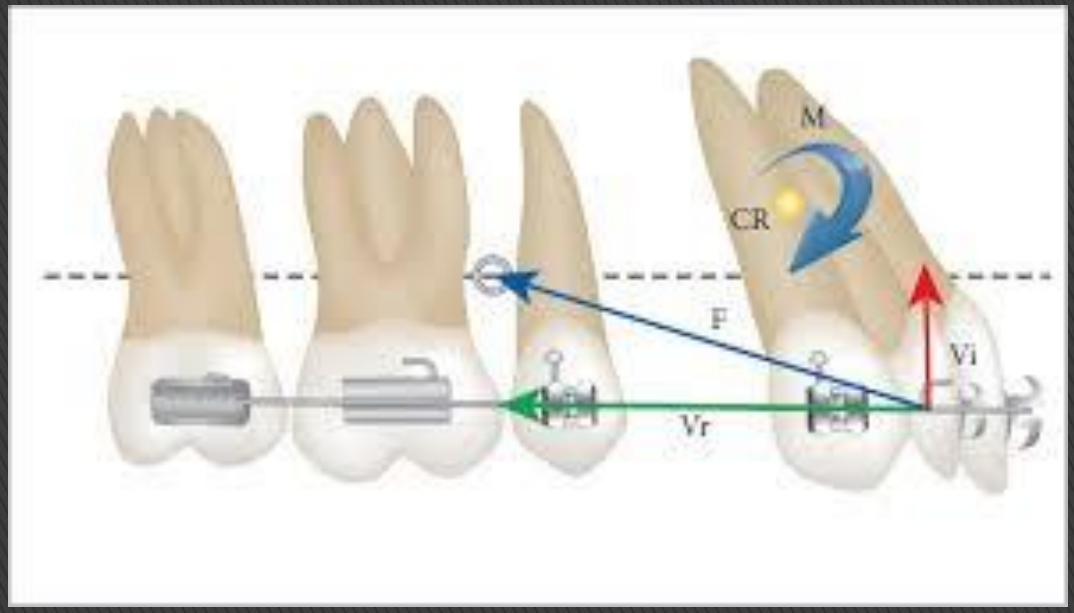
Paul G.B. Johnson, DDS
Midwest Periodontics
Sioux Falls, SD

Periodontics is simple.

Unlike…

Endodontics

Orthodontics

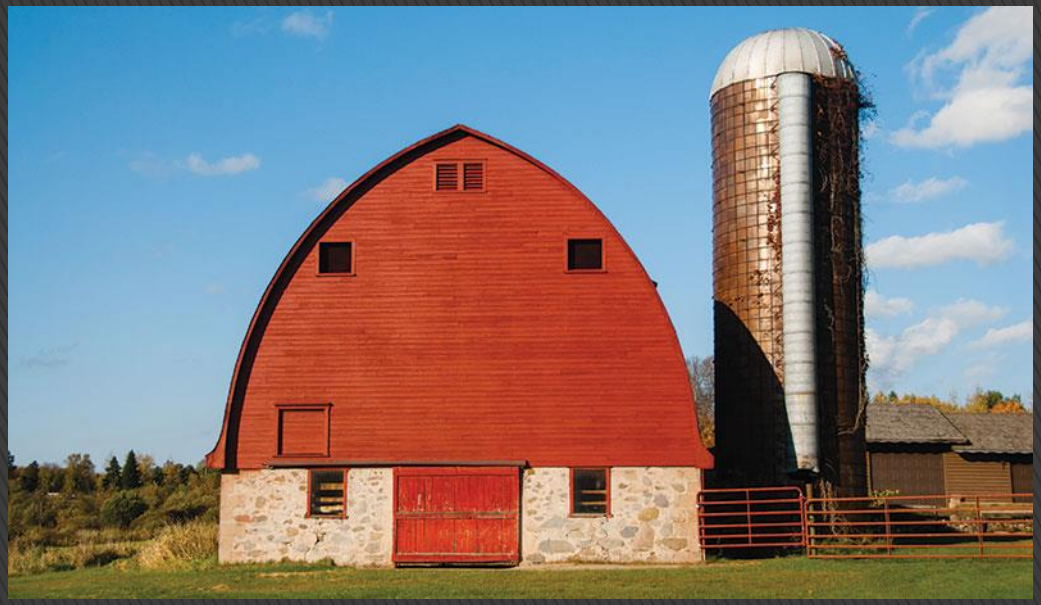


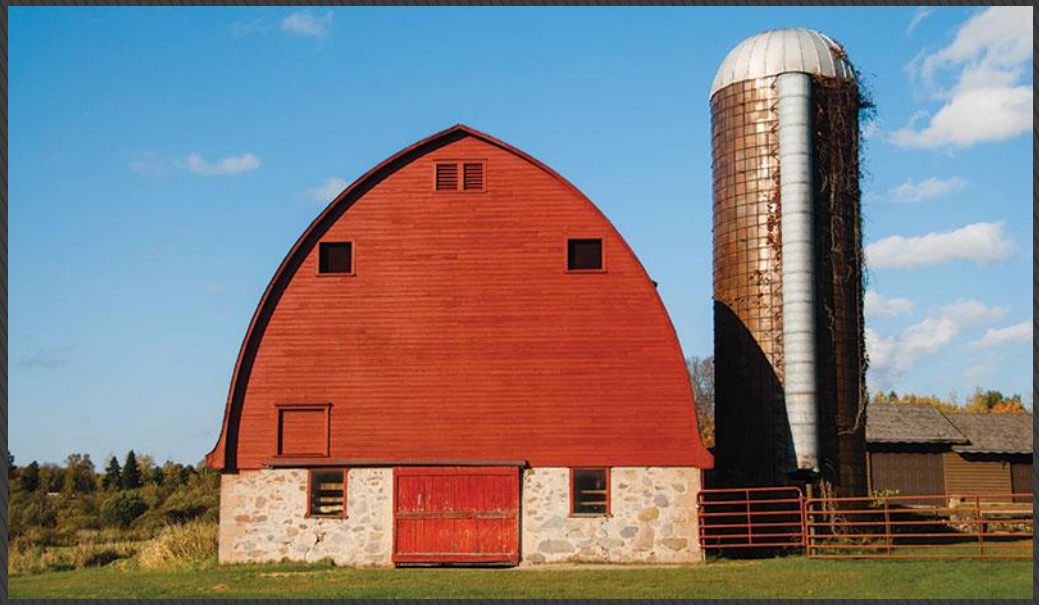
Oral Surgery

Pediatrics

Pediatrics

Periodontics





What is periodontal disease?

- peri” – around
- “dont” – tooth

It can manifest in many forms.

