Functional Lab Tests for the Podiatric Physician 2026

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Board Certified in Family Practice
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No conflict of interest

1967

CBC with Diff

WBC
RBC
HB
Hematocrit
Platelet Count
Abs. Neutrophils
Abs. Lymphocytes
Abs. Monocytes
Abs. Eosinophils
Abs. Bacophills

CMP

BuN
Creatinine
EGFR
BUN | Creatinine Ratio
Sodium
Potassium
Chloride
CO2
Calcium
Albumin
Total Bilirubin
Alkaline
Phosphatase
AST
ALT

2026

CBC with Diff

WBC
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CMP

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CO2
Calcium
Albumin
Total Bilirubin
Alkaline
Phosphatase
AST
ALT

HS- CRP
High Sensitivity C-reactive protein

1. Marker of Systemic Inflammation and Cardiovascular Risk

Low Risk < 1mg/L

Intermediate Risk 1-3mg/L

High Risk > 3mg/L

2. Marker of Infection and Acute Inflammation

Pneumonia

Appendicitis

Sepsis

C-reactive protein is a mediator of cardiovascular disease

Radjesh J Bisoendial, S Matthijs Boekholdt, Menno Vergeer, Erik S G Stroes, John J P Kastelein

Heart Journal 2010 September 20

Affiliations Expand

Abstract

C-reactive protein is postulated to embody an index that can reflect cardiovascular risk and can be used to independently predict major cardiovascular events and mortality. On the other hand, credible experimental data have become available that demonstrate the abundant presence of C-reactive protein in atherosclerotic lesions and, moreover, identify C-reactive protein as an initiator of several pathogenic pathways that can cause atherogenic changes. Consequently, there has been a paradigm shift in which C-reactive protein is no longer regarded as merely an indicator of cardiovascular risk, but increasingly considered a direct partaker in the pathogenesis of atherosclerotic cardiovascular disease. These data underscore the need to explore risk-reducing interventions that selectively inhibit C-reactive protein activity as a novel strategy to prevent clinical manifestations of atherosclerosis.

HS- CRP
High Sensitivity C-reactive Protein

3. Marker of Metabolic and Adipose Tissue Inflammation

Obesity

Insulin Resistance

Type 2 Diabetes

4. Prognostic Indicator of Various Diseases

Cardiovascular

Cancer

Rheumatoid Diseases

Homocysteine

< 11.4 umol / L

1. Intermediate in Methionine and Cysteine Metabolism

Involved in cme- carbon metabolism

High levels affect downstream methylation capacity

2. Substitute for Methylation Reactions

When re-methylated to methionine it forms S - Adenosylmethionine (SAM) the principal universal methyl donor for DNA, RNA, Protein, Lipid and Neurotransmitter Methylation

Methylation

1. Transfers Methyl Groups Via S - Adenosyl Methionine (SAM) From Methionine to Deverse

Targets -> DNA, Proteins, Lipids

2. After Donation, S - Adenosylhomocysteine (SAH) Forms and Converts to Homocysteine

3. Homocysteine Resumes Methionine or Forms Cysteine, Maintaining Methylation and Redox Balance

Meta-Analysis

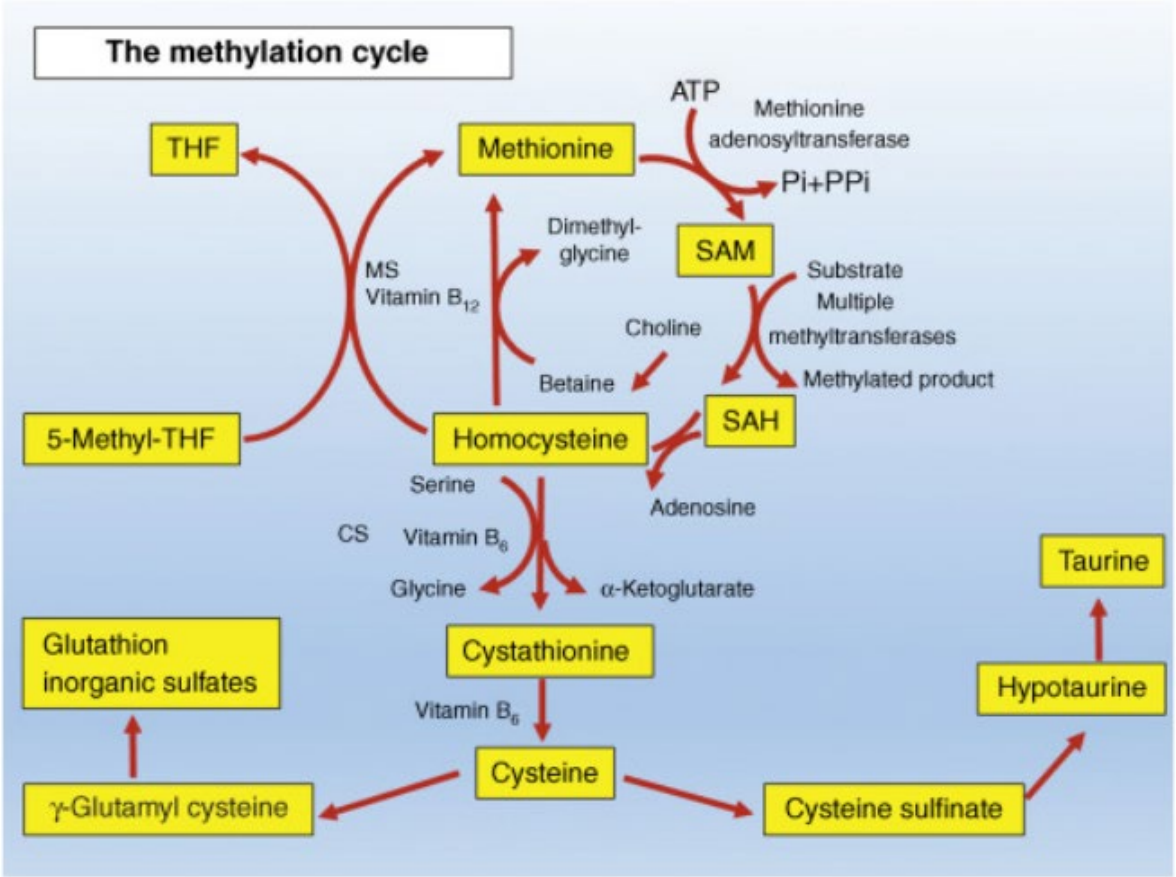
. 2019 Aug 20;140(8):645-657.
doi: 10.1161/CIRCULATIONAHA.118.039357. Epub 2019 Aug 19.

