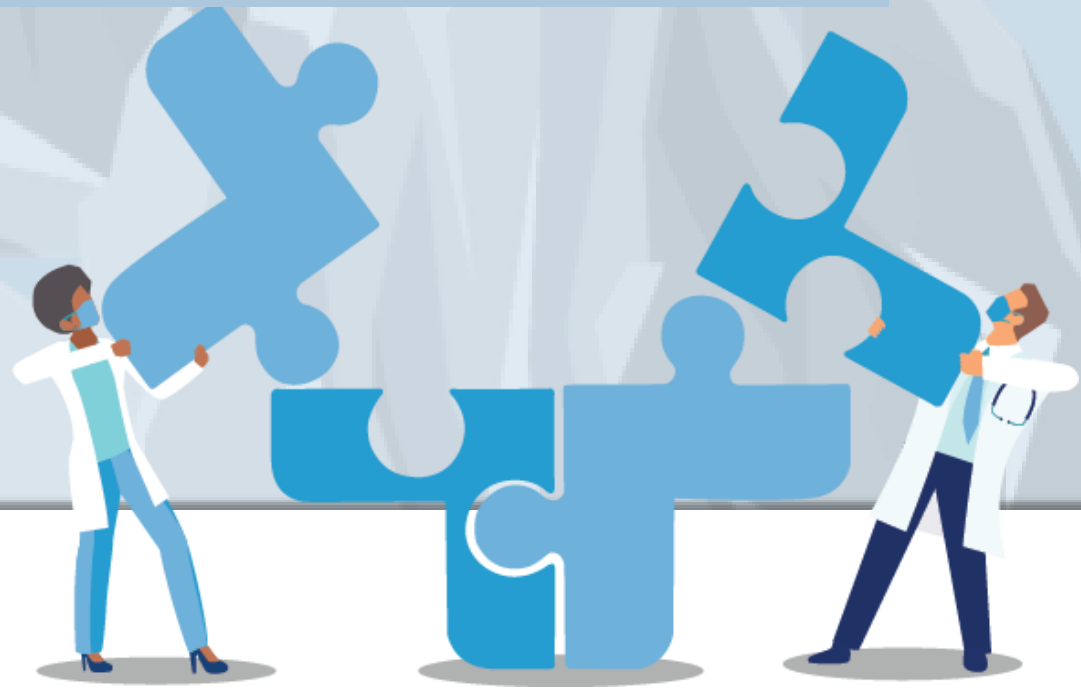
Wound Bed Preparation to Optimize Topical Therapy

1:00pm - 1:30pm Saturday November 12,2022

Gregory Bohn, MD, UHM/ABPM, MAPWCA, FACHM, FAAWC

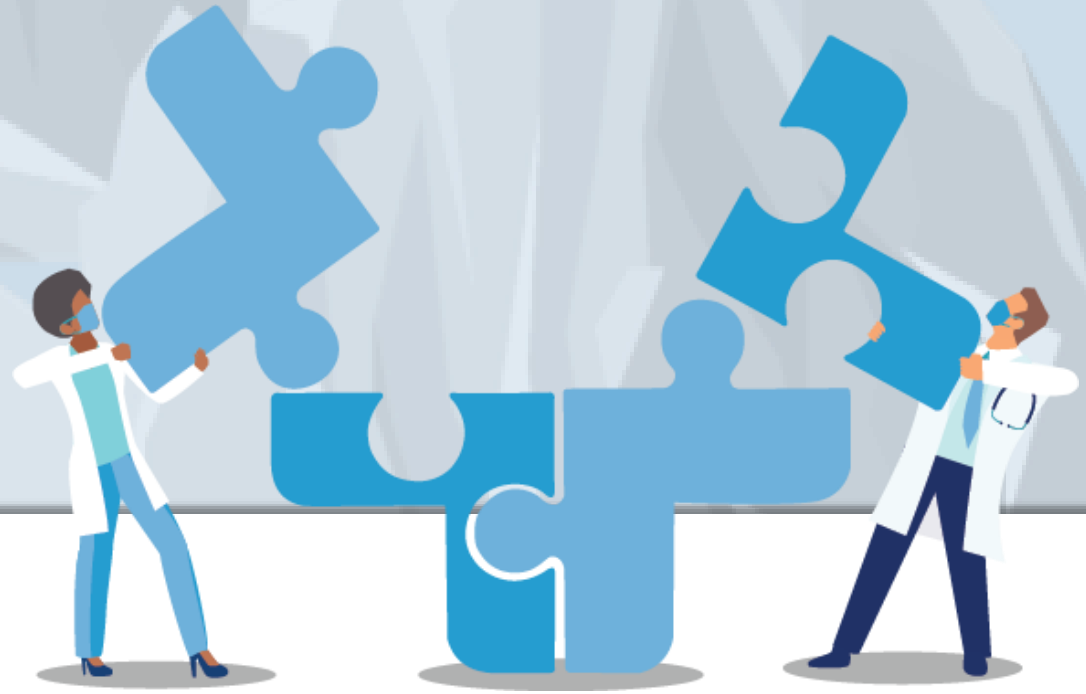


AAWC Association for the Advancement of Wound Care
2022 Annual Conference
Technology in Wound Care

Faculty Disclosures

Consultant:

- Aroa
- ULURU
- Urgo
- Arch Technologies



Objectives

~~Identify Critical Concepts Related to Destruction of ECM~~

Better understand the interplay between Biofilm, MMP production and ECM destruction

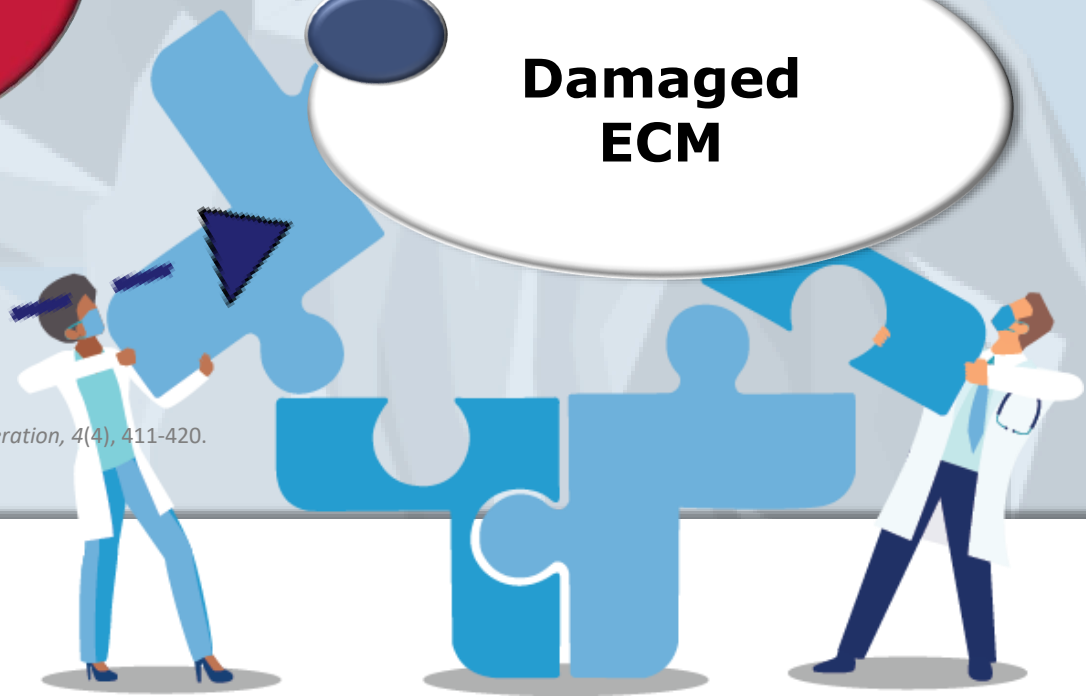
Consider updating treatment approach to reflect change in the model of Chronic Wound Pathophysiology

Increased Bioburden

Chronic Wounds

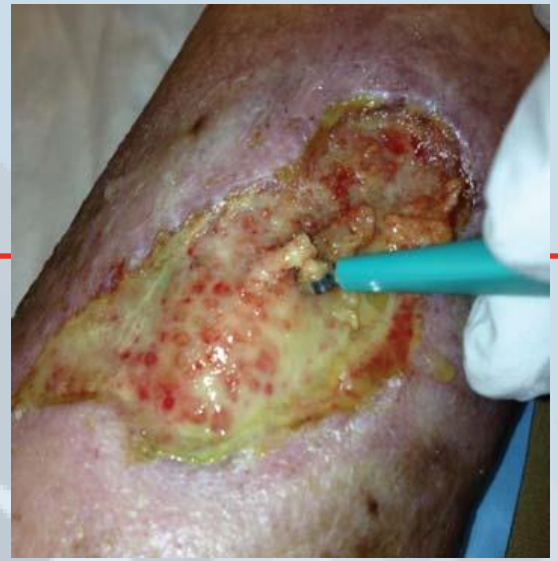
Increased Protease

Damaged ECM



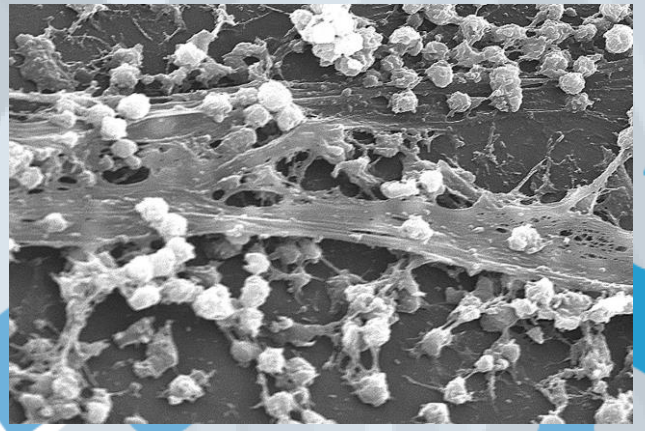
Mast, B., & Schultz, G. (1996). Interactions of cytokines, growth factors, and proteases in acute and chronic wounds. *Wound Repair and Regeneration, 4*(4), 411-420.

Biofilm in Chronic Wounds



Chronic wounds - 78.2 % have chronic biofilm

Acute wounds – 6.0 %



Malone M, Barjnsholt T, McBain AJ, James GA, Stoodley P, Leaper D, Tachi M, Shultz G, Swanson T, Wolcott RD "The prevalence of biofilms in chronic wounds: A systematic review and meta-analysis of published data" Journal of Wound Care, 2017 Jan 2;26(1):20-25.
James G, et al; Biofilms in chronic wounds. Wound Repair and Regeneration: 16(1) 2008 p 37-44.