Healthy Mouth

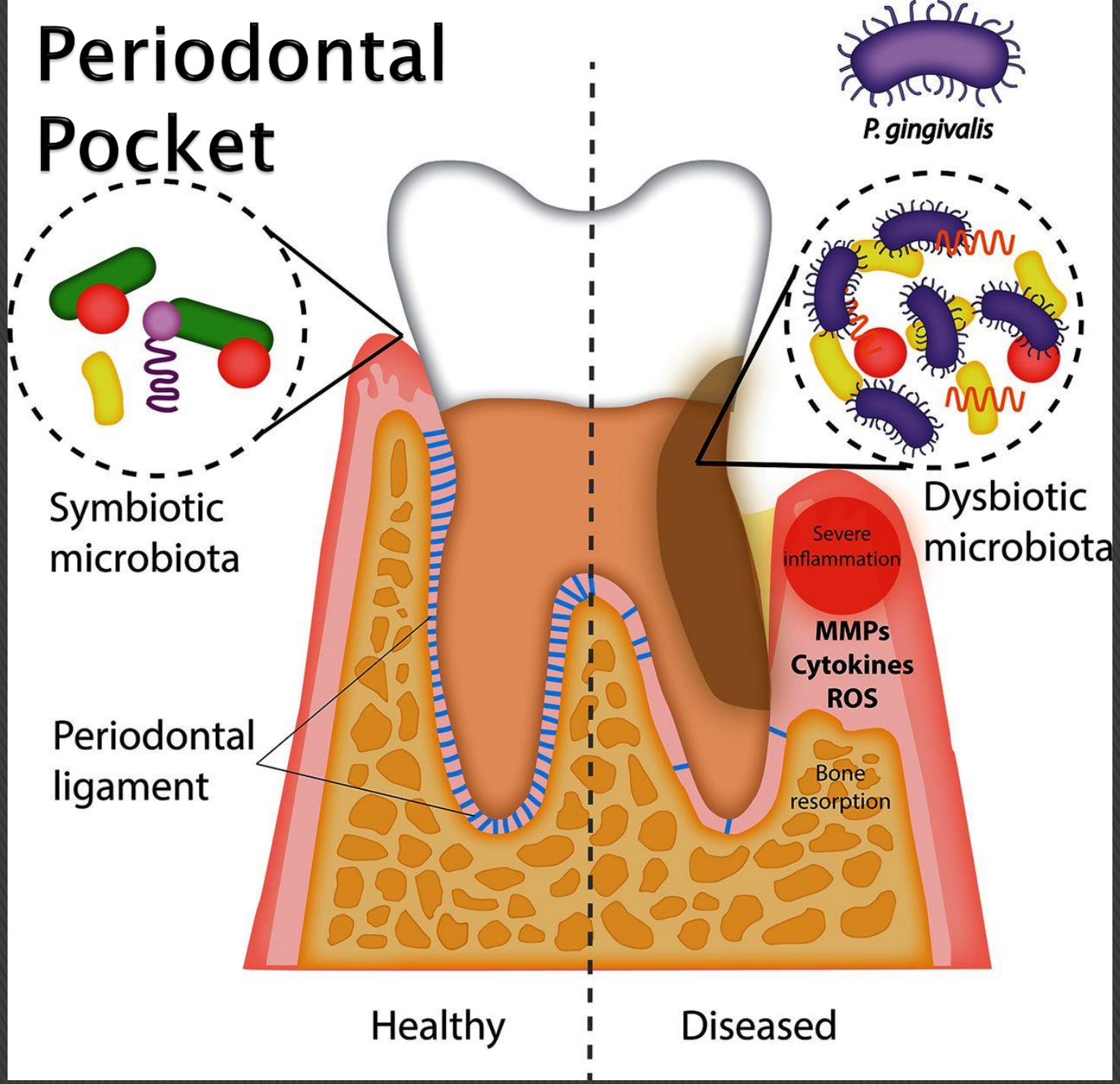
Gingivitis

ANUG

Periodontitis

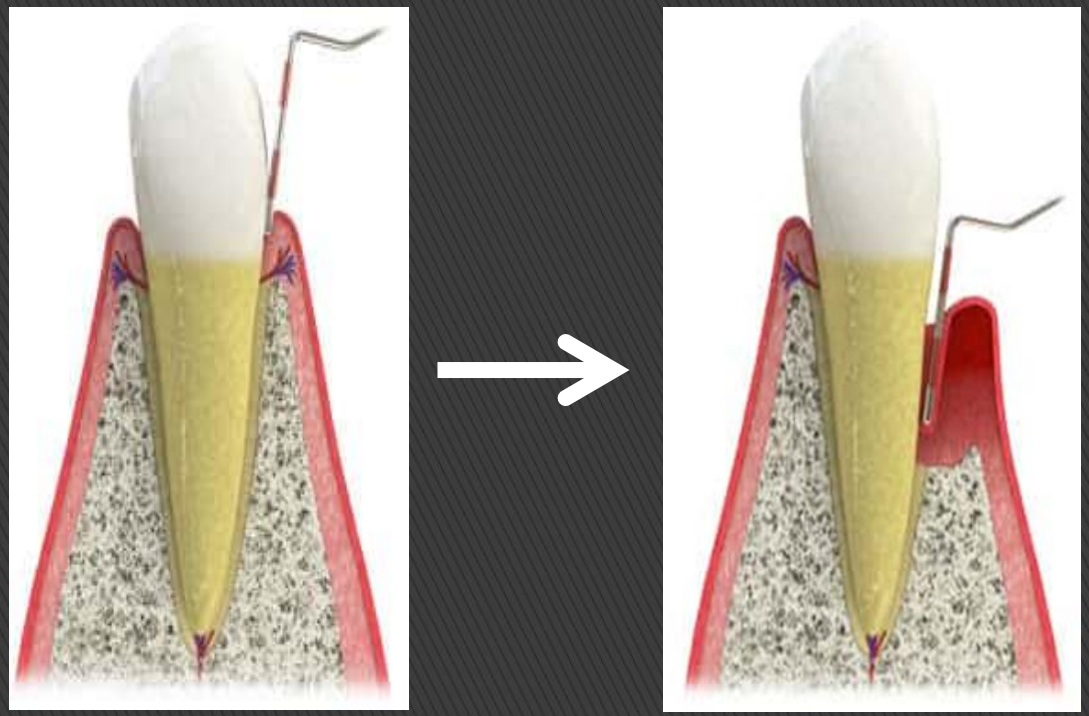
Diagnosing Gingivitis and Periodontitis

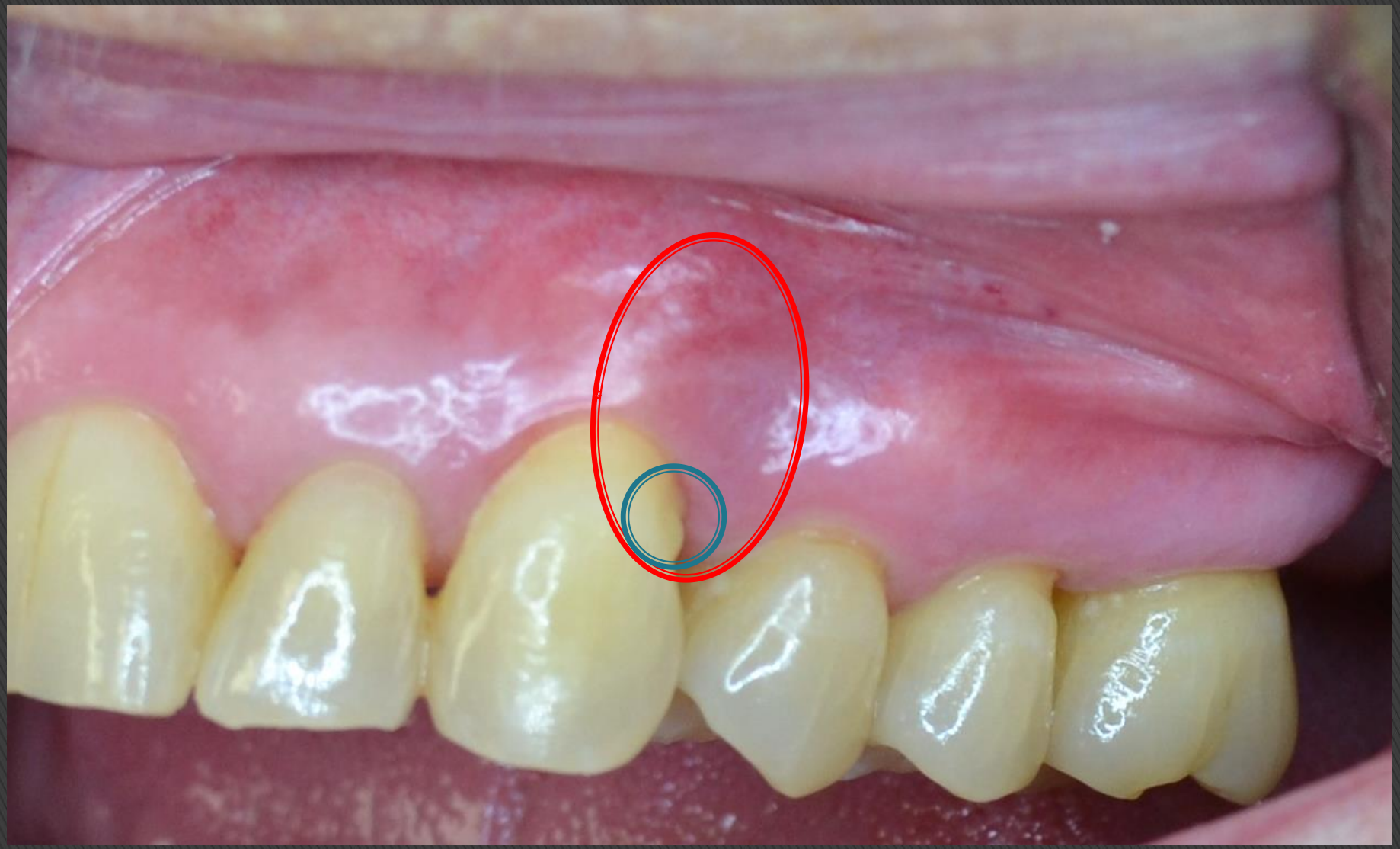
Periodontal Pocket

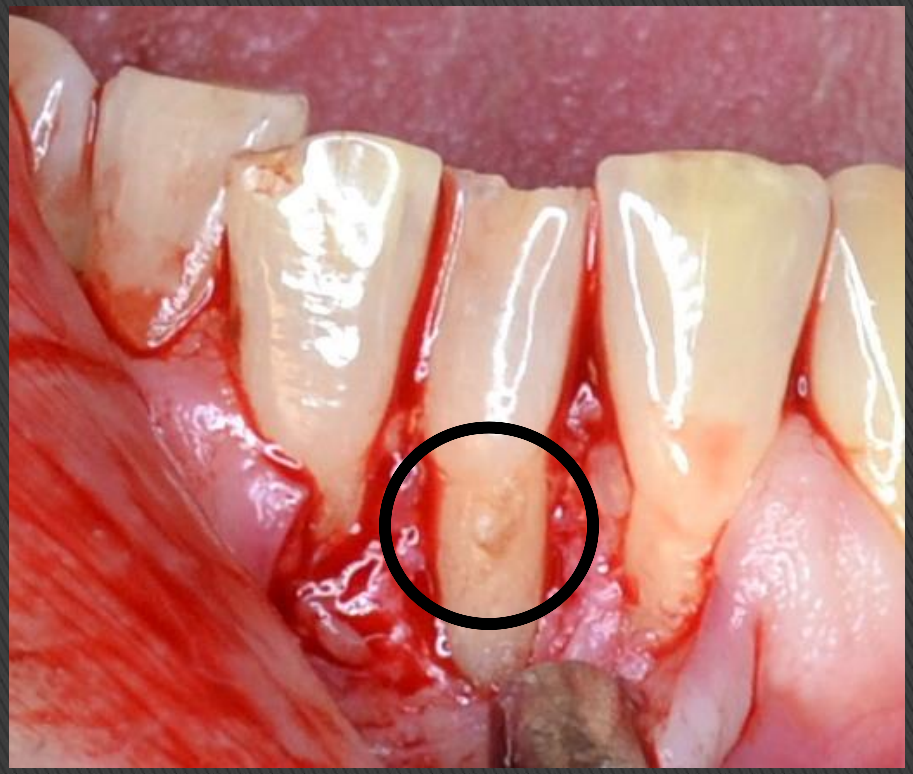


Periodontitis

- Attachment loss
- Pockets of infection
- Bone loss
- Loose teeth
- Malodor
- Bleeding
- Suppuration







Sub gingival calculus

Diagnosing with X-ray

This walks through the door…

Accurate diagnosis followed by treatment

-Removal of hopeless teeth

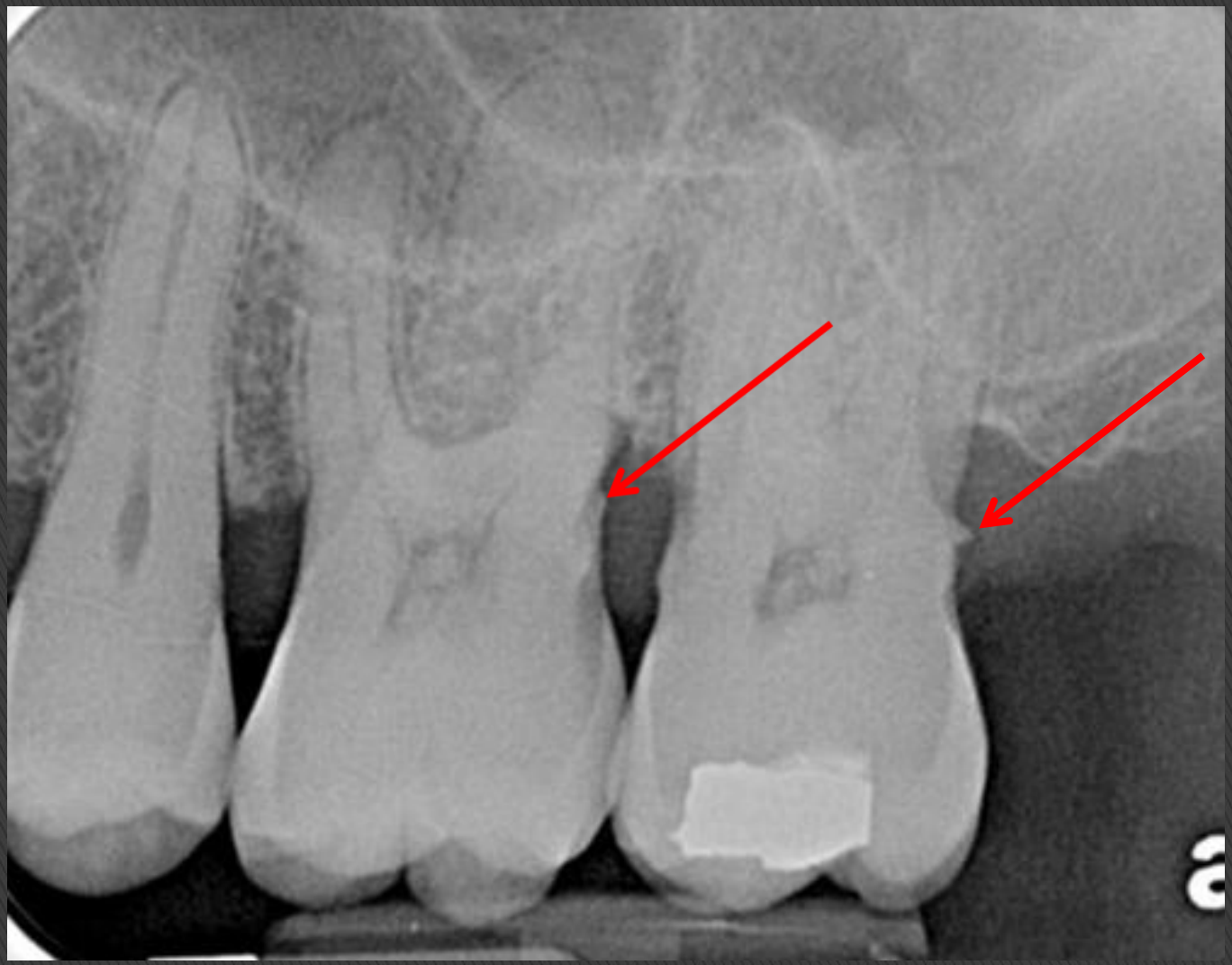
-Scaling and root planing

-Bone grafting

-Tissue grafting

-Complete replacement of tooth with a dental implant (we lost ☹)