Blood Leukocyte DNA Methylation Predicts Risk of Future Myocardial Infarction and Coronary Heart Disease

Golareh Agha[1], Michael M Mendelson[2,3,4], Cavin K Ward-Caviness[5,6], Roby Joehanes[2,7], TianXiao Huan[8], Rahul Gondalia[9], Elias Salfati[10], Jennifer A Brody[11], Giovanni Fiorito[12], Jan Bressler[13], Brian H Chen[14], Symen Ligthart[15], Simonetta Guarrera[12], Elena Colicino[16], Allan C Just[16], Simone Wahl[17], Christian Gieger[17], Amy R Vandiver[18], Toshiko Tanaka[14], Dena G Hernandez[19], Luke C Pilling[20], Andrew B Singleton[19], Carlotta Sacerdote[21], Vittorio Krogh[22], Salvatore Panico[23], Rosario Tumino[24], Yun Li[25], Guosheng Zhang[26], James D Stewart[27], James S Floyd[11], Kerri L Wiggins[11], Jerome I Rotter[28], Michael Multhaup[29], Kelly Bakulski[30], Steven Horvath[31], Philip S Tsao[32], Devin M Absher[33], Pantel Vokonas[34], Joel Hirschhorn[35,36], M Daniele Fallin[37], Chunyu Liu[38], Stefania Bandinelli[17], Eric Boerwinkle[39,40], Abbas Dehghan[41], Joel D Schwartz[42], Bruce M Psaty[42,43], Andrew P Feinberg[44], Lifang Hou[45], Luigi Ferrucci[46], Nona Sotoodehnia[47], Giuseppe Matullo[12], Annette Peters[48,49], Myriam Fornage[50], Themistocles L Assimes[51], Eric A Whitsel[52], Daniel Levy[2,3], Andrea A Baccarelli[1]
Affiliations Expand

Background: DNA methylation is implicated in coronary heart disease (CHD), but current evidence is based on small, cross-sectional studies. We examined blood DNA methylation in relation to incident CHD across multiple prospective cohorts.

Conclusion: Methylation of blood-derived DNA is associated with risk of future CHD across diverse populations and may serve as an informative tool for gaining further insight on the development of CHD.


The methylation cycle
ATP
Methionine adenosyltransferase
Pi+PPi
THF
Methionine
SAM
Dimethyl-glycine
MS
Vitamin B_{12}
Substrate
Multiple
methyltransferases
Methylated product
Choline
Betaine
5-Methyl-THF
Homocysteine
SAH
Adenosine
Serine
CS
Vitamin B_6
Glycine
α-Ketoglutarate
Taurine
Glutathion
inorganic sulfates
Cystathionine
Hypotaurine
Vitamin B_6
Cysteine
γ-Glutamyl cysteine
Cysteine sulfinate

Why Methylation Matters

Gene Regulation: DNA Methylation

Patterns help determine which genes are “on” or “off” in a cell

Epigenetics:

Methylation can change in response to age, diet, environment and disease

Metabolism + Detoxification:

Methylation removes toxins

NeuroBiology:

