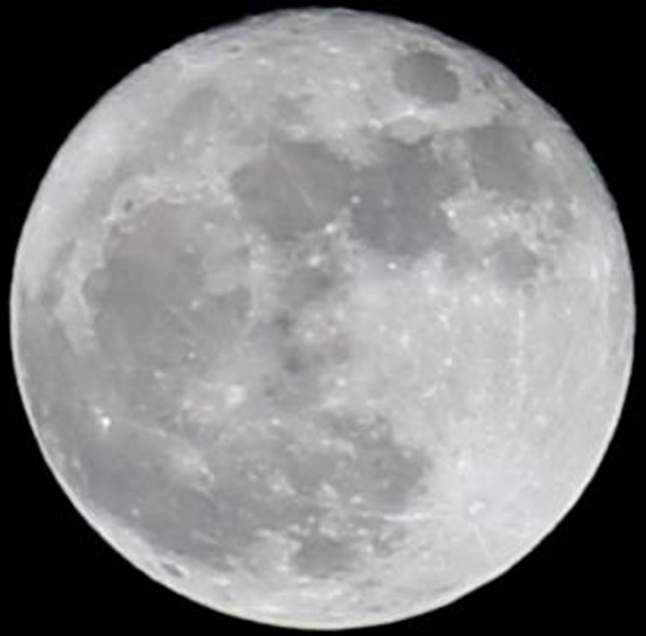
Wound Healing and Repair



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Clinical Relevance

- Understanding the literature
- Various products and therapeutics – Rx and OTC
  - Client Questions
  - New products
- Surgery Consults
  - Bandaging

Studying This Topic

- Once you have studied the basic structure and function:
  - Epithelium - keratinization
  - Dermis – collagen
  - BMZ
  - Vessels and nerves
  - Adhesion molecules
  - Cytokines
- NOW JUST PUT IT IN MOTION!

Let's Build

- Construction
  - Numerous parts
  - Simultaneous
  - Overlap
- What about your practice???

The Building/Healing Process – Scarring?

- Multiple redundancies, rapid inflammation
- For the dermis - scar formation is the end-point
- Inflammation required in order for this to happen rapidly
- Scar formation may be the price we have to pay!

Is it Wound Healing or Repair?

- Def: Repair

  "To restore to a sound or healthy state"

  (fibroblasts)

  Vs.

- Def: Heal

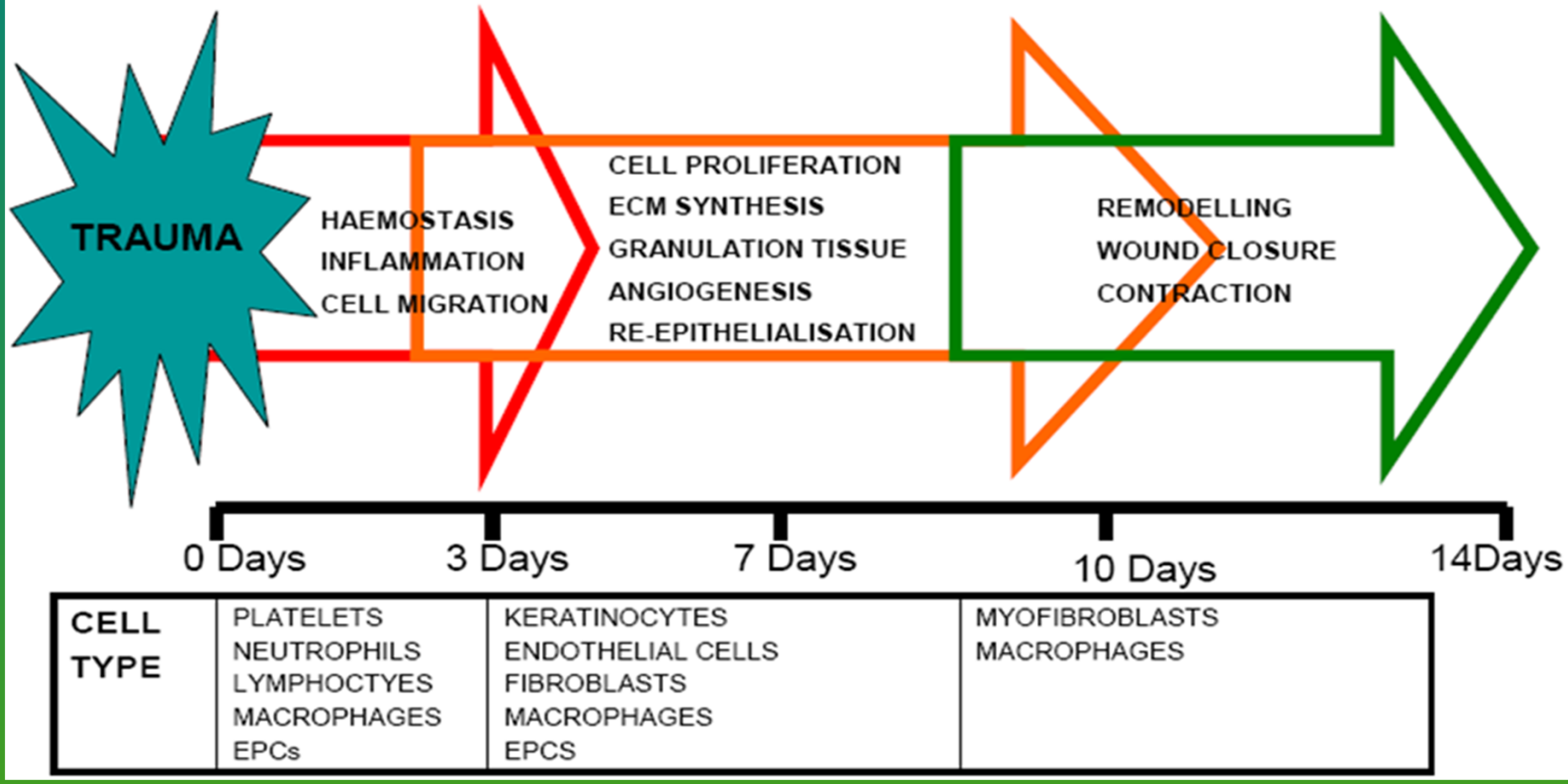
  "To restore to original purity or integrity"

  (keratinocytes & endothelia)

Introduction

- Four Phases (or is it three???)
  - Coagulation/Inflammation
  - Tissue Formation
  - Scarring/Remodeling

Stages of Normal Cutaneous Wound Healing

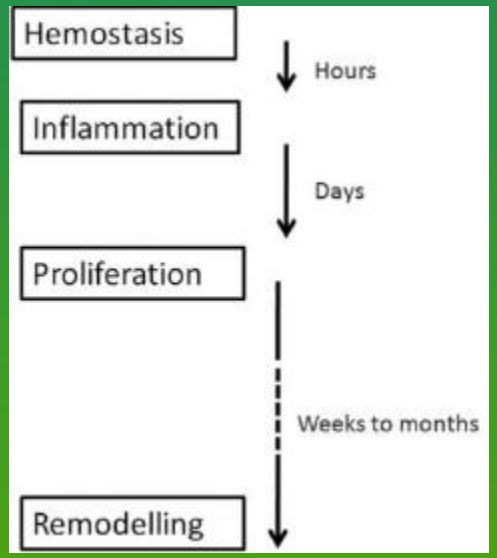


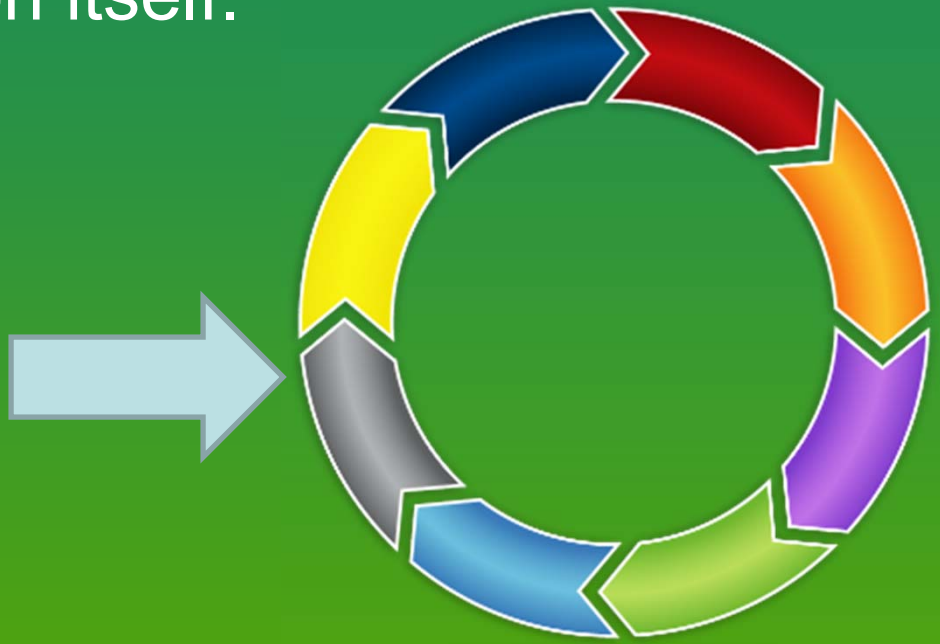
CELL TYPE	PLATELETS NEUTROPHILS LYMPHOCTYES MACROPHAGES EPCs	KERATINOCYTES ENDOTHELIAL CELLS FIBROBLASTS MACROPHAGES EPCS	MYOFIBROBLASTS MACROPHAGES

- EPCS = Endothelial Progenitor Cells
- **From www.intechopen.com**

When things go wrong..

- Normal wound healing has a mostly linear progression through the phases
- Chronic wounds go through the processes in a jumbled more cyclic fashion with the cycle continuing to feed on itself.

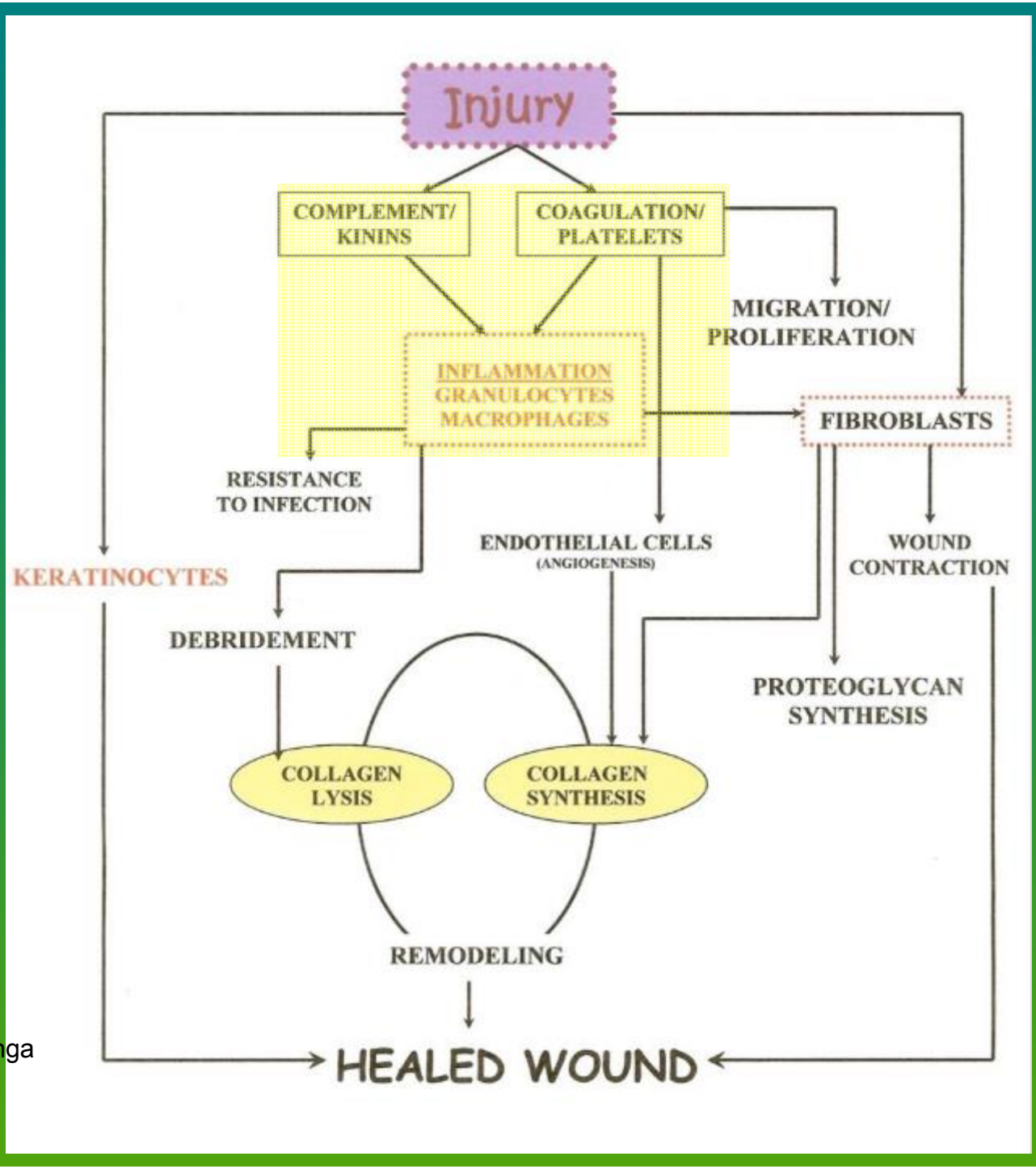




Introduction

- Coagulation/Inflammation
  - Platelets
  - Neutrophils
  - Macrophages
  - Mast Cell
- Tissue Formation
  - Macrophages
  - Fibroblasts
  - Keratinocytes
  - Endothelial cell
  - Mast Cell

- Scarring/Remodeling
  - Myofibroblasts
    - Enzymes
      - MMP's
      - Serine proteases
  - Mast Cell
  - T-cells



Courtesy V. Falanga

Proc AAD 2003

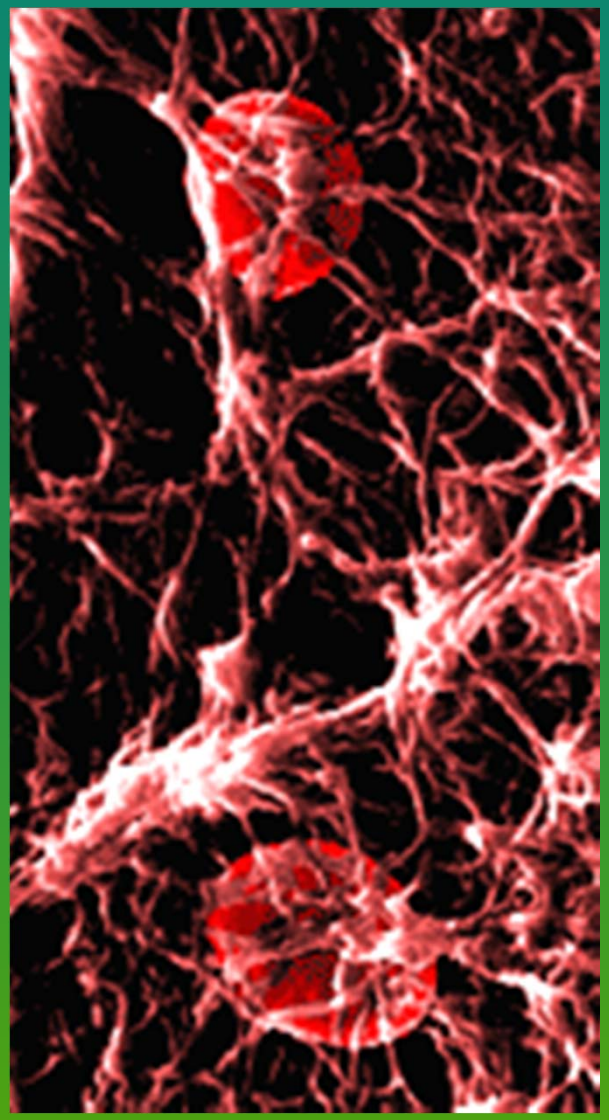
Coagulation/Inflammation

Important processes

- Fibrin Clot & Platelet Aggregation
- Neutrophil cleansing of the wound
- Macrophage debridement and cleansing

What does the clot do?

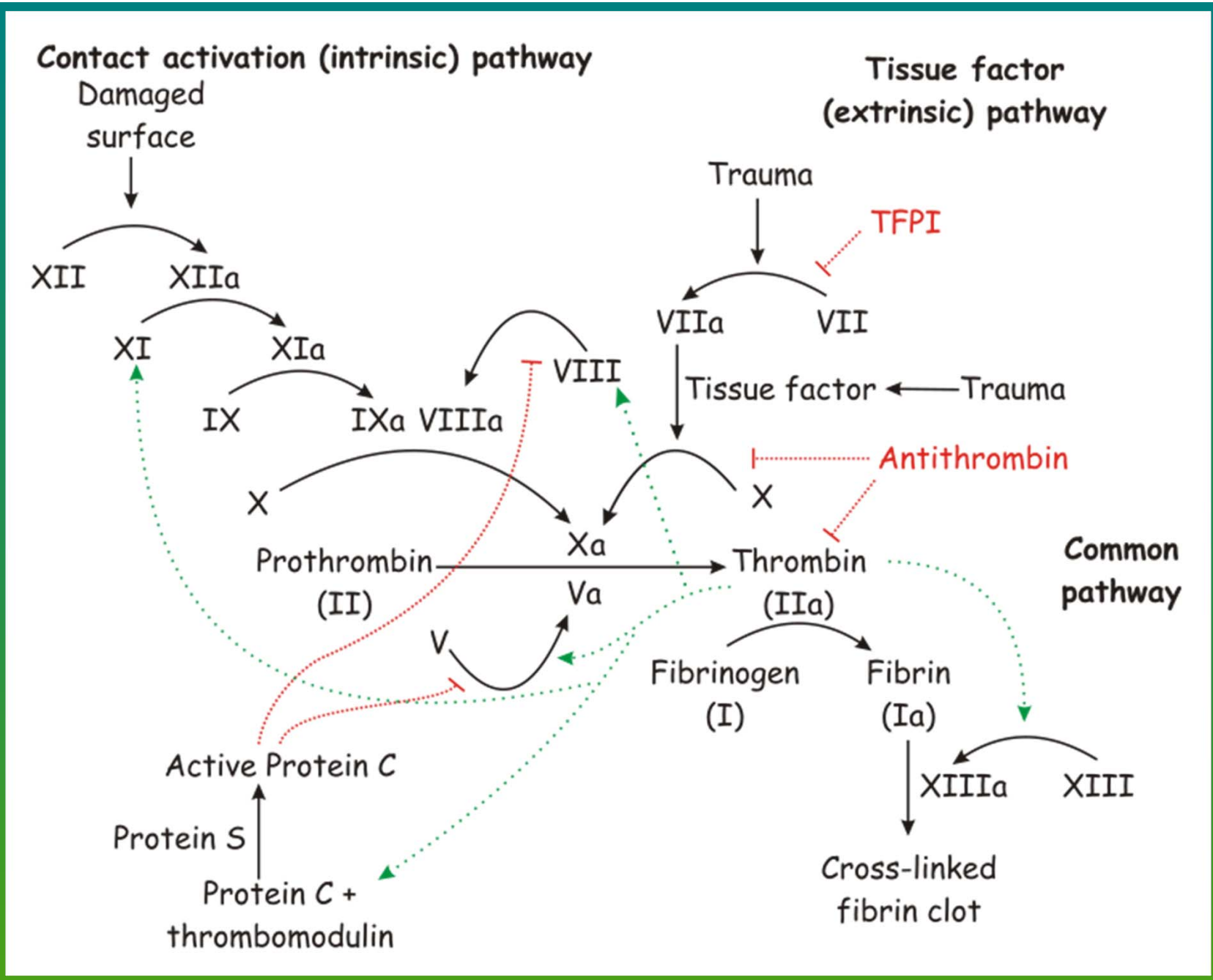
- The clot
  - Allows for movement of inflammatory cells and newly forming vessels into the wound
  - Provides scaffold for organization of provisional matrix



Cellsalive.com

What starts the process?