Chronic Wound Pathophysiology

Repeated Tissue Injury

Ischemia
and
Bioburden-Biofilm

EPA and BPA indicators

Prolonged Elevated Inflammation

↑Neutrophils
↑Macrophages
↑Mast cells

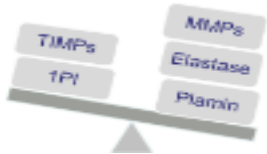
Destruction of Essential Proteins

↑ECM degradation
↓Growth factors/receptors
↓Cell migration
↓Cell proliferation

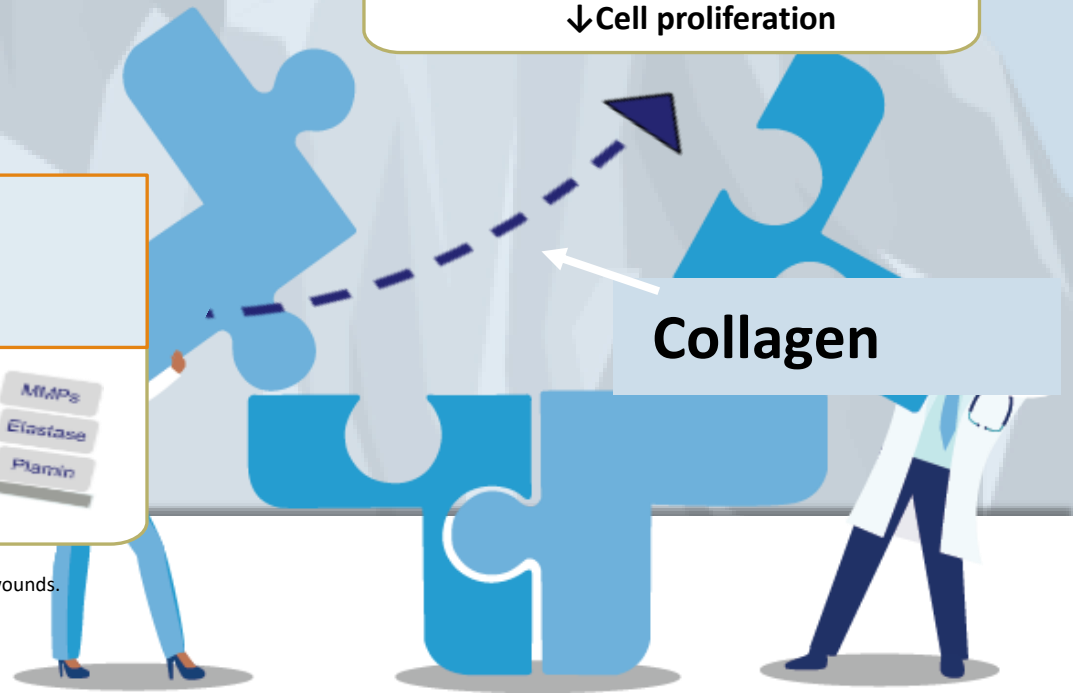
Biofilm Strategy

Imbalanced Proteases and Inhibitors

↑Proteases
↓ Inhibitors



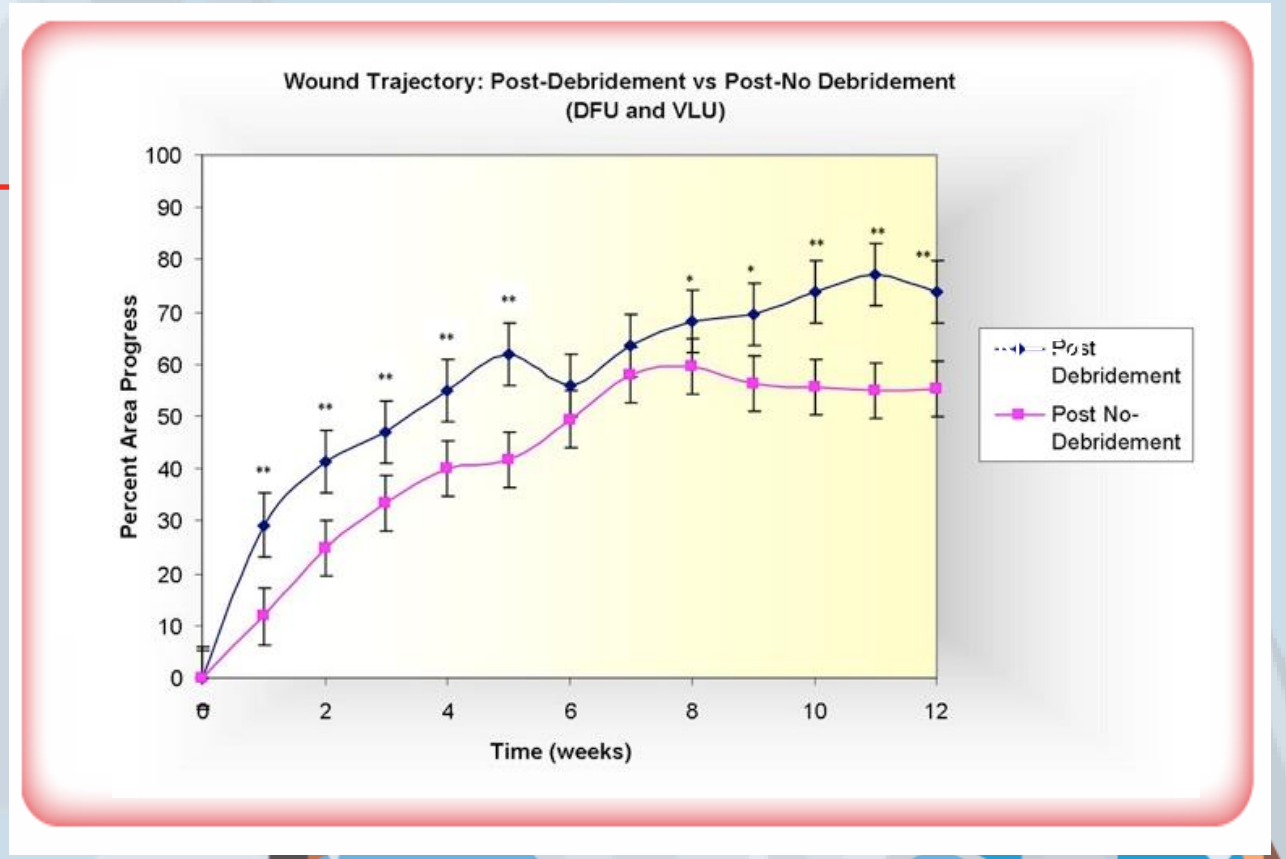
Collagen



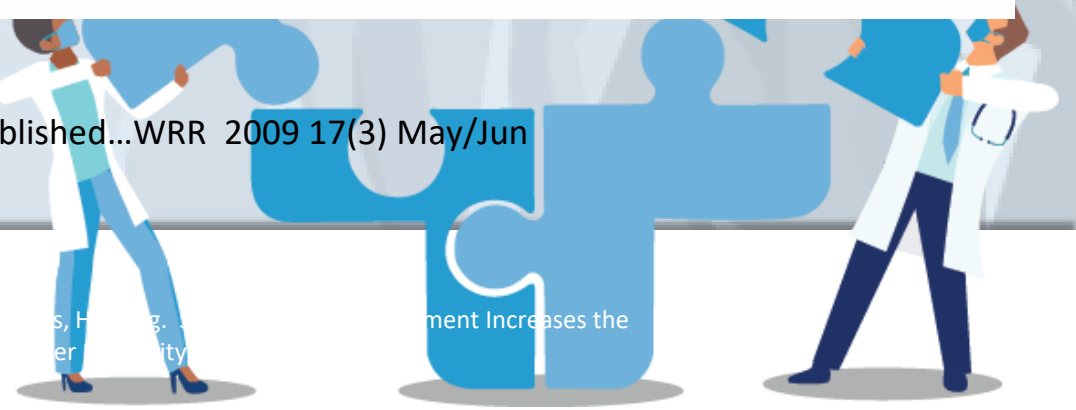
Mast, B., & Schultz, G. (1996). Interactions of cytokines, growth factors, and proteases in acute and chronic wounds. *Wound Repair and Regeneration, 4*(4), 411-420.

Debridement Frequency and Healing DFU/VLU

- Wounds serially debrided within the first four weeks of the treatment period had a median wound area reduction 54% higher than wounds that were not debrided.
- Wounds that *eventually healed* and those that did not ***both*** experienced **greater area reduction** following visits **with debridement**

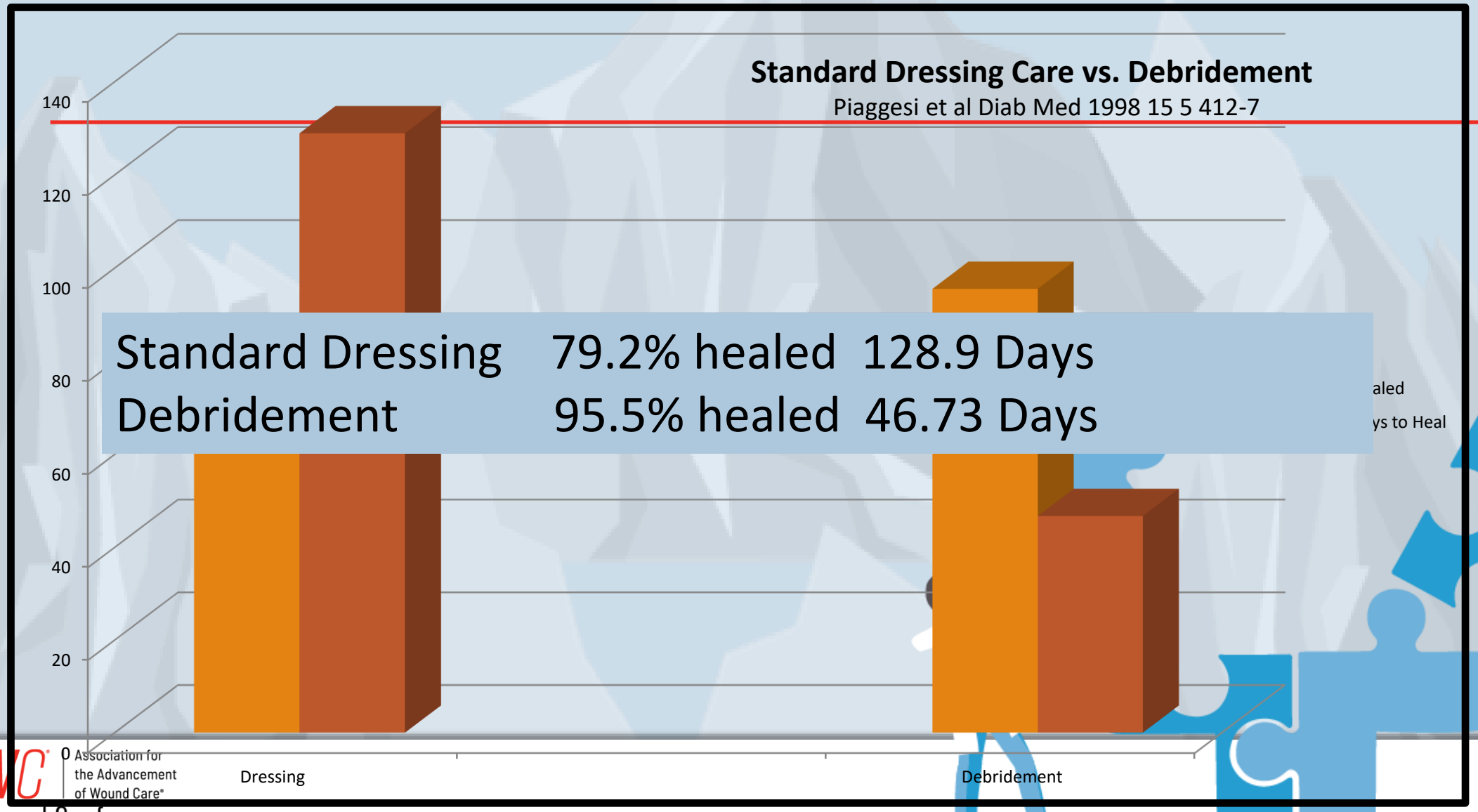


Full study just published…WRR 2009 17(3) May/Jun



ment Increases the

Debridement vs Dressing Treatment



Proactive Therapy

- Enables early and aggressive implementation of Broad-spectrum therapy/ treatment plan early from day 1.
- Resolve inflammation.
- Balance Protease and breakdown of ECM/Healing
- Build tissue restore ECM for tissue development.
- Target both acute and chronic wounds with early intervention

Bohn, G. A., G. S. Schultz, B. A. Liden, M. N. Desvigne, E. J. Lullove, I. Zilberman, M. B. Regan, M. Ostler, K. Edwards, G. M. Arvanitis and J. F. Hartman (2017). "Proactive and Early Aggressive Wound Management: A Shift in Strategy Developed by a Consensus Panel Examining the Current Science, Prevention, and Management of Acute and Chronic Wounds." Wounds 29(11): S37-S42.