Methylation influences neurotransmitter synthesis and receptor function

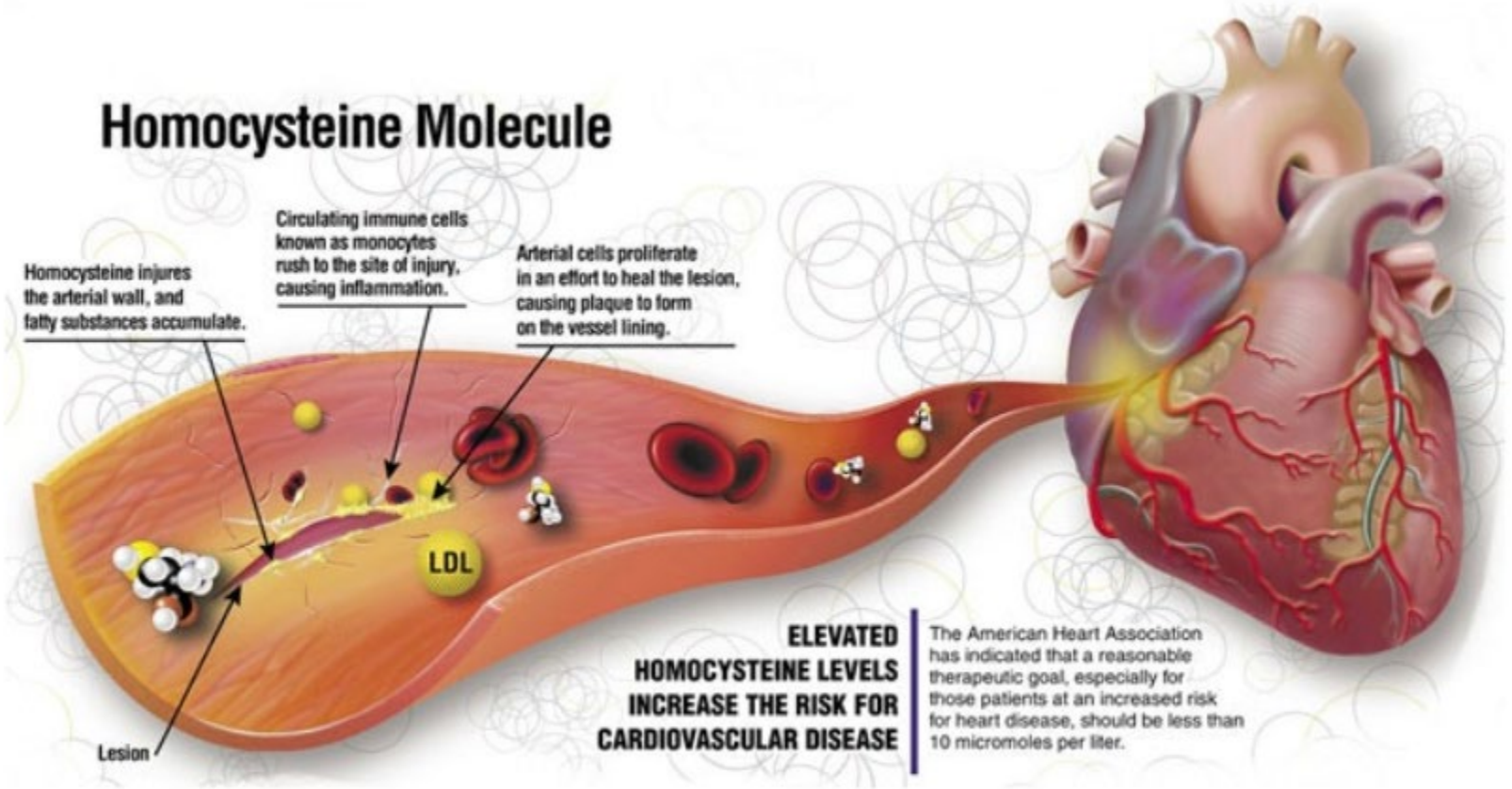
Homocysteine Continued

1. Vascular Disease:

Elevated homocysteine level is associated with vascular endothelial dysfunction, prothrombotic states and arterial disease

2. Antioxidant Defense:

The transsulfuration pathway generates cysteine which is a precursor to Glutathione


Homocysteine Molecule
Homocysteine injures the arterial wall, and fatty substances accumulate.
Circulating immune cells known as monocytes rush to the site of injury, causing inflammation.
Arterial cells proliferate in an effort to heal the lesion, causing plaque to form on the vessel lining.
LDL
Lesion
ELEVATED HOMOCYSTEINE LEVELS INCREASE THE RISK FOR CARDIOVASCULAR DISEASE
The American Heart Association has indicated that a reasonable therapeutic goal, especially for those patients at an increased risk for heart disease, should be less than 10 micromoles per liter.

Homocysteine and coronary heart disease

Robert Clarke, Sarah Lewington
Vascular Medicine 2014 November 2

Abstract

Children with homocystinuria have markedly elevated plasma homocysteine concentrations and increased risks of stroke and coronary heart disease (CHD). Supplementation with folic acid, vitamin B6, and vitamin B12 lower homocysteine levels and such therapy is remarkably effective in delaying the occurrence of vascular events in affected individuals. The relevance, if any, of moderately elevated homocysteine levels to cardiovascular disease in the general population is uncertain. The results of retrospective studies of homocysteine and risk of cardiovascular disease (where blood is collected after the onset of disease) indicate that CHD or stroke patients invariably have higher homocysteine levels than age-matched controls. In contrast, the results of prospective studies (where blood is collected before onset of disease) show much weaker associations of homocysteine with cardiovascular disease. This article examines the background, epidemiological evidence relating homocysteine with vascular disease, and effects of vitamin supplements on homocysteine concentrations. Large-scale clinical trials of folic acid-based vitamin supplements are currently in progress to test whether lowering blood homocysteine levels can reduce the risks of CHD and stroke.