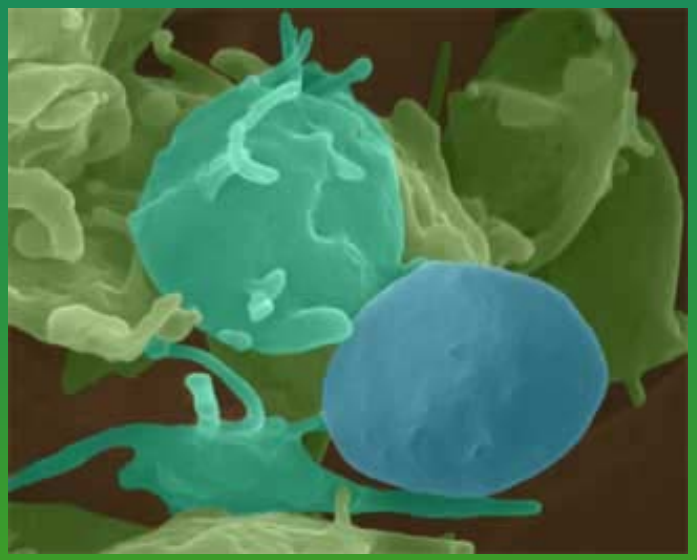
- Clotting results from:
  - Surface activation of Hageman factor XII
  - Tissue “Pro-coagulant” released from the damaged cells
  - Coagulations factors released from activated platelets and endothelial cells



- **The Fibrin Clot**

Platelets

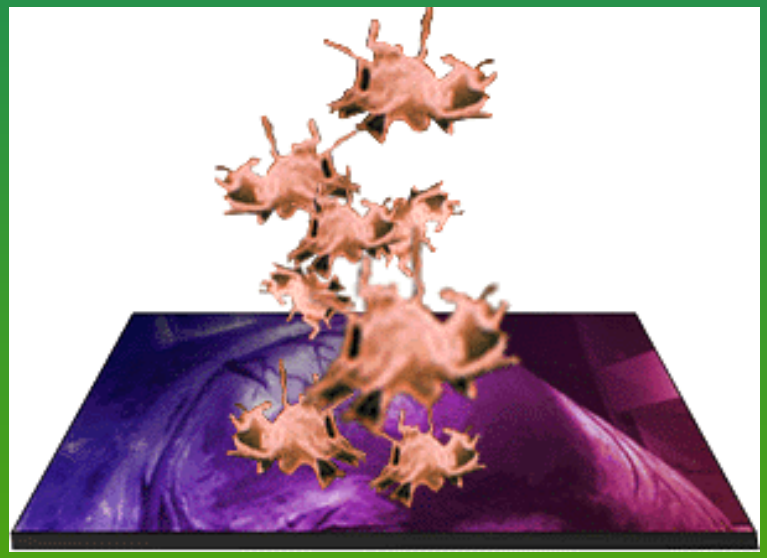
- Platelets
  - Platelets become activated when exposed to thrombin and fibrillar collagen
  - Once activated they release
    - Fibrinogen
    - Fibronectin
    - Thrombospondin
    - Von Willebrands Factor



www.bus.lsu.edu

Platelets – Cytokine profile

- Platelets also release:
  - PDGF
  - TGF-β1
    - *Most important storage factor for this cytokine*
  - PAF
  - Serotonin



sln.fi.edu/biosci/ blood/platelet.html

How do we stop the clot??

- Stimuli for formation is diluted
- Adjacent vessel production of prostacyclin and protein C which inhibits clot formation
  - Degrade Factors V and VIII
- Adjacent vessel release of plasminogen activator
  - Clot lysis
    - Convert plasminogen to plasmin

Inflammation

Initiation of Inflammation

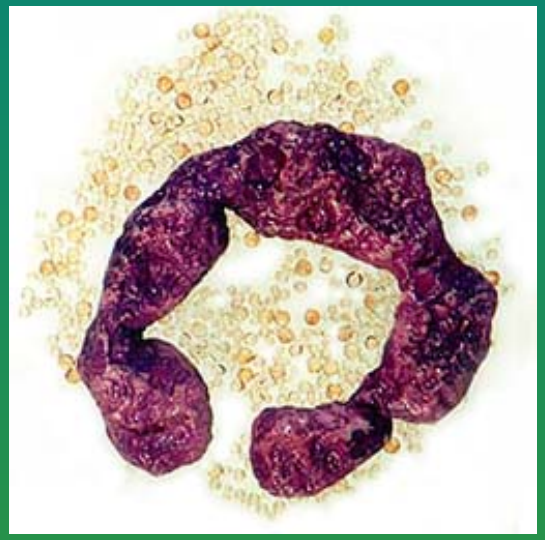
- Activation of Hageman factor will lead to generation of bradykinin fragments which will initiates the complement cascades
- Complement cascades will release C3a and C5a which will call neutrophils and monocytes and increase vessel permeability
- The neutrophils, macrophages and mast cells will further attract more.

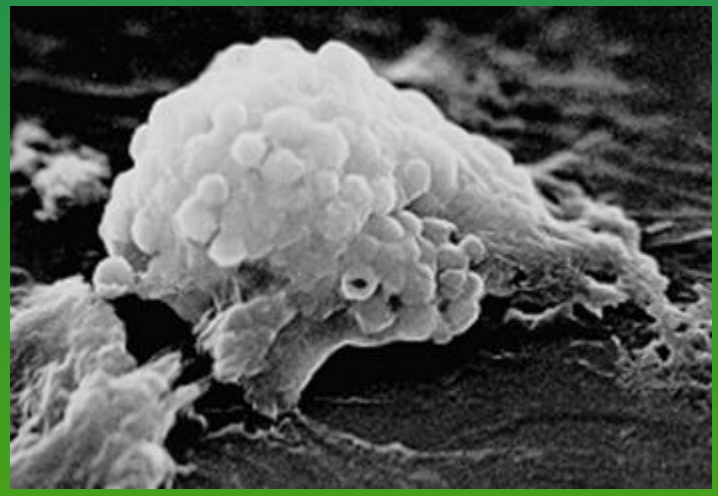
Inflammation

- Neutrophils
  - Start acting first
  - Chemoattractants for neutrophils
    - IL-8
    - Gro
    - Kallikrein
    - FDP's and fibrinopeptides
    - Platelet released cytokines
    - Bacterial proteins (bacterial cleavage)

Inflammation

- Neutrophils Con't
  - Neutrophils normally "roll" along the endothelial cells
    - Sialyl Lewis X carbohydrate residue binding to E- or P-selectin on endothelial cells
  - Chemoattractant factors simulate LFA-1 expression on neutrophils
    - Adheres to I-CAM and V-CAM on endothelial cells





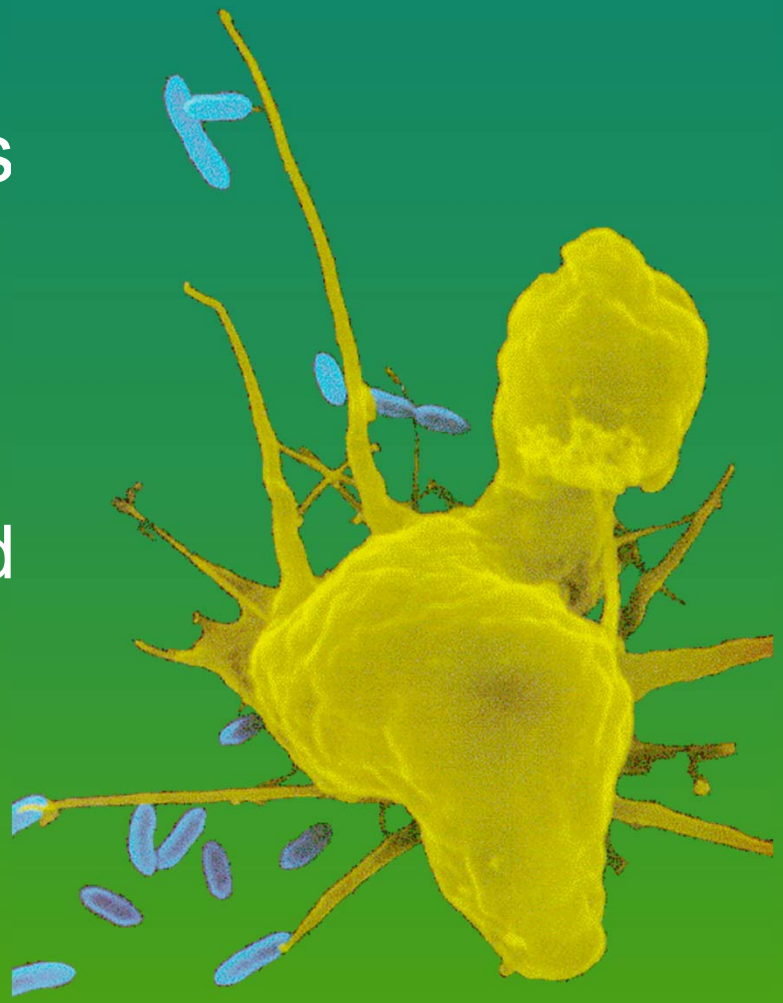
www.medfak.ni.ac.yu

Inflammation - Neutrophils

- Neutrophils Con't - Primary Role:
  - Phagocytosis and intracellular killing of contaminating bacteria
  - Persistently contaminated wounds
  - Role in sterile wounds?
    - Release cytokines for early activation of local fibroblasts

Inflammation - Macrophages

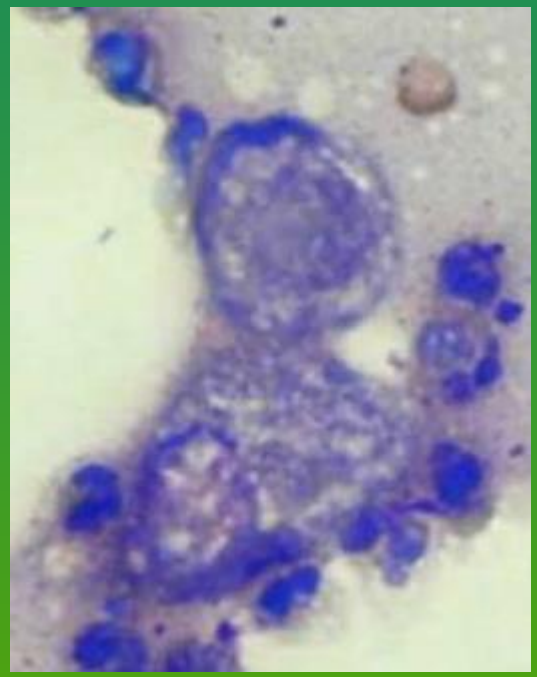
- Monocytes/Macrophages
  - Arrival represents transition from inflammation to repair
  - Within 24-48 hours should replace neutrophils as predominant cell



www.people.vcu.edu

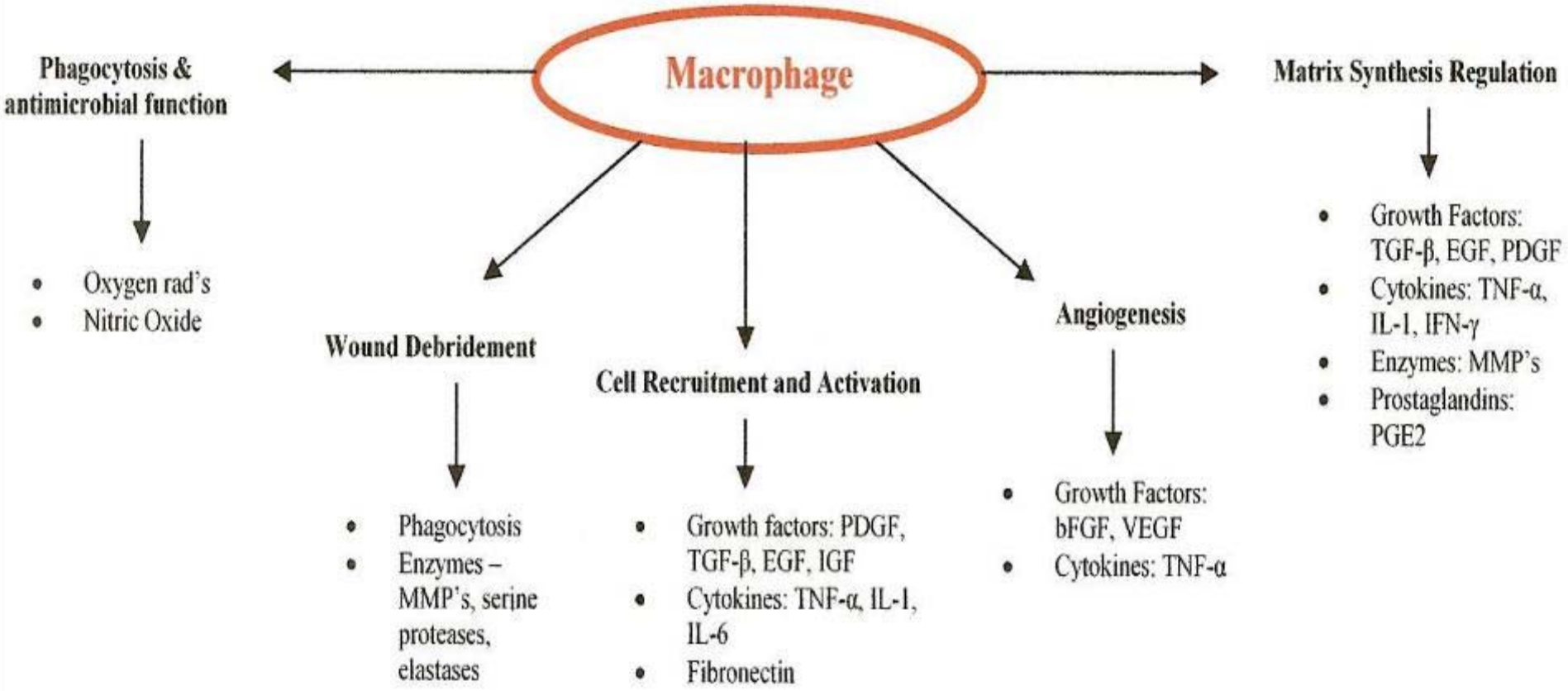
Macrophages

- Chemoattractants for Monocytes/Macrophages:
  - Same as neutrophils – Plus:
    - Collagen fragments
    - Elastin fragments
    - Fibronectin
    - TGF-β1



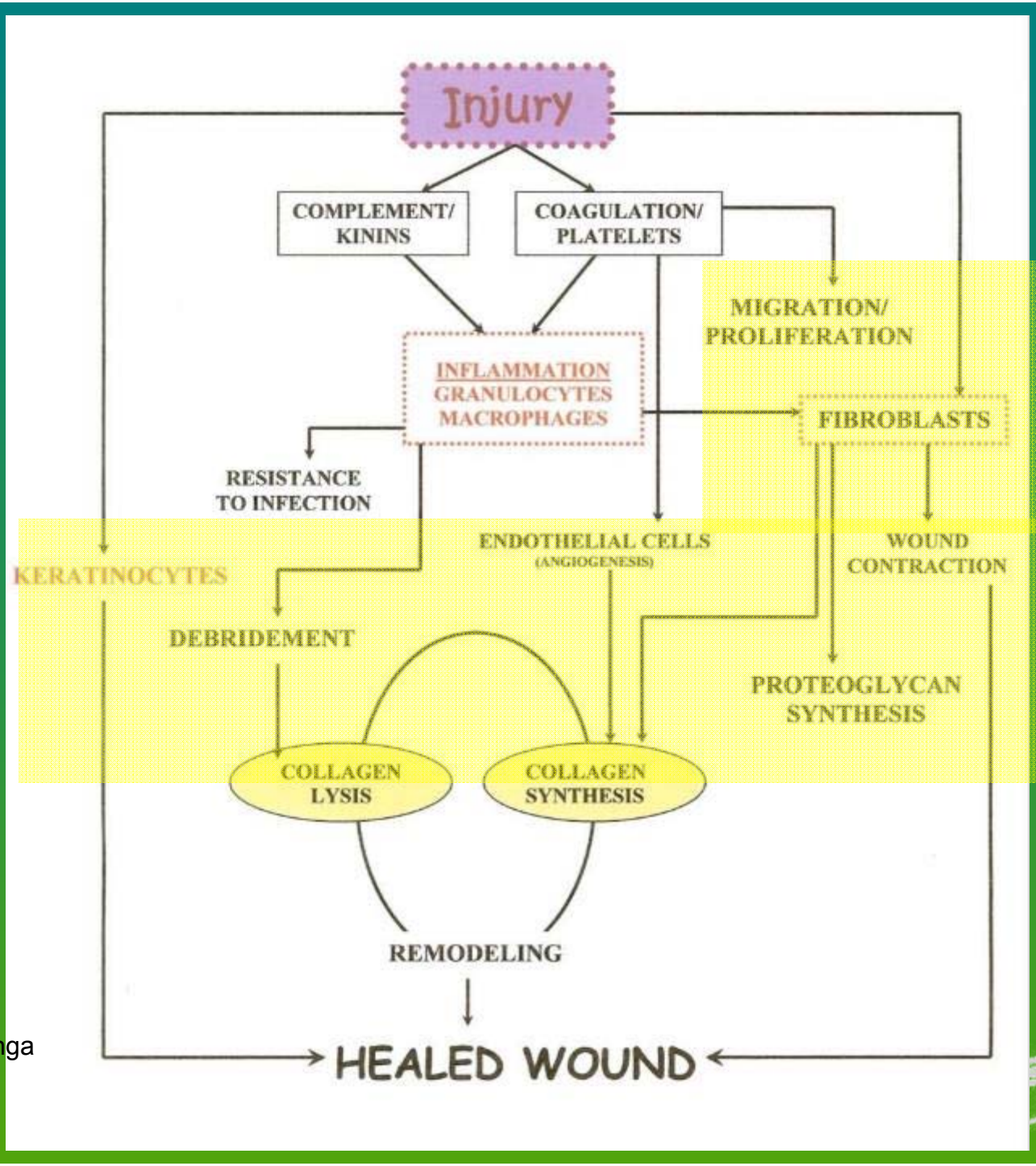
Inflammation

- Actions include
  - Phagocytosis and killing of bacteria
  - Removal of effete neutrophils
  - Scavenge tissue debris
  - Growth Factor release recruit and activate fibroblasts
    - PDGF
    - FGF
    - TGF-β1
- Removed via apoptosis


Macrophage
Phagocytosis & antimicrobial function
Oxygen rad's
Nitric Oxide
Wound Debridement
Phagocytosis
Enzymes – MMP's, serine proteases, elastases
Cell Recruitment and Activation
Growth factors: PDGF, TGF-β, EGF, IGF
Cytokines: TNF-α, IL-1, IL-6
Fibronectin
Angiogenesis
Growth Factors: bFGF, VEGF
Cytokines: TNF-α
Matrix Synthesis Regulation
Growth Factors: TGF-β, EGF, PDGF
Cytokines: TNF-α, IL-1, IFN-γ
Enzymes: MMP's
Prostaglandins: PGE2

Is Inflammation Necessary?