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Proactive and Early Aggressive Wound Management:

A Shift in Strategy Developed by a Consensus Panel Examining the Current Science, Prevention, and Management of Acute and Chronic Wounds

Gregory A. Bohn, MD; Gregory S. Schultz, PhD; Brock A. Liden, DPM; Michael N. Desvigne, MD; Eric J. Lullove, DPM; Igor Zilberman, DPM; Mary B. Regan, PhD, RN; Marta Ostler, PT; Karen Edwards, MSS, RN, BSN, WOCN; Georgia M. Arvanitis, PhD; and Jodi F. Hartman, MS

Abstract: Normal wound healing is accomplished through a series of well-coordinated, progressive events with overlapping phases. Chronic wounds are described as not progressing to healing or not being responsive to management in a timely manner. A consensus panel of multidisciplinary wound care professionals was assembled to (1) educate wound care practitioners by identifying key principles of the basic science of chronic wound pathophysiology, highlighting the impact of metalloproteinases and biofilms, as well as the role of the extracellular matrix; and (2) equip practitioners with a systematic strategy for the prevention and healing of acute injuries and chronic wounds based upon scientific evidence and the panel members' expertise. An algorithm is presented that represents a shift in strategy to proactive and early aggressive wound management. With proactive management, adjunct therapies are applied preemptively to acute injuries to reduce wound duration and risk of chronicity. For existing chronic wounds, early aggressive wound management is employed to break the pathophysiology cycle and drive wounds toward healing. Reducing bioburden through debridement and bioburden management and using collagen dressings to balance protease activity prior to the use of advanced modalities may enhance their effectiveness. This early aggressive wound management strategy is recommended for patients at high risk for chronic wound development at a minimum. In their own practices, the panel members apply this systematic strategy for all patients presenting with acute injuries or chronic wounds.

Key words: chronic wounds, wound healing, acute wounds, prevention, wound management, bioburden

Wounds 2017;29(11 Suppl):S37–S42.

BACKGROUND

Prior to a discussion of chronic wounds, a thorough understanding of the normal physiology involved with wound healing is crucial. Normal wound healing is achieved through a series of well-coordinated, progressive events designed to restore the barrier function and mechanical integrity of the skin.[1,2] Wound healing involves biochemical and biomechanical interactions between cells and their microenvironment, of which the extracellular matrix (ECM) with its diverse collection of structural, adhesive, and resilient biomolecules is the primary component. Because the interactions between the cells and ECM are reciprocal and dynamic (ie, continually changing in response to cues from their microenvironment), the term *dynamic reciprocity* was developed to indicate the ongoing, bidirectional interactions between cells and the ECM.[2–4] This concept of dynamic reciprocity provides a context from which to understand developmental processes, tumor growth, and wound healing.[2,3,5–7]

The molecular events associated with wound healing commonly are categorized into 4 phases and are summarized in **Figure 1**. The first phase (vascular response/hemostasis phase) begins upon disruption of blood vessels, which leads to a series of molecular events designed to stop blood loss. These events include vasoconstriction, formation of a platelet plug, and coagulation, during which cells respond to changes in the ECM and vice versa.[1,2,4,8] The second phase (inflammatory) is characterized by the sequential influx of immune cells that have a range of functions, including removal of bacteria, debris, and devitalized tissue.[1,2,4,8] The repair, or proliferative, phase involves the formation of granulation tissue, new blood vessels, macrophages, fibroblasts, and loose connective tissues.[1,2,4,8] Early contraction and reepithelialization also occur during this third phase of wound healing. During the fourth phase (remodeling/maturation), myofibroblasts interact with collagen bundles and growth factors to contract the wound.[1,2,4,8–10] Metalloproteinases (MMPs) are released by macrophages, endothelial cells, and epidermal cells and, besides removing damaged ECM and bacteria, play a necessary role in remodeling the early matrix.[1,2,4,8] Myofibroblasts and fibroblasts replace the early matrix with stronger type I collagen.[2,4] This slow remodeling of collagen, including formation of bundles and crosslinks, progresses to scar formation over several months.[2]

Disclosure: Dr. Bohn is a consultant for Acelity (San Antonio, TX) and Medline (Northfield, IL) and a consultant and speaker for Hollister Incorporated (Libertyville, IL). Dr. Schultz provides research support for Bovie Medical (Purchase, NY) and CorMedix (Bedminster, NJ); is a consultant for Exoxemis, Inc (Little Rock, AR), Organogenesis (Canton, MA), and Hollister Incorporated; provides research support and is a consultant for Medline and Smith & Nephew (London, UK). Dr. Liden is a consultant for Calgon Carbon (Downingtown, PA), Osteosolutions (South Croydon, UK), Tissue Regenix (London, UK), and Vivex (Miami, FL); and a consultant and speaker for Hollister Incorporated. Dr. Desvigne is a consultant for Regenesis Biomedical Inc (Scottsdale, AZ), Board Member of Wound Research Foundation, and a consultant and speaker for Acelity, Smith & Nephew, Hollister Incorporated, and Tissue Regenix. Dr. Lullove is a consultant for Cumberland Pharmaceuticals (Nashville, TN), Hollister Incorporated, Osiris Therapeutics (Columbia, MD), and Skye Biologics (El Segundo, CA). Dr. Zilberman, Ms. Ostler, and Ms. Edwards are speakers for Hollister Incorporated. Dr. Regan is an employee of Hollister Incorporated. Dr. Arvanitis is a consultant for Hollister Incorporated. Editorial support and honorariums for panel members were provided by Hollister Incorporated. Evelyn Quintin, RN, BSN, CWCN, CWS, provided assistance in figure preparation.

Step Down Therapy

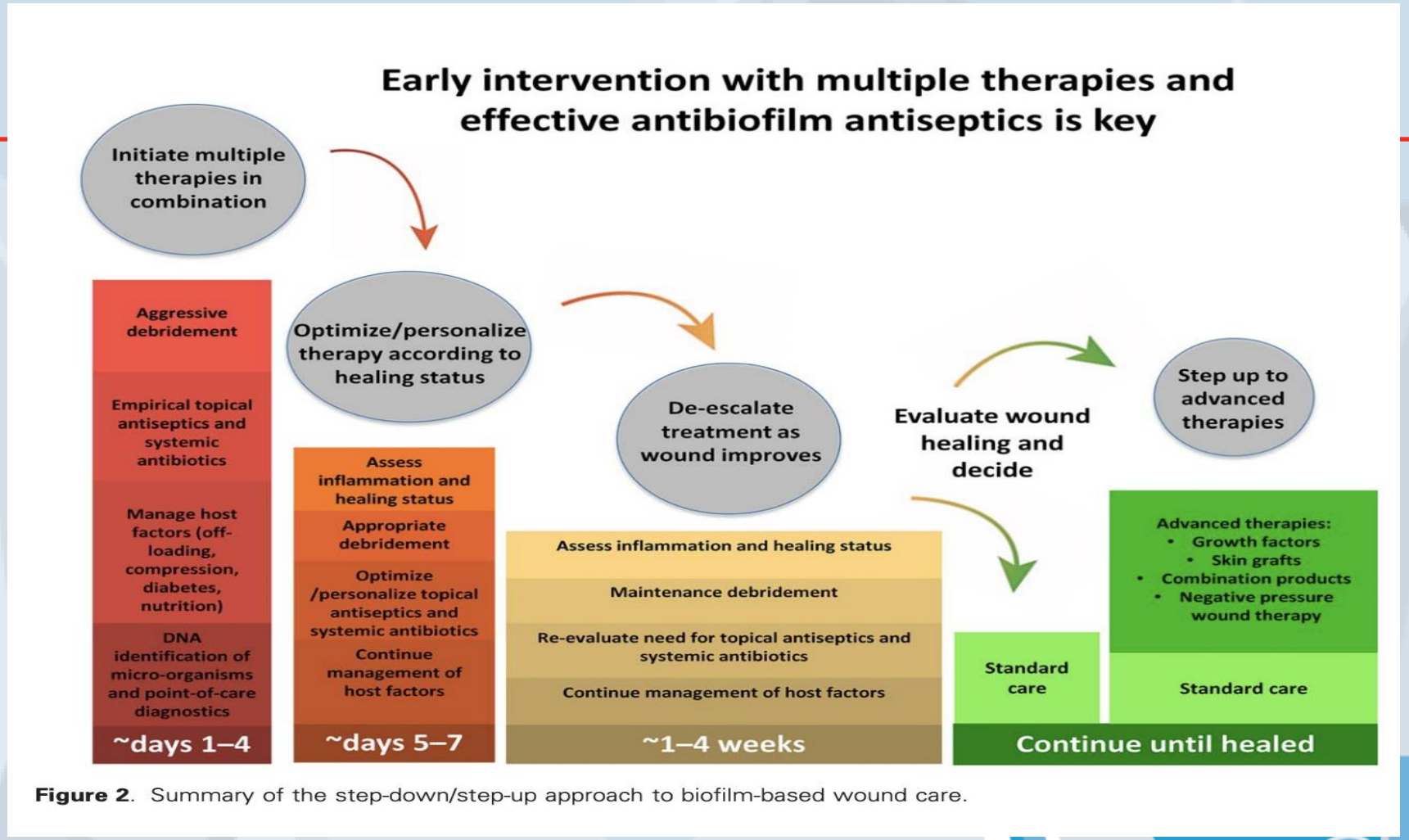

Figure 2. Summary of the step-down/step-up approach to biofilm-based wound care.

Schultz G et al; Consensus guidelines for the identification and treatment of biofilms in chronic nonhealing wounds Wound Rep Reg (2017) 25 744–757