Cortisol

A.M. = 4 - 22 meq / L
P.M. = 3 - 17

Total & Free

A steroid Hormone produced by the Adrenal Glands

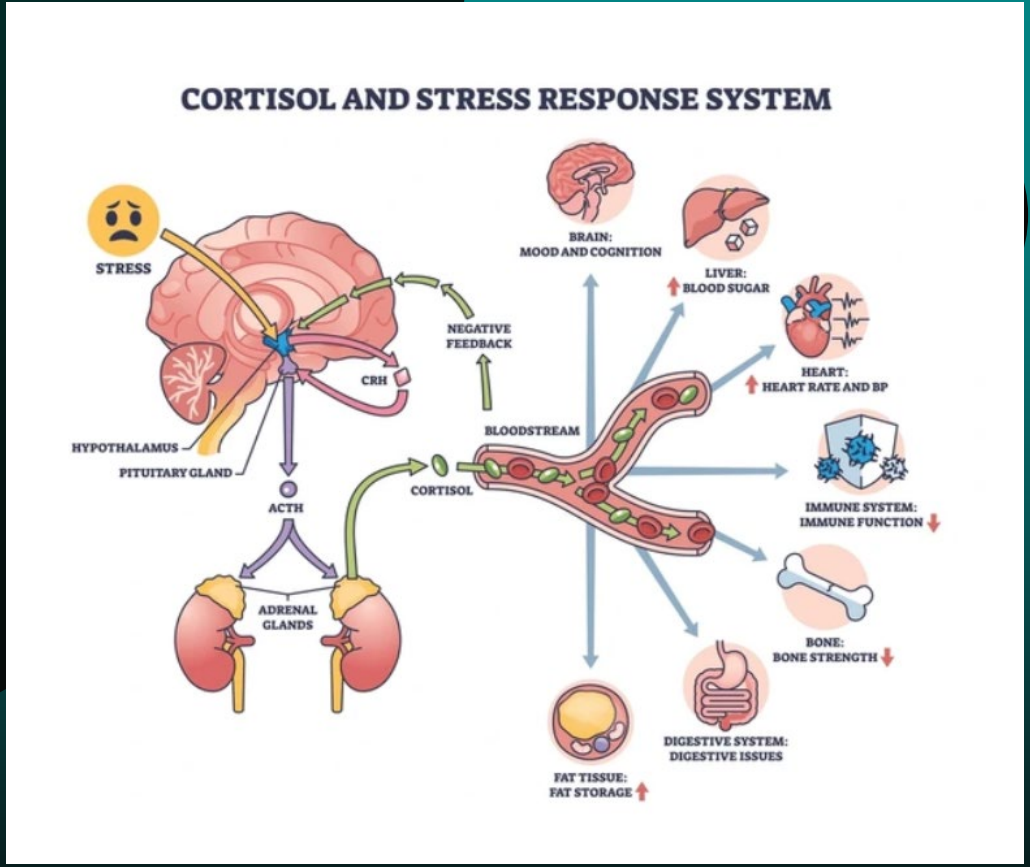
Key Roles:

- Regulates Metabolism -> Promotes Gluconeogenesis
- Maintains Blood Pressure
- Manages Blood Sugar Levels
- Reduces Inflammation

Peaks in memory, lowest at night

Cortisol Reflects HPA Axis Status

Low Level = Adrenal Insufficiency
High Levels = Cushing's Syndrome
Stress Response
Exogenous Steroid Effect


CORTISOL AND STRESS RESPONSE SYSTEM
STRESS
HYPOTHALAMUS
PITUITARY GLAND
CRH
ACTH
ADRENAL GLANDS
NEGATIVE FEEDBACK
CORTISOL
BLOODSTREAM
BRAIN: MOOD AND COGNITION
LIVER: BLOOD SUGAR
HEART: HEART RATE AND BP
IMMUNE SYSTEM: IMMUNE FUNCTION
BONE: BONE STRENGTH
DIGESTIVE SYSTEM: DIGESTIVE ISSUES
FAT TISSUE: FAT STORAGE

Review

Adv Wound Care (New Rochelle) Adv Wound Healing
2019 July 5

Stress-Induced Hormones Cortisol and Epinephrine Impair Wound Epithelization

Olivera Stojadinovic[1], Katherine A Gordon[1], Elizabeth Lebrun[1], Marjana Tomic-Canic[1]
Affiliations Expand

Abstract

Background: Stress-induced disruption of hormonal balance in animals and humans has a detrimental effect on wound healing.

The problem: After the injury, keratinocytes migrate over the wound bed to repair a wound. However, their nonmigratory phenotype plays a role in pathogenesis of chronic wounds. Despite many therapeutic approaches, there is a dearth of treatments targeting the molecular mechanisms mediated by stress that prevent epithelization.

Basic/clinical science advances: Recent studies show that epidermal keratinocytes synthesize stress hormones. During acute wound healing, cortisol synthesis in the epidermis is tightly controlled. Further, a key intermediate molecule in the cholesterol synthesis pathway, farnesyl pyrophosphate (FPP), can bind glucocorticoid receptor (GR) and activate GR. Additionally, keratinocytes express beta-2-adrenergic-receptor (β2AR), a receptor for the stress hormone epinephrine. Importantly, migratory rates of keratinocytes are reduced by cortisol, FPP, epinephrine, and other β2AR agonists, thus indicating their role in the inhibition of epithelization. Topical inhibition of local glucocorticoid and FPP synthesis, as well as treatment with β2AR antagonists promotes wound epithelization.

Clinical care relevance: Modulation of local stress hormone production may represent an important therapeutic target for wound healing disorders.

DHEA- S
Dehydroepiandrosterone

61 - 442 m<g / dL

DHEA produced in the zona reticularis of the adrenal cortex

DHEA serrer a circulating precursor

For peripheral production of
Testosterone
Dihydrotestosterone
Estradiol

Levels are age and sex dependent

DHEA- S

Low DHEA Symptoms

- Decreased libido
- Fatigue
- Depression
- Decrease muscle mass find bone density

Impact on Metabolism and Body Composition

Associated with

- Fat Distribution
- Insulin Sensitivity
- Lipid Profiles

. Epub 2018 Feb 24.