- How essential is inflammation?
  - Early studies show failure to heal with steroids and anti-macrophage serum
  - More recent studies have healing without an inflammatory infiltrate (knock out)
  - Exaggerated inflammation detrimental to healing
  - Fetal cells are less permissive to neutrophil diapedesis than adult cells



Courtesy V. Falanga

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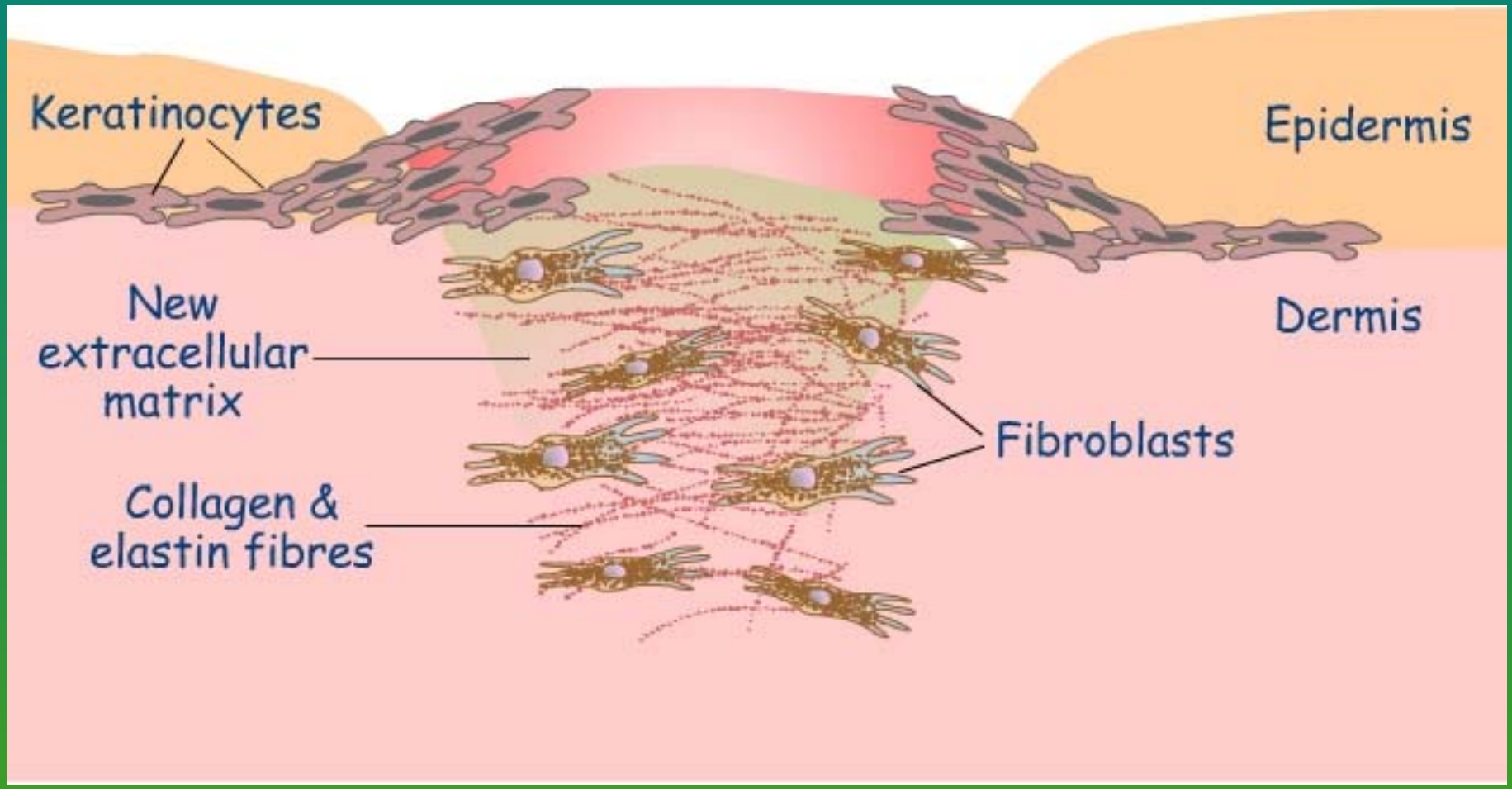

Keratinocytes
Epidermis
New extracellular matrix
Dermis
Fibroblasts
Collagen & elastin fibres

Proliferation and Tissue Formation

- Factors affecting/guiding this phase:
  - Tissue hypoxia (Negative pressure vs. hyperbaric O2 therapy)
  - Adhesion proteins
  - ECM components
  - Action of inflammatory cells
- These factors guide the activation and migration of fibroblasts, keratinocytes and endothelial cells

Effects of Hypoxia on Healing

- Effects of hypoxia/low oxygen tension (Negative pressure therapy):
  - Activate fibroblasts and endothelial cells
  - Stimulate macrophage to release angiogenic factors (bFGF)
  - Bandaging techniques help us enhance this effect
- Cyclical oxygenation/hypoxia – oxygen radicals

Effects of Hyperoxygenation

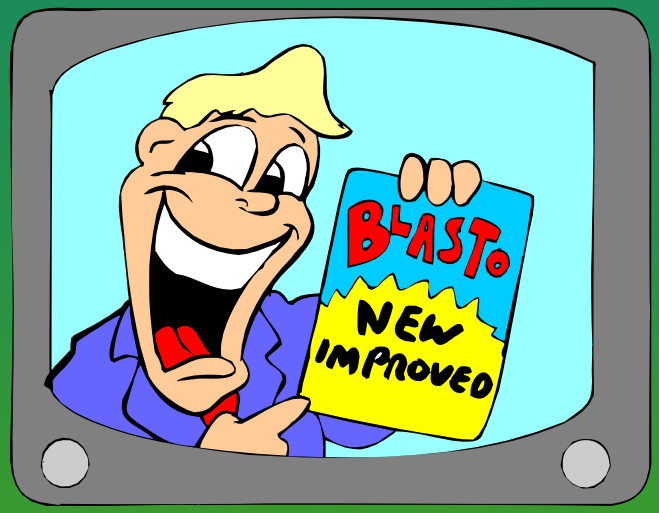
- Oxygen is required for all protein synthesis (collagen, elastin, cellular replication) as well as cell movement.
- Oxygen is also required for NO synthesis – regulation of angiogenesis.
- Reduction of Radical Oxygen Species (ROS).
  - Superoxide anion radical is the one-electron reduction product of oxygen.
    - Drives endothelial cell signaling such as required during angiogenesis
    - H2O2 – breakdown product – supports cell migration and proliferation through redox reactions as well as mediate formation of hypochlorous acid in neutrophils.
  - NADPH oxidases represent one major source of superoxide anion radicals at the wound-site - oxidases in phagocytic cells help fight infection.

Fibroplasia - Dermis

- Fibroblast is the main cell of this stage:
  - Formation of granulation tissue and re-collagenation of the dermal ECM
  - Substantial phenotypic changes
    - Determined by the ECM and surrounding growth factors and cytokines
  - Some evidence of sub-populations of fibroblasts rather than phenotype change (like mast cells)

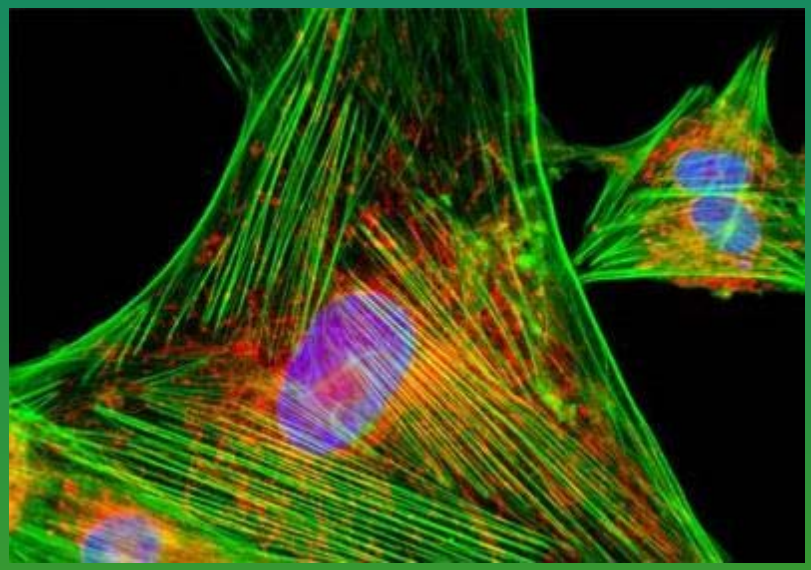
The Fibroblast - activation

- Resting in a collagen-rich ECM
  - Biosynthetically inactive
  - Integrin receptors α2
- Start changing phenotype
  - When exposed to initial provisional matrix
  - When exposed to PDGF and TGF-β1



The Fibroblast - activation

- When within the fibrin clot:
  - Integrin receptors α3, α5 & αv upregulated
  - Actino-myosin cytoskeleton
  - Produce hyaluronan (HA) and RHAMM (HA receptor)
  - Fibronectin
  - Release serine proteases and MMP's



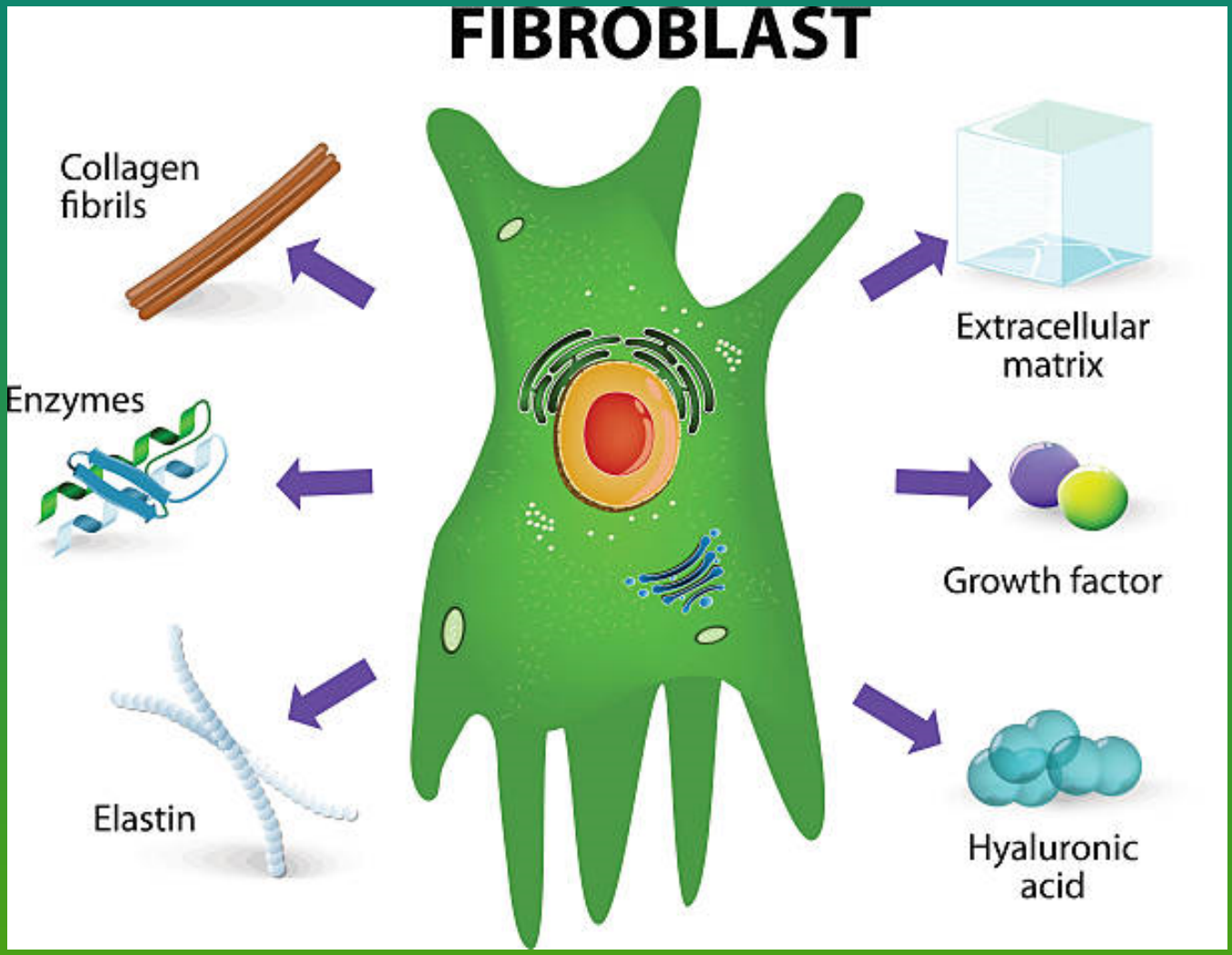
micro.magnet.fsu.edu

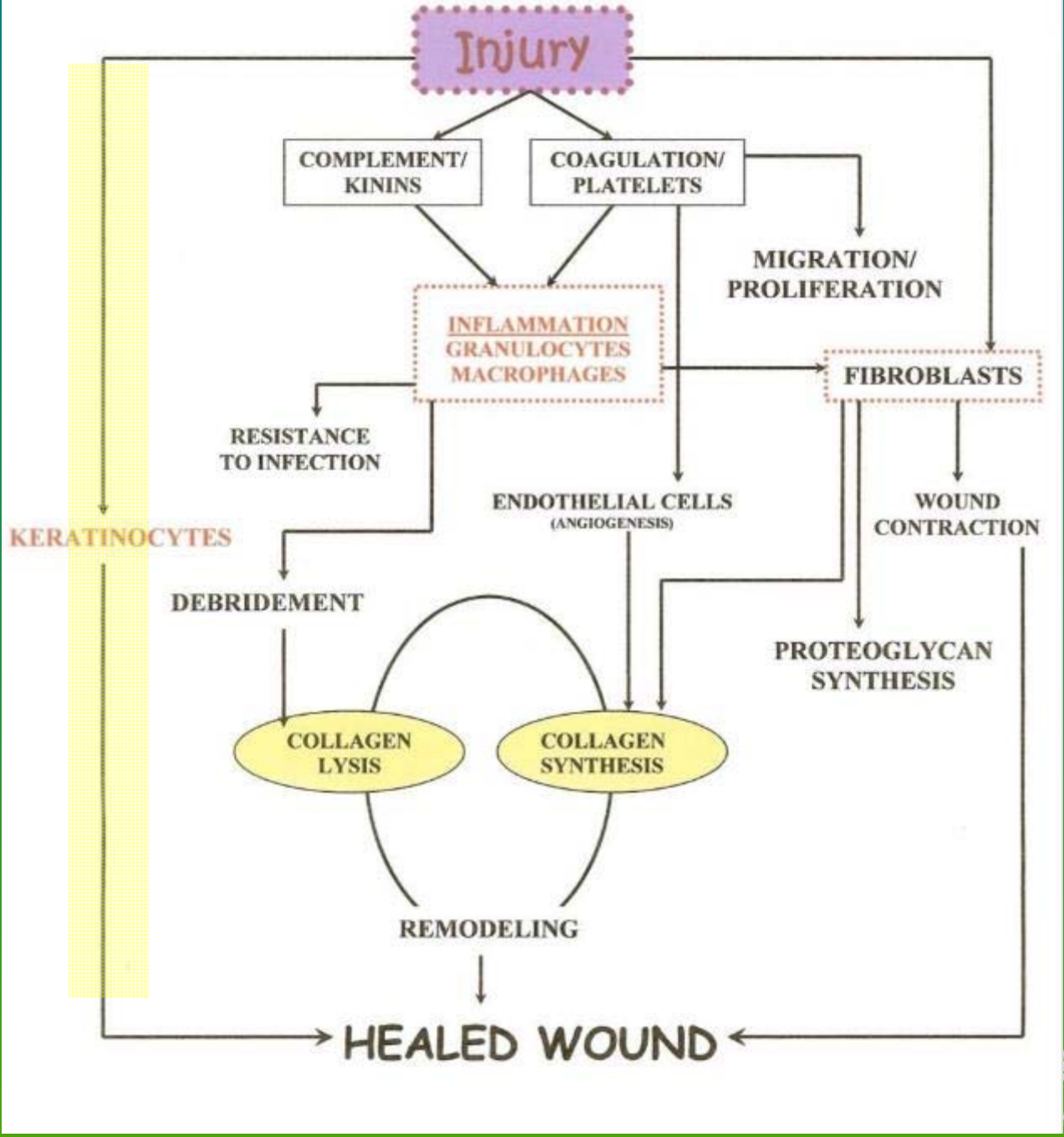
Fibroplasia

- In the first phase fibroblasts produce a “loose” ECM of fibronectin and HA
  - Golgi apparatus and plasma membrane

- Secondly – Collagen and proteoglycans (RER production)
  - IL-4 potent stimulator of collagen synthesis switch (Mast cells)

Fibroplasia





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Proliferation and Tissue Formation
Keratinization

- Reestablishment of epithelial cover
  - Starts within hours of injury
  - Start from either wound edge or from remaining adnexal structures
  - Continuous action throughout the phases of healing

Proliferation and Tissue Formation
Keratinization

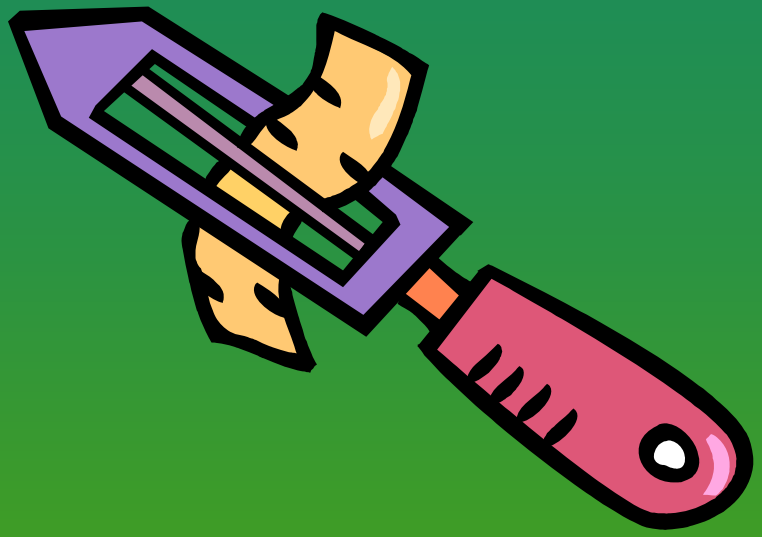
- Keratinocytes migrate in “leap frog” fashion
- Keratinocytes adherent to ECM stay stationary and migrating keratinocytes are the overlying ones

Proliferation and Tissue Formation
Keratinization

- Keratinocytes do not migrate over intact BMZ
  - Erosions – fibrin and fibronectin will penetrate the BMZ
- Provisional matrix KC's can migrate over:
  - Collagen I
  - Collagen III
  - Collagen V
  - Fibronectin
  - Fibrin
  - Tenascin
  - Vitronectin

Proliferation and Tissue Formation
Keratinization

- Migrating KC's cannot bind to denatured collagen or denatured fibrin - wound dissection – "peeling off" the eschar.



