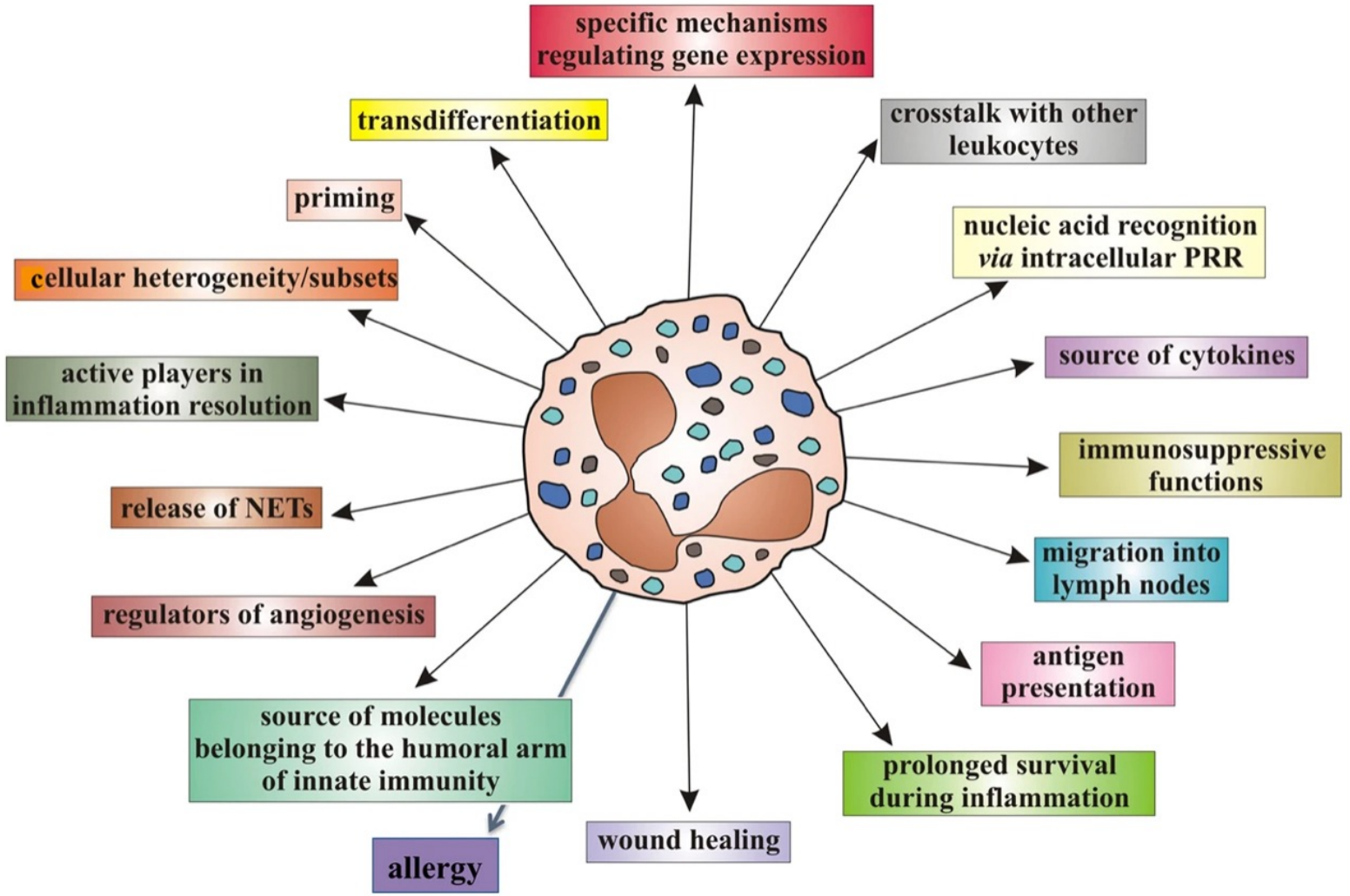


- Neutrophil extracellular traps (NETs) protect against infection, in particular by large pathogens, but they are also implicated in the pathology associated with a growing number of immune-mediated conditions.
- NET formation is triggered by innate immune receptors through downstream intracellular mediators that include reactive oxygen species (ROS), produced by NADPH oxidase or mitochondria, which activate myeloperoxidase (MPO), neutrophil elastase (NE) and protein-arginine deiminase type 4 (PAD4) to promote chromatin decondensation.
- NETosis is induced in response to microbial cues and endogenous danger signals and must be tightly regulated to prevent excessive tissue damage during acute inflammation or chronic inflammatory and autoimmune disease. Microorganism size, microbial virulence factors and cytokines are regulators of NETosis.
- NETs have several immune-modulatory functions that have been implicated in disease. They can prime other immune cells to induce sterile inflammation or potentiate autoimmunity by stimulating interferon responses owing to NET-associated oxidized DNA and antimicrobial peptides.
- NETs can also occlude the vasculature by promoting thrombosis and obstruct important organ areas, capture metastatic tumours and delay wound healing in diabetes.