Dehydroepiandrosterone and Bone

Shuanhu Zhou[1], Julie Glowacki[1]
Affiliations Expand

- PMID: 30029729

- DOI: 10.1016/bs.vh.2018.01.005

Abstract

In humans, dehydroepiandrosterone (DHEA), secreted mainly from the adrenal cortex, and its sulfate ester, DHEAS, are the most abundant circulating steroids. DHEA/DHEAS possess pleiotropic effects in human aging, bone, metabolic diseases, neurologic function/neurodegenerative diseases, cancer, immune system and disorders, cardiovascular diseases, diabetes, muscle function, sexual dysfunction, and other health conditions. The age-related reduced levels of DHEA and DHEAS are associated with bone mineral density measures of osteopenia and osteoporosis. Clinical, epidemiological, and experimental studies indicate that DHEA replacement therapy may be beneficial for bone health through its inhibition of skeletal catabolic IL-6 and stimulation of osteoanabolic IGF-I-mediated mechanisms. Studies with primary cultures of human bone marrow-derived mesenchymal stem cells (hMSCs) were used to show that DHEA stimulates osteoblastogenesis. The in vitro stimulation of both osteoblastogenesis and IGF-I gene expression by DHEA in hMSCs requires IGF-I receptor, PI3K, p38 MAPK, or p42/44 MAPK signaling pathways. The in vitro inhibition of IL-6 secretion in hMSCs by DHEA was more consistent and extensive than by estradiol or dihydrotestosterone. In summary, evidence from us and others indicates that DHEA may be useful for treating bone diseases through its inhibition of skeletal catabolic IL-6 and stimulation of anabolic IGF-I-mediated mechanisms.

Vitamin D 30-100ng/mL

Calciferol - A Fat-Soluble Secosteroids

Exists on 2 Forms:

VIT D2 - Ergocalciferol:

Found on plant sources

VIT D3 - Ergocalciferol:

Produced on the skin in response to sun exposure (UVB)

Activation:

VIT D is inactive when ingested or produced on the skins

Converted in the liver to 25 - hydroxy D then on the kidney to the active form 1, 25 dihydroxy 2D calcitriol

Function:

Handles calcium and phosphorus for bone health

Influences immune function

Muscle performances

Cell growth

Modulates gene expression

Review

J Investig Med

. 2019 Aug;59(6):881-6.

Vitamin D and the immune system

Cynthia Aranow[1]
Affiliations Expand

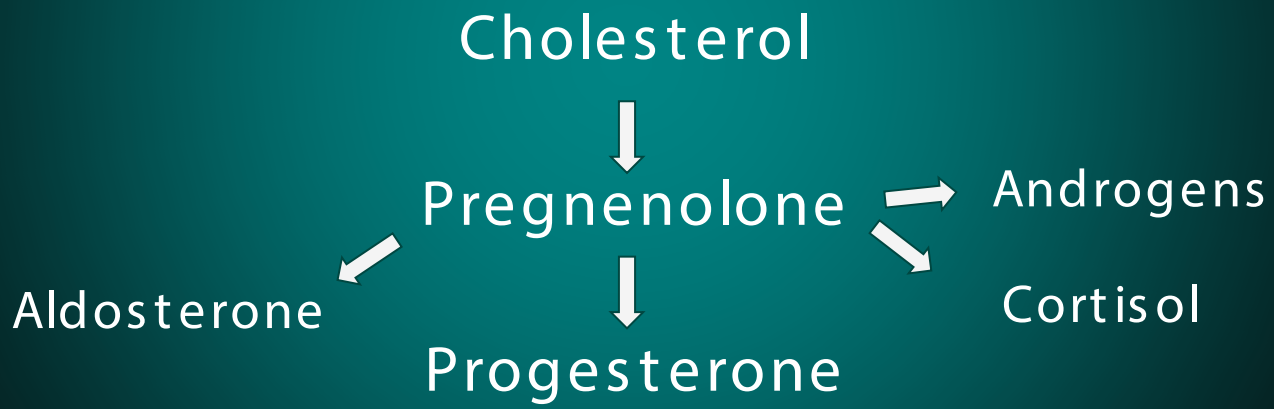
Abstract

It is now clear that vitamin D has important roles in addition to its classic effects on calcium and bone homeostasis. As the vitamin D receptor is expressed on immune cells (B cells, T cells, and antigen-presenting cells), and these immunologic cells are all capable of synthesizing the active vitamin D metabolite, vitamin D has the capability of acting in an autocrine manner in a local immunologic milieu. Vitamin D can modulate the innate and adaptive immune responses. Deficiency in vitamin D is associated with increased autoimmunity and an increased susceptibility to infection. As immune cells in autoimmune diseases are responsive to the ameliorative effects of vitamin D, the beneficial effects of supplementing vitamin D-deficient individuals with autoimmune disease may extend beyond the effects on bone and calcium homeostasis.