Once migration has started – BMZ will follow in "zipper" like fashion

Proliferation and Tissue Formation
Keratinization

- Phenotypic changes for migration
  - Retract tonofilaments - actin (detachment)
  - Dissolve desmosomes and hemidesmosomes (detachment)
  - Increase gap junctions (enhanced cellular communication)
  - Peripheral actin filaments and formation of lamellipodia (motor apparatus)

→

Proliferation and Tissue Formation
Keratinization

- Phenotypic changes for migration (con't)
  - KC's express keratins 5&14 as well as 6&16
  - Express serine proteases (plasminogen activators eg.) and MMP's (collagenases) (dissection through the wound)

→

Proliferation and Tissue Formation
Keratinization

- Phenotypic changes for migration (con't)
  - Express integrins that allow attachment to the provisional matrix
    - α5β1 and αvβ6: attachment to fibronectin
    - αvβ5: attachment to vitronectin
    - α2β1: attachment to collagen matrix

Proliferation and Tissue Formation
Keratinization

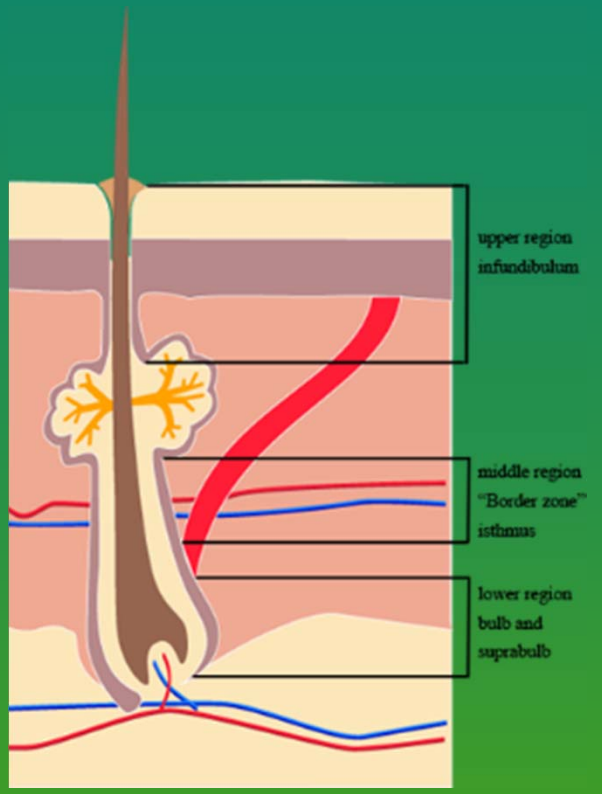
- Primary signal that stops KC migration is reconstitution of laminin within the BMZ
  - Large glycoprotein, prevent direct contact between KC's and collagens within BMZ and dermis
- Proliferation begins 1-2 days after injury
  - Likely absence of neighbor cells and growth factors are primary stimulators for proliferation
  - Proliferation and migration are independent of each other (TGF-β1 promotes migration – no effect on proliferation)

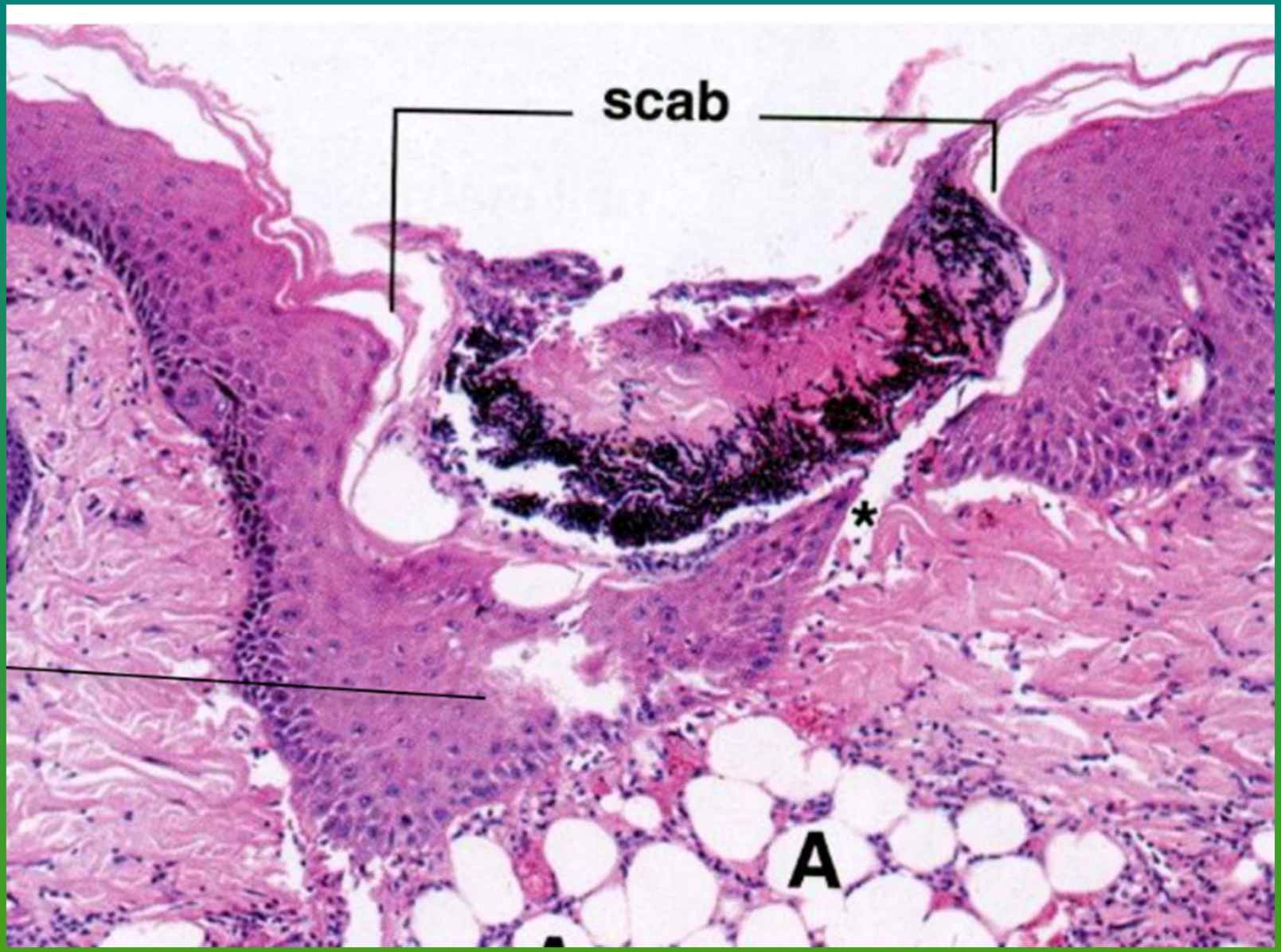
Proliferation and Tissue Formation
Keratinization – Hair Follicle

- Humans: Haired skin heals faster than non-haired skin
- Mouse studies: HF contributes during acute phase of healing

Proliferation and Tissue Formation Keratinization – Hair Follicle

- Bulge Region:
  - 25% of KC's in newly healed mouse wound from bulge
  - Destined to become transient amplifying cells
  - Lost several weeks later
- Infundibulum/Upper Isthmus:
  - Become permanent stem cell residents of epidermis




scab
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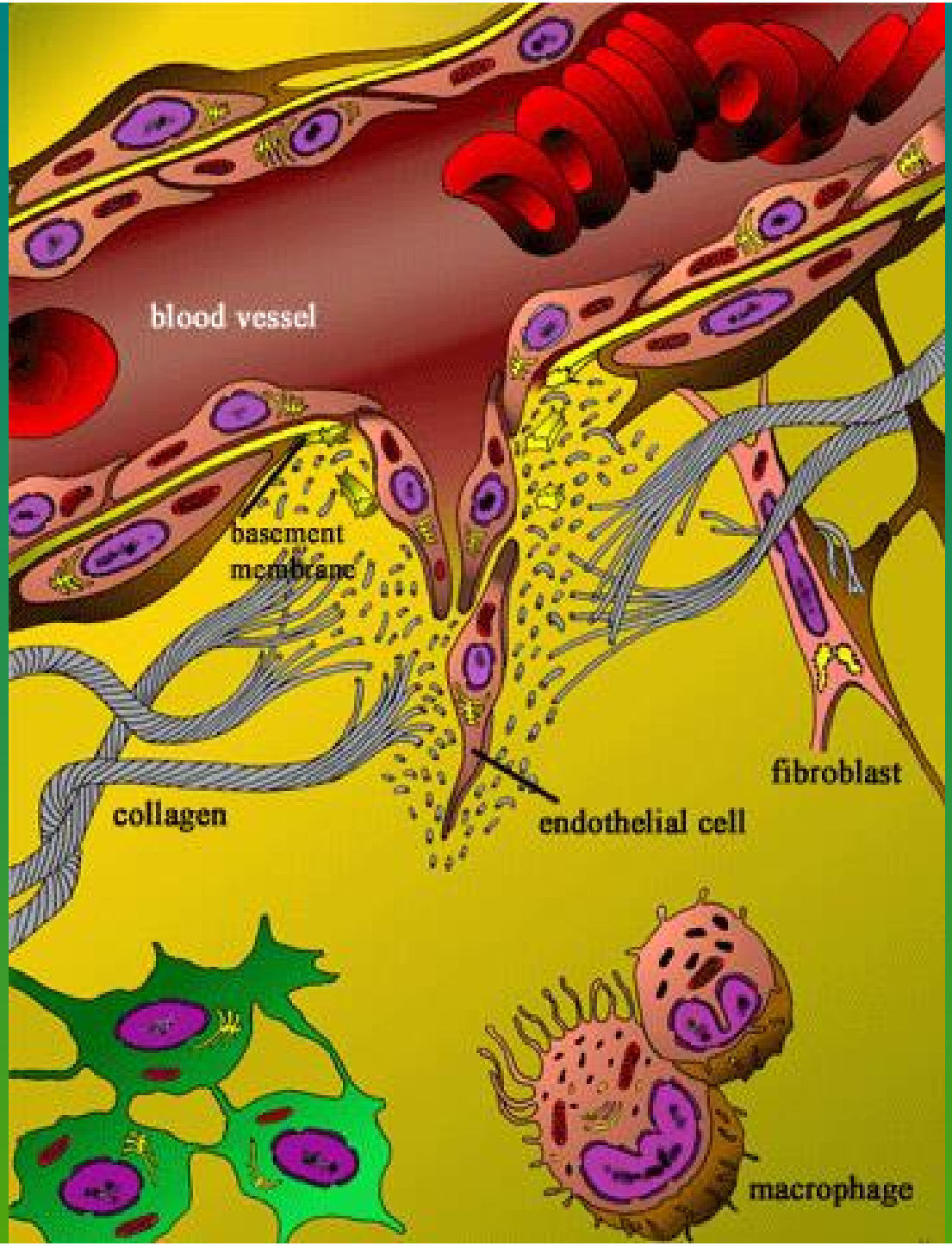
A

Proliferation and Tissue Formation Neovascularization

- Angiogenesis depends on 4 related events:
  - Mitogenic stimulation
  - Endothelial cell phenotype alteration
  - Chemoattractant-driven migration
  - Appropriate extracellular matrix

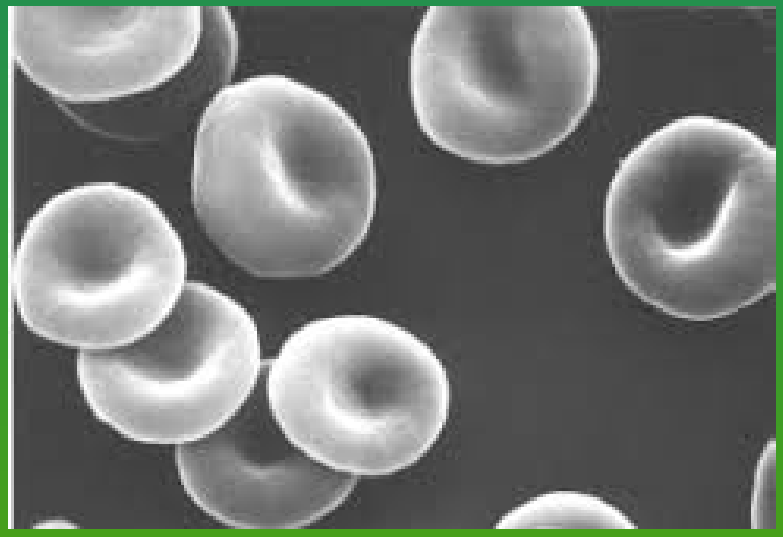
Proliferation and Tissue Formation Neovascularization

- Macrophages (and platelets) release basic fibroblast growth factor (bFGF).
  - This will result in MMP (collagenase I) production by endothelial cells which will degrade the BM – allowing migration
  - bFGF stimulates migration of endothelial cells out of the fragmented BM


blood vessel
basement membrane
fibroblast
collagen
endothelial cell
macrophage

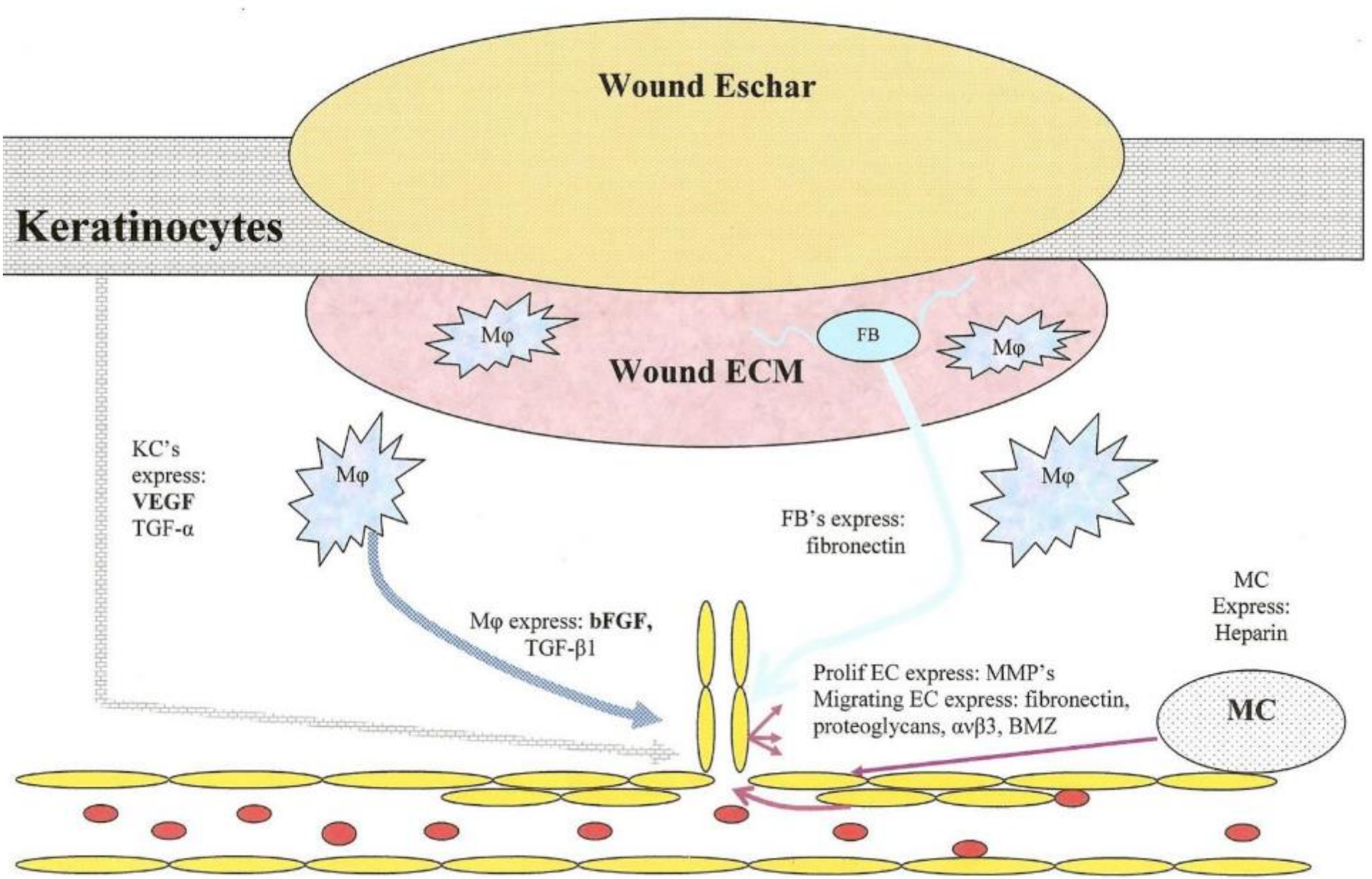
Proliferation and Tissue Formation Neovascularization

- Endothelial chemoattractants:
  - Keratinocytes and platelets:
    - vascular endothelial growth factor (VEGF), TGF-α
  - Macrophages and platelets:
    - bFGF, TGF-β1
  - ECM/fibroblasts:
    - fibronectin
  - Mast cells:
    - heparin



Proliferation and Tissue Formation Neovascularization

- Endothelial cells' actions:
  - Produce their own ECM (initially) – fibronectin and proteoglycans
  - Express αvβ3
    - Adhesion to fibrinogen, fibrin, and Factor VIII (vWF)
  - Produce their own BM


Wound Eschar
Keratinocytes
Mφ
FB
Mφ
Wound ECM
KC's
express:
VEGF
TGF-α
Mφ
Mφ
FB's express:
fibronectin
MC
Express:
Heparin
Mφ express: bFGF,
TGF-β1
Prolif EC express: MMP's
Migrating EC express: fibronectin,
proteoglycans, αvβ3, BMZ
MC

Proliferation and Tissue Formation Reinnervation

- Not a lot of published material
  - Scar tissue and grafts do not regain the innervation of the original tissue
  - Experimental dennervation did have an effect on rat wound healing

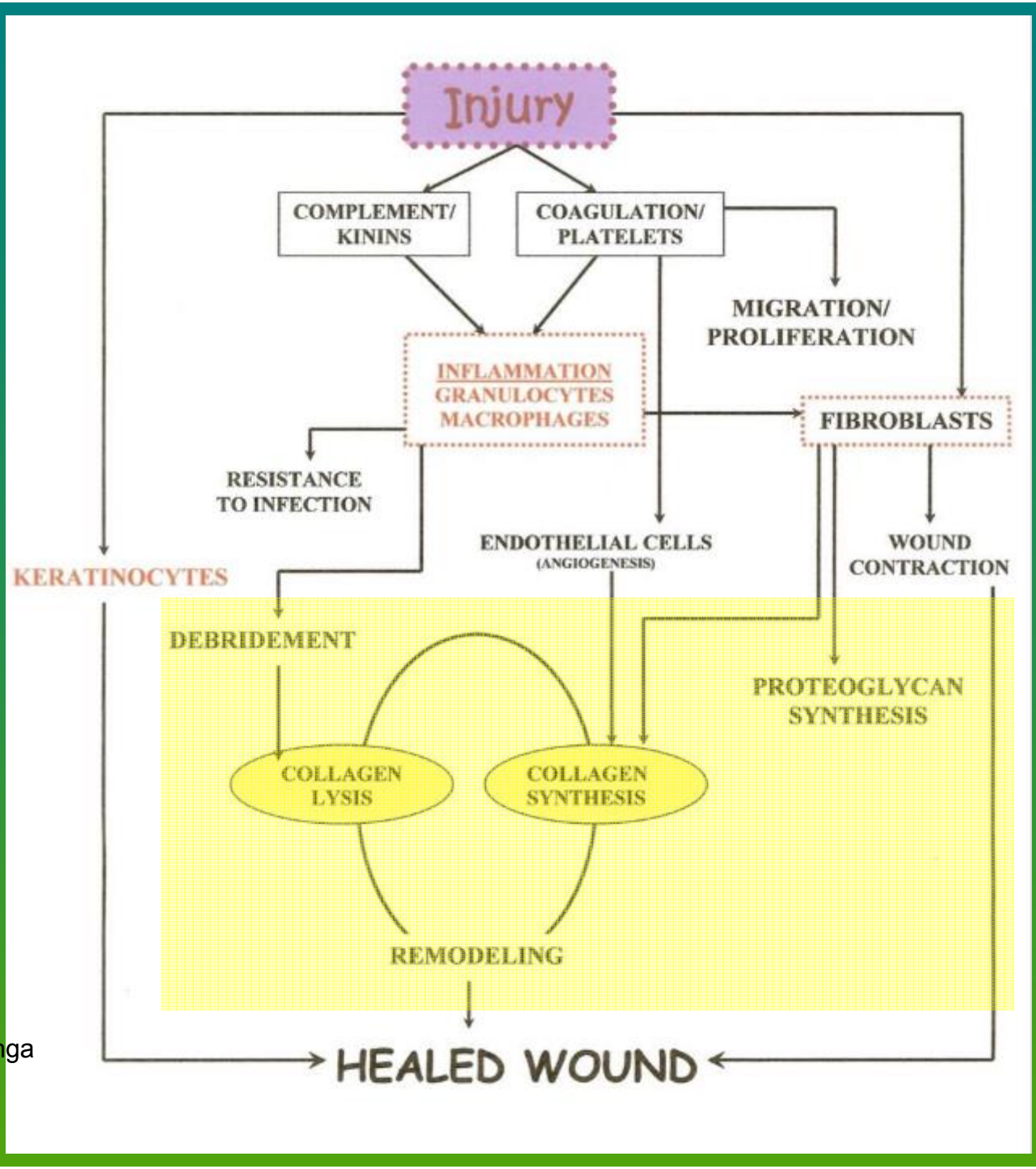
www.bitspin.net

Proliferation and Tissue Formation Reinnervation

- Healing adult wounds associated initially with hyperinnervation
  - 300% increase in skin innervation density
    - NGF may mediate
    - Topical NGF accelerate healing in mice – both normal and diabetic