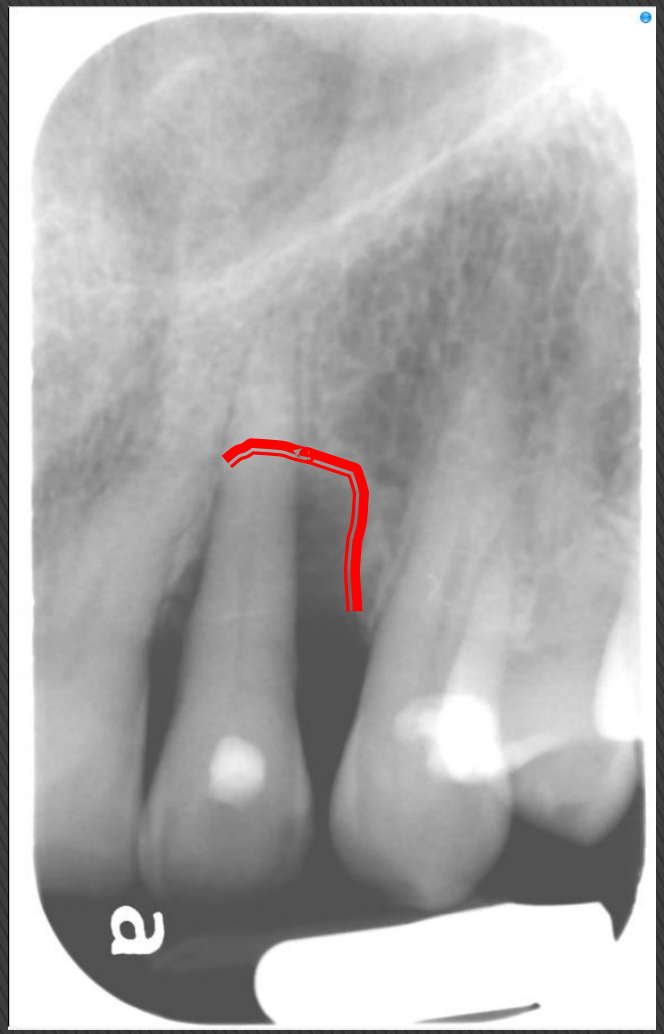
Calculus

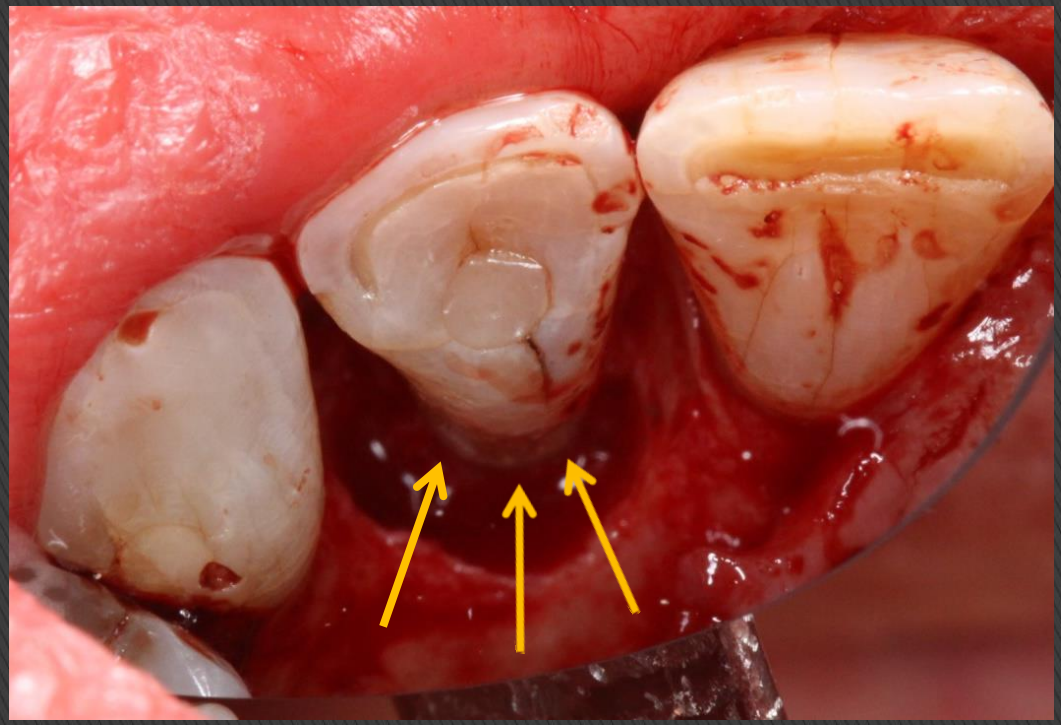


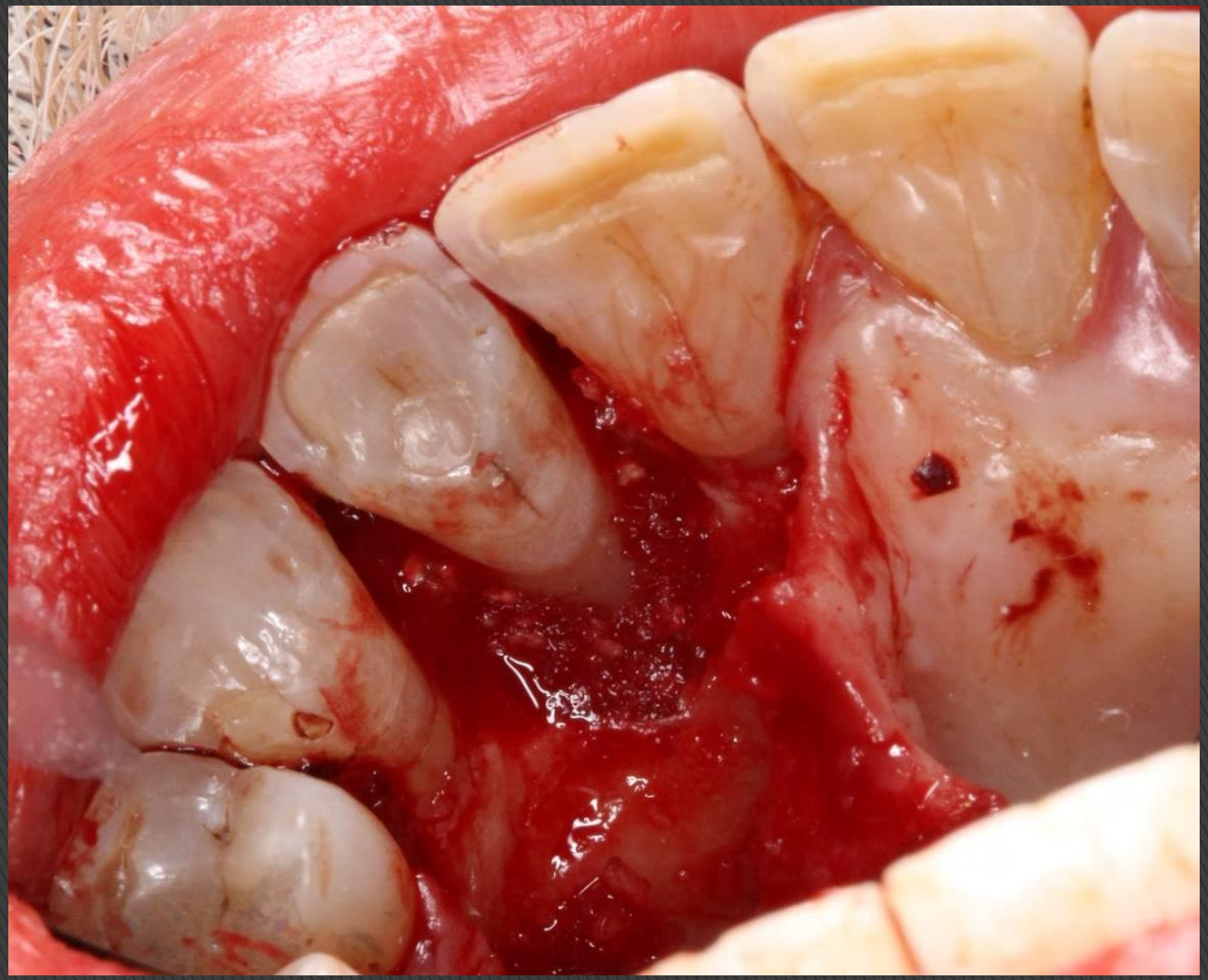
Scaling and Root Planing

ANUG

Bone Graft

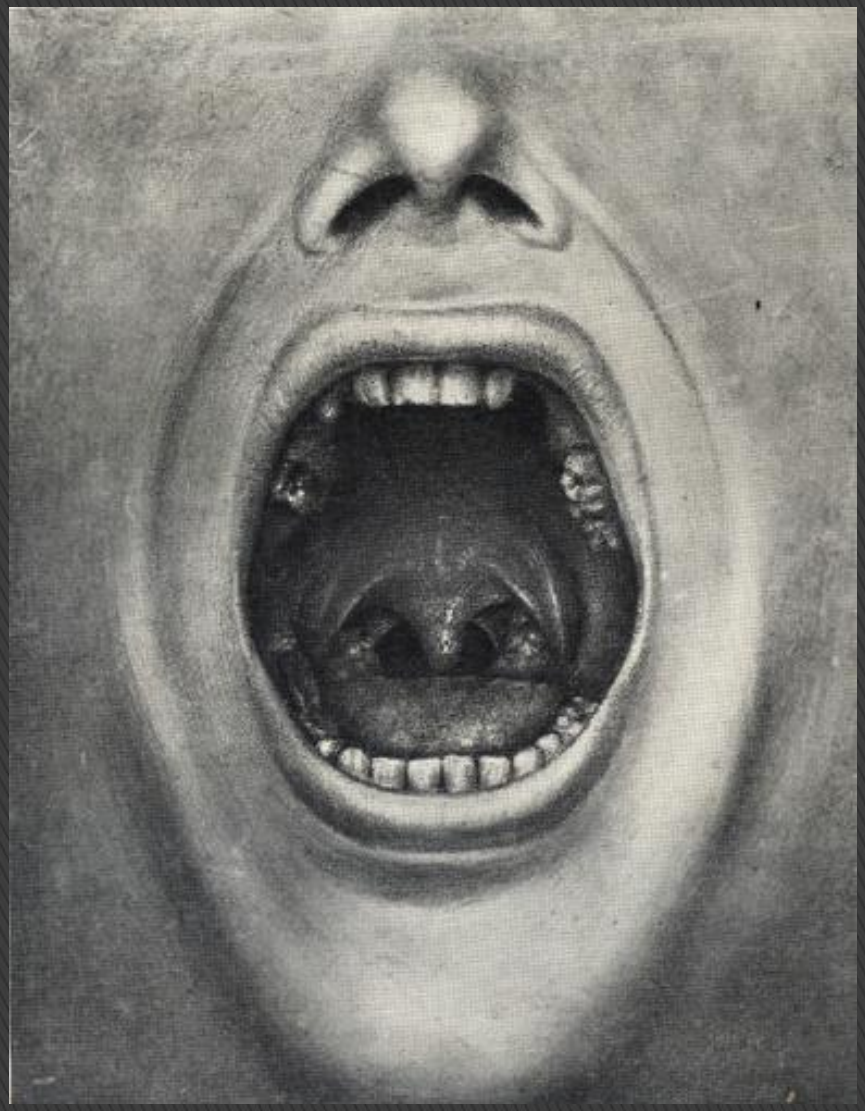






6 months

Is periodontal disease a potential risk factor for systemic diseases?



Proposed systemic relationships:

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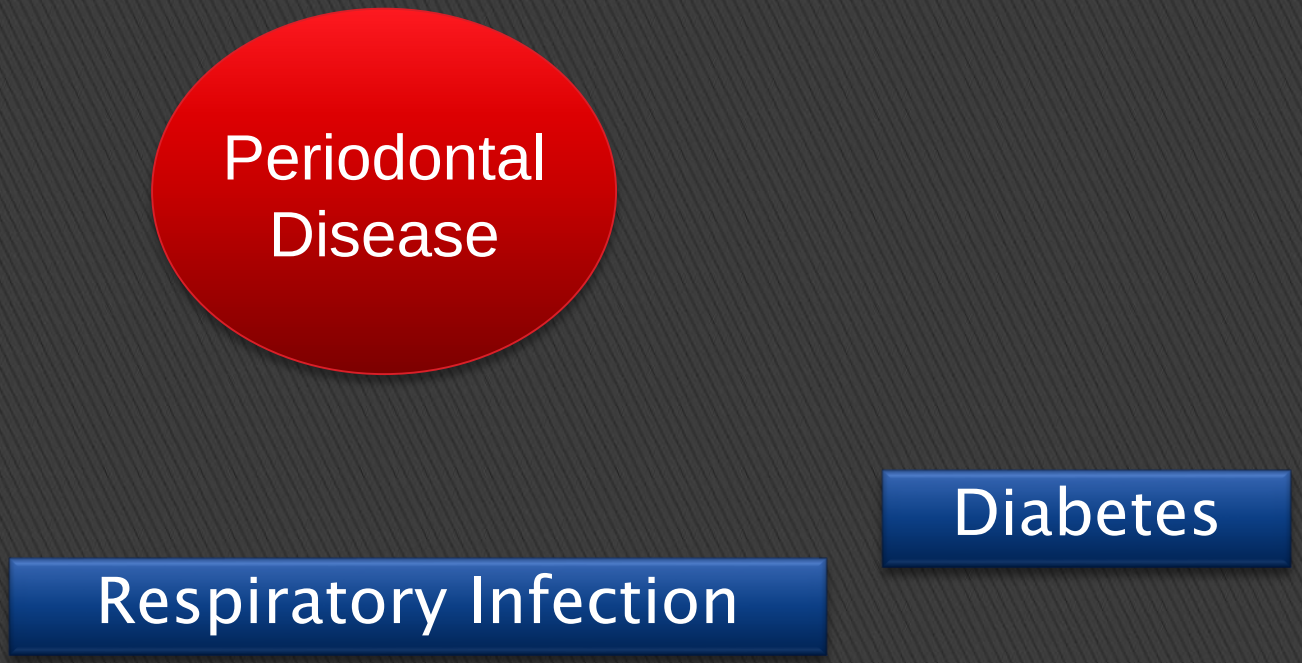