Pregnenolone 22-237 ng/dL

(Mother of all Hormones)

1. Endogenous Synthesis (Body's Natural Production)
   - Primary Site: Adrenal Cortex
   - Starting Material: Cholesterol
   - Cholesterol -> Pregnenolone via enzyme p450 in the mitochondria



Pregnenolone

Functions

- The fundamentals precursor for all downstream steroid hormones
- Acts as a neurosteriod that modulates neuronal signaling cognition, mood and stress responses
- Modulates adrenal and gonadal hormone production
- Levels decline with age
  - Will affect
    - Energy
    - Libido
    - Metabolic regulation
- Pregnenolone steal - syndrome

Pregnenolone promotes degradation of key proteins in immune signaling to suppress inflammation

Subathra Murugan[1], Padmaja Jakka[1,2], Swapna Namani[1,3], Varadendra Mujumdar[1], Girish Radhakrishnan[1]
Affiliations Expand

Abstract

Pregnenolone is a steroid hormone precursor that is synthesized in various steroidogenic tissues, in the brain, and in lymphocytes. In addition to serving as the precursor for other steroid hormones, pregnenolone exerts its own effect as an anti-inflammatory molecule to maintain immune homeostasis in various inflammatory conditions. Pregnenolone and its metabolic derivatives have been shown to have beneficial effects in the brain, including enhancing memory and learning, reversing depressive disorders, and modulating cognitive functions. A decreased level of pregnenolone has been observed in neuroinflammatory diseases, which emphasizes its role in neuroprotection and neuroregeneration. Although the anti-inflammatory property of pregnenolone was recognized several decades ago, its mechanism of action remains unknown. Here we report that pregnenolone promotes ubiquitination and degradation of the TLR2/4 adaptor protein TIRAP and TLR2 in macrophages and microglial cells. Pregnenolone and its metabolites suppressed the secretion of tumor necrosis factor α and interleukin-6 mediated through TLR2 and TLR4 signaling. Pregnenolone has been reported to induce activation of cytoplasmic linker protein 170, and this protein has recently been shown to promote targeted degradation of TIRAP. We observed enhanced degradation of TIRAP and TLR4 suppression by cytoplasmic linker protein 170 in the presence of pregnenolone. Our experimental data reveal novel nongenomic targets of pregnenolone and provide important leads to understand its role in restoring immune homeostasis in various inflammatory conditions.

Insulin - Like Growth Factor 1

IGF - 1

- IGF - 1 is produced primarily in the liver
- Additional sources are bone, muscle, fat, other tissue
- Production is regulated by growth hormone

Regulation of IGF - 1 Production

- Adequate Protein intake
- Insulin can modulate production
- Estrogen + testosterone influence IGF - 1 levels
- Thyroid hormones and inflammatory cytokines can modify IGF - 1

IGF- 1

Physical Functions

- Stimulate longitudinal bone growth, muscle development, organ growth
- Encourage protein synthesis, muscle repair
- Influences glucose + lipid metabolism
- Supports neuronal growth

Clin Exp Rheumatol

. 2025 Jun;43(6):1122-1127.
doi: 10.55563/clinexprheumatol/g17lb8. Epub 2025 Mar 18.

Lower circulating IGF-1 levels in fibromyalgia: meta-analysis highlighting potential pathogenic role

Young Ho Lee[1], Gwan Gyu Song[2]

Objectives: To evaluate the relationship between circulating insulin-like growth factor (IGF-1) levels and fibromyalgia (FM).

Methods: Meta-analyses were performed to compare serum/plasma IGF-1 levels in patients with FM and healthy controls and in patients with FM according to subgroups based on region, sample size, data type, publication year, and matched variables (age, sex, and/or BMI).

Results: Twelve studies from eleven reports including 512 patients with FM and 308 controls were selected. IGF-1 levels were not found to be decreased in the FM group (standardized mean differences (SMD) =-0.347, 95% confidence interval [CI]: -0.747 to 0.053, p=0.089. However, sensitivity analysis showed that results of one study significantly affected the pooled SMD (SMD =-0.458, 95% CI: -0.822 to -0.093, p=0.014), indicating that the results of this meta-analysis were unstable. Additionally, the SMD changed to be significant after adjusting for publication bias (SMD =-0.513, 95% CI: -0.924 to -0.102). Stratification according to data type showed a significantly lower IGF-1 level in the FM group with original data (SMD =-0.458, 95% CI: -0.857 to -0.060, p=0.024). Stratification by publication year revealed a significantly lower IGF-1 level in the FM group by recent year (year >2012) (SMD =-0.679, 95% CI: -1.066 to -0.293, p=0.001).

Conclusions: Our meta-analysis demonstrated that IGF-1 levels were significantly lower in patients with FM, suggesting that IGF-1 might play an important role in the pathogenesis of FM.

Fasting Insulin

M
2-20 NU / mL

The concentration of insulin in the blood after an overnight FAST (8-12 hours)

Why it's Measured

- Assess insulin resistance and metabolic health
- Helps evaluate risk for Type 2 Diabetes and fatty liver
- Complement fasting glucose and HbA1c
- Monitor conditions affection, insulin production and sensitivity

Fasting Insulin 2-25 uU/mL

Interpet Results

Elevated Fasting Insulin
- Insulin resistance

Low Fasting Insulin
- Insulin deficiency or reduced beta cell function

Practical Uses

- Screening high risk populations
- Guiding life style or pharmacologic intervention
- Contextualizing glucose abnormalities and diabetes risk beyond fasting glucose + HbA1c
- Using HOMA-IR calculation. Homeostatic Model Assesment of insulin Resistance . HOMA = fasting insulin xFasting glucose /405

International Journal of Cardiology
Volume 172, Issue 2, 15 March 2020, Pages 450-455

Elevated fasting serum insulin level predicts future development of hypertension

Author links open overlay panel Sung Keun Park [a e 1], Ju Young Jung [a 1], Won Joon Choi [b], Yun Hong Kim [b], Hyun Soo Kim [b], Woo Taek Ham [c],

Abstract

Background

Studies have investigated clinical association between fasting insulin level and hypertension. However, it is still debatable whether elevated fasting insulin actually increases the risk of hypertension with the passage of time. Thus, this study was aimed at investigating the association between baseline fasting insulin level and the development of hypertension.