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MMP Degrades Collagen

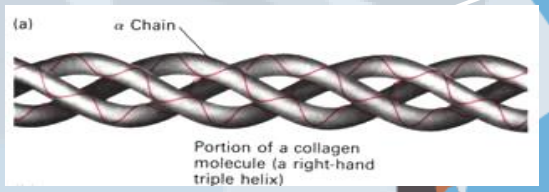
stromelysin
(MMP3)

pro-collagenase
(inactive)
pro-MMP1
pro-MMP8
pro-MMP13

collagenase
(active)
MMP1
MMP8
MMP13

gelatinase
MMP2
MMP9

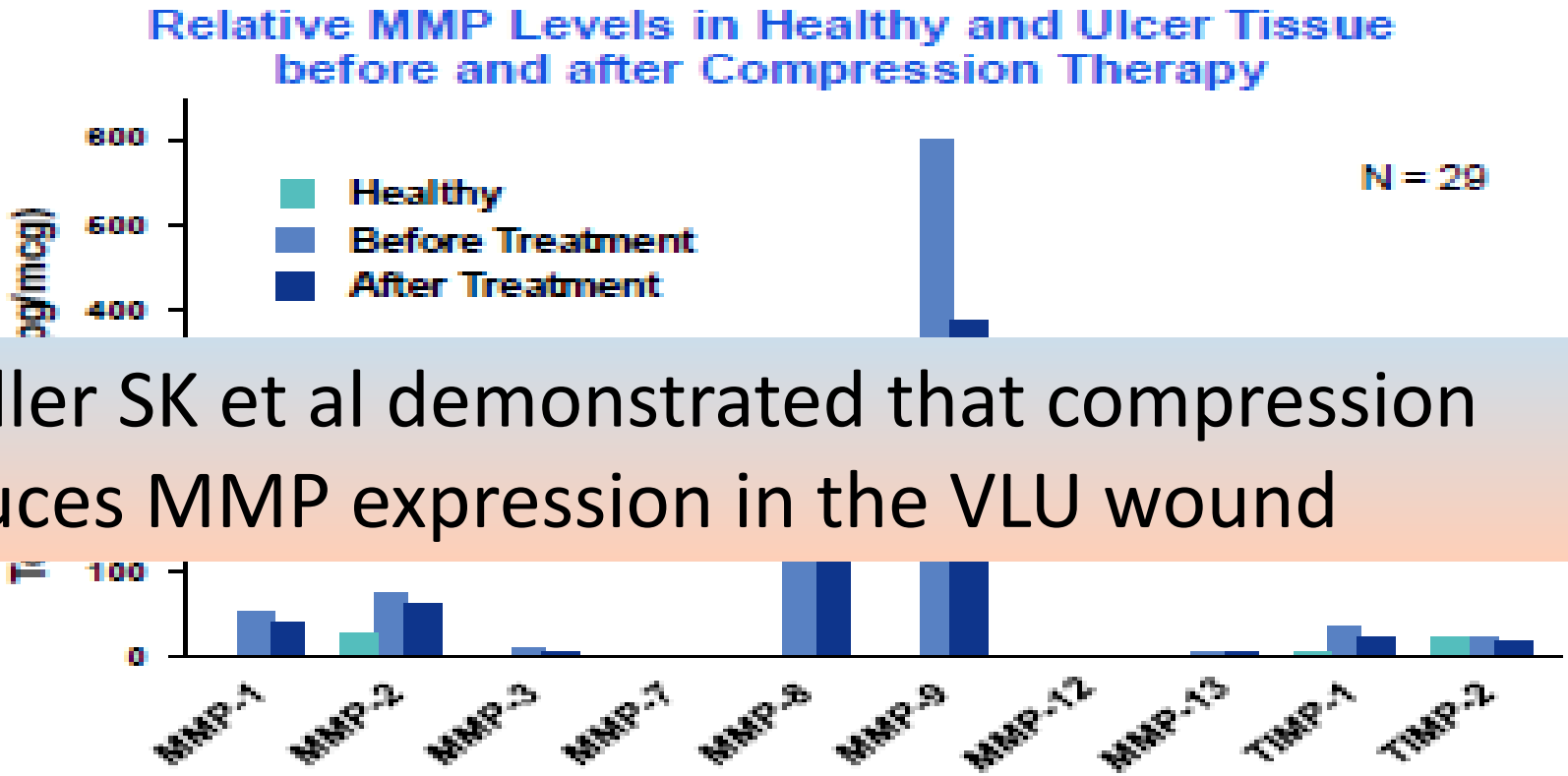
collagen fibril



cleaved collagen fibril

collagen peptides

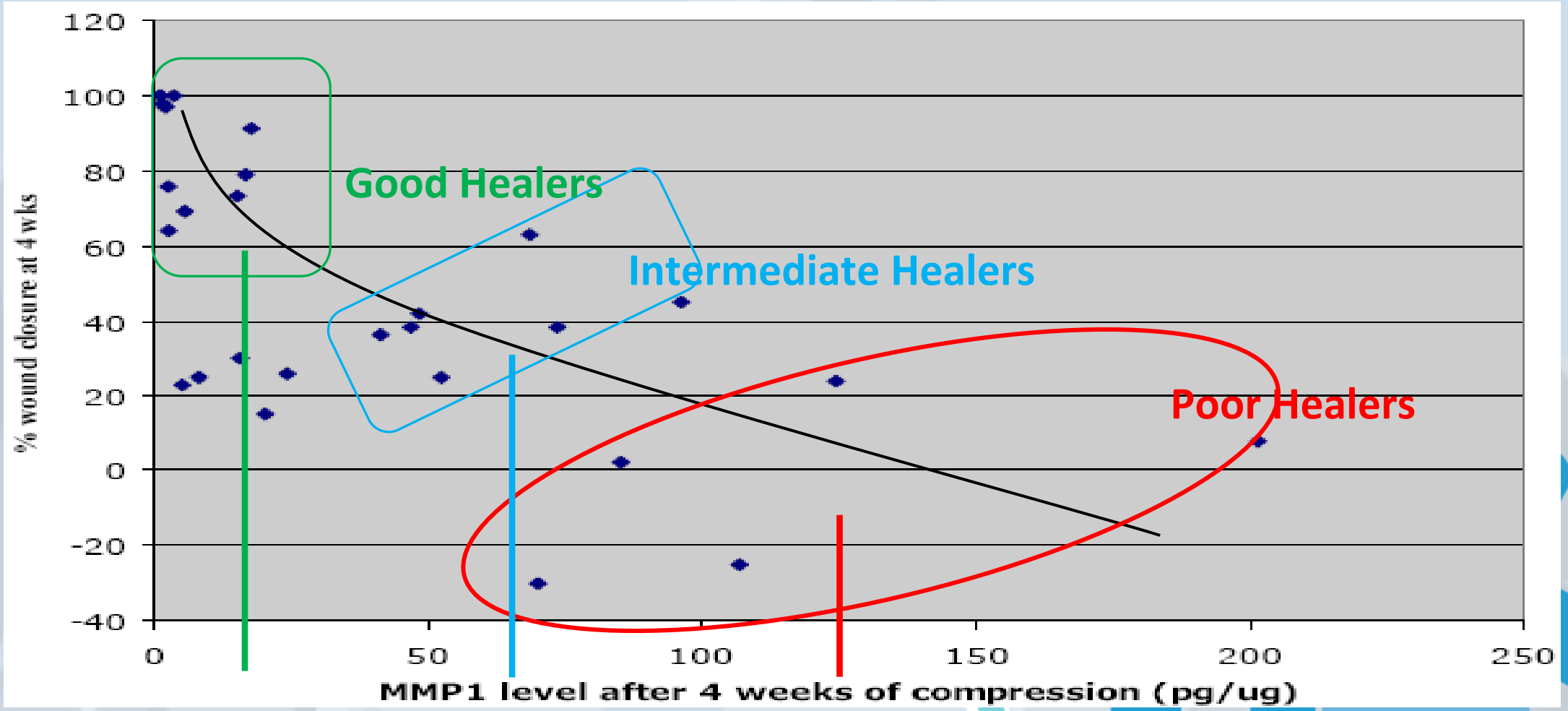
Venous Leg Ulcers are Inflammatory



Beidler SK et al demonstrated that compression reduces MMP expression in the VLU wound

MMP = matrix metalloproteinase; TIMP = tissue inhibitor of metalloproteinase.
Beidler SK, et al. *Wound Repair Regen.* 2008;16(5)642-648. Beidler SK, et al. *J Vasc Surg.* 2009;49(4):1013-1020.

MMP-1 in Venous Ulcers



Beidler, S. K., Douillet, C. D., Berndt, D. F., Keagy, B. A., Rich, P. B., & Marston, W. A. (2008). Multiplexed analysis of matrix metalloproteinases in leg ulcer tissue of patients with chronic venous insufficiency before and after compression therapy. *Wound Repair and Regeneration, 16*(5), 642-648.

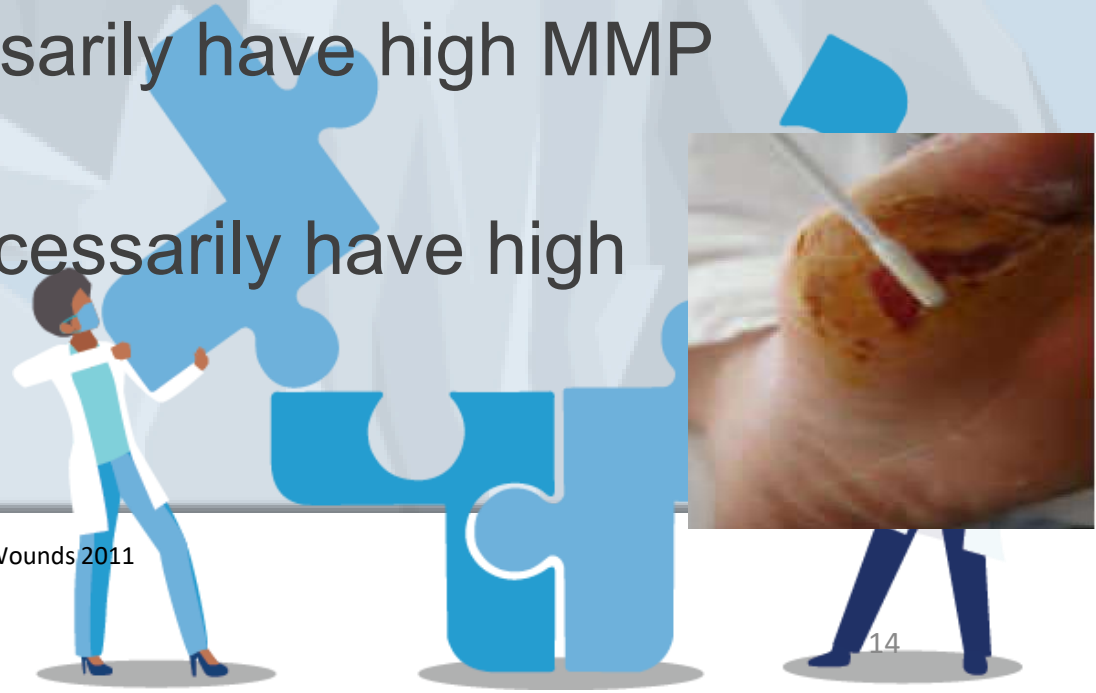
Elevated protease activity and non healing wounds

28% of chronic wounds have elevated protease activity (EPA) as defined by their test (Threshold)

Positive EPA Indicator Chronic Wounds have 90% probability that they won't heal

Wounds with high elastase did not necessarily have high MMP levels

Wounds with high MMP levels did not necessarily have high elastase levels



Serena, Cullen , et al Protease Activity Levels Associated with Healing Status of Chronic Wounds 2011

Multiple proteases contribute to Non Healing

Individual protease is not causative of the excessive protease activity (EPA) Collective of different MMPs

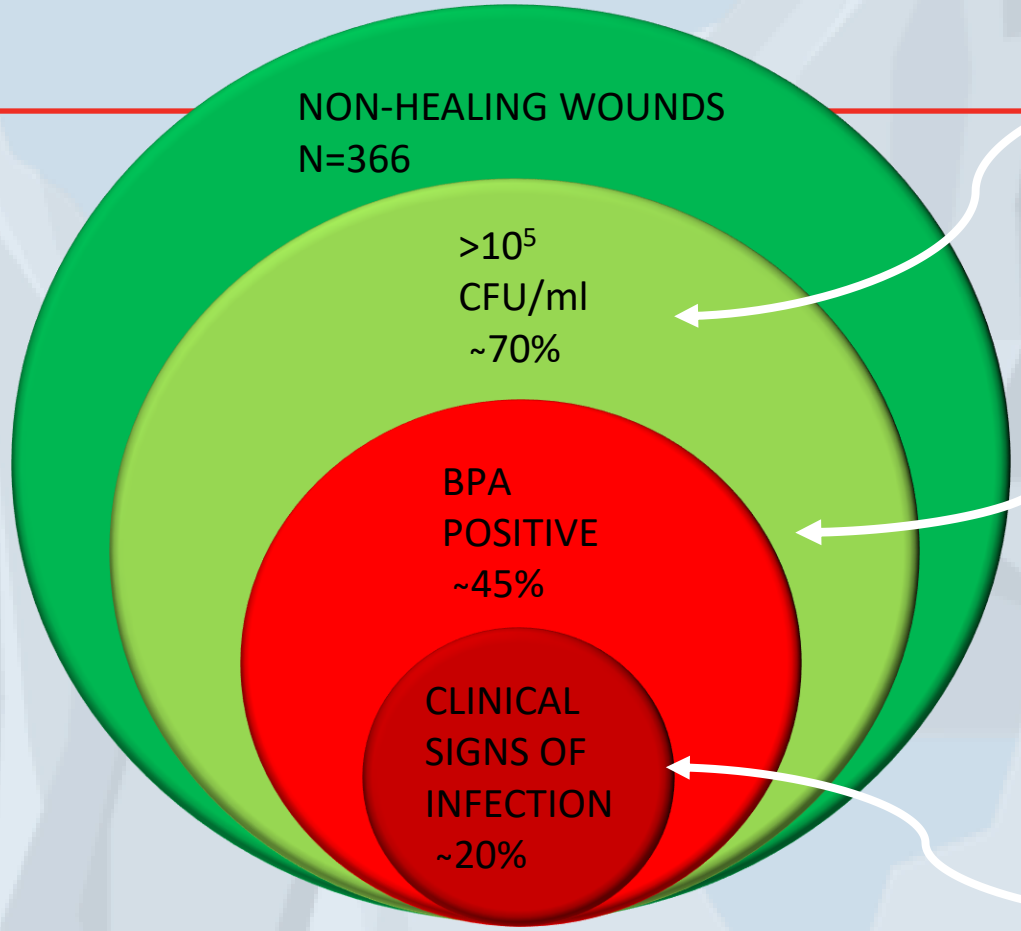
A wound does not need to have high levels of all proteases to be non-healing.