Proliferation and Tissue Formation

- Effects of nerve derived factors on wound healing:
  - Substance P enhances release of IL-1 and TNF-α from mast cells
  - Substance P and K stimulate DNA synthesis in cultured skin fibroblasts
  - Vasoactive intestinal peptide (VIP) enhances KC migration
  - Substance P and calcitonin gene-related peptide (CGRP) are increased in hypertrophic scars



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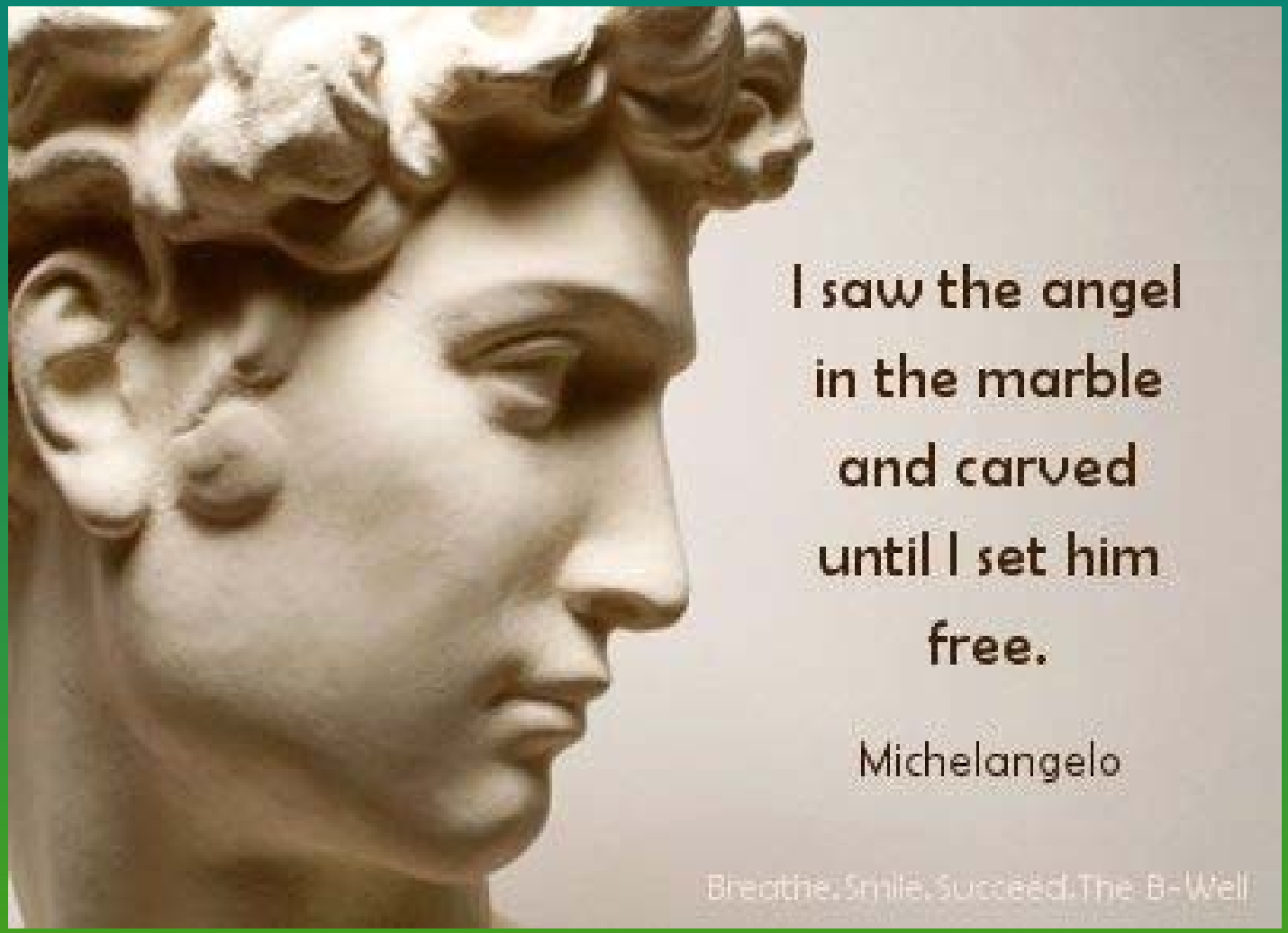

I saw the angel
in the marble
and carved
until I set him
free.
Michelangelo
Breathe. Smile. Succeed. The B-Well

Remodeling

- Remodeling of the ECM
  - Process that continues for months
  - Reorganization of the tissue
  - High degree of overlap with other phases of wound healing
  - Scar attains approximately 70% of the strength of the original tissue - repaired
  - Apoptosis and action of MMP's are essential for necessary cellular reduction in the wound

Extracellular Matrices

- Roles of ECM:
  - Form structure and strength
    - Cell-matrix interactions
    - Cellular phenotype
    - Cell polarity
  - Cell adhesion and migration
    - Scaffold for migration
    - Cell recruitment: inflammatory cells, epithelial cells, endothelial cells
  - Interactions with other components in matrix:
    - Growth factors
    - Proteases and MMP's

Extracellular Matrices

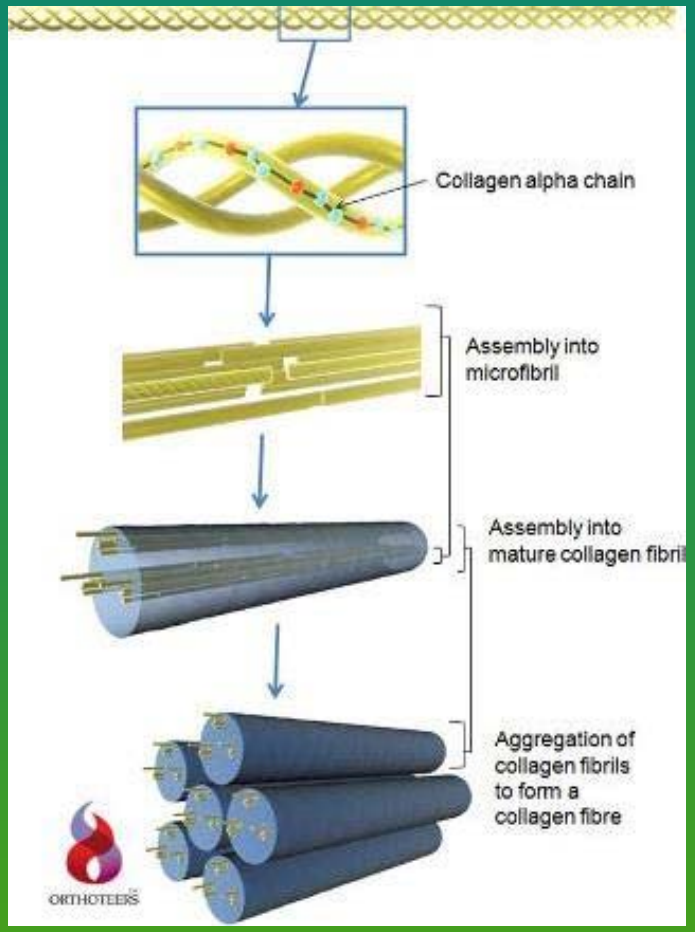
- ECM review:
  - Fibrin Clot
    - Derived from fibrinogen
    - Fibrin fibrils interact with platelets via α2β3
    - Fibrin, with PDGF induce fibroblasts to express fibronectin integrins

Extracellular Matrices - Fibronectin

– Fibronectin
  - Second provisional matrix
  - Multifunctional cell adhesion protein found in blood and other tissues
  - Binds fibrin and found even in the early fibrin clot
  - Supports movement of fibroblasts, keratinocytes and endothelial cells
  - Opsonizes ECM debris so can be phagocytosed
  - Template for collagen fibril organization

Extracellular Matrices - Collagen

- Collagen I & III are the primary collagens in wound repair
- Collagen III is first present and synthesized and increased in granulation tissue
- Collagen I is made later – starting at about day 5
- Type V collagen increased during angiogenesis and in highly vascular scars



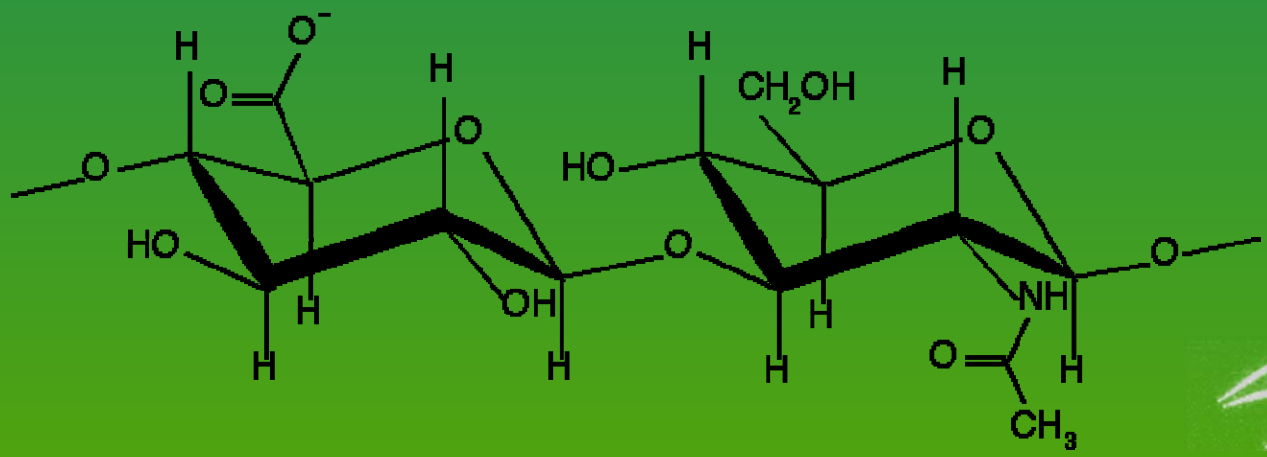
Extracellular Matrices - Collagen

- Collagens (con't):
  - Intact collagen affects phenotype and function of various cell types
    - Primarily via regulation of expression of integrin receptors
  - By week three – 20% of final wound strength
    - Now additional strength gained by remodeling – not new collagen deposition


Glycosaminoglycans
Proteoglycans
Their role as ECM's

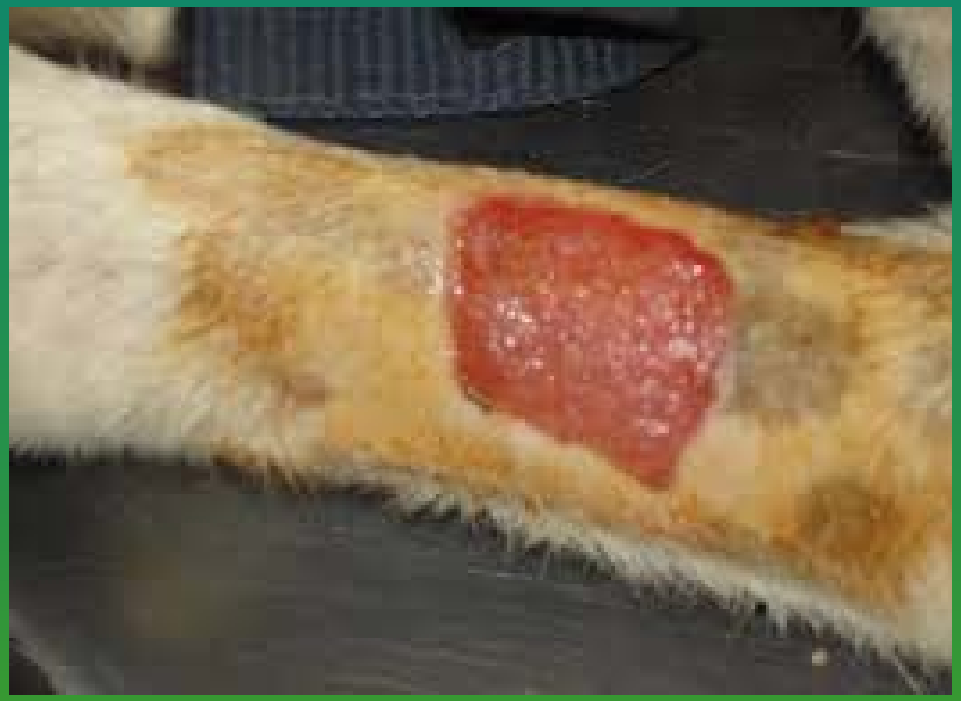
Extracellular Matrices – GAG's

- Glycosaminoglycans – (GAG)
  - All GAG's are repeating hexosamine and an acidic sugar
  - Highly polar and attract water
  - Shock absorption, lubrication, storage
  - Hyaluronan and heparin
    - Proteoglycans (Sulfated GAG macromolecules: dermatan sulfate, chondroitin sulfate, keratin sulfate, heparin sulfate



Extracellular Matrices - HA

- Hyaluronan - hyaluronic acid (HA)
  - HA unique – synthesized at plasma membrane (large molecule)
  - Linear polymer of repeating N-acetyl glucosamine-glucuronic acid disaccharides



Healthy granulation tissue in a surgically created wound. HA gives the “glistening” appearance of the wound

Extracellular Matrices - HA

- Component of early granulation tissue
- Concentration increases early then drops between days 5-10 (hyaluronidase) and then remains stable while proteoglycan (sulfated GAG's) concentration increases

- Fetal wounds do not decrease their HA levels

Extracellular Matrices - HA

- Two HA receptors:
  - CD44 – widely distributed
    - Involved in attachment, uptake and breakdown of HA
    - Fibroblasts from hypertrophic scars and scleroderma shown to have increased CD44 on their membranes
  - RHAMM – found on macrophages and fibroblasts
    - Essential for motility of macrophages and fibroblasts

Extracellular Matrices Proteoglycans

- Proteoglycans
  - Contain a protein core to which one or more GAG's are covalently bound
  - Huge range of versatile molecules

Extracellular Matrices Proteoglycans

– Roles of Proteoglycans:
  - Water storage
  - Extracellular organization
  - Growth factor storage
  - Promotion of growth factor expression and binding
  - Enzyme and autocoid storage in granules
  - Regulation of blood coagulation

Extracellular Matrices Proteoglycans

- Serglycine from mast cells
  - Regulation of coagulation. Interacts with AT-III and inhibits factor Xa (activated thrombokinase) and thrombin (factor IIa)
  - Binds histamine and cationic proteases which affect vasopermeability and wound debridement
  - Activate matrix metalloproteinases

Extracellular Matrices Proteoglycans

- Chondroitin-4-sulfate (C4S) and Dermatan Sulfate (DS)
  - Produced by mature scar fibroblasts
  - Structural
  - Regulate collagen fibrillogenesis
  - C4S likely facilitates collagen deposition during matrix formation and remodeling phase
    - C4S is elevated in hypertrophic scars

Extracellular Matrices Proteoglycans

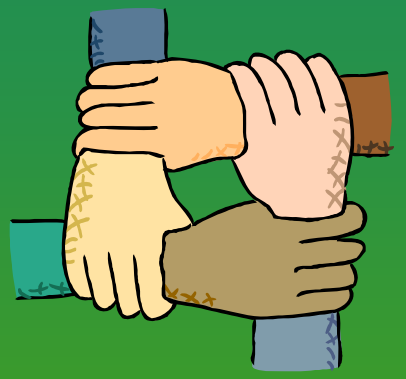
- Versican
  - Promote migration – decreases adhesion
- Decorin
  - Inhibit growth of cells *in-vitro*
- Syndecan family
  - Modulated during wound repair
  - Likely involved in migration, proliferation and gene expression

Remodeling

- Wound Contraction
  - Transition to the 3rd and final phase of wound repair
  - Centripetal or concentric decrease in size of an open wound
  - Dogs – clinical contraction 5-9 days after injury

Remodeling

- Wound Contraction (con't)
  - TGF-β1 no longer produced, but is still present in the wound as contraction begins
  - Fibroblasts assume myofibroblast phenotype
    - Large actin bundles
    - Cell-cell links with adherens jxns
    - Cell-matrix links with integrins
      - α5β1 to fibronectin
      - α2β1 to collagen

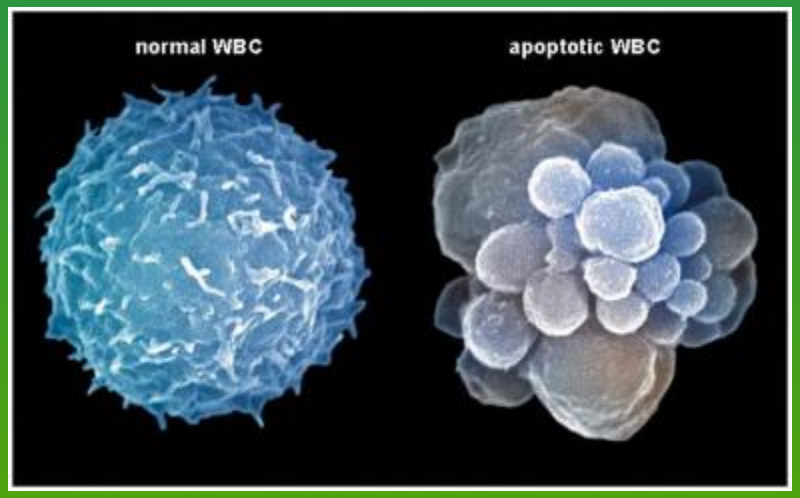


Remodeling

- Wound Contraction (con't)
  - Contraction exerts transmission of force equally across the wound
  - Platelet and macrophage isoforms of PDGF stimulate contraction – fibroblast isoform does not
  - Full thickness wounds contract about 40%
    - Partial thickness contracts much less

Remodeling

- Wound Contraction (con't)
  - – 10th day – fibroblasts undergo apoptosis
    - Transition to acellular scar
    - Impaired or decreased apoptosis likely plays a role in hypertrophic scars
    - Human diabetics have increased apoptosis (generalized) – also resulting in impaired healing (diabetic foot ulcers)