Conclusion

The risk of hypertension was in proportion to the baseline fasting insulin level. In addition, hyperinsulinemia was an independent risk factor for the future development of hypertension. These findings suggest the value of fasting insulin level as an early predictor of hypertension.

Vitamin B - Cobalamin

200 - 1100 pg / mL

VIT B12 refers to a group of related corrinoids

Methylcobalamin and adenosylcobalamin are the biologically active cofactor forms

Dietary Sources:

- Meat
- Fish
- Diary
- Eggs

Absorption Pathway:

1. Stomach
   B12 binds to intrinsic factor produced by parietal call
2. The B12 - IF complex binds to cubilin receptors in the terminal ileum enabling absorption into enterocytes
3. Inside cells, B12 is released and transported on the blood bound to transcobalamin 2

VIT B12

B 12 is required for proper DNA synthesis

B 12 is needed for myelin sheath integrity

B 12 helps convert homocysteine to methionine

B 12 supports normal RBC production

Deficiency of VIT B 12

- Disrupts methylation and energy production
  - Leading to
    - Anemia
    - Neuropathy
    - Cognitive changes
    - Elevated homocysteine

Why Order B - 12 levels

Suspected deficiency:
symptoms: fatigue, pallor, glossitis, neuropathy, balance problems, memory/ cognition changes

Vitamin B_{12} is a factor for induced cellular plasticity and tissue repair

Marta Kovatcheva[1], Elena Melendez[2], Dafni Chondronasiou[2], Federico Pietrocola[2,3], Raquel Bernad[2], Adrià Caballe[2], Alexandra Junza[4,5], Jordi Capellades[4,6], Adrián Holguín-Horcajo[7,8], Neus Prats[2], Sylvere Durand[9,10], Meritxell Rovira[7,8], Oscar Yanes[4,5], Camille Stephan-Otto Attolini[2], Guido Kroemer[9,10,11], Manuel Serrano[12,13,14]
Affiliations Expand

Abstract

Transient reprogramming by the expression of OCT4, SOX2, KLF4 and MYC (OSKM) is a therapeutic strategy for tissue regeneration and rejuvenation, but little is known about its metabolic requirements. Here we show that OSKM reprogramming in mice causes a global depletion of vitamin B_{12} and molecular hallmarks of methionine starvation. Supplementation with vitamin B_{12} increases the efficiency of reprogramming both in mice and in cultured cells, the latter indicating a cell-intrinsic effect. We show that the epigenetic mark H3K36me3, which prevents illegitimate initiation of transcription outside promoters (cryptic transcription), is sensitive to vitamin B_{12} levels, providing evidence for a link between B_{12} levels, H3K36 methylation, transcriptional fidelity and efficient reprogramming. Vitamin B_{12} supplementation also accelerates tissue repair in a model of ulcerative colitis. We conclude that vitamin B_{12}, through its key role in one-carbon metabolism and epigenetic dynamics, improves the efficiency of in vivo reprogramming and tissue repair.

Lipid Panel

Lipoprotein (a) Apolipoprotein B
Total cholesterol, Triglycerides
HDL, LDL

Lipoprotein (a)

- Genetically inherited
- Special type of LDL
- Carries fat and cholesterol throughout the bloodstream
- Contains a unique protein - Apolipoprotein (a)

- Stickier
- Causes inflammation
- Forms plaques

- Levels are stable throughout life
- Not influenced by lifestyle changes or major lipid lowering therapies…..PCSK9 may reduce 20%

Lipoprotein (a)

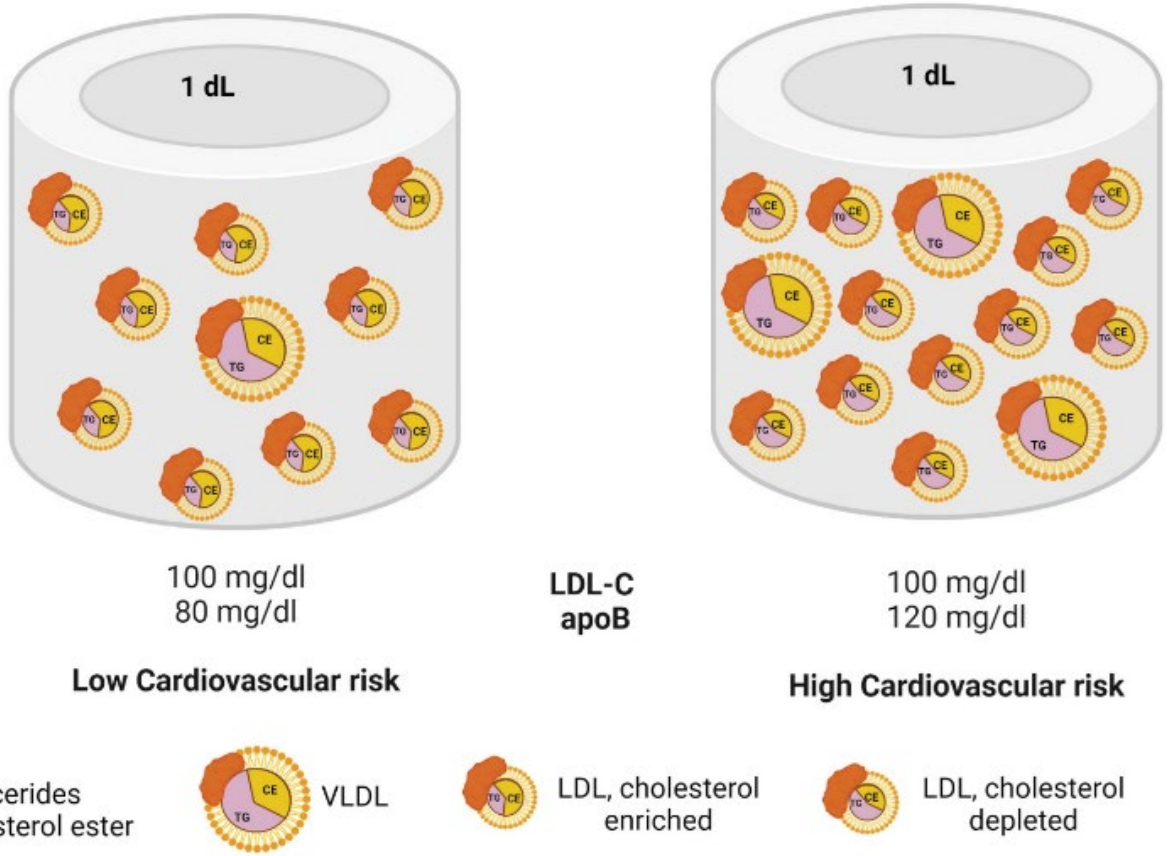
- Is an independent risk factor for cardiovascular disease
- High levels can cause plaque buildup
- High levels cause inflammation
- High levels can cause blood clotting