Individual proteases seem to be able to compensate for one another in providing a highly proteolytic wound environment.

This highlights the need to measure multiple proteases in order to determine if proteolytic activity is causing a problem in the wound and preventing it from healing

Serena, Cullen , et al Protease Activity Levels Associated with Healing Status of Chronic Wounds 2011

Clinical utility of detecting bacterial pathogenesis



Reliance on culture can over-diagnose infection

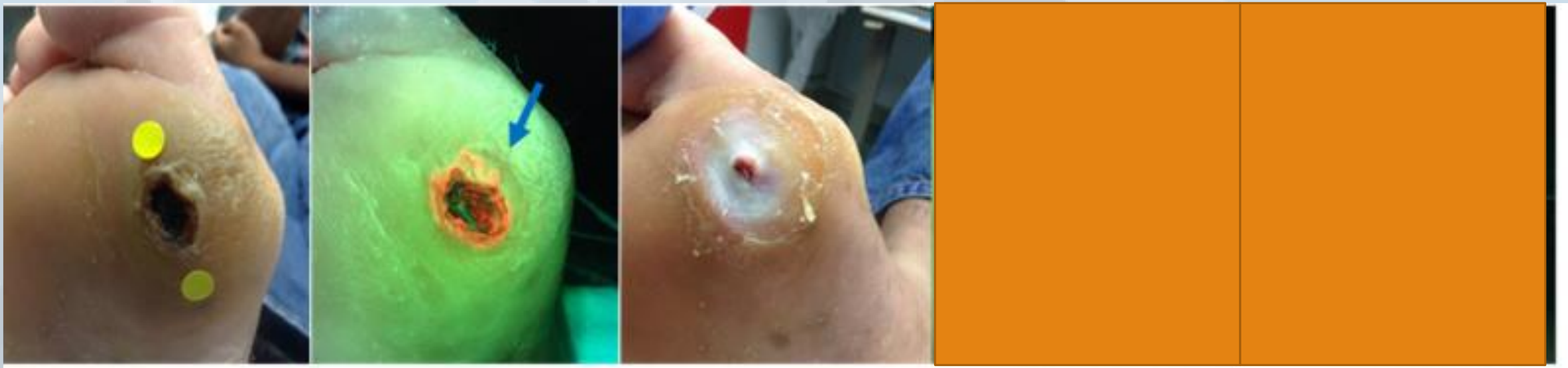
Testing for Bacterial Protease can more reliably diagnosis infective risk

Reliance on clinical signs can under-diagnose infection

1. Serena, et al, Bacterial proteases: A marker for a 'state of pathogenesis' in chronic wounds, 2015
2. Armstrong, Bauer, Bohn, Principles of Best Diagnostic Practice in Tissue Repair and Wound Healing: An Expert Consensus Diagnostics 2021, 11, 50.

Fluorescent Imaging

Directed selective debridement



Initial assessment

After image informed debridement

After subsequent image informed debridement

Serena et,al; Guidelines for Point-of-Care Fluorescence Imaging for Detection of Wound Bacterial Burden Based on Delphi Consensus Diagnostics 2021 11 1219.

Point of Care Protease Assessment

Can wound assessment of protease activity direct treatment of the chronic wound if elevated?

28% of wounds have EPA Require collagen to balance

What about the other 72% that don't have high EPA Still need ECM replacement

Are wounds with Low protease levels non healing for other reasons?

Would a Collagen Dressing improve healing?

Would a a ECM be beneficial?

Serena, Cullen , et al Protease Activity Levels Associated with Healing Status of Chronic Wounds 2011

Sequential Degradation of the ECM

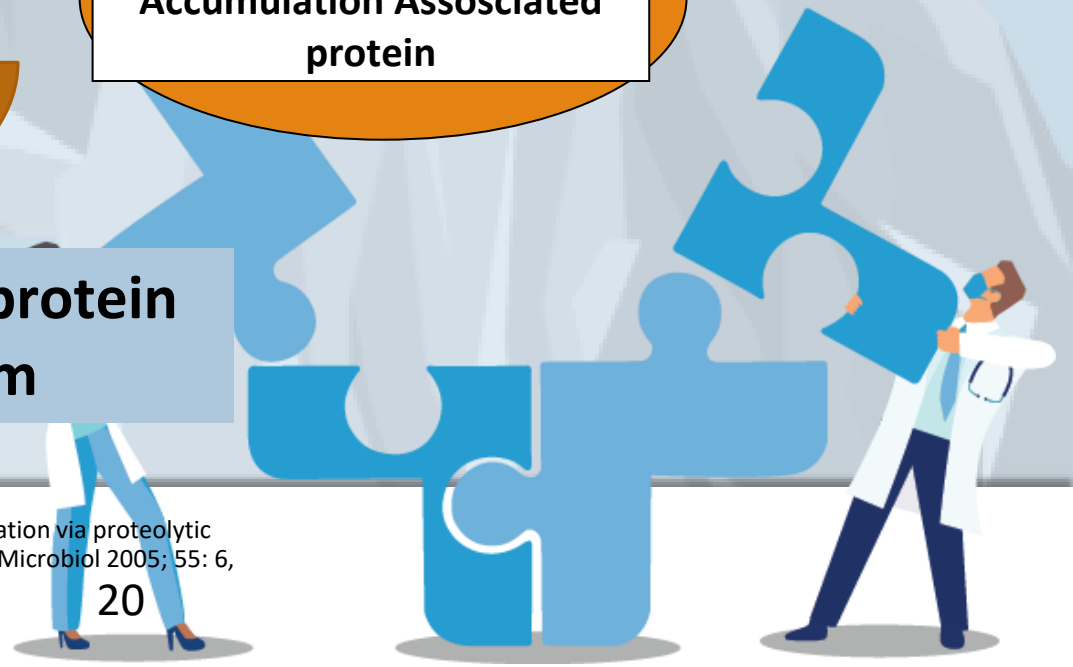
Protease Activity in Wound Promotes Biofilm

Biofilm Hijacking to attract WBCs

Biofilm attracts WBCs and impairs WBC clearing

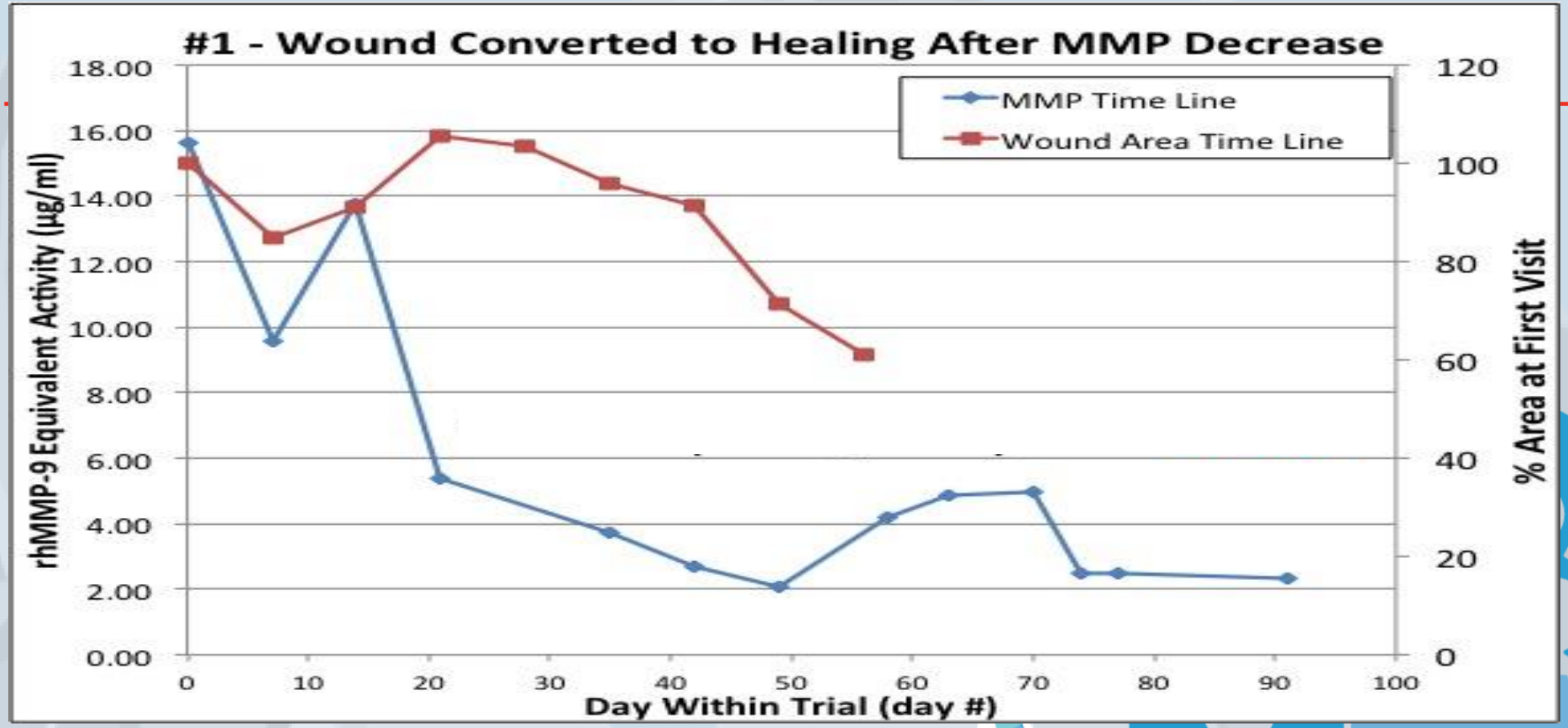
Protease activates Accumulation Assosciated protein

Accumulation-associated protein (Aap) supports Biofilm



Rohde, H., Burdelski, C., Bartscht. K. et al. Induction of Staphylococcus epidermidis biofilm formation via proteolytic processing of the accumulation associated protein by staphylococcal and host proteases. Mol Microbiol 2005; 55: 6, 1883-1895