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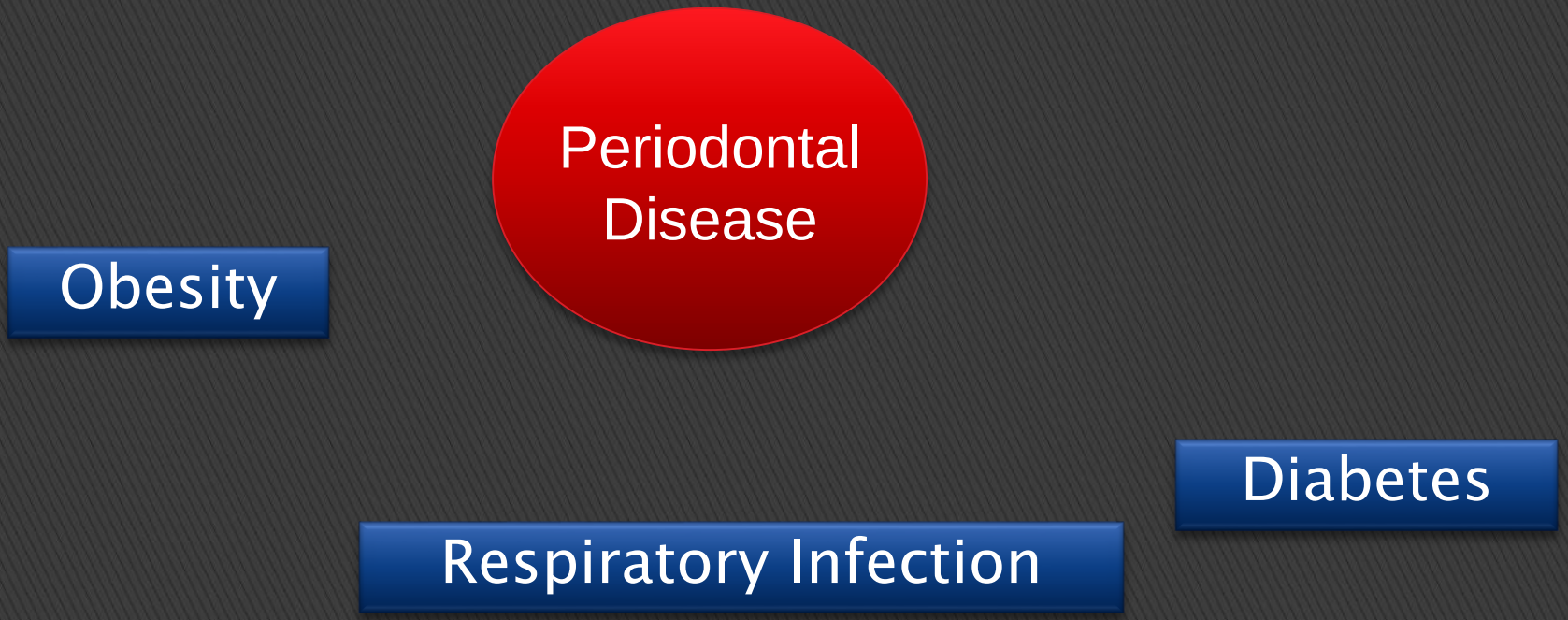

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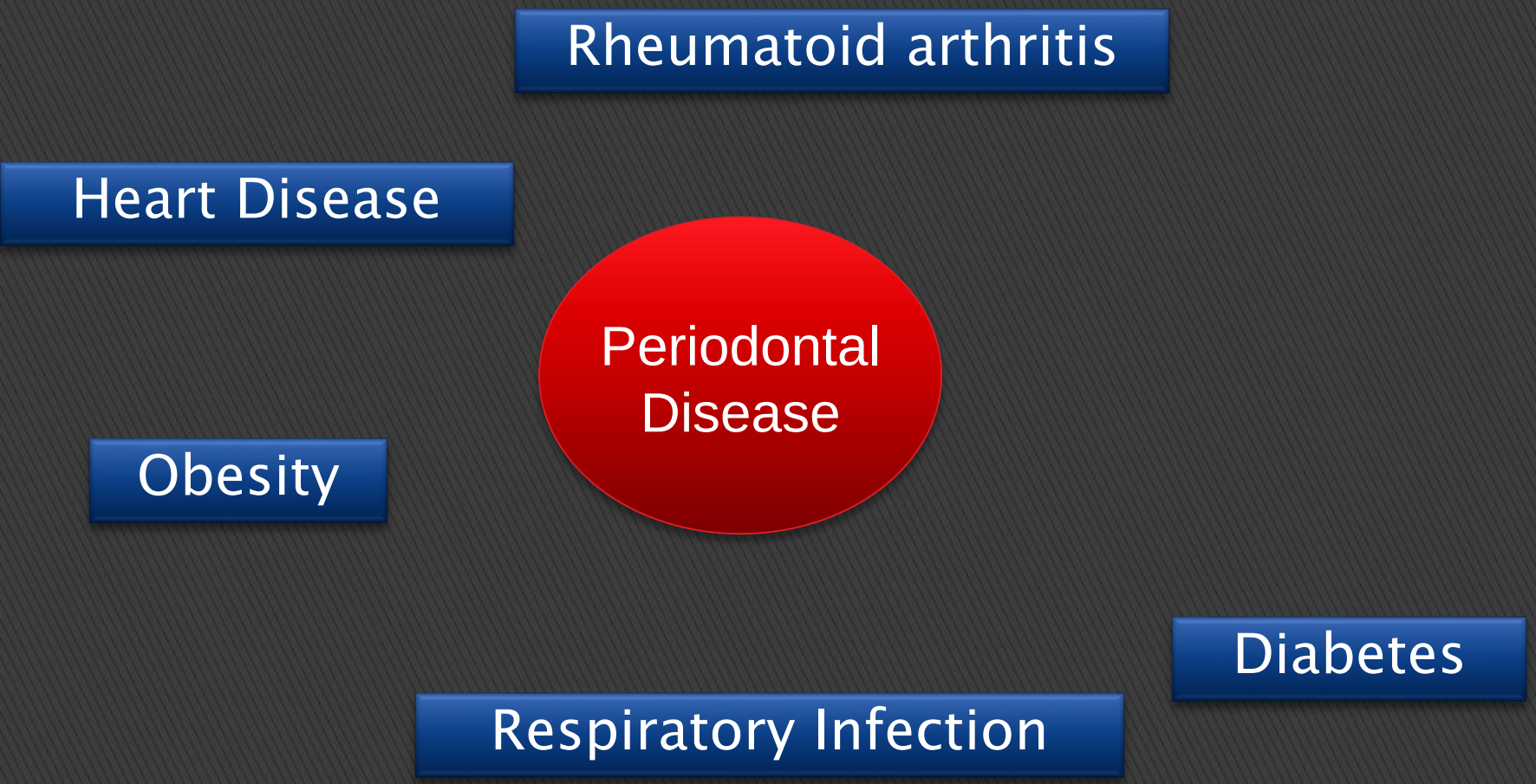
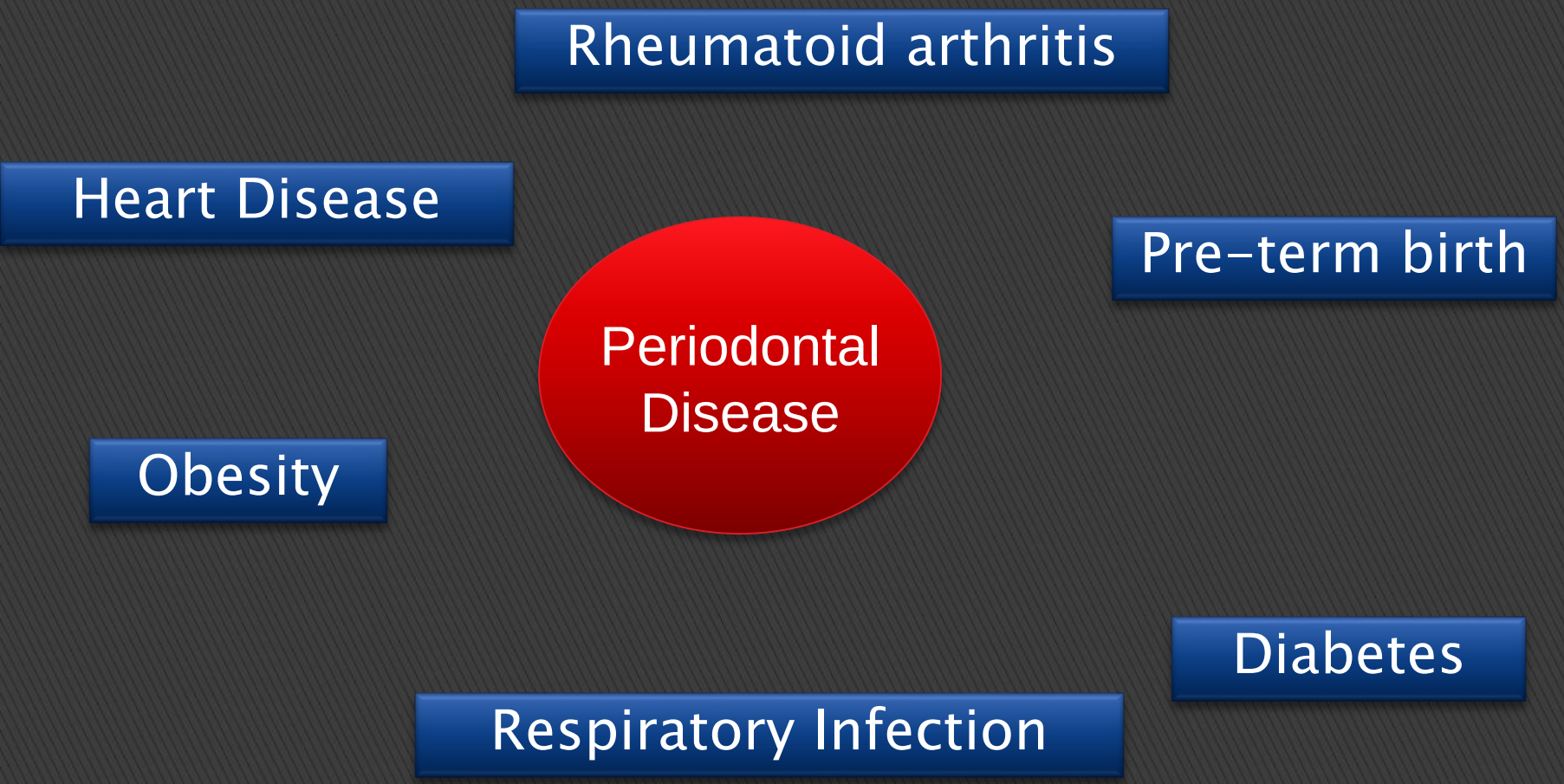
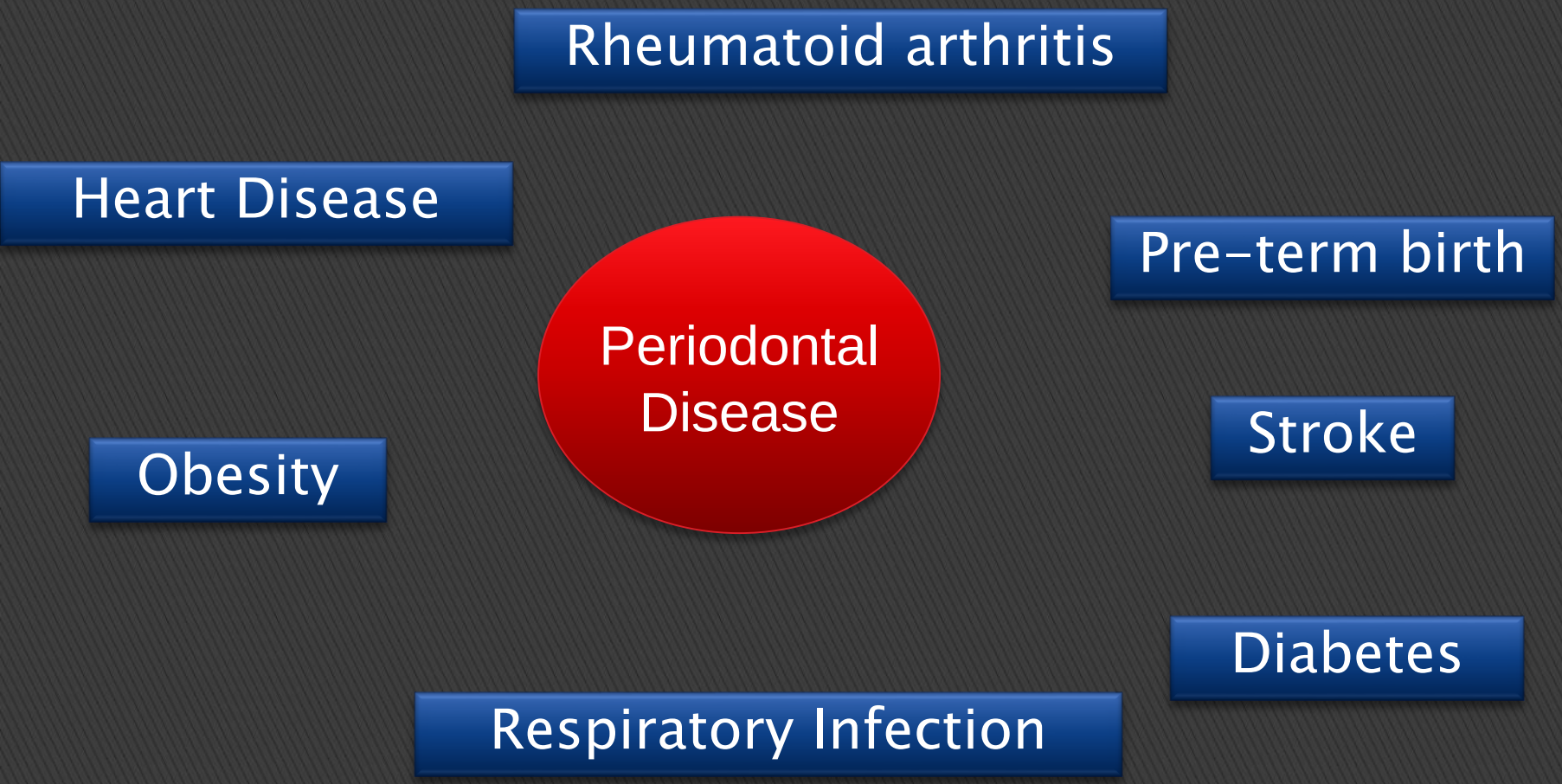
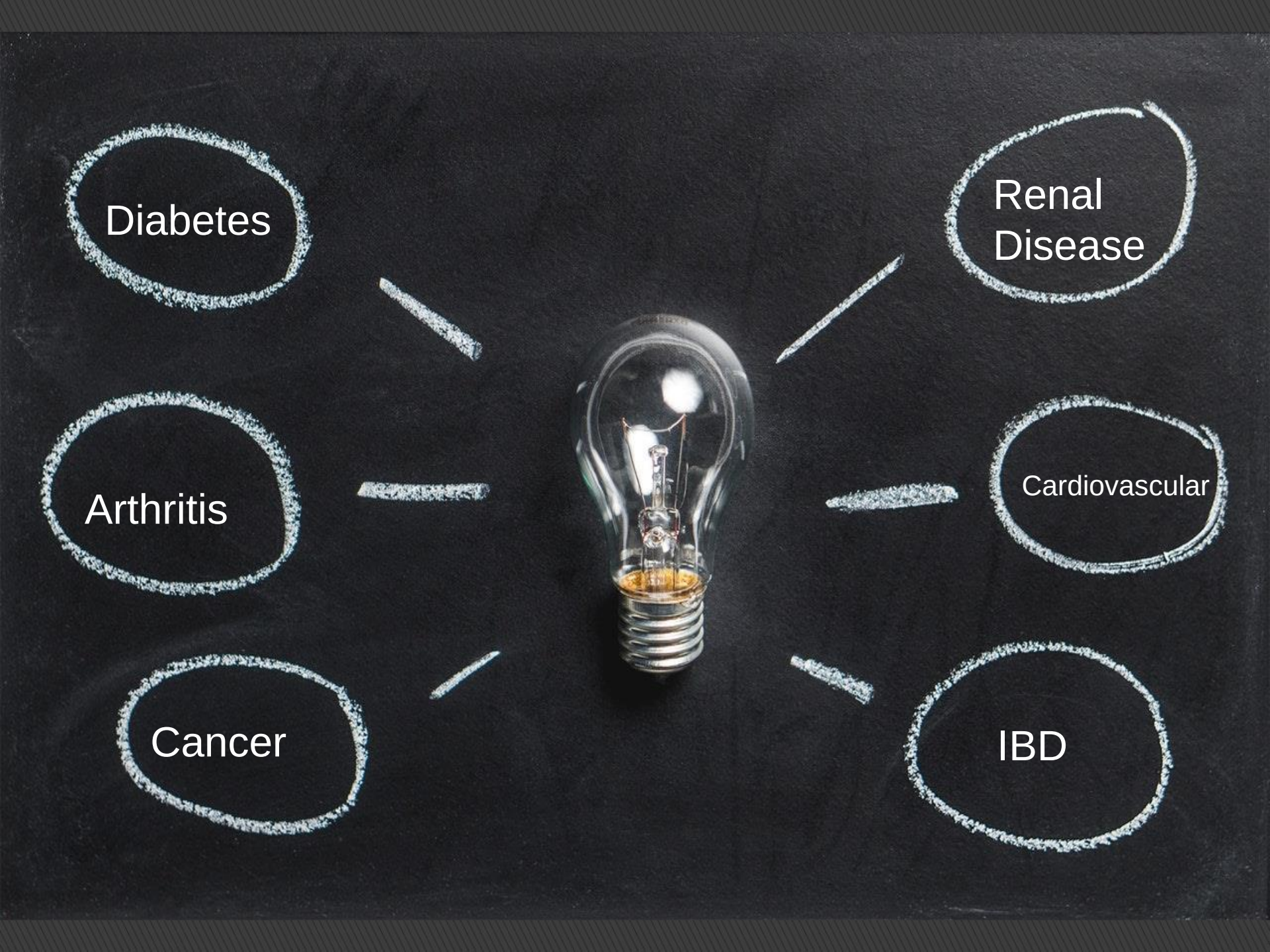