https://www.ncbi.nlm.nih.gov/pmc/articles/PMC9687310/

# Biologic Activity of Neutrophils




https://www.nature.com/articles/s41423-020-00581-9

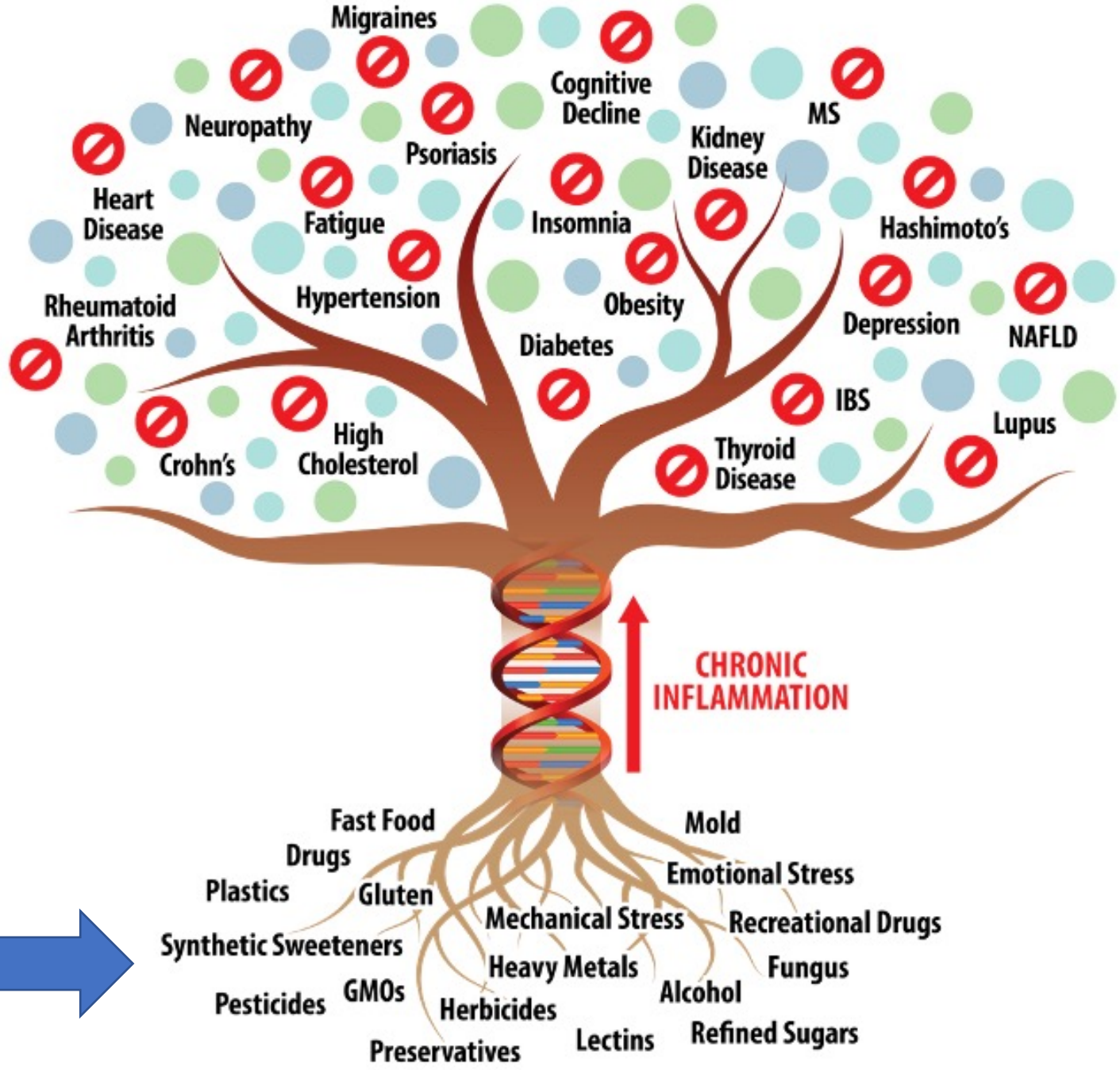
| | Test | Current Result and Flag | | Previous Result and Date | Units | Reference Interval |
|---|---|---|---|---|---|---|
| | WBC [01] | 7.1 | | | x10E3/uL | 3.4-10.8 |
| ▼ | **RBC** [01] | **3.54** | **Low** | | x10E6/uL | 3.77-5.28 |
| ▼ | **Hemoglobin** [01] | **9.6** | **Low** | | g/dL | 11.1-15.9 |
| ▼ | **Hematocrit** [01] | **31.8** | **Low** | | % | 34.0-46.6 |
| | MCV [01] | 90 | | | fL | 79-97 |
| | MCH [01] | 27.1 | | | pg | 26.6-33.0 |
| ▼ | **MCHC** [01] | **30.2** | **Low** | | g/dL | 31.5-35.7 |
| | RDW [01] | 15.3 | | | % | 11.7-15.4 |
| | Platelets [01] | 301 | | | x10E3/uL | 150-450 |
| | Neutrophils [01] | 80 | | | % | Not Estab. |
| | Lymphs [01] | 10 | | | % | Not Estab. |
| | Monocytes [01] | 6 | | | % | Not Estab. |
| | Eos [01] | 3 | | | % | Not Estab. |
| | Basos [01] | 0 | | | % | Not Estab. |