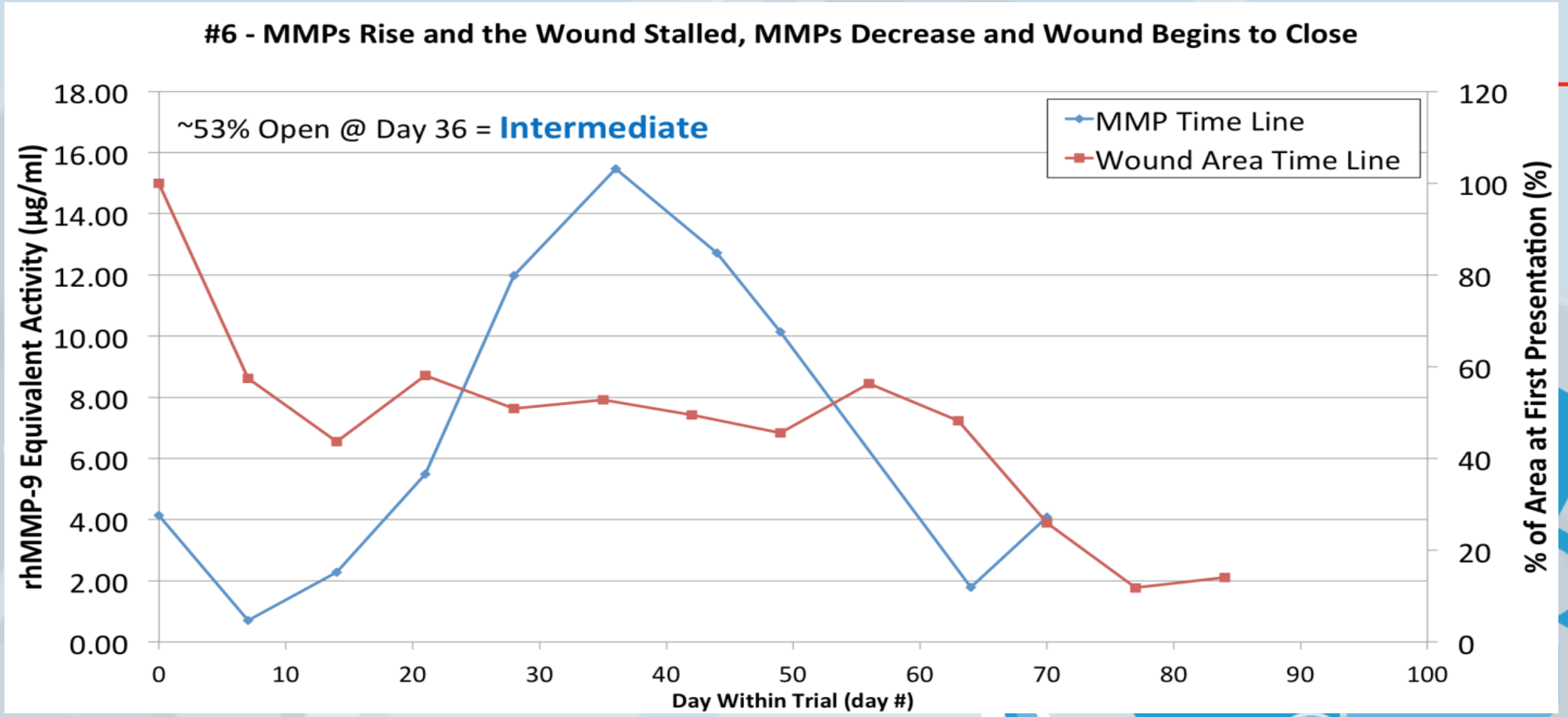
Protease Activity in Wound Promotes Biofilm Change in MMP level Precedes Wound Change



Bohn, Ovine-Based Collagen Matrix Dressing: Next-Generation Collagen Dressing for Wound Care February 2016 Advances in Wound Care 5(1):1-10

Gibson D, Yang Q, Schultz G Description of MMP Activity Levels and Wound Surface Area Changes in Venous Leg Ulcers. SAWC Spring 2014

Serial MMP levels assay may help to assess effectiveness of intervention / treatment



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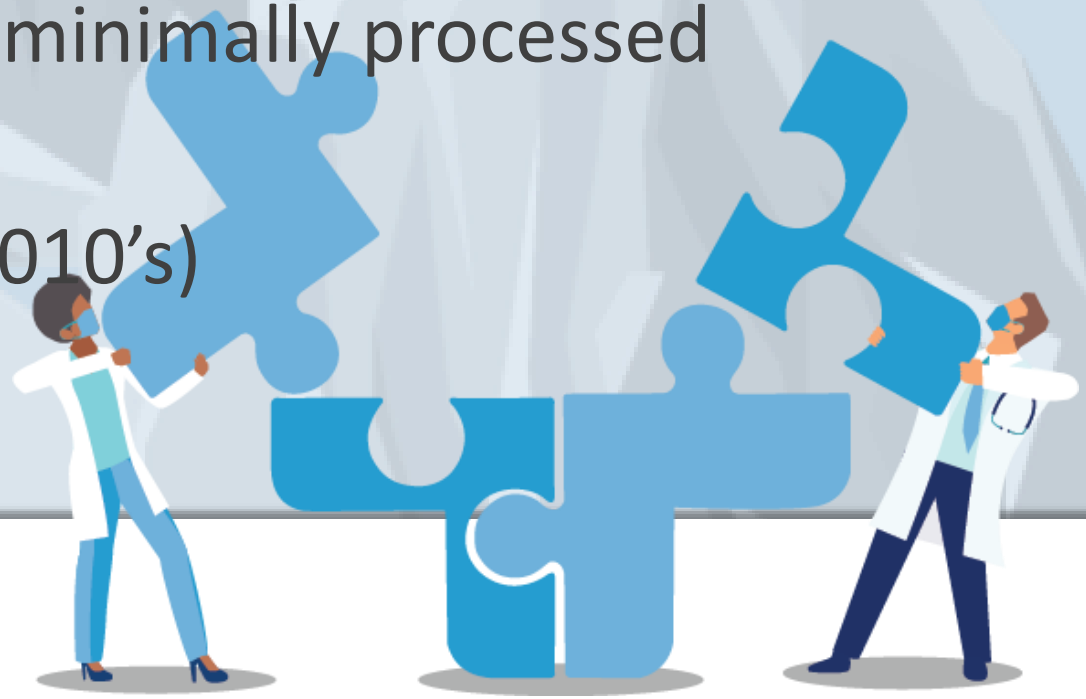
Collagen Dressing

Oxidized Cellulose (Surgicel) used in surgery to stop bleeding. (1990's)

Oxidized Cellulose (ORC) impregnated with processed collagen (Gelatin) as a wound dressing (2000's)

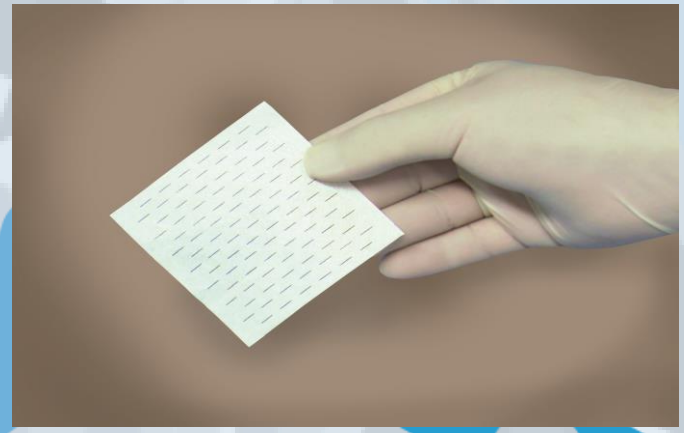
ECM Collagen demonstrated value of minimally processed collagen source (2010's)

Biofilm management and detection (2010's)



Collagen Extra Cellular Matrix

- To reduce excess MMP activity, collagen dressings act as a sacrificial substrate[1]
- Intact, native extracellular matrix promotes tissue granulation[2] and epithelialization for final wound closure[3]
- Extracellular Matrix regulates cellular function and next phenotype expression[4].

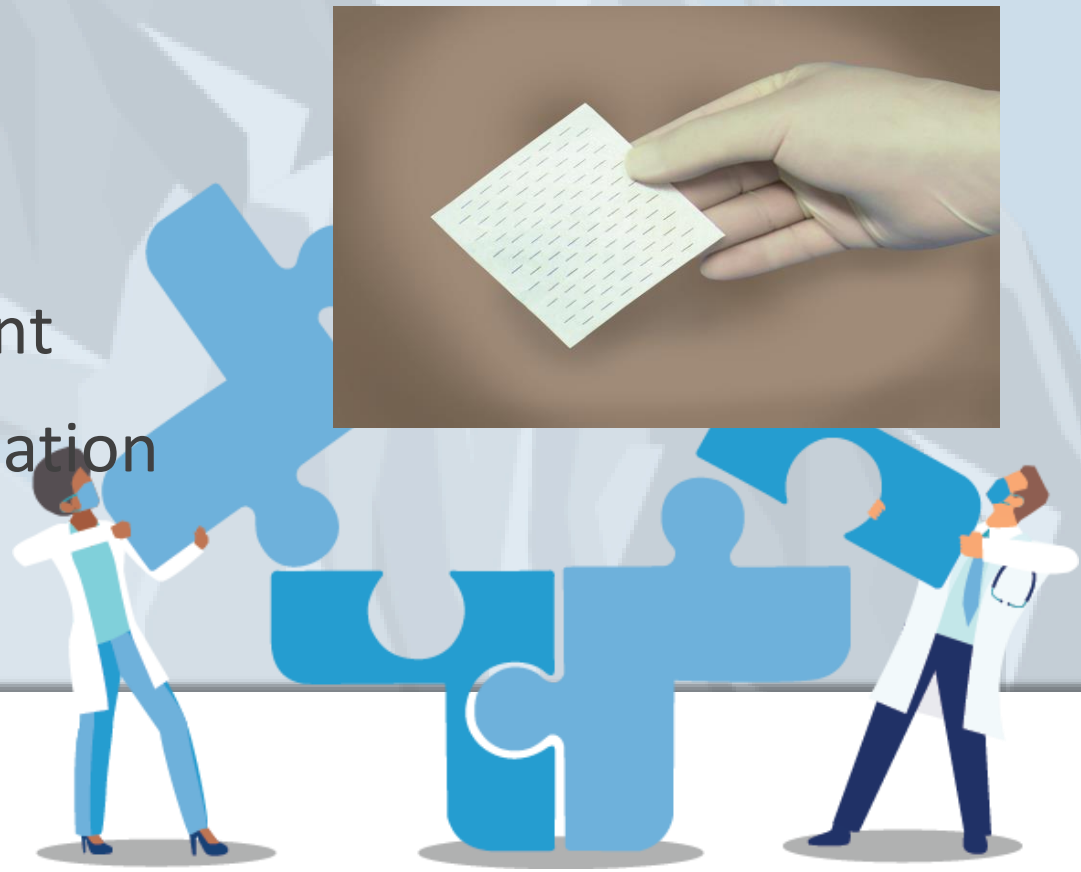


1.Schultz, G., Ladwig, G., & Wysocki, A. (2005). Extracellular matrix: Review of its roles in acute and chronic wounds. *World Wide Wounds*. Retrieved from http://www.worldwidewounds.com/2005/august/Schultz/Extrace-Matric-Acute-Chronic-Wounds.html
2.Tonnesen MG et al. Angiogenesis in Wound Healing. The Society for Investigative Dermatology, Inc. Vol 5, 1; 2000.
3. Pastar I et al. Epithelialization in Wound Healing: A Comprehensive Review. Adv in Skin and Wound Care, Vol 3, 7; 2014.
4.) Schultz, Davidson, Krisner et al. Dynamic Reciprocity in the Wound Microenvironment Wound Repair Regeneration 2011 Mar 19 (2) 134-148