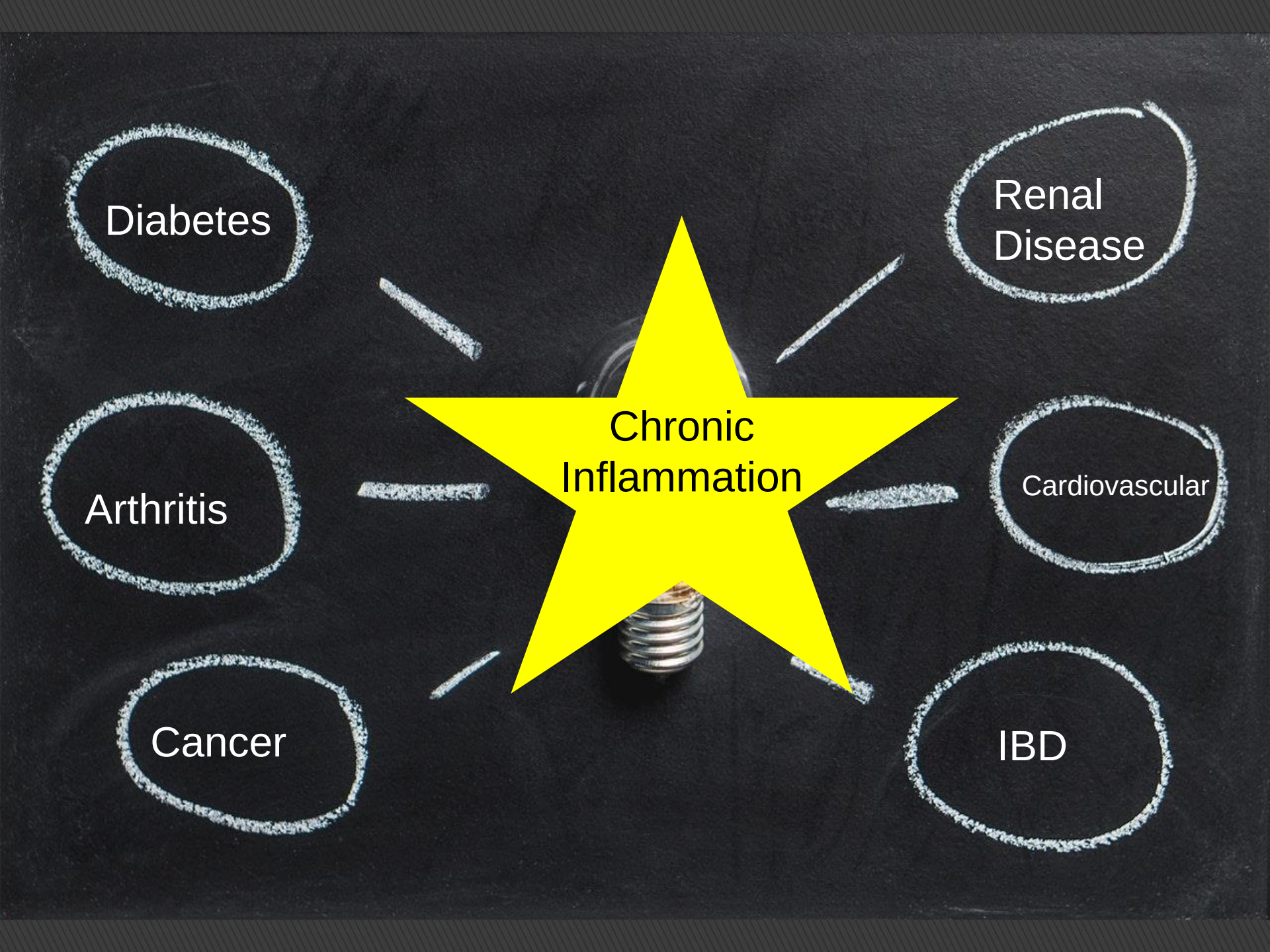
Proposed systemic relationships:

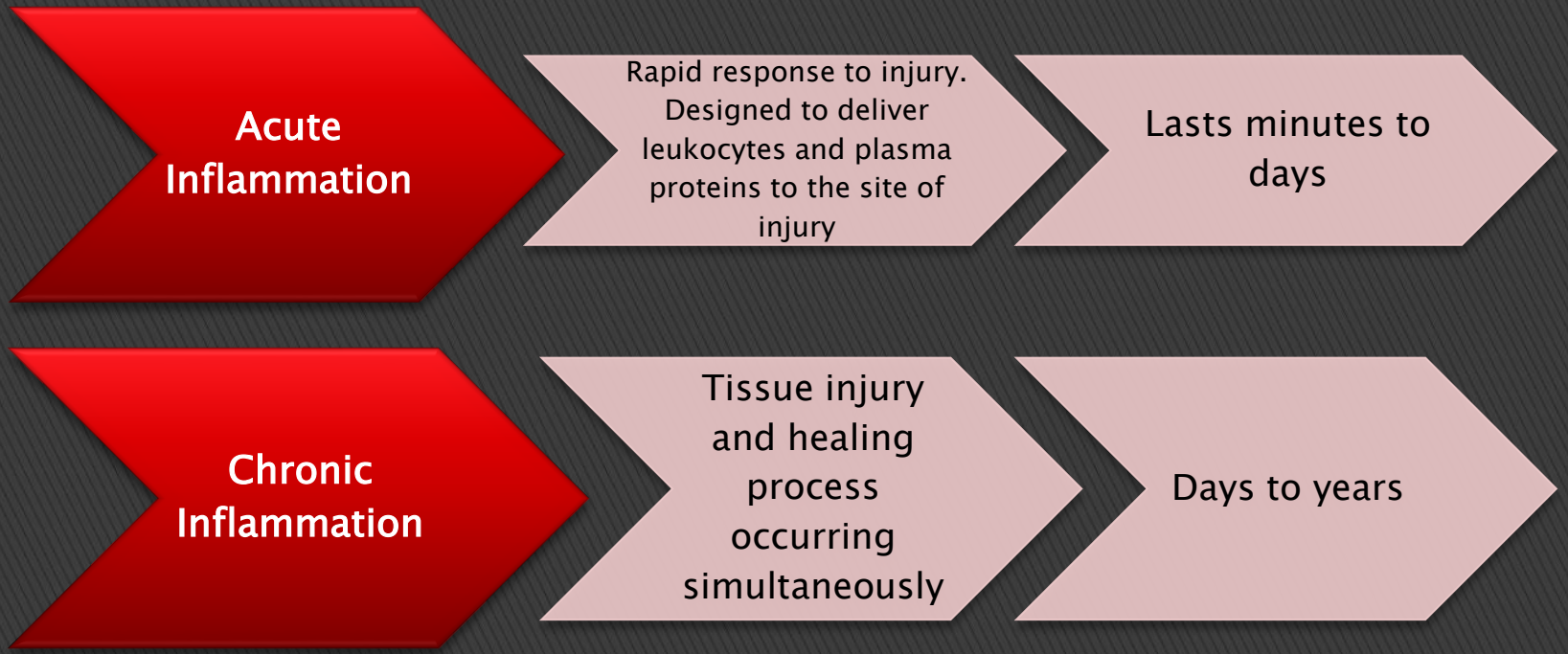


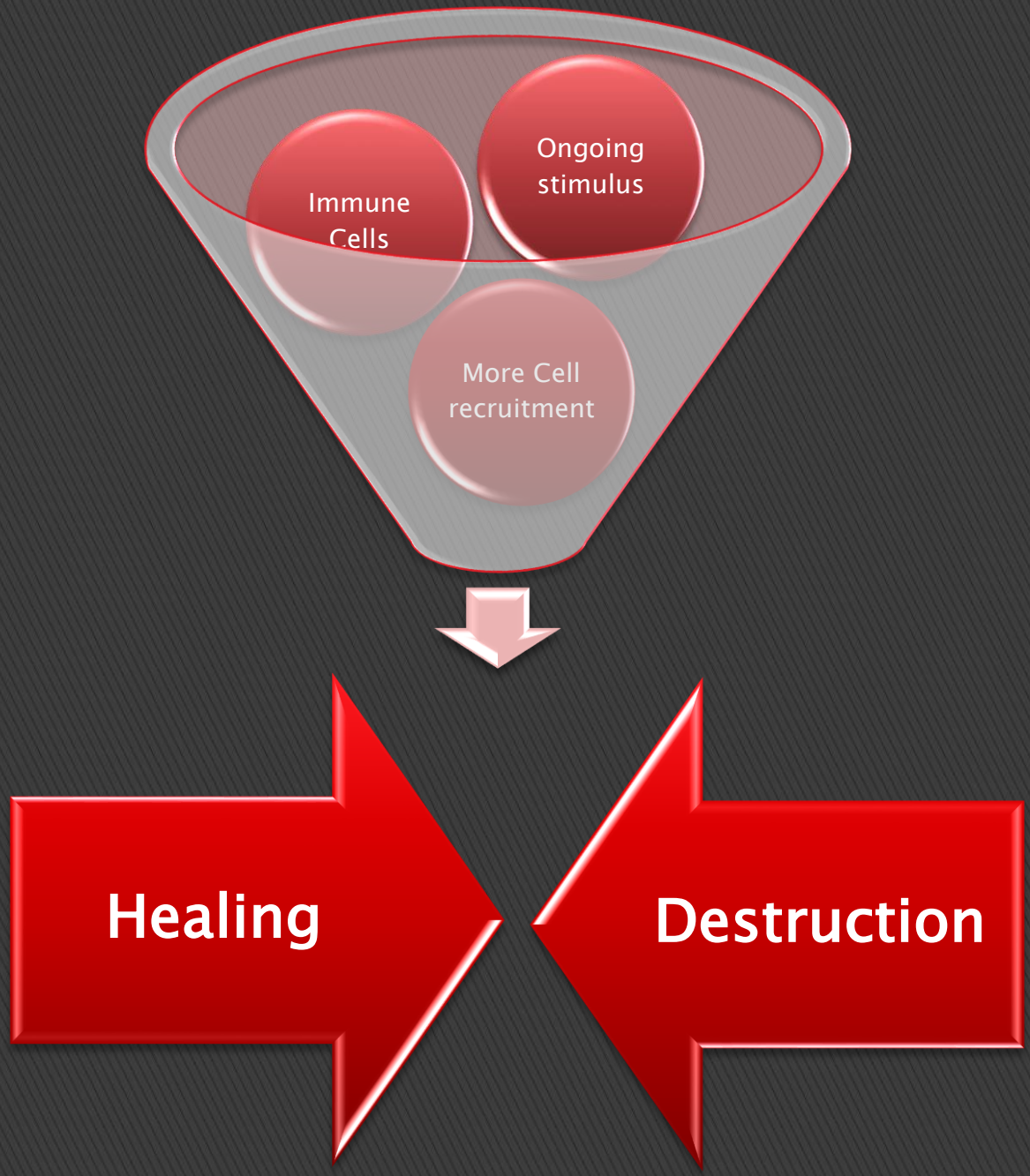
Proposed systemic relationships:

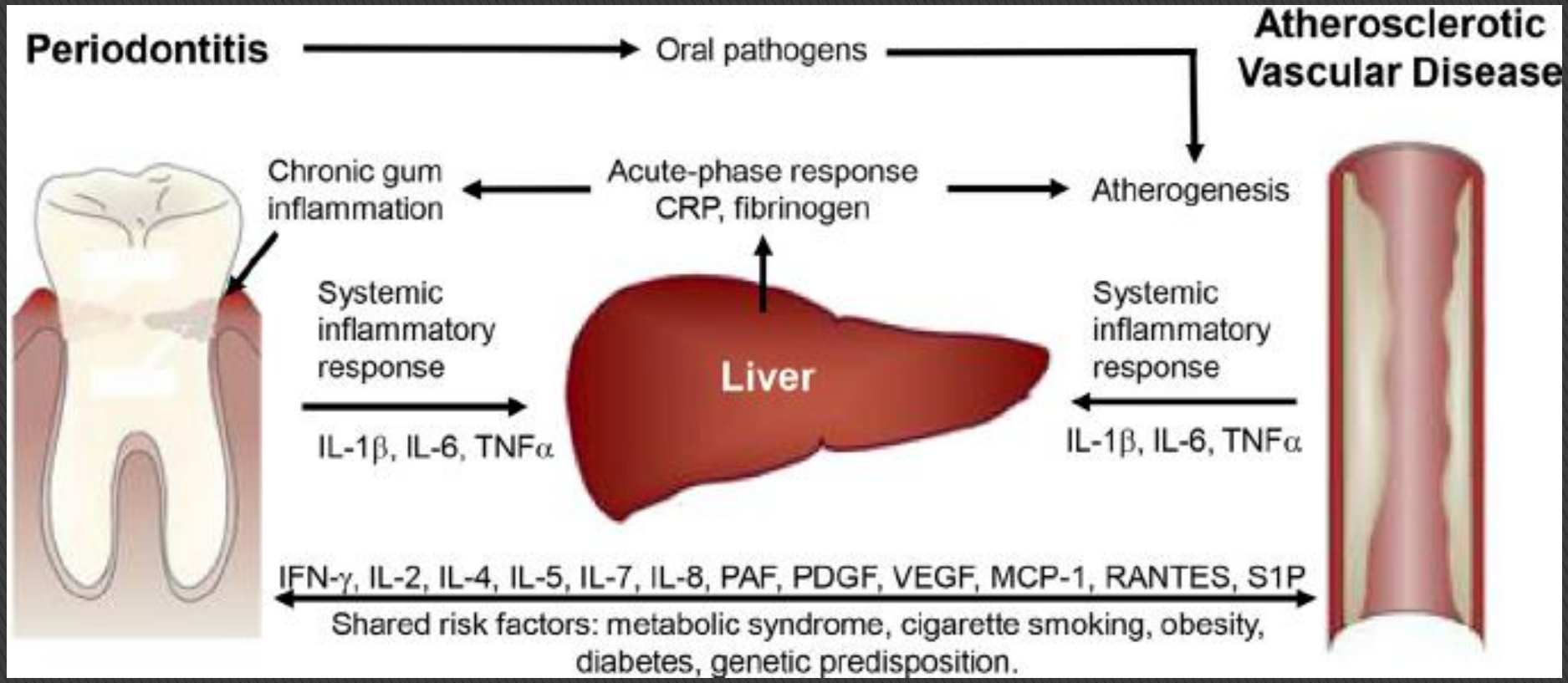


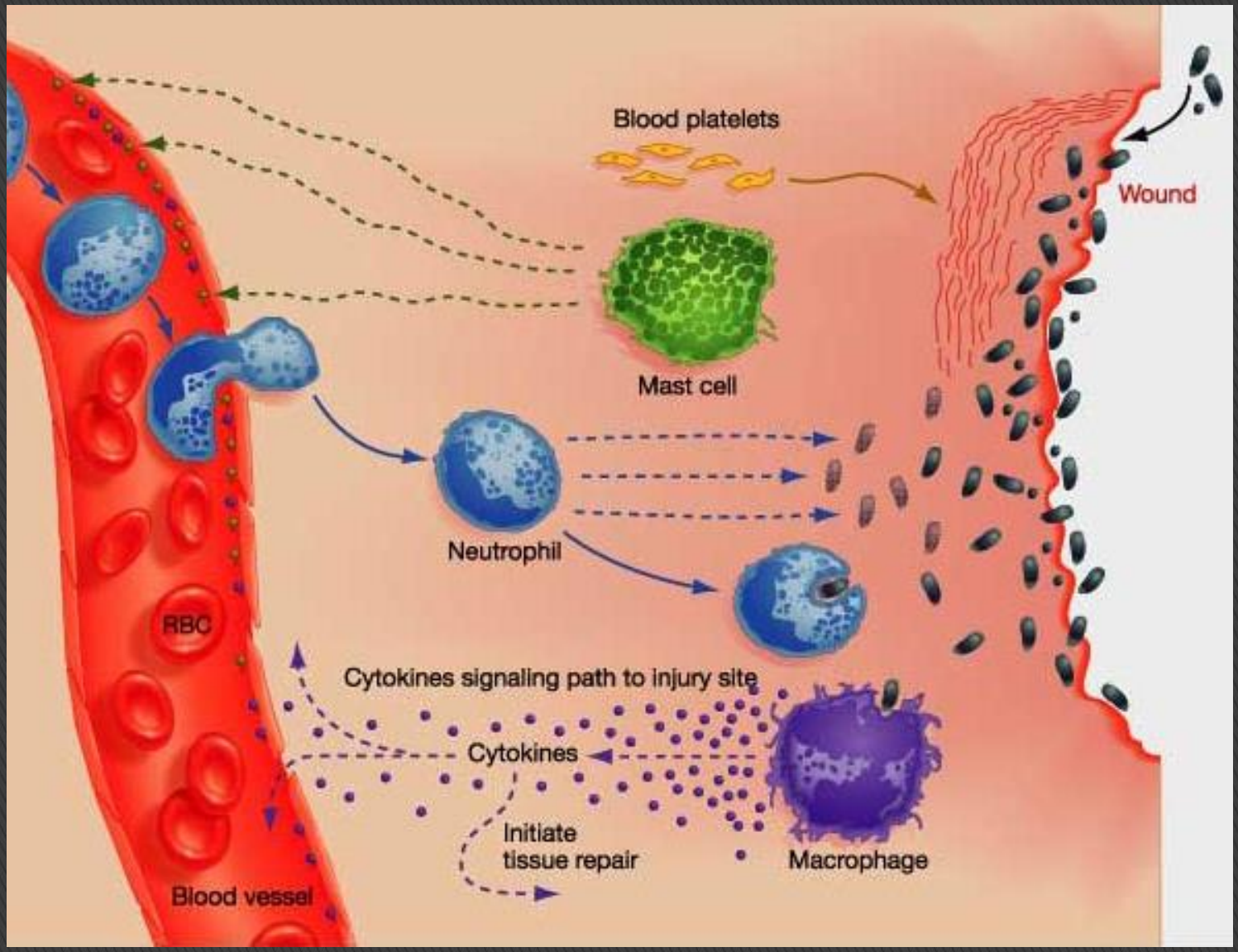

Diabetes
Renal Disease
Arthritis
Cardiovascular
Cancer
IBD


Diabetes
Renal Disease
Chronic Inflammation
Arthritis
Cardiovascular
Cancer
IBD


Acute Inflammation
Rapid response to injury. Designed to deliver leukocytes and plasma proteins to the site of injury
Lasts minutes to days
Chronic Inflammation
Tissue injury and healing process occurring simultaneously
Days to years


Immune Cells
Ongoing stimulus
More Cell recruitment
Healing
Destruction


Periodontitis
Oral pathogens
Atherosclerotic Vascular Disease
Chronic gum inflammation
Acute-phase response CRP, fibrinogen
Atherogenesis
Systemic inflammatory response
IL-1β, IL-6, TNFα
Liver
Systemic inflammatory response
IL-1β, IL-6, TNFα
IFN-γ, IL-2, IL-4, IL-5, IL-7, IL-8, PAF, PDGF, VEGF, MCP-1, RANTES, S1P
Shared risk factors: metabolic syndrome, cigarette smoking, obesity, diabetes, genetic predisposition.