| | Test | Current Result and Flag | | Previous Result and Date | Units | Reference Interval |
|---|---|---|---|---|---|---|
| | Iron Bind.Cap.(TIBC) | 326 | | | ug/dL | 250-450 |
| | UIBC [01] | 307 | | | ug/dL | 118-369 |
| ▼ | **Iron** [01] | **19** | **Low** | | ug/dL | 27-139 |
| | Ferritin [01] | 99 | | | ng/mL | 15-150 |

| | Test | Current Result and Flag | | Previous Result and Date | Units | Reference Interval |
|---|---|---|---|---|---|---|
| | WBC [01] | 5.7 | | | x10E3/uL | 3.4-10.8 |
| ▼ | **RBC** [01] | **3.35** | **Low** | | x10E6/uL | 3.77-5.28 |
| | Hemoglobin [01] | 11.5 | | | g/dL | 11.1-15.9 |
| ▼ | **Hematocrit** [01] | **33.2** | **Low** | | % | 34.0-46.6 |
| ▲ | **MCV** [01] | **99** | **High** | | fL | 79-97 |
| ▲ | **MCH** [01] | **34.3** | **High** | | pg | 26.6-33.0 |
| | MCHC [01] | 34.6 | | | g/dL | 31.5-35.7 |
| | RDW [01] | 12.8 | | | % | 11.7-15.4 |
| ▲ | **Platelets** [01] | **526** | **High** | | x10E3/uL | 150-450 |
| | Neutrophils [01] | 50 | | | % | Not Estab. |
| | Lymphs [01] | 37 | | | % | Not Estab. |
| | Monocytes [01] | 7 | | | % | Not Estab. |
| | Eos [01] | 5 | | | % | Not Estab. |
| | Basos [01] | 1 | | | % | Not Estab. |

| Test | Current Result and Flag | Previous Result and Date | Units | Reference Interval |
|---|---|---|---|---|
| Iron Bind.Cap.(TIBC) | 278 | | ug/dL | 250-450 |
| UIBC [01] | 193 | | ug/dL | 118-369 |
| Iron [01] | 85 | | ug/dL | 27-139 |
| Iron Saturation | 31 | | % | 15-55 |

| | | | | | | |
|---|---|---|---|---|---|---|
| | AST (SGOT) [01] | 29 | | | IU/L | 0-40 |
| ▲ | **ALT (SGPT)** [01] | **35** | **High** | | IU/L | 0-32 |


Migraines
Neuropathy
Psoriasis
Cognitive Decline
MS
Kidney Disease
Heart Disease
Fatigue
Insomnia
Hashimoto's
Hypertension
Obesity
Depression
NAFLD
Rheumatoid Arthritis
Diabetes
IBS
Lupus
Crohn's
High Cholesterol
Thyroid Disease
CHRONIC INFLAMMATION
Fast Food
Mold
Drugs
Emotional Stress
Plastics
Gluten
Mechanical Stress
Recreational Drugs
Synthetic Sweeteners
Heavy Metals
Fungus
Pesticides
GMOs
Alcohol
Herbicides
Refined Sugars
Lectins
Preservatives

# The Wedge Protocol