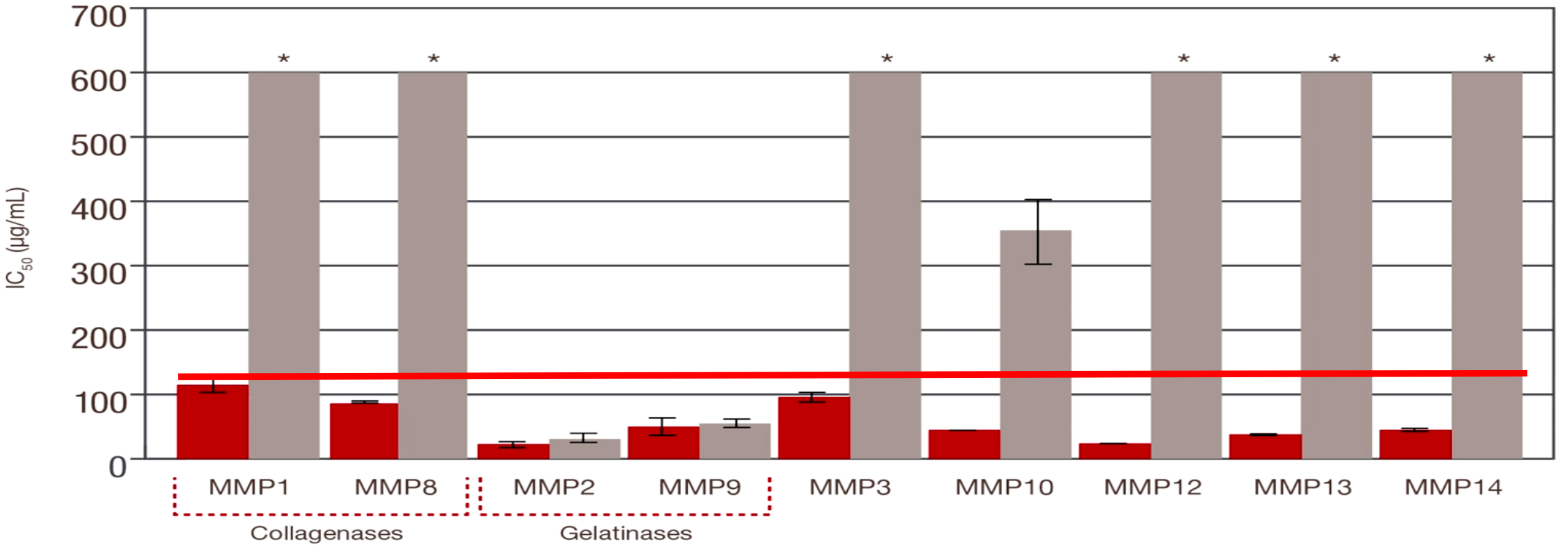
Collagen Extra Cellular Matrix

Cell-extracellular matrix interactions:

- Guide and regulate cellular morphology
- Cellular differentiation
- Migration
- Proliferation
- Cellular survival during tissue development
- Angiogenesis and granulation tissue formation
- Chronic wound healing



Collagen ECM dressing
Broad-spectrum MMP reduction

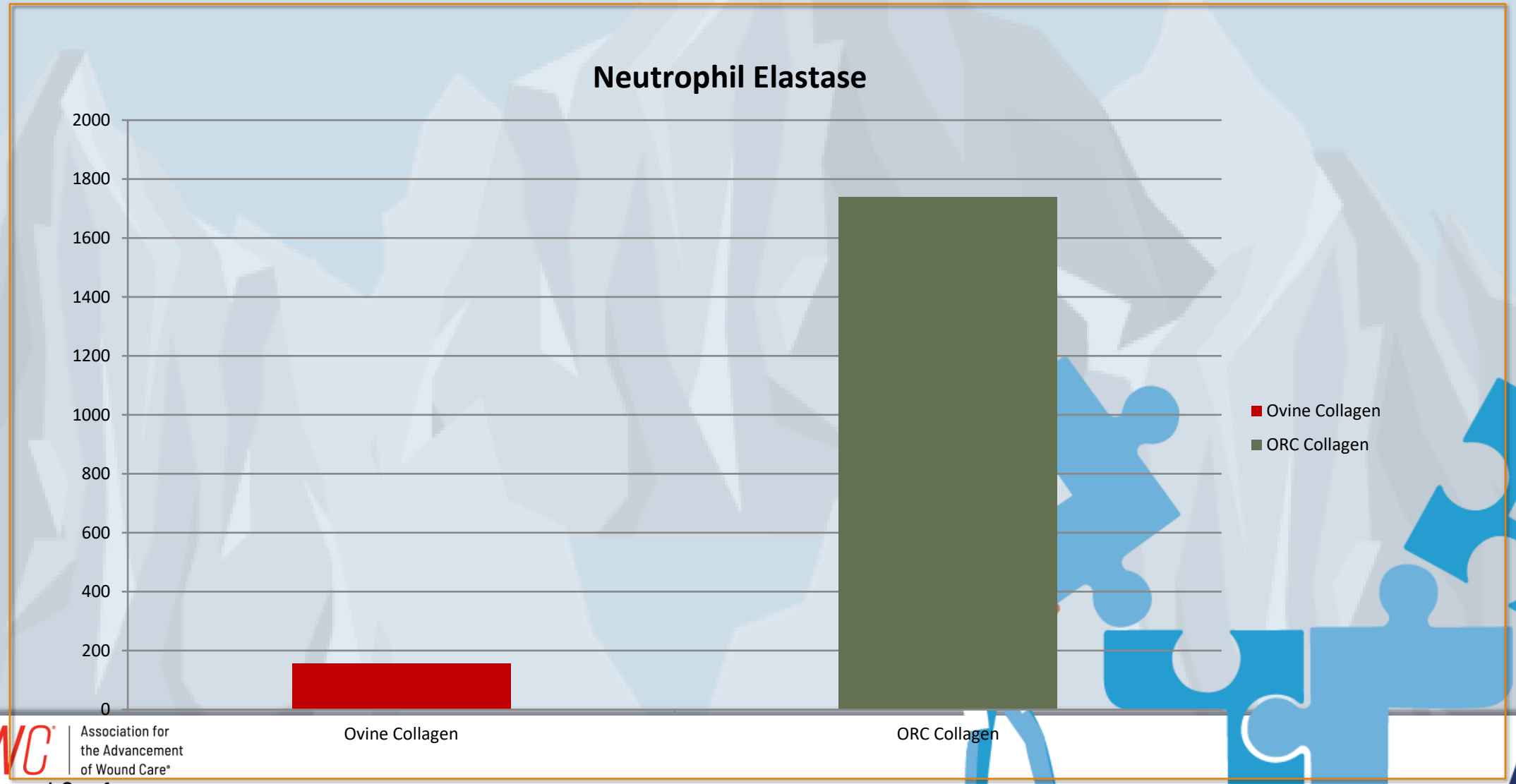


Error represents standard error from triplicate experiments.

* Indicates samples where the IC_{50} was estimated to be approximately 600 µg/ml, or greater.

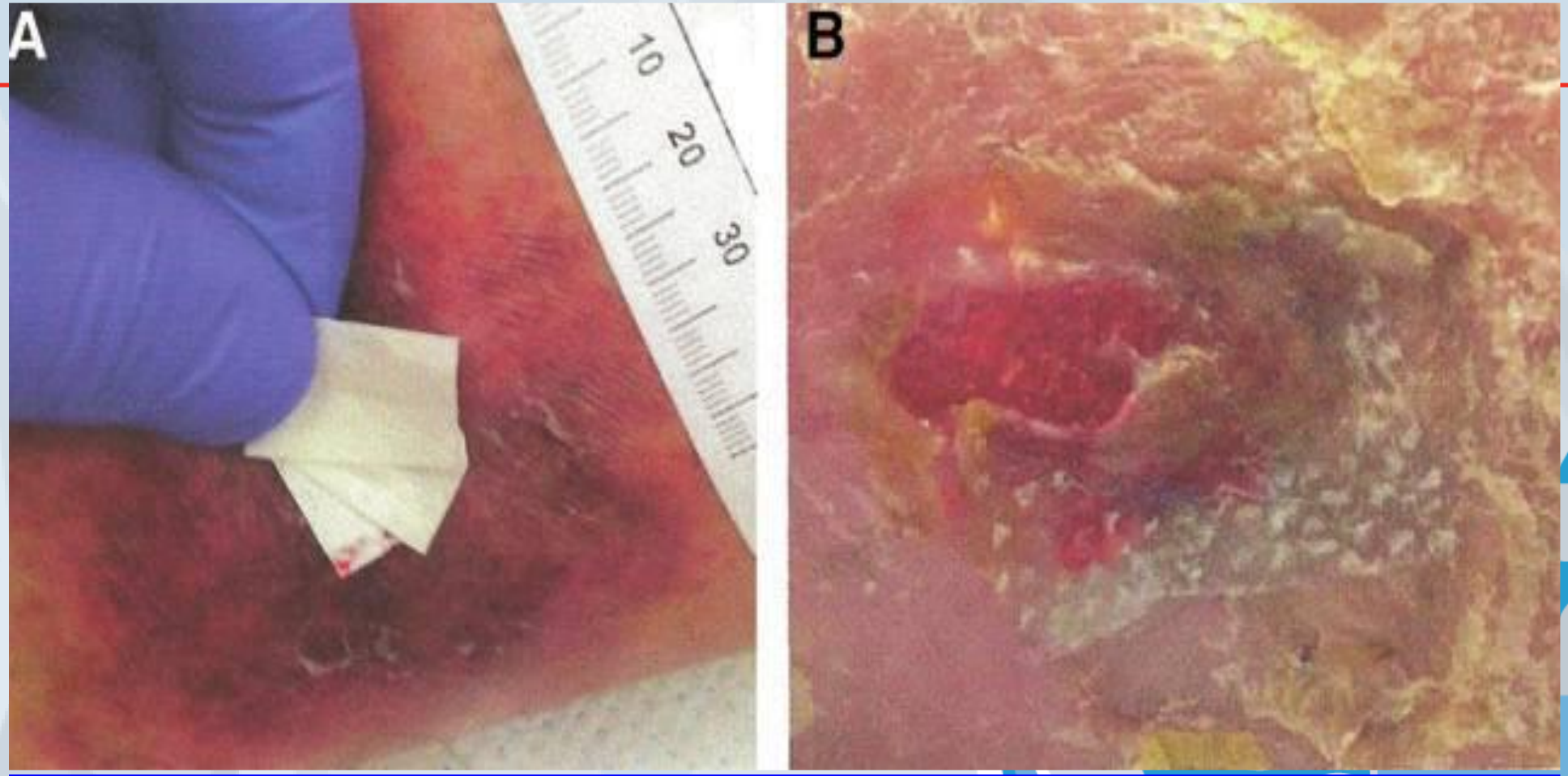
Negron L, Lun S, May BC. Ovine forestomach matrix biomaterial is a broad spectrum inhibitor of matrix metalloproteinases and neutrophil elastase. Int Wound J. 2012 Nov 1.