Blood platelets
Wound
Mast cell
Neutrophil
RBC
Cytokines signaling path to injury site
Cytokines
Initiate
tissue repair
Macrophage
Blood vessel


Periodontal Disease
and
Diabetes Mellitus

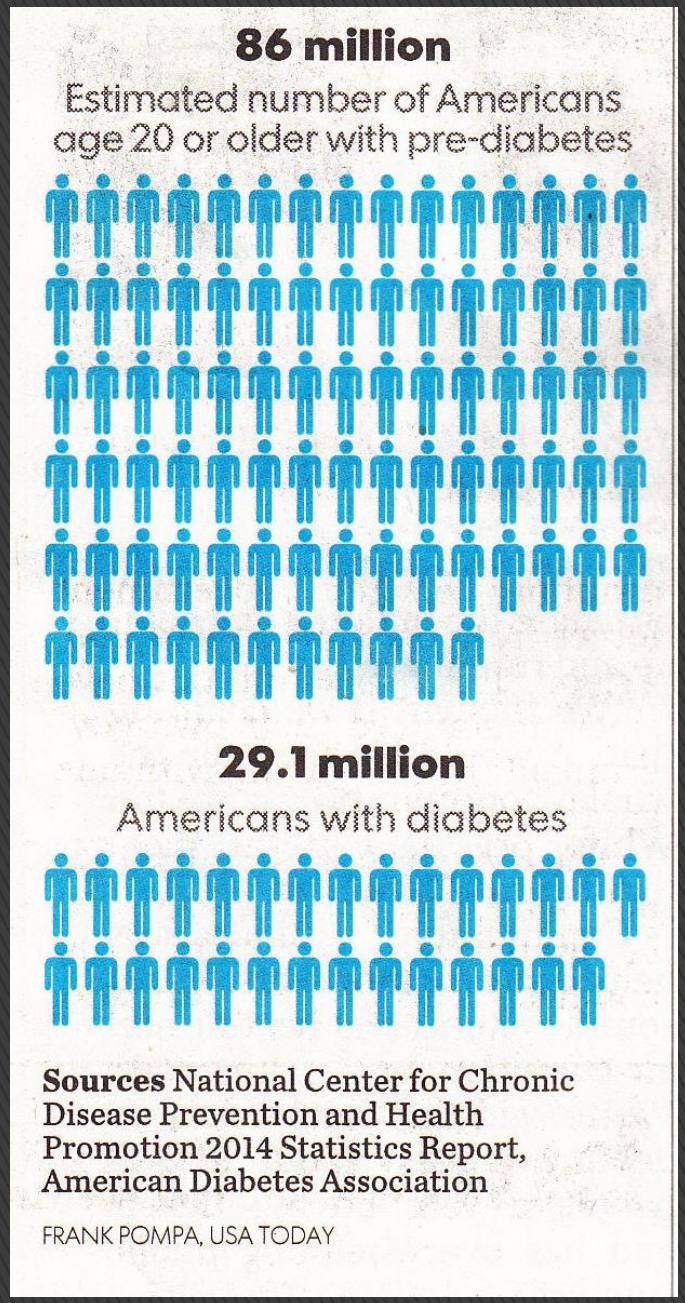
USA TODAY — ARGUS LEADER
MONDAY, SEPTEMBER 15, 2014

NATION

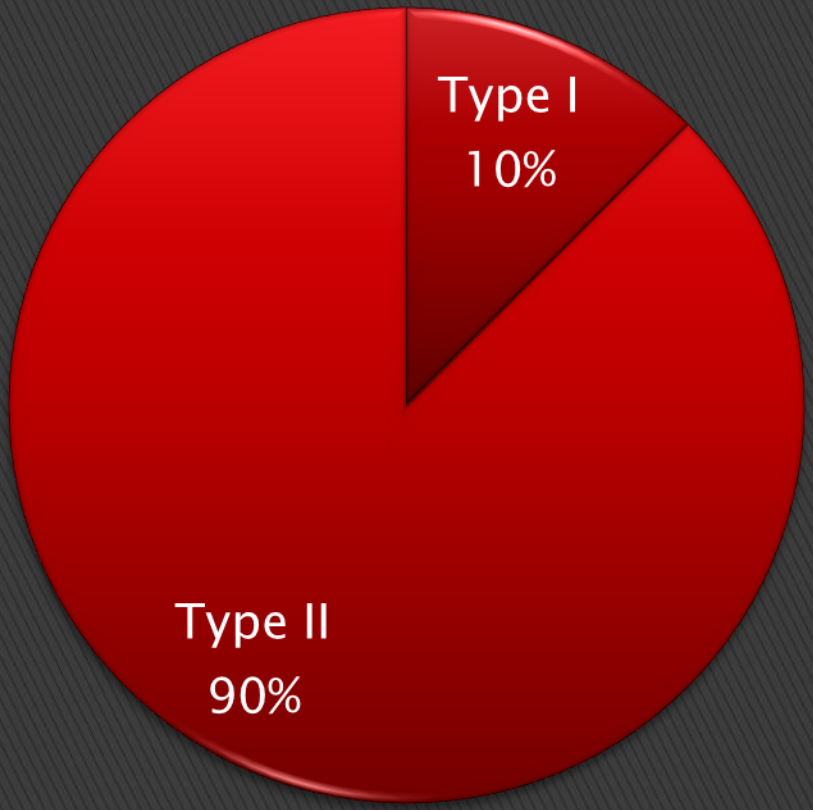
USA moving closer to facing diabetes crisis

PRE-DIABETES PROBLEM

The total cost of diagnosed diabetes hit $245 billion in the United States in 2012. A breakdown:



Type I vs. Type II: Prevalence

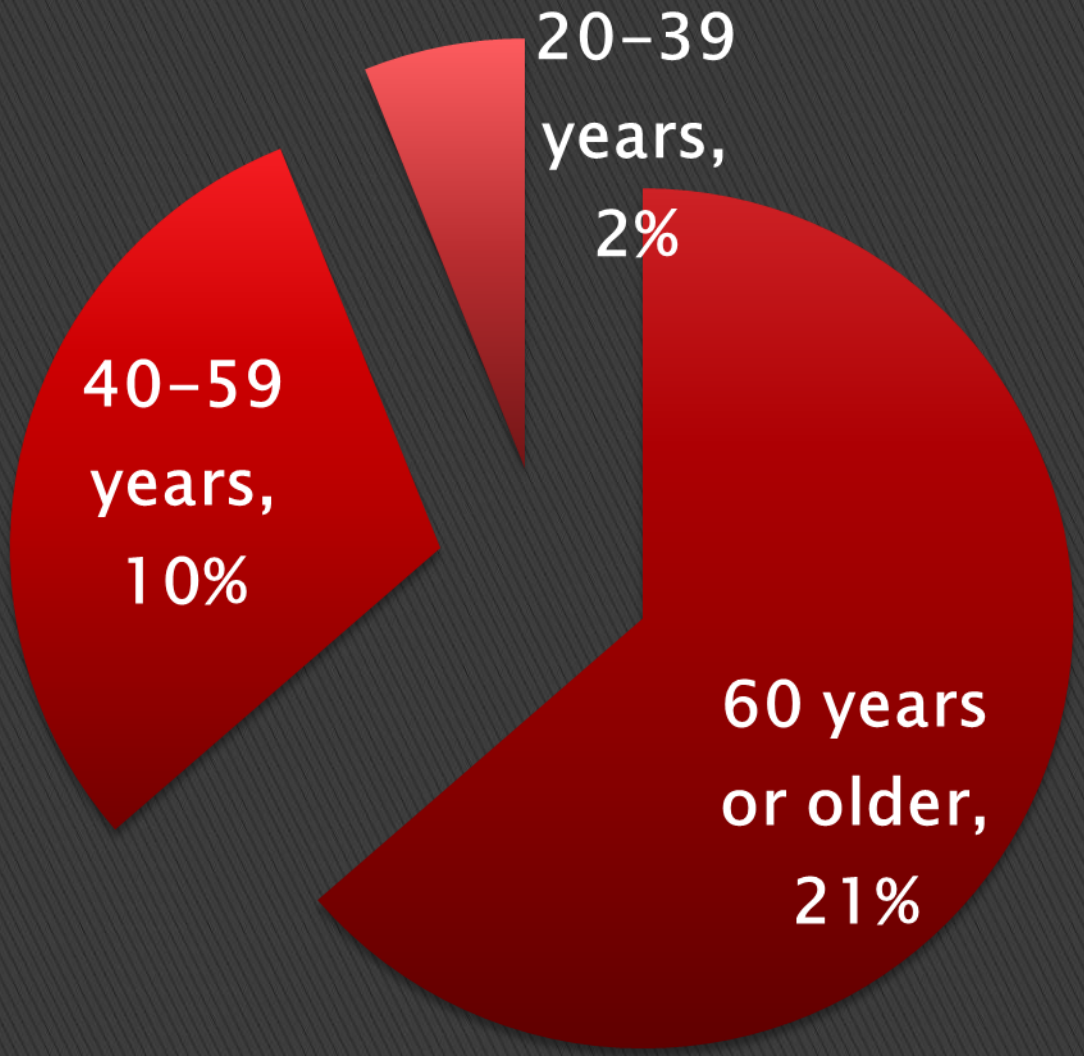


Mealey, 2006

Diabetes and Race?

- 1.7 – 2.2 times more common in Hispanic, Black, Native Americans, Alaska Native, and Asian-American populations

What About Diabetes and Age?



Type I

- Autoimmune destruction of beta cells in pancreas. Total loss of insulin.
- Glucose unable to enter target cells and results in sustained hyperglycemia.
- Exogenous Insulin required

Type II

- Insulin resistance. Not absence
- Patients retain ability to secrete insulin, but production diminishes over time
- Patients may remain undiagnosed for years

Classic Diabetic Complications

1) Retinopathy (Eye Disease)
2) Nephropathy (Kidney Disease)
3) Neuropathy (Nerve Damage)
4) Disease of large blood vessels
   - -Heart Disease
   - -Stroke
   - -Peripheral Vascular Disease
5) Altered Wound Healing
6) Periodontal Disease

Mealy, BL

Proposed mechanisms:

Host inflammatory response is different in diabetics

There is impaired PMN chemotaxis

Mealey, 2006
Leeper, 1985 PMN function is inhibited

Proposed mechanisms:

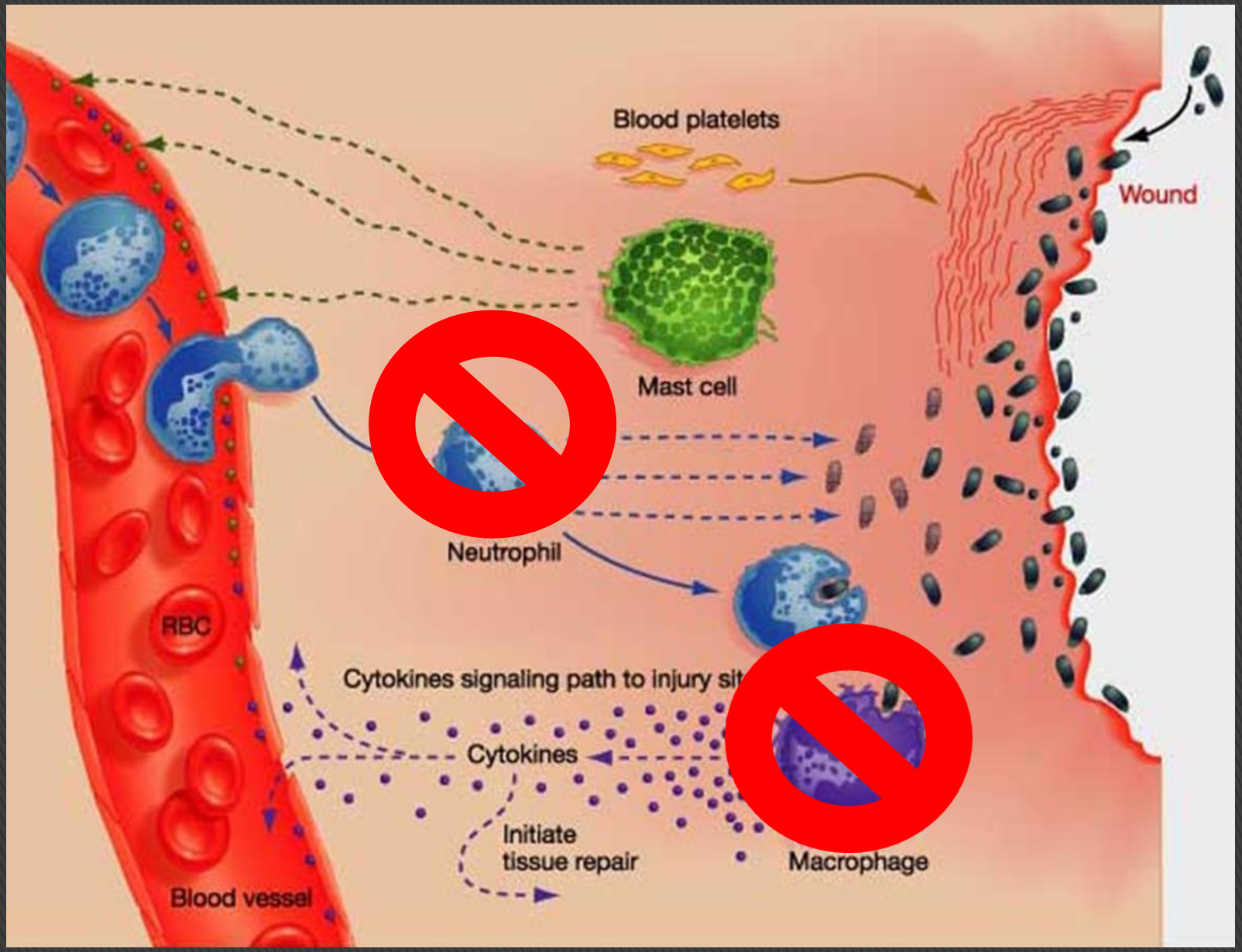
Host inflammatory response is different in diabetics

A1C values greater than 8% had 2x more pro-inflammatory mediators than A1C values below 8% (IL-1beta)

Hyperglycemic state causes inhibition of osteoblast proliferation and collagen production (altered wound healing)

Advanced Glycation End Products (AGEs); proteins that become irreversibly glycated in hyperglycemic states...