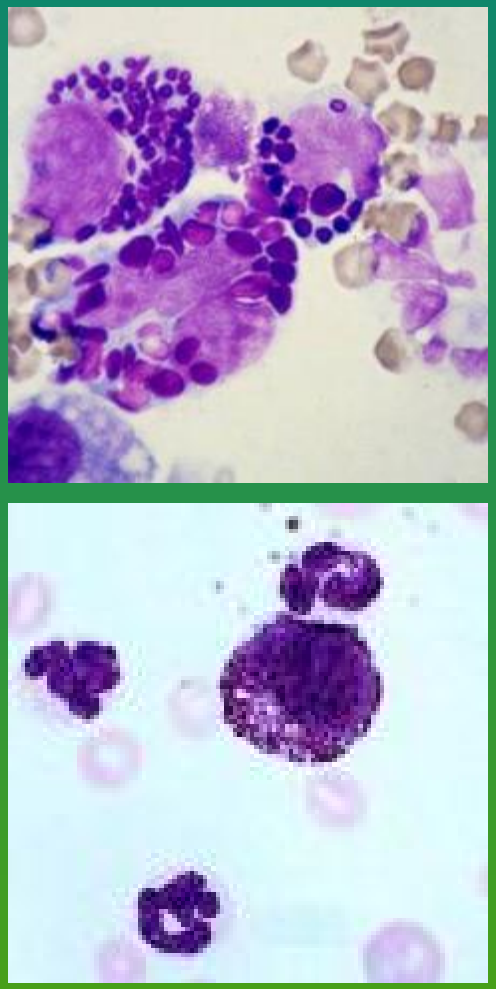


Remodeling

- Collagen Lysis
  - MMP's are primary enzymes
  - In horses: study by Schwartz et al revealed that distal limb wounds have a significantly decreased MMP-1 (collagenase I) and increased TGF-β1 protein and mRNA as well as increased myofibroblasts and collagen I

Mast Cell - Review

- The Mast Cell
  - Involved in all levels of wound healing
  - Located near endothelial cells and nerves adjacent to the wound



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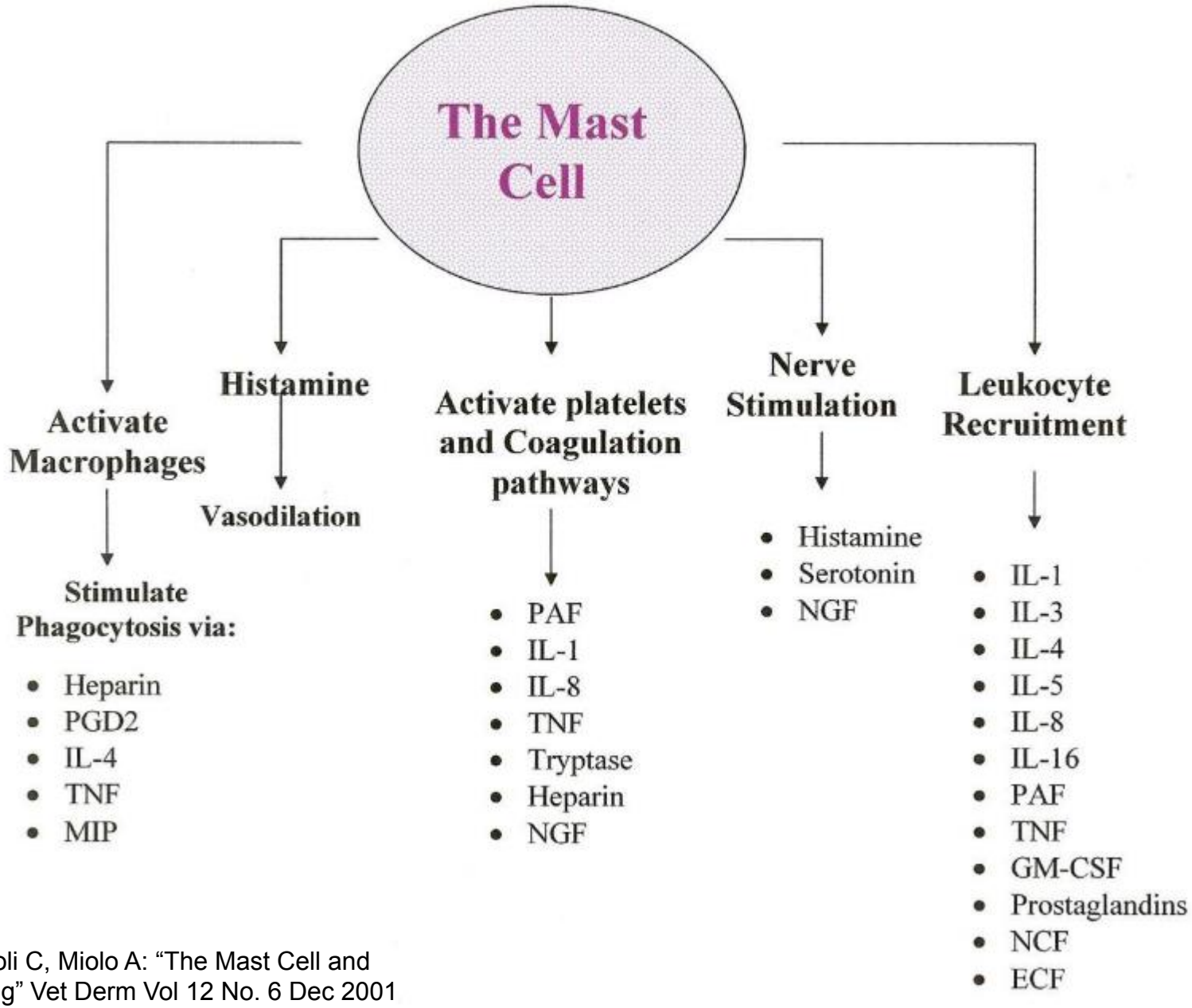
Mast Cell - Review

- Resides adjacent to local microcirculation and nerve endings
- Mobile and will migrate in response to stimuli
  - Physical stimulus – sunlight, trauma
  - Immunogenic – IgE, complement, cytokines and growth factors
  - Neurogenic - neuropeptides
- Cytoplasm filled with metachromatic granules containing biologically active mediators

Mast Cell - Review

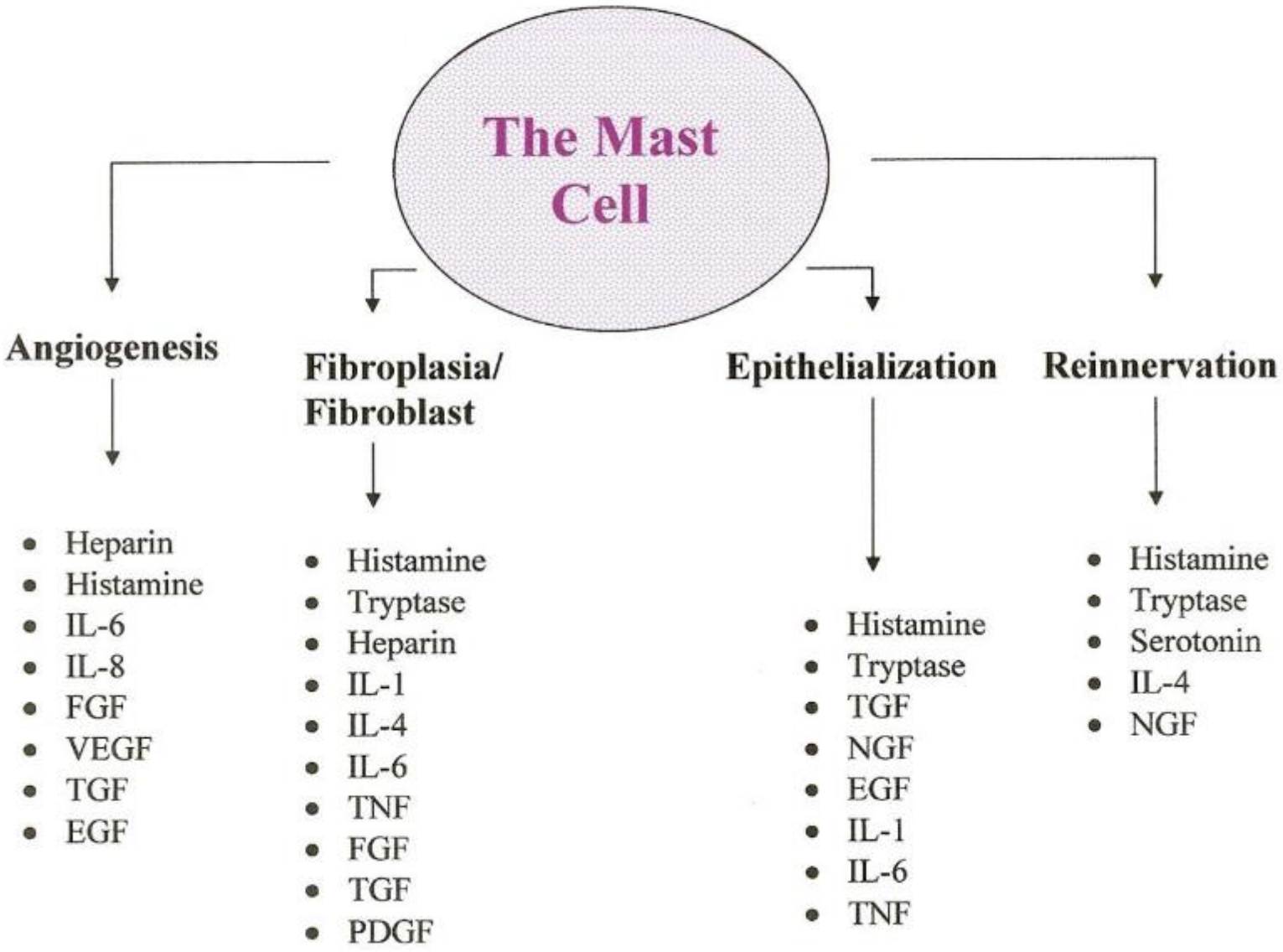
- Canine Mast Cell Mediators
  - α-Chymase
  - Tryptase
  - Heparin
  - Protease-3 (MCP-3)
  - Gelatinase (MMP-9)
  - Cathepsin C
  - Histamine
  - TNF
  - IL-4
  - IL-5
  - bFGF
  - TGF-β
  - PDGF
  - SCF
  - PDG2

Mast Cell and the Inflammatory Phase



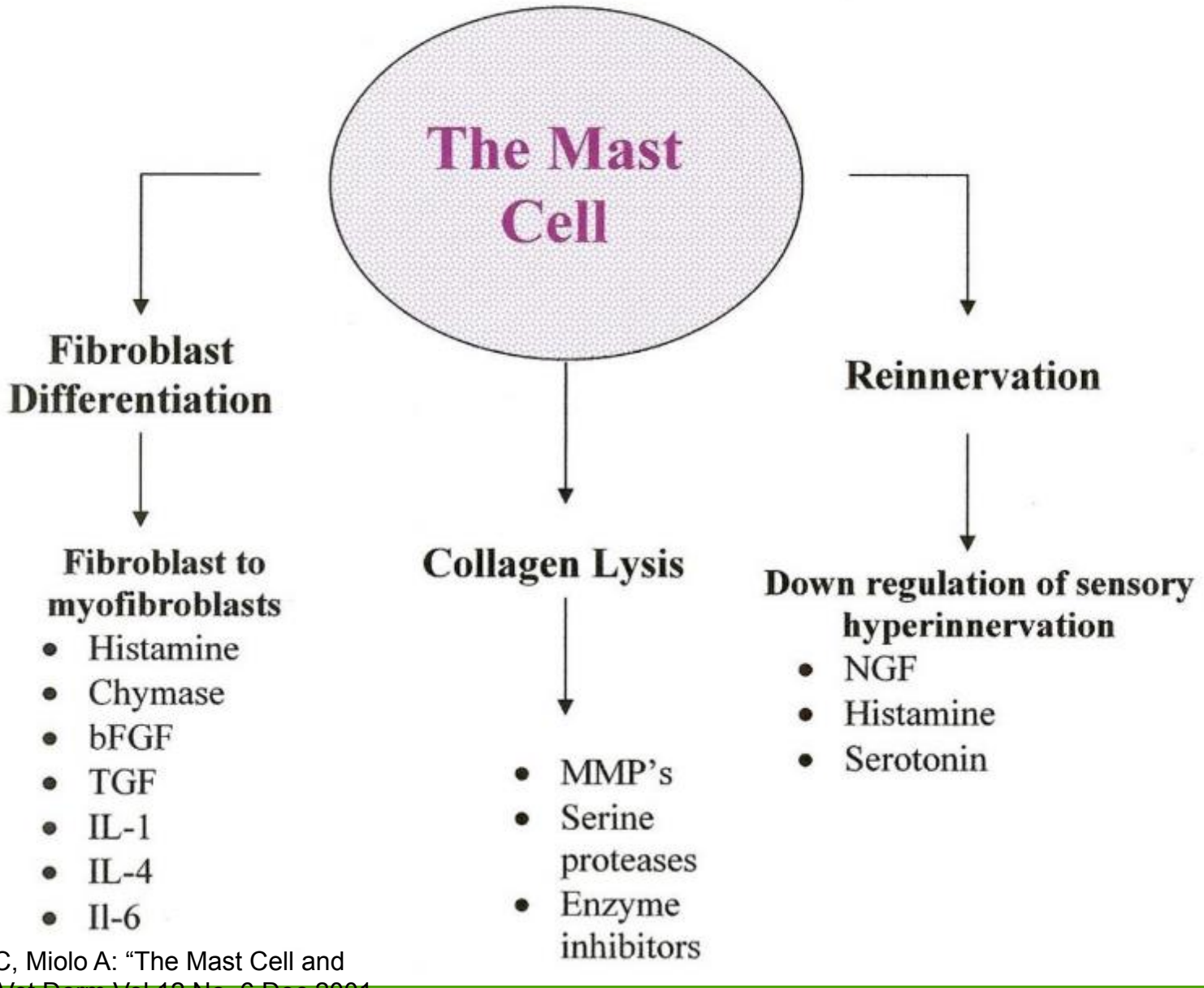
Taken from Noli C, Miolo A: "The Mast Cell and Wound Healing" Vet Derm Vol 12 No. 6 Dec 2001

Mast Cell and the Proliferative Phase



Taken from Noli C, Miolo A: "The Mast Cell and Wound Healing" Vet Derm Vol 12 No. 6 Dec 2001

Mast Cell and Remodeling



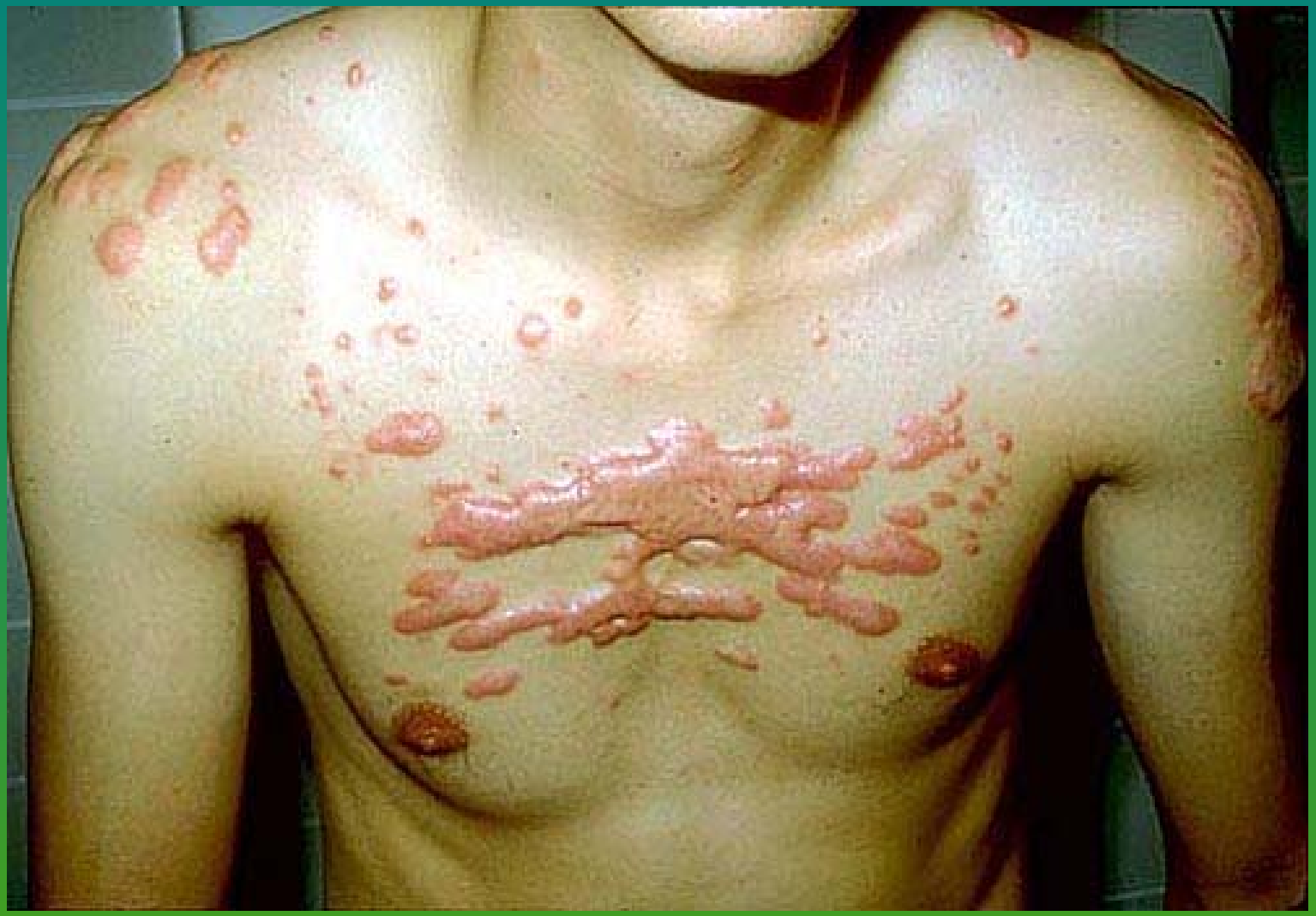
Taken from Noli C, Miolo A: "The Mast Cell and Wound Healing" Vet Derm Vol 12 No. 6 Dec 2001

Pathology

- Wound healing studied as straight forward process
- Complications **usually** occur
  - Infection, thrombosis, ischemia, repeated trauma, etc.