Reference Range: Risk

Optimal < 75 nmol / L

Moderate 75 - 125

High > 125


1 dL
1 dL
100 mg/dl
80 mg/dl
LDL-C
apoB
100 mg/dl
120 mg/dl
Low Cardiovascular risk
High Cardiovascular risk
TG: Triglycerides
CE: Cholesterol ester
VLDL
LDL, cholesterol enriched
LDL, cholesterol depleted

Apolipoprotein B

- Is a large protein that serves as the main structural scaffold for several atherogenic lipoproteins

- 2 Types:
    - ApoB - 100 ,

      VLDL
      - LDL
      - Lipo (a)

      Apo B – 48
      Chylomicrons

- High ApoB is a better predictor of cardiovascular risk then LDL

Reference Range:

Optimal < 90 mg / dL

Moderate 90 - 129

High > 130

Apolipoprotein B: Bridging the Gap Between Evidence and Clinical Practice

Diana De Oliveira-Gomes, MD; Parag H. Joshi , MD, MHS; Eric D. Peterson , MD, MPH; Anand Rohatgi , MD, MSCS; Amit Khera , MD, MSc; Ann Marie Navar, MD, PhD

ABSTRACT: Despite data suggesting that apolipoprotein B (apoB) measurement outperforms low-density lipoprotein cholesterol level measurement in predicting atherosclerotic cardiovascular disease risk, apoB measurement has not become widely adopted into routine clinical practice. One barrier for use of apoB measurement is lack of consistent guidance for clinicians on how to interpret and apply apoB results in clinical context. Whereas guidelines have often provided clear lowdensity lipoprotein cholesterol targets or triggers to initiate treatment change, consistent targets for apoB are lacking. In this review, we synthesize existing data regarding the epidemiology of apoB by comparing guideline recommendations regarding use of apoB measurement, describing population percentiles of apoB relative to low-density lipoprotein cholesterol levels, summarizing studies of discordance between low-density lipoprotein cholesterol and apoB levels, and evaluating apoB levels in clinical trials of lipid-lowering therapy to guide potentia

SUMMARY OF RESULTS AND APOB TARGETS The totality of data suggests that apoB may be a superior marker not only of risk of ASCVD but also of the benefit of lipid-lowering therapy compared with LDL-C. ApoB testing has practical benefits, including that patients do not need to fast, and is more accurate than LDL-C testing in patients with high triglyceride or very low LDL-C levels. Guidelines are increasingly recommending more aggressive lipid reduction; therefore, obtaining precise lipid measurements is essential. This has led many to call for the preferential use of apoB over LDL-C testing

Apolipoprotein B Particles and Cardiovascular DiseaseA Narrative Review

1. Allan D. Sniderman, MD[1]; George Thanassoulis, MD[1]; Tamara Glavinovic, MD, FRCPC[2]

JAMA Cardiol
Published Online: October 23, 2019
2019;4;(12):1287-1295. doi:10.1001/jamacardio.2019.3780

Abstract

Importance The conventional model of atherosclerosis presumes that the mass of cholesterol within very low-density lipoprotein particles, low-density lipoprotein particles, chylomicron, and lipoprotein (a) particles in plasma is the principal determinant of the mass of cholesterol that will be deposited within the arterial wall and will drive atherogenesis. However, each of these particles contains one molecule of apolipoprotein B (apoB) and there is now substantial evidence that apoB more accurately measures the atherogenic risk owing to the apoB lipoproteins than does low-density lipoprotein cholesterol or non–high-density lipoprotein cholesterol.

Conclusions and Relevance Apolipoprotein B unifies, amplifies, and simplifies the information from the conventional lipid markers as to the atherogenic risk attributable to the apoB lipoproteins.

Pass - Through Lab Tests

A billing arrangement between a medical practice and a laboratory

Who is involved?

- The doctors office
- The laboratory
- The patient or insurance plan

How it works?

- Doctor orders labs or draws blood for lab work
- Lab reports results to doctor
- Payment
  1. Lab bills the payment
  2. The practice bills the patient or insurance

Example:

- Blood draws in office for a CBL
- Lab charges $12.00
- Pt's insurance charged $24.00