Mealey, 2006 A1C value/inflammation
Sakurai, 2003 AGE/RAGE
Leeper, 1985 PMN function is inhibited


Blood platelets
Wound
Mast cell
Neutrophil
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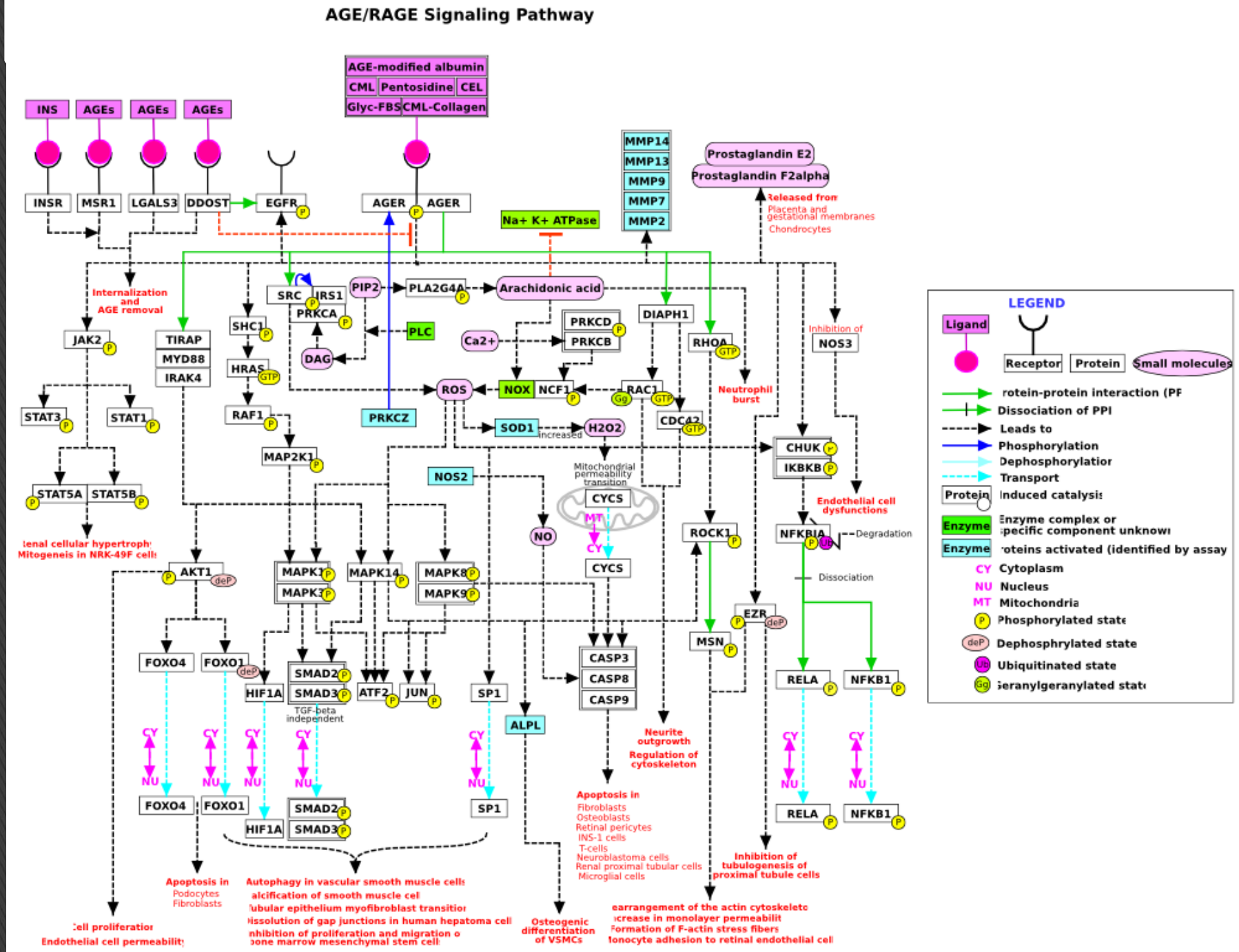
Macrophage
Blood vessel


AGE/RAGE Signaling Pathway
AGE-modified albumin
CML
Pentosidine
CEL
Glyc-FBS
CML-Collagen
INS
AGEs
AGEs
AGEs
INSR
MSR1
LGALS3
DDOST
EGFR
AGER
AGER
Na+ K+ ATPase
MMP14
MMP13
MMP9
MMP7
MMP2
Prostaglandin E2
Prostaglandin F2alpha
Released from
Placenta and gestational membranes
Chondrocytes
Internalization and AGE removal
SRC
IRS1
PRKCA
PIP2
PLA2G4A
Arachidonic acid
PLC
DAG
Ca2+
PRKCD
PRKCB
DIAPH1
RHOA
GTP
Inhibition of
NOS3
JAK2
TIRAP
MYD88
IRAK4
SHC1
HRAS
GTP
RAF1
STAT3
STAT1
PRKCZ
ROS
NOX
NCF1
RAC1
Gg
GTP
Neutrophil burst
SOD1
increased
H2O2
CDC42
GTP
CHUK
IKBKB
MAP2K1
NOS2
Mitochondrial permeability transition
CYCS
MT
CY
CYCS
NO
Endothelial cell dysfunctions
NFKBIA
Ub
Degradation
ROCK1
Dissociation
STAT5A
STAT5B
enal cellular hypertroph
Mitogeneis in NRK-49F cell
AKT1
deP
MAPK1
MAPK3
MAPK14
MAPK8
MAPK9
EZR
deP
MSN
FOXO4
FOXO1
deP
SMAD2
SMAD3
TGF-beta independent
HIF1A
ATF2
JUN
SP1
CASP3
CASP8
CASP9
RELA
NFKB1
CY
NU
ALPL
Neurite outgrowth
Regulation of cytoskeleton
Apoptosis in
Fibroblasts
Osteoblasts
Retinal pericytes
INS-1 cells
T-cells
Neuroblastoma cells
Renal proximal tubular cells
Microglial cells
Inhibition of tubulogenesis of proximal tubule cells
Apoptosis in
Podocytes
Fibroblasts
Autophagy in vascular smooth muscle cells
alcification of smooth muscle cel
ubular epithelium myofibroblast transitio
issolution of gap junctions in human hepatoma cell
nhibition of proliferation and migration o
one marrow mesenchymal stem cell
ell proliferatio
Endothelial cell permeabilit
Osteogenic differentiation of VSMCs
earrangement of the actin cytoskeleto
crease in monolayer permeabilit
ormation of F-actin stress fibers
onocyte adhesion to retinal endothelial cel
LEGEND
Ligand
Receptor
Protein
Small molecules
rotein-protein interaction (PF
Dissociation of PPI
Leads to
Phosphorylation
Dephosphorylatior
Transport
Protein
nduced catalysis
Enzyme
nzyme complex or
pecific component unknow
Enzyme
oteins activated (identified by assay
CY Cytoplasm
NU Nucleus
MT Mitochondria
P Phosphorylated state
deP Dephosphrylated state
Ub Ubiquitinated state
Gg eranylgeranylated stat

Studies of diabetes and periodontal disease

Number of bleeding sites improved as glycemic control improved (Mealey, 2006)

Less inflammation = less bleeding

Number of bleeding sites improved as glycemic control improved (Mealey, 2006)

No statistical significance regarding amount of plaque, bleeding, calculus present did not differ among diabetics and non-diabetics (Khader, 2006)

Diabetics responded differently to the presence of ‘local factors’.

- Number of bleeding sites improved as glycemic control improved (Mealey, 2006)
- No statistical significance regarding amount of plaque, bleeding, calculus present did not differ among diabetics and non-diabetics (Khader, 2006)
- Average pocket pocket depth was signficantly different among diabetics versus non-diabetics (Khader, 2006).

Repair process is inhibited in diabetics

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- No statistical significance regarding amount of plaque, bleeding, calculus present did not differ among diabetics and non-diabetics (Khader, 2006)
- Average pocket pocket depth was signficantly different among diabetics versus non-diabetics (Khader, 2006).
- 6-fold increased risk of worsening glycemic control over time compared to diabetic subjects without periodontal disease (Taylor, 1996).

Greater bacterial load = greater inflammation?

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- No statistical significance regarding amount of plaque, bleeding, calculus present did not differ among diabetics and non-diabetics (Khader, 2006)
- Average pocket pocket depth was signficantly different among diabetics versus non-diabetics (Khader, 2006).
- 6-fold increased risk of worsening glycemic control over time compared to diabetic subjects without periodontal disease (Taylor, 1996).
- Periodontal disease may induce elevated systemic chronic inflammatory state. So when diabetics and non-diabetics are hit with a viral or bacterial infection it is shown that insulin resistance increases. Treating perio may restore insulin sensitivity.

Bacteremia is very common. Especially after brushing. Disease is like an open wound.

Number of bleeding sites improved as glycemic control improved (Mealey, 2006)

No statistical significance regarding amount of plaque, bleeding, calculus present did not differ among diabetics and non-diabetics (Khader, 2006)

Average pocket pocket depth was signficantly different among diabetics versus non-diabetics (Khader, 2006).

6-fold increased risk of worsening glycemic control over time compared to diabetic subjects without periodontal disease (Taylor, 1996).

Periodontal disease may induce elevated systemic chronic inflammatory state. So when diabetics and non-diabetics are hit with a viral or bacterial infection it is shown that insulin resistance increases. Treating perio may restore insulin sensitivity.

Reduction of P. *Gingiivalis* was associated with a significant reduction in levels of A1C (Grossi, 1997)

In a study of Pima Indians of Southern Arizona,15 years of age or older, periodontitis was diagnosed in 60% of diabetics and 36% of non-diabetics.

Nelson RG, et al; Diabetes Care 1990

Periodontal therapy produced a significant reduction in A1c levels

Vergnes, 2010
-Evid based dent

Why do diabetics get worse periodontal disease than non-diabetics?

- A difference in the bacteria?
  - **No, BUT a dysbiosis may be present.**
- Vascular changes?
  - **Yes**
- Changes in the immune response?
  - **Yes**
- Changes in wound healing?
  - **Yes**

Cianciola, 1982

A two-way relationship exists between diabetes and periodontal disease. Specific etiology is hard to support.

A two-way relationship exists between diabetes and periodontal disease. Specific etiology is hard to support.

Periodontal Disease and Rheumatoid Arthritis

Oral Health Basics

- The number one enemy of your teeth is plaque. This is a sticky, colorless substance that forms on teeth, especially above and below the gums. It's similar in consistency to mayonnaise.

  The more you can get rid of plaque itself, the fewer cavities you are likely to get. Removing plaque can help reduce gingivitis and your chances of getting periodontal disease.

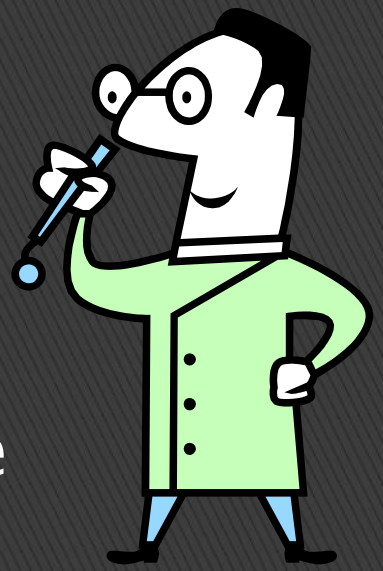
Diabetes and Oral Health

- Keep blood glucose down
- Eat healthy!
- CLEAN your teeth twice per day
  - Use a flouride toothpaste
  - Avoid sugary or starchy snacks prior to bed
- Use floss, interproximal brush, or a waterpik to clean between your teeth.
- Take dentures out at night
- See your dentist (or friendly neighborhood periodontist) at least twice per year to monitor for any signs of periodontal disease.

Importance of Professional Dental Care

- Visit your dentist twice a year

- Some people with periodontal disease will need to have their teeth Professionally cleaned every three to four months.



Any brush works, even manual ones. Just use it twice per day and make sure the bristles are soft!

Dry Mouth

Possible Causes:

Medication side-effects

- About 400 medications have dry mouth as a side effect: Treatments for high blood pressure, allergies, and depression, and many other conditions

Chemotherapy or radiation treatments

Systemic disease – Sjrögren's

Dry Mouth – Management

Physician may change medications.
Drink extra water.
Protect teeth with fluoride.
Use a saliva substitute.

Avoid:

- Sugary snacks or drinks
- Beverages with caffeine or alcohol
- Mouthwashes with alcohol
- Tobacco

Conclusion

Diabetics with marginal or poor control should be screened and treated for periodontal disease to help them keep their teeth, but also to very possibly improve their glycemic control.