Pathology

- Human Keloids vs. Hypertrophic Scars
  - Keloids:
    - Black individuals predisposed
    - Invasive
    - Abundance of collagen III
    - Problem with proliferative phase
  - Hypertrophic Scars:
    - Genetic factors – but not clearly defined
    - Do not tend to be invasive
    - Decreased collagen lysis – abundance of collagen I
    - Problem with remodeling phase
  - Both show mast cell hyperplasia



Pathology

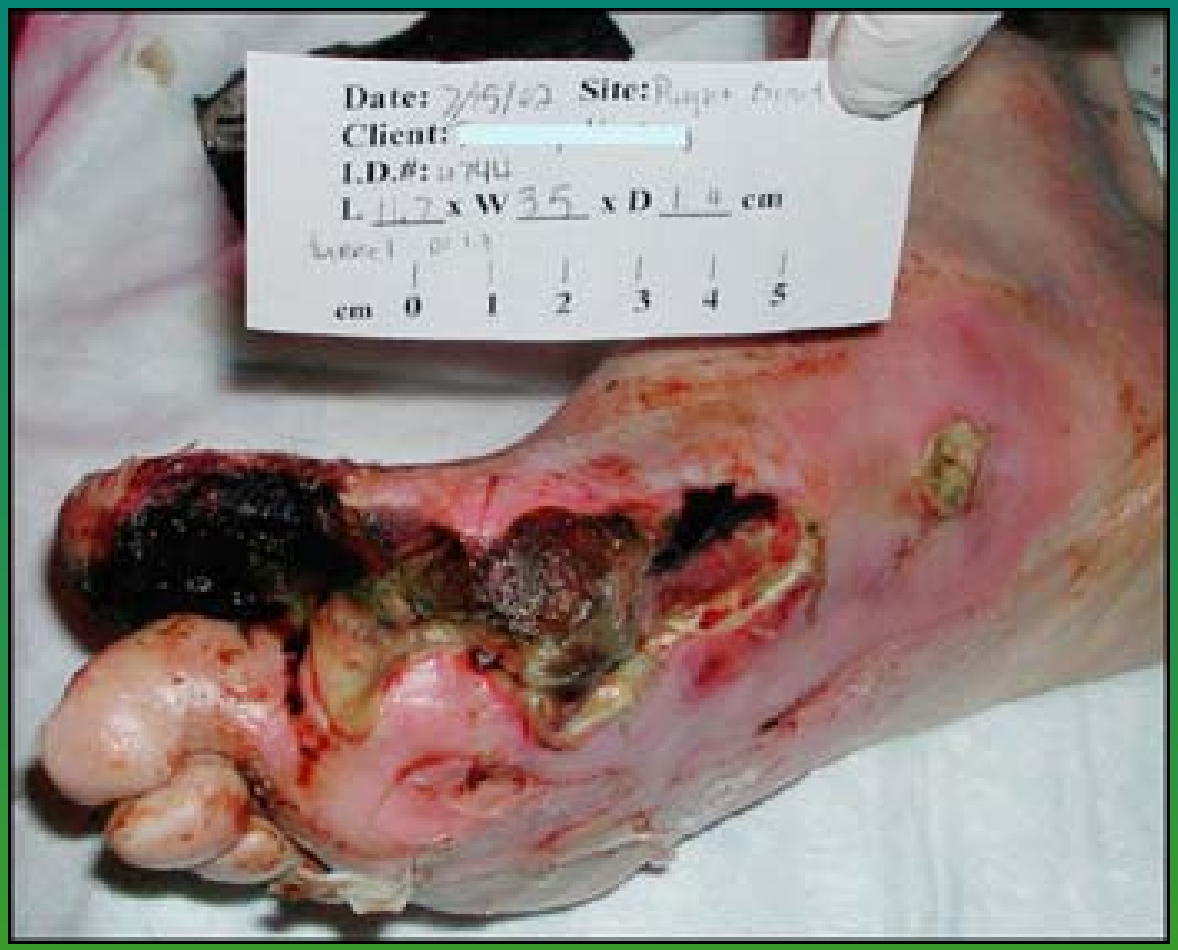
- Equine exuberant granulation tissue
  - Most commonly compared to keloid
    - They are invasive
    - Lacks epithelial cover (unlike keloid)
    - TGF-β1 is increased and induces general protein synthesis – this does not occur in keloids
      - In keloids TGF-β1 induces 12-fold increase in ECM matrix formation – but not general protein increase
    - Various studies looking at anti TGF-β1 and –β3 serum application to these wounds

Pathology

- Equine exuberant granulation tissue (con't)
  - Hypoxia (bandaging) induces the phenomenon
  - Application of corticosteroids can decrease excessive inflammation and retard fibroblasts – however will also retard epithelialization, thus extending the problem

Pathology

- Human diabetic ulcers
  - Chronic non-healing wound
  - Wound “stuck” in proliferative phase with excessive ECM
  - Cells possibly altered – unresponsive to growth factors – debridement
  - Hyperglycemia reduces MMP’s
  - Macrophages show decreased cytokine release
  - …PHOTO WARNING….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Wound Dressings

- Ideal Dressing:
  - Capable of retaining humidity while debris is removed
    - Impermeable to bacteria
    - Allow gas exchange
  - Free of particulates or toxic component
  - Provide protection from trauma while preventing self removal

Wound Dressings

- Ideal Dressing – Con't
  - Removable without causing further damage or pain
  - Doesn't require frequent changing
  - Provides thermal insulation
  - Cost effective and long shelf life

Wound Dressings

- Dressing Types: Each has advantages/disadvantages based on the wound type and location
  - Low-Adherent Dressings
  - Semi-permeable films
  - Hydrocolloids
  - Hydrogels
  - Alginates
  - Foam Dressings
  - Antimicrobial dressings

Wound Dressings

- Low Adherent Dressings
  - Inexpensive
  - Easily available
  - Allows exudate to absorb through a secondary dressing
    - Maintaining a moist wound bed
  - Prevent dressing from adhering to the wound

Wound Dressings

- Semi-permeable films:
  - Sterile plastic sheets of polyurethane coated acrylic adhesive
  - Primary wound cover
  - Not on highly exudative wounds
  - Impermeable to fluids and bacteria – but permeable to air and water vapor
  - Flexible – good for difficult sites

Wound Dressings

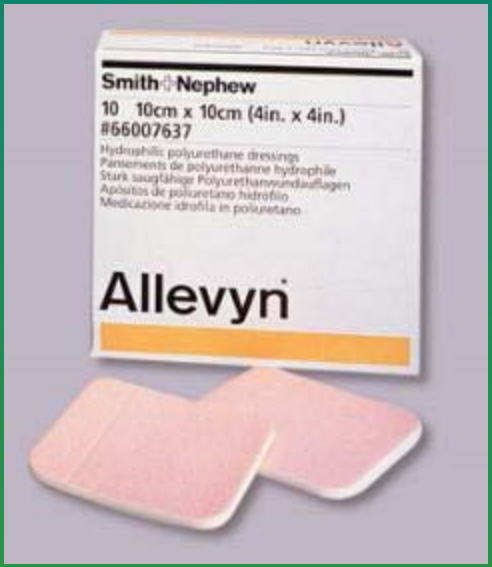
- Hydrocolloids
  - Colloids
    - Carboxymethylcelluose
    - Gelatin
    - Pectin
    - Elastomers
  - Forms gel on wound surface
    - Gel can be mistaken for pus
  - Water/air permeable – rehydrate wounds/eschars
  - Good for highly exudative wounds

Wound Dressings

- Hydrogels
  - Insoluble polymers with 96% water content
    - Provide water to wound
    - Amorphous hydrogels are most commonly used
  - Polymers are only partially hydrated, dressing can still absorb wound exudate
  - Facilitate self debridement (autolysis)

Wound Dressings

- Foam Dressings
  - Polyurethane or silicone foam
    - Vapor permeable
    - Thermal insulation



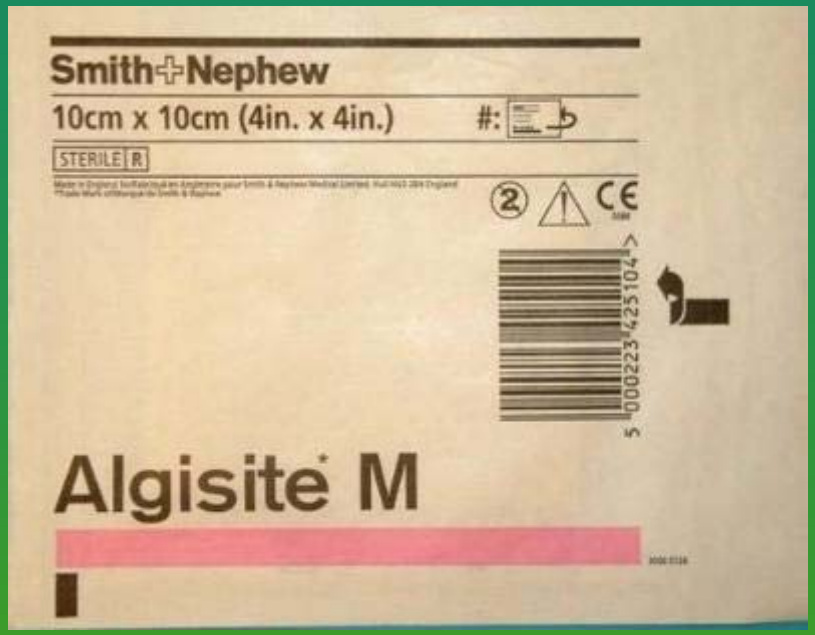
  - Hydrophilic wound contact with hydrophobic backing
    - Highly absorbent
  - Available as cavity dressing
    - Small chips of hydrophillic polyurethane foam in a perforated polymeric film
    - Contain exudate

Wound Dressings

- Antimicrobial dressings:
  - Silver
  - Povidone iodine
  - Cadexomer iodine
  - Metronidazole

Wound Dressings

- Alginates:
  - Calcium and Sodium salts of alginic acid found in brown seaweed: *Phaeophyceae*
    - Some all 100% calcium
    - Some 80:20 calcium:sodium
    - All rich in mannuronic or guluronic acid
  - When contact wound fluid forms hydrophillic gel
  - Can be washed off (mannuronic acid) easily or peeled (glucuronic) off
  - Absorb 15 to 20 times their weight in fluid
    - Excellent for exudative wounds

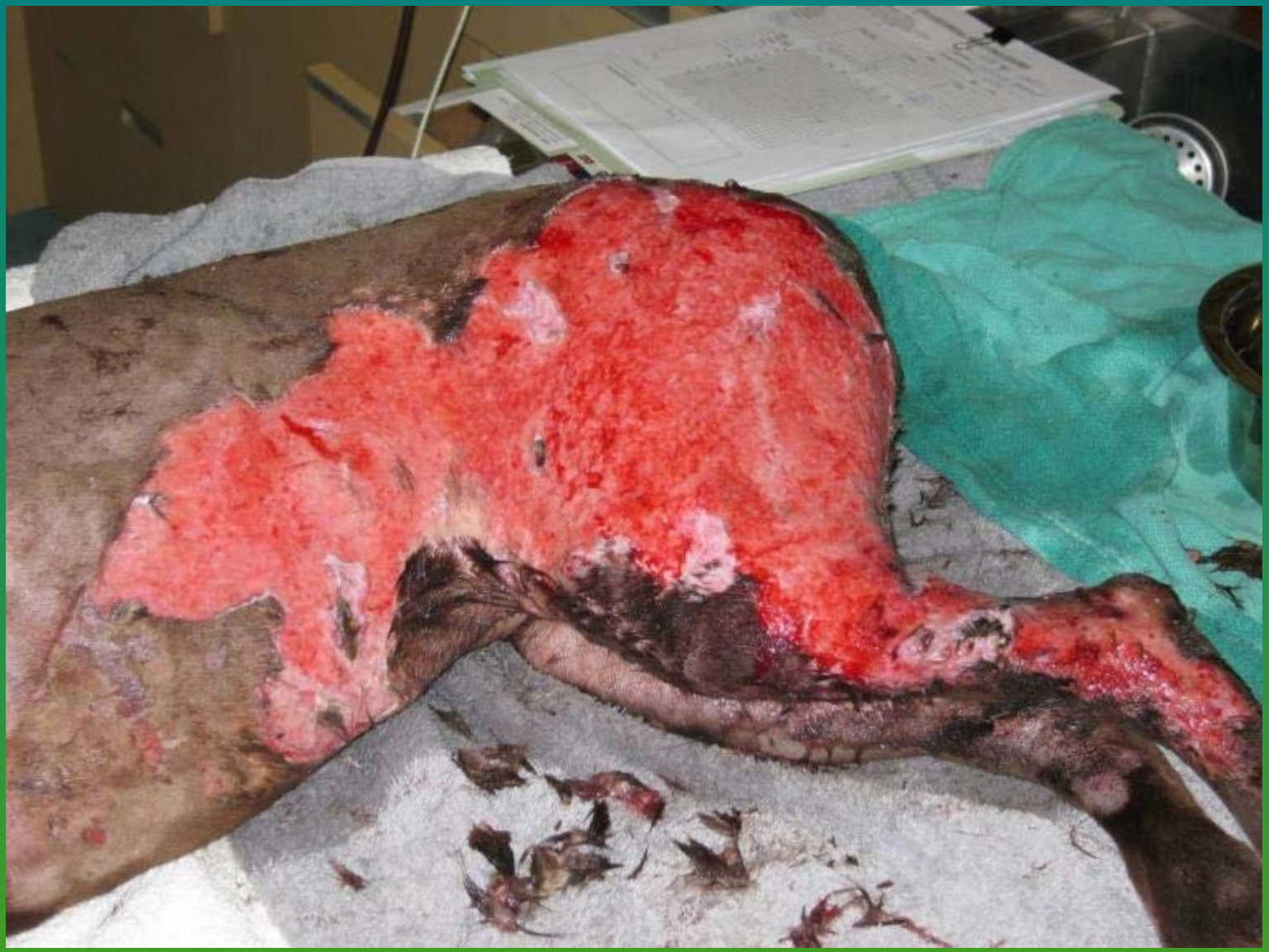


Coco Greene

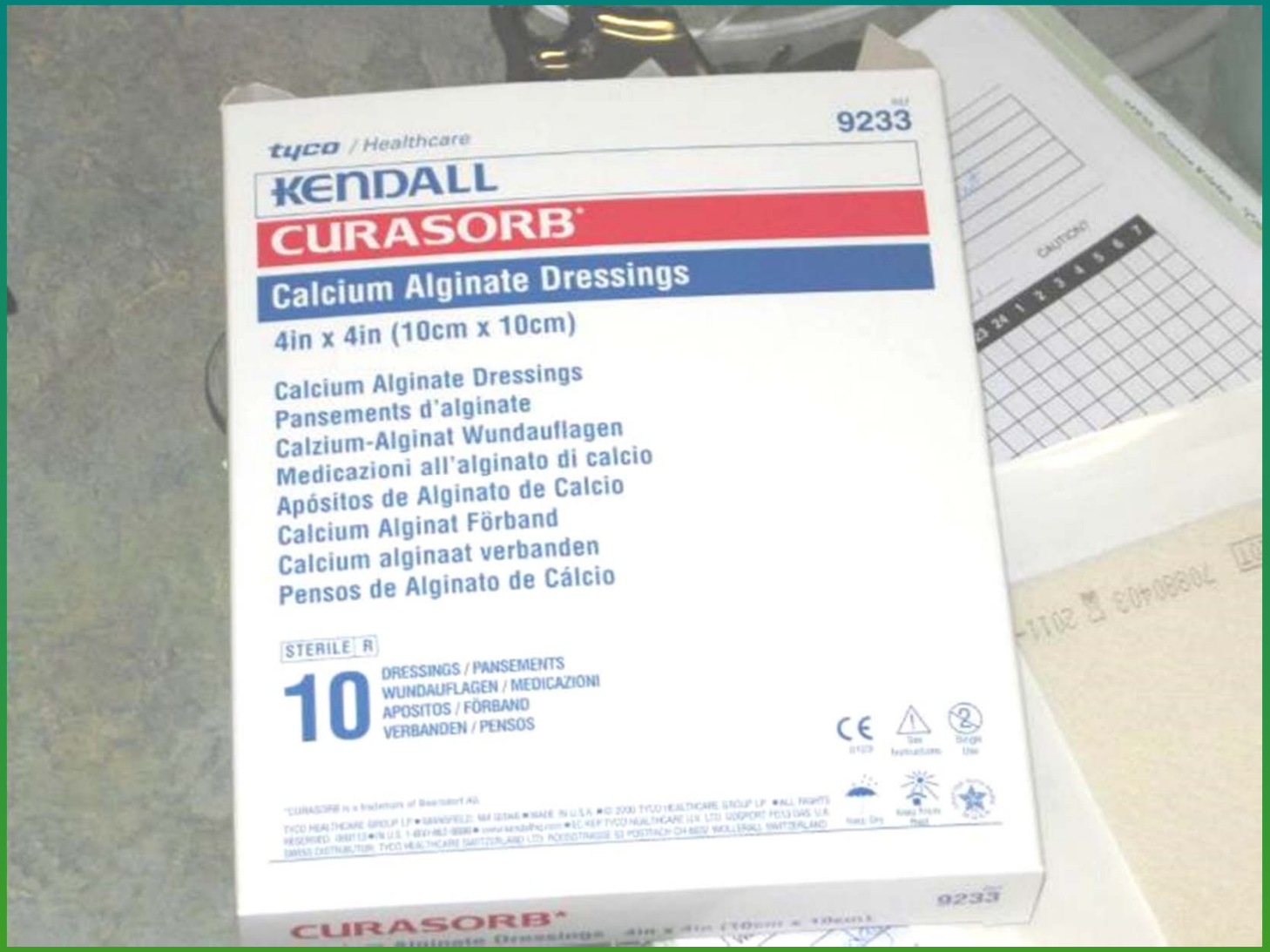
- 4 mo FI Lab
- Thanksgiving day – oil from turkey fryer
- Presented to our surgeons on day 4 after the injury.

Day 4

Day 4



Day 5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Calcium Alginate Dressings
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Calcium Alginate Dressings
Pansements d'alginate
Calzium-Alginat Wundauflagen
Medicazioni all'alginato di calcio
Apósitos de Alginato de Calcio
Calcium Alginat Förband
Calcium alginaat verbanden
Pensos de Alginato de Cálcio
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DRESSINGS / PANSEMENTS
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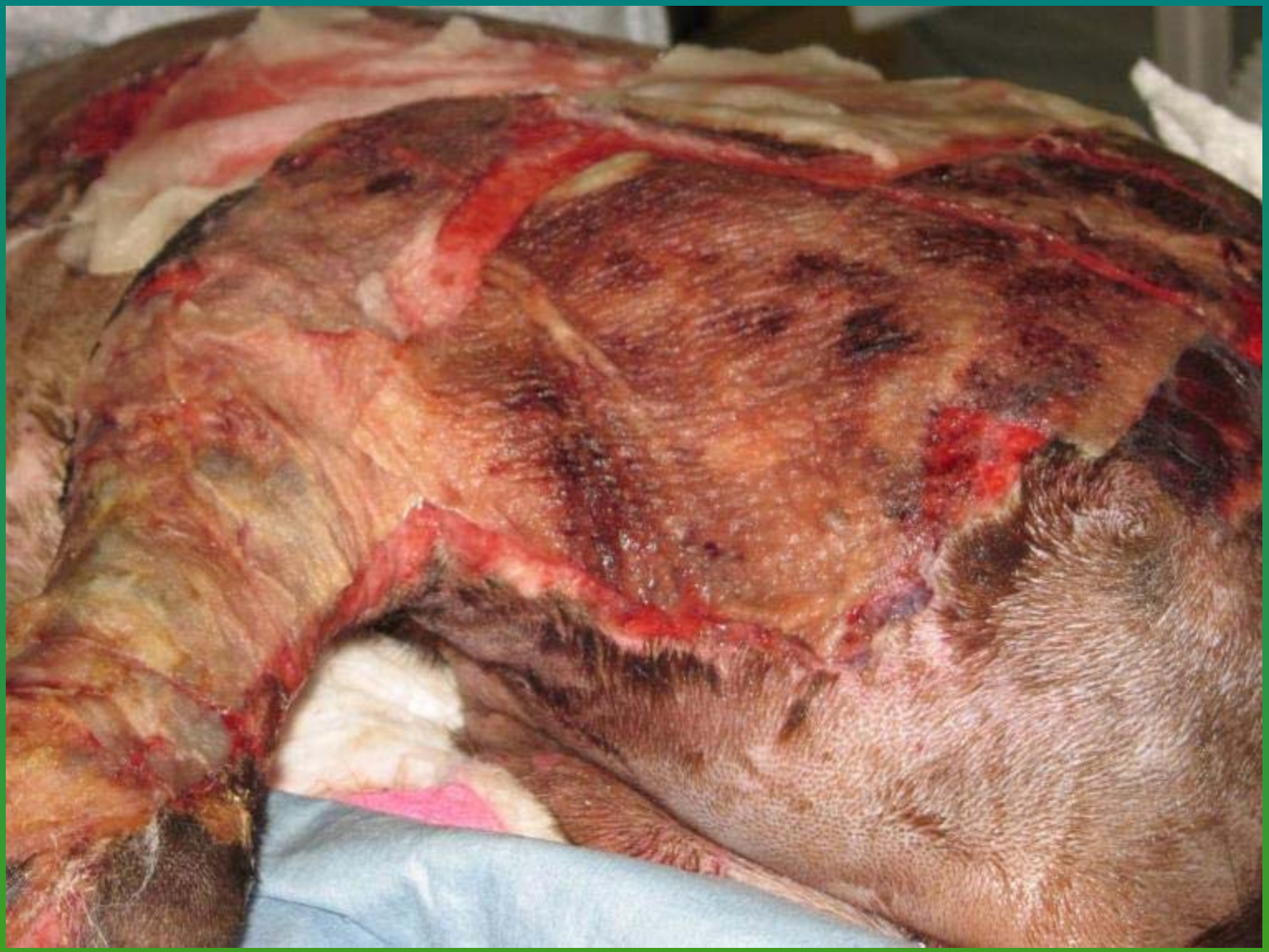
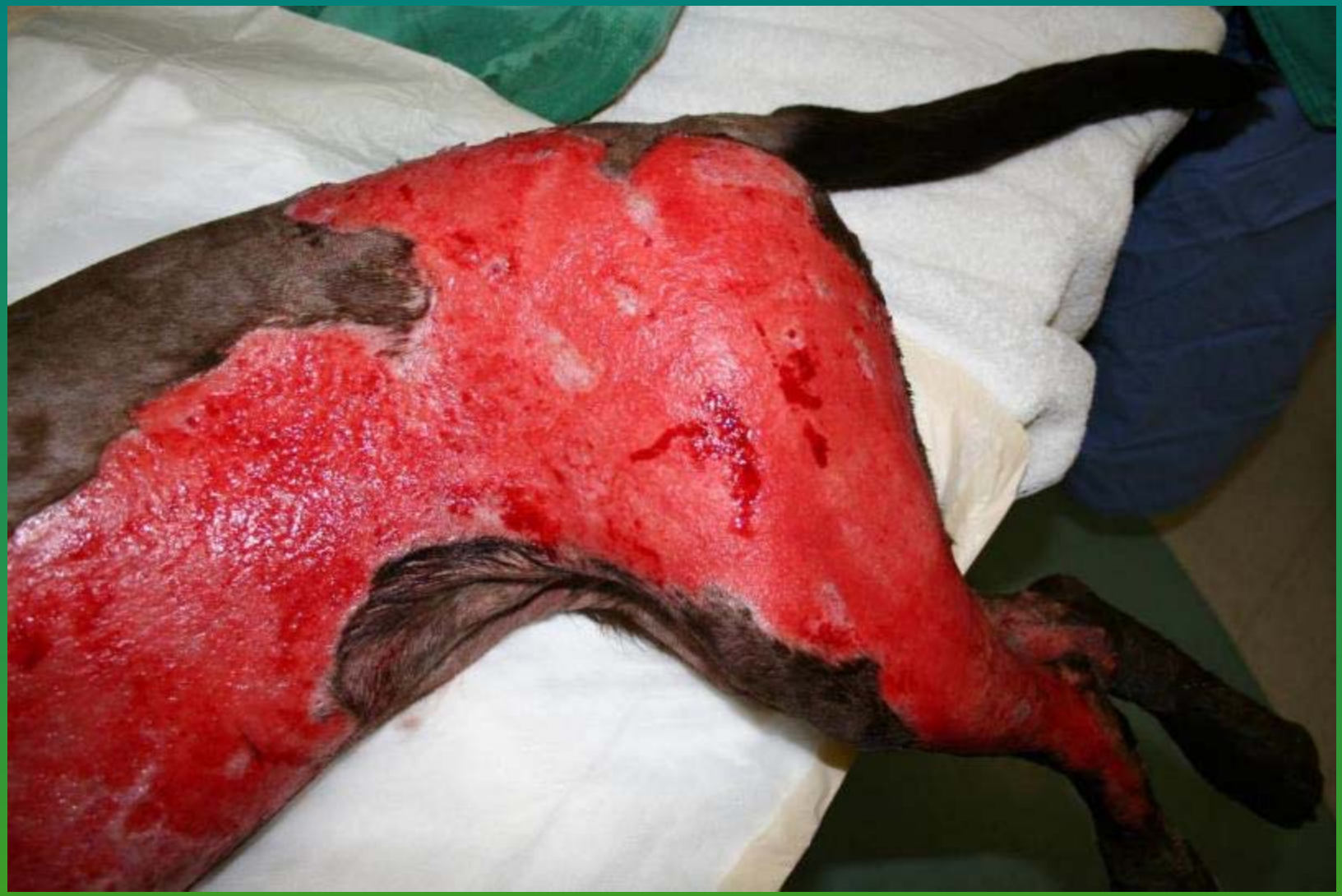
Day 5

Day 5

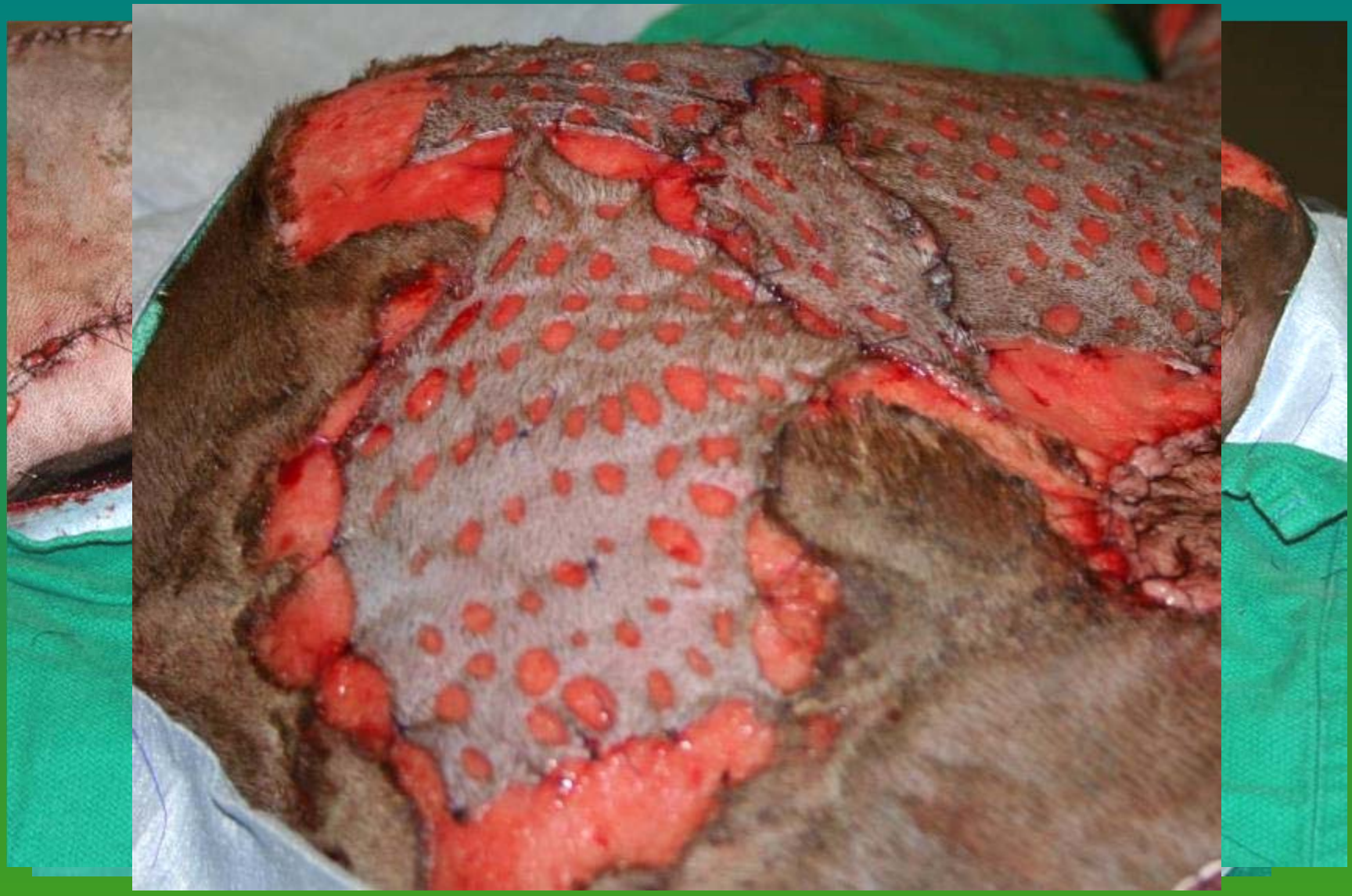


Day 5



Day 8

Day 9



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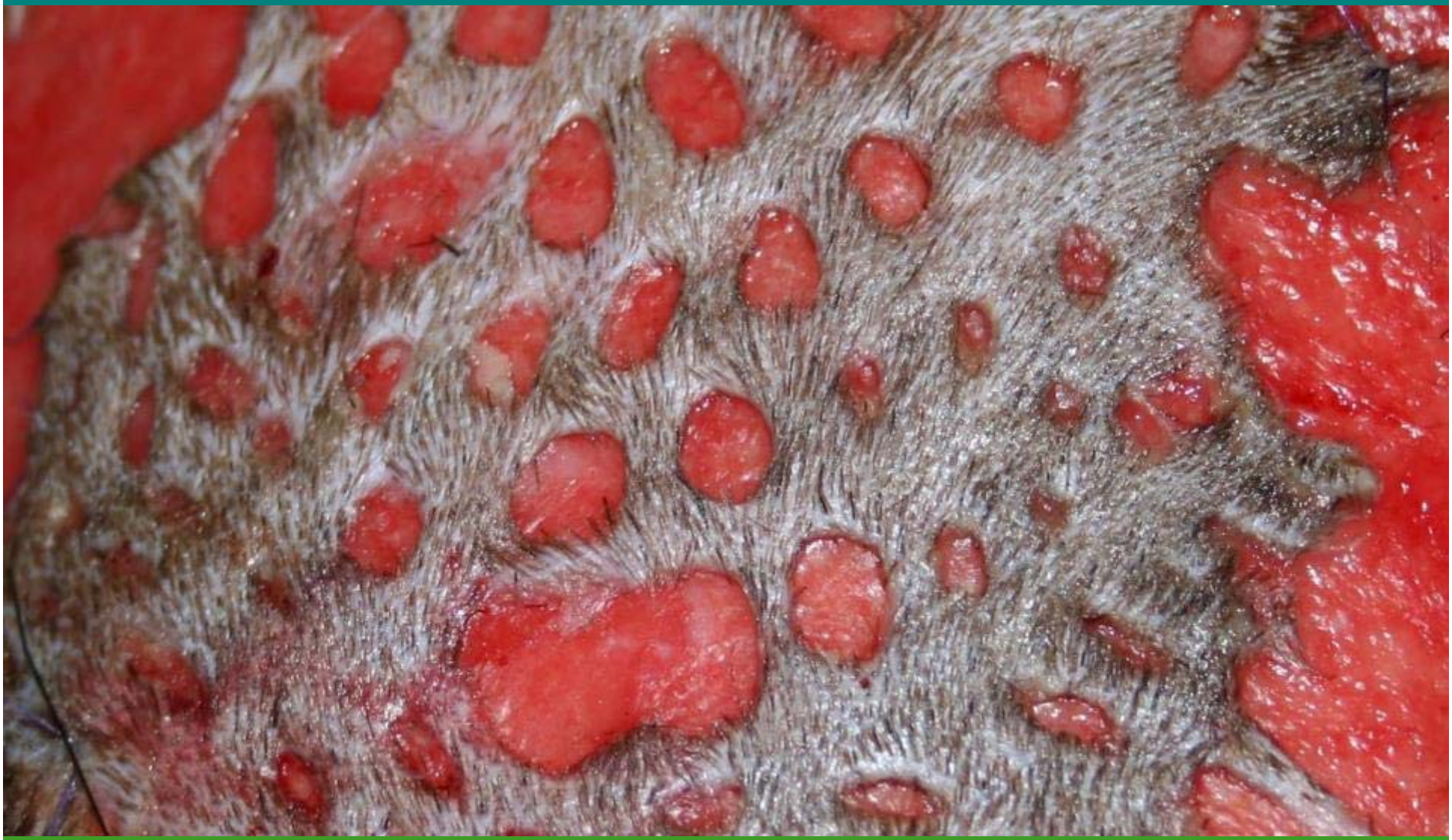
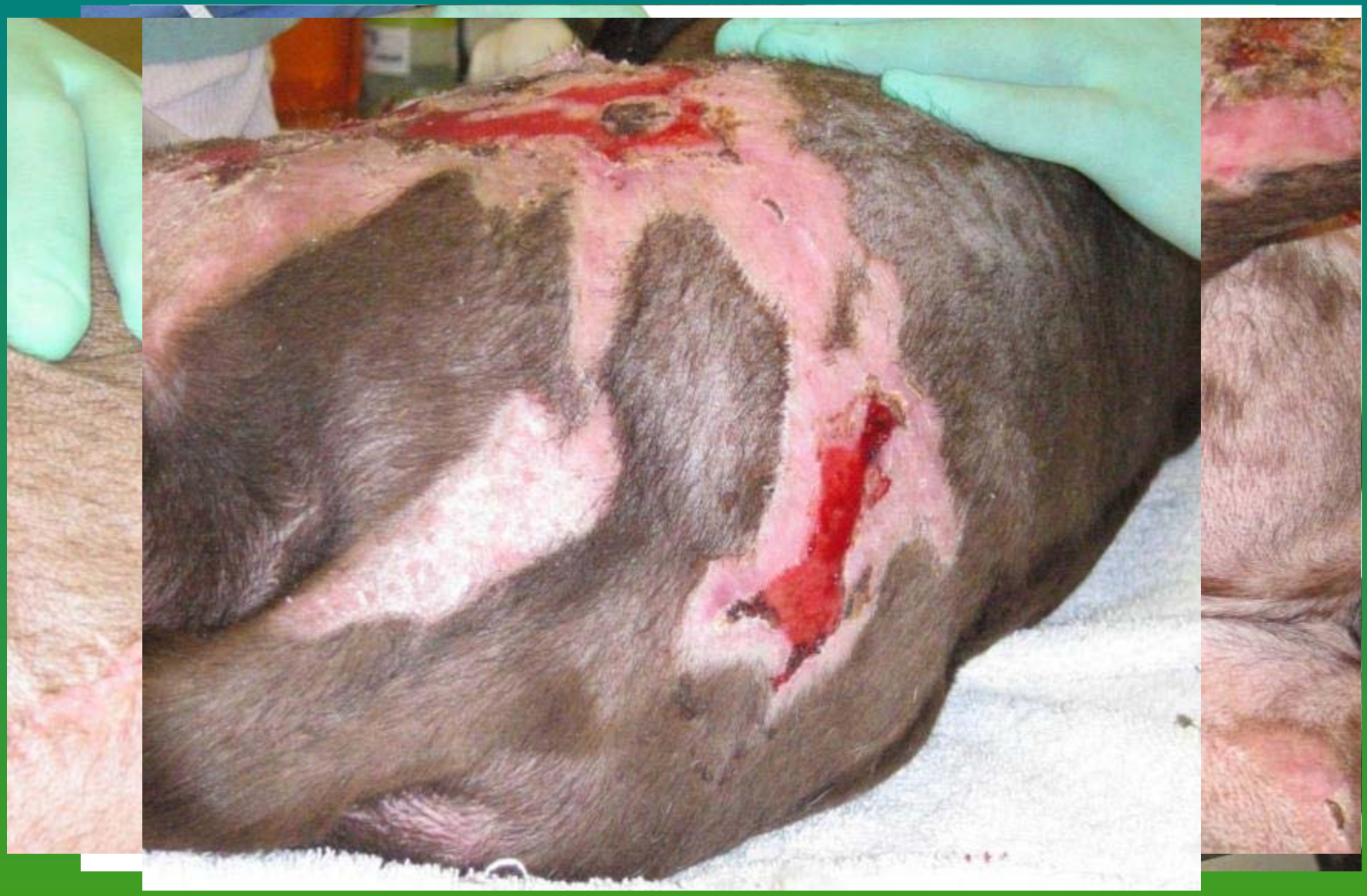
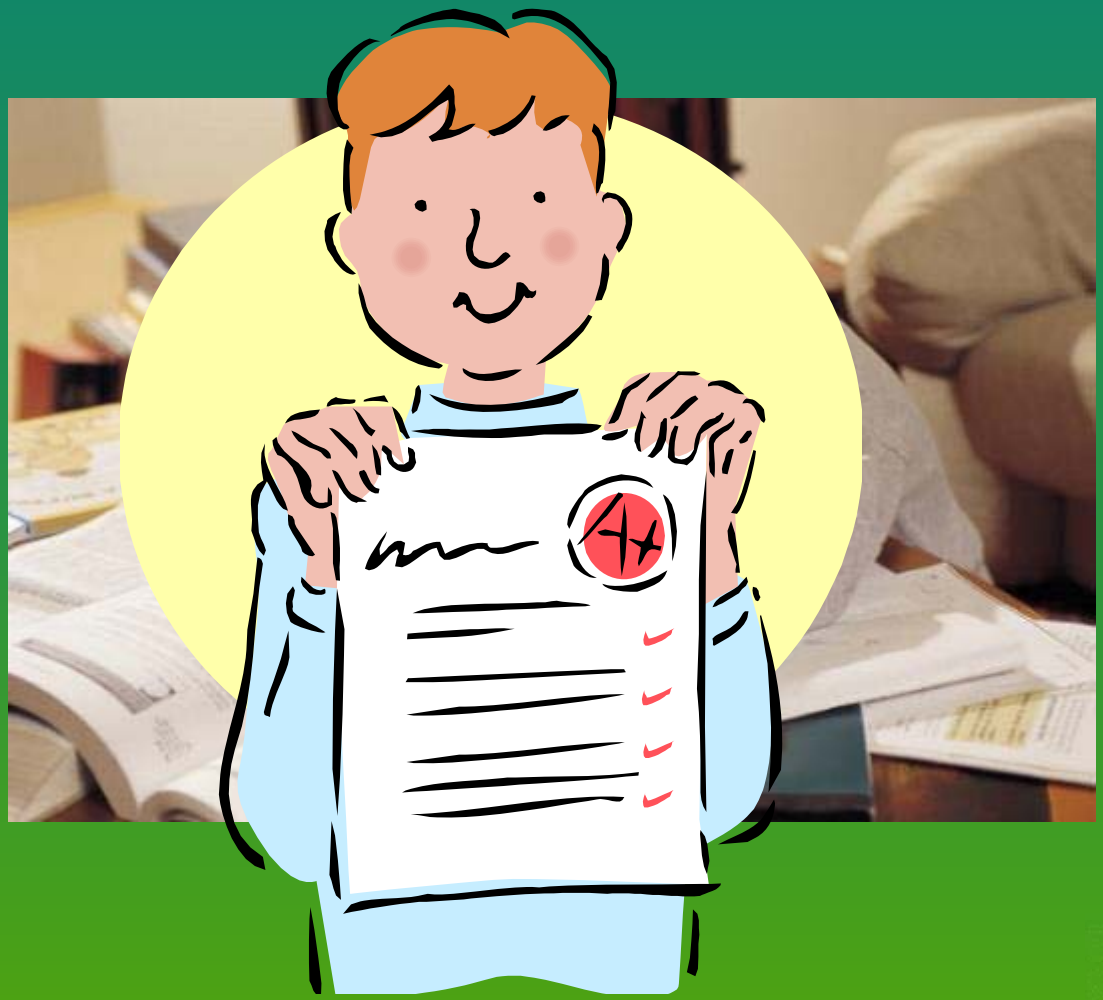


Day 11



Day 25

GOOD LUCK



THEY’RE SINGING!!!!