Buffering of Neutrophil Elastase

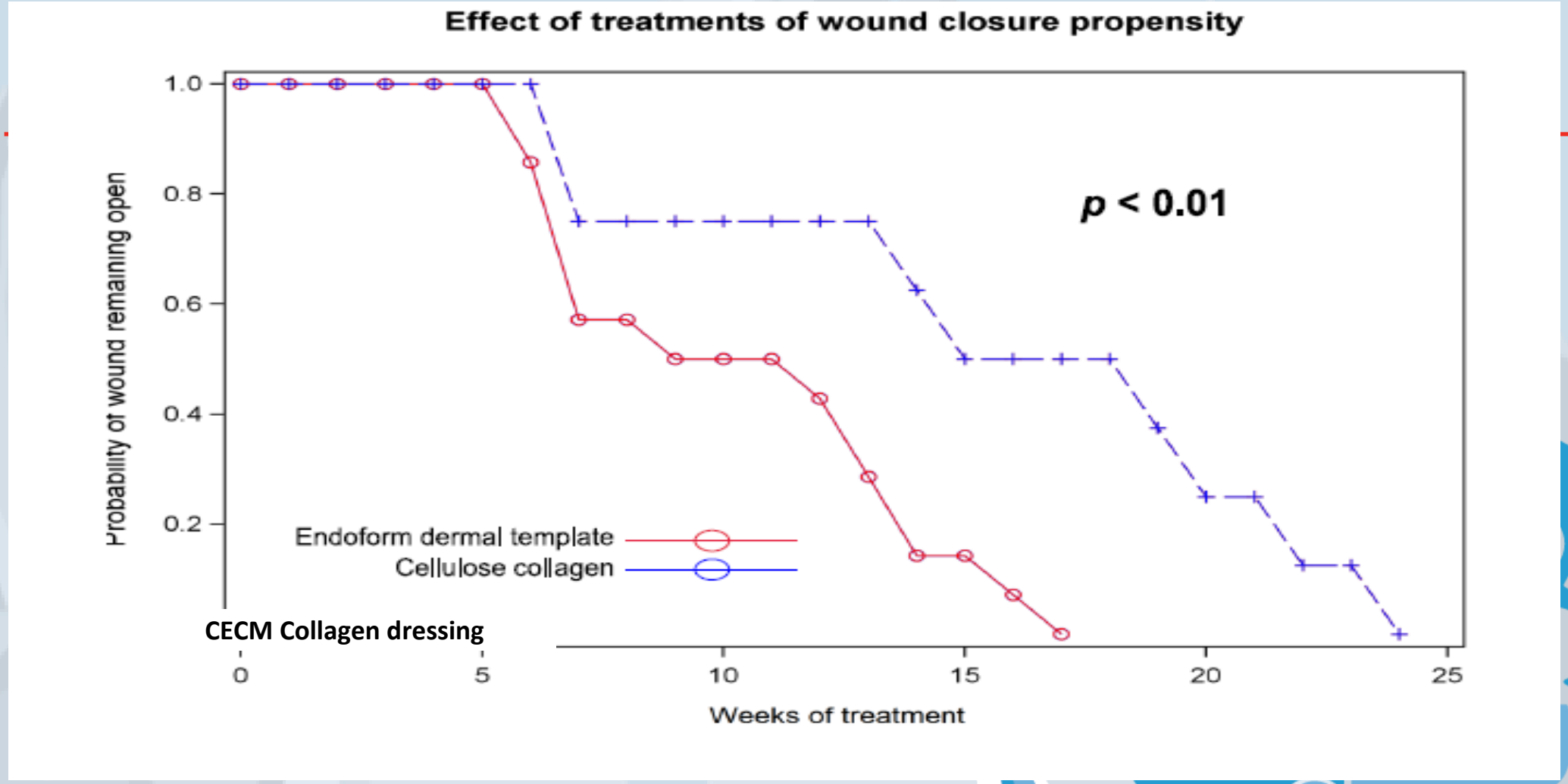


. Negron L, Lun S, May BC. Ovine forestomach matrix biomaterial is a broad spectrum inhibitor of matrix metalloproteinases and neutrophil elastase. Int Wound J. 2012 Nov 1

Dosing to provide sufficient Collagen for duration of treatment episode.

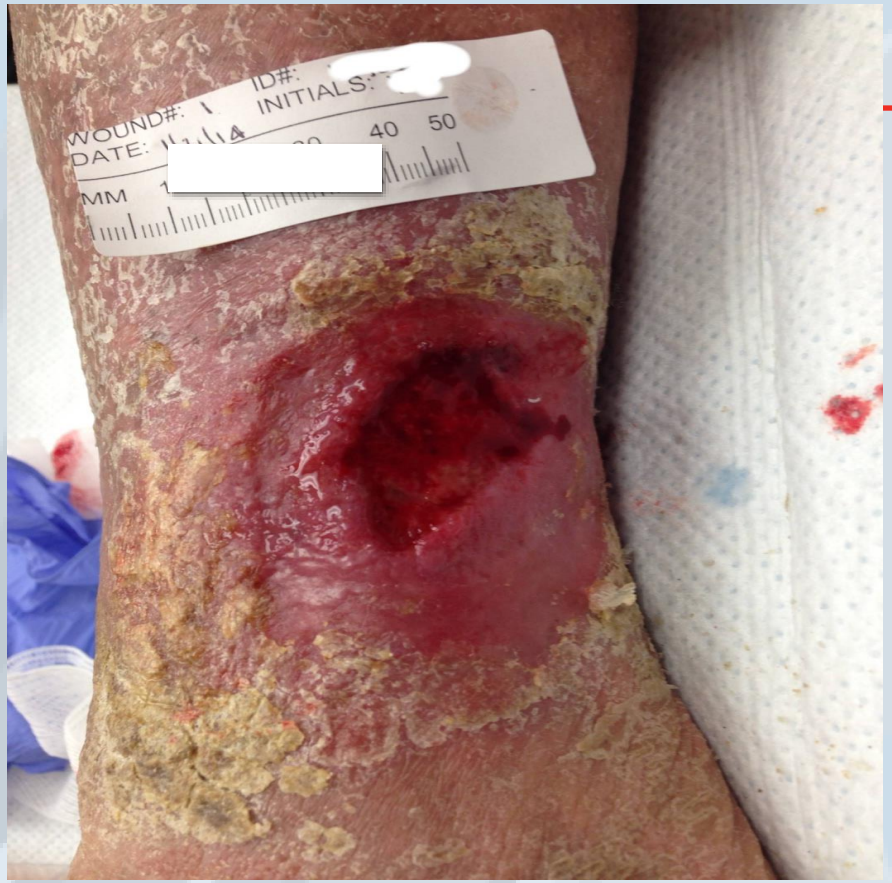


VLU Wounds closed faster with CECM Collagen dressing



Bohn G A New Ovine Collagen Dressing Demonstrates Cost Effectiveness in the Treatment of Venous Leg Ulcers SAWC Spring 2013 Denver CO

Venous Ulcer Combining Protease Management and Biofilm Management Strategy

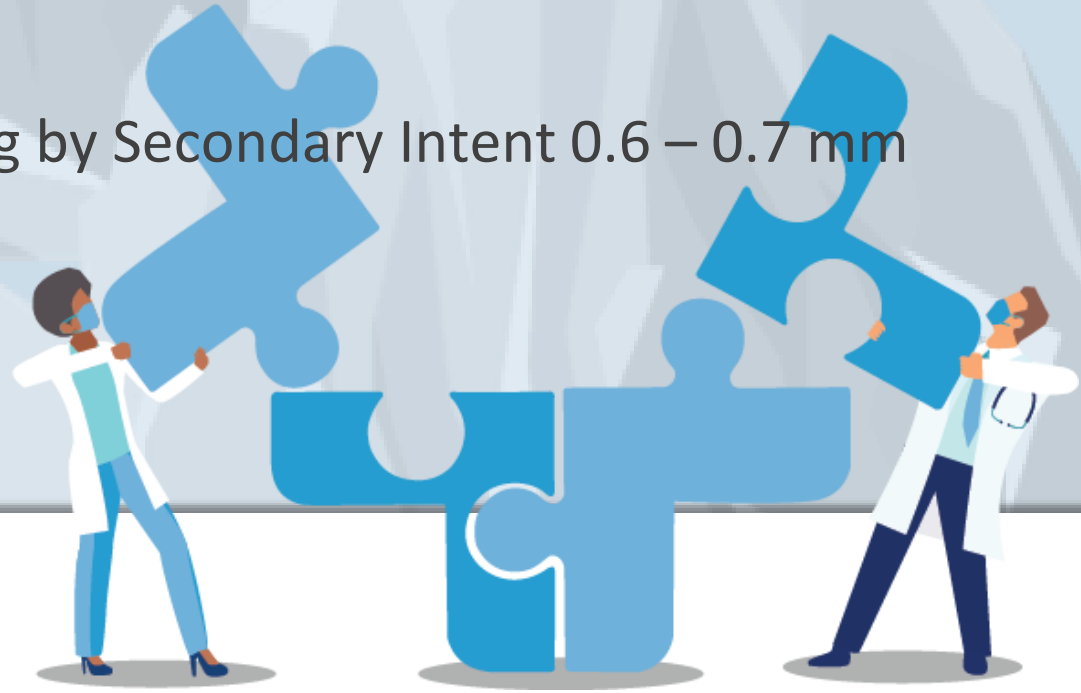


68 yo female with a painful VLU present for 9 months

Size 3.5 cm x 4.1 cm = 14.35 sq cm

Large Size Negative risk factor for healing at 12 weeks

Rate of healing by Secondary Intent 0.6 – 0.7 mm per day

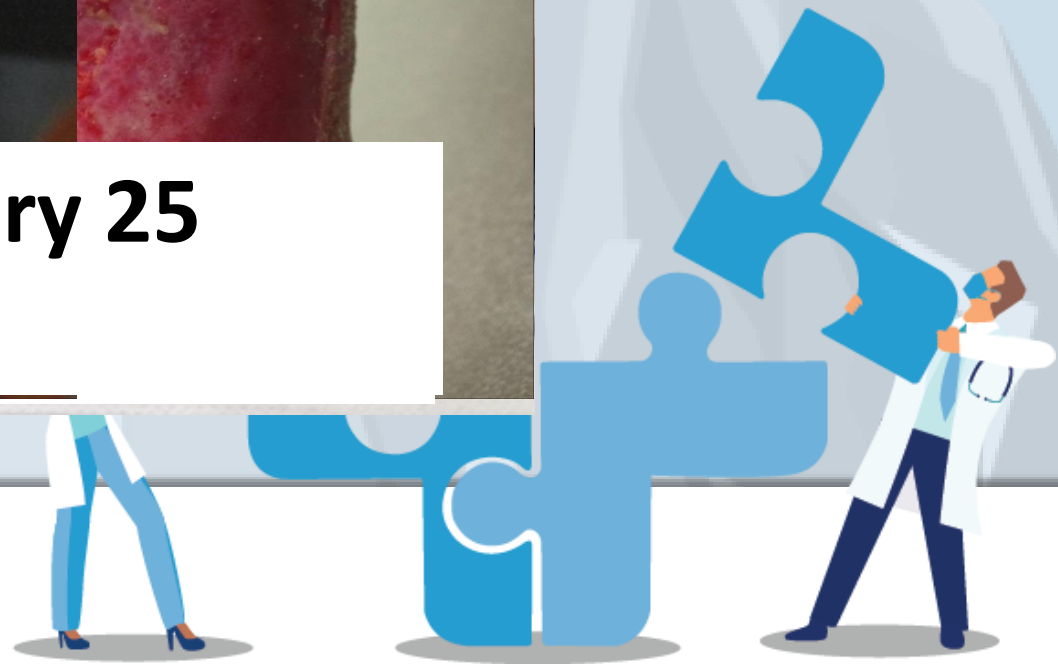


www.medetec.co.uk/book%20abstracts/wound-healing-mechanisms.pdf accessed 2/28/2017

Venous Ulcer Healing by Secondary Intention With Collagen

January 21 to February 25

35 days



CECM Collagen in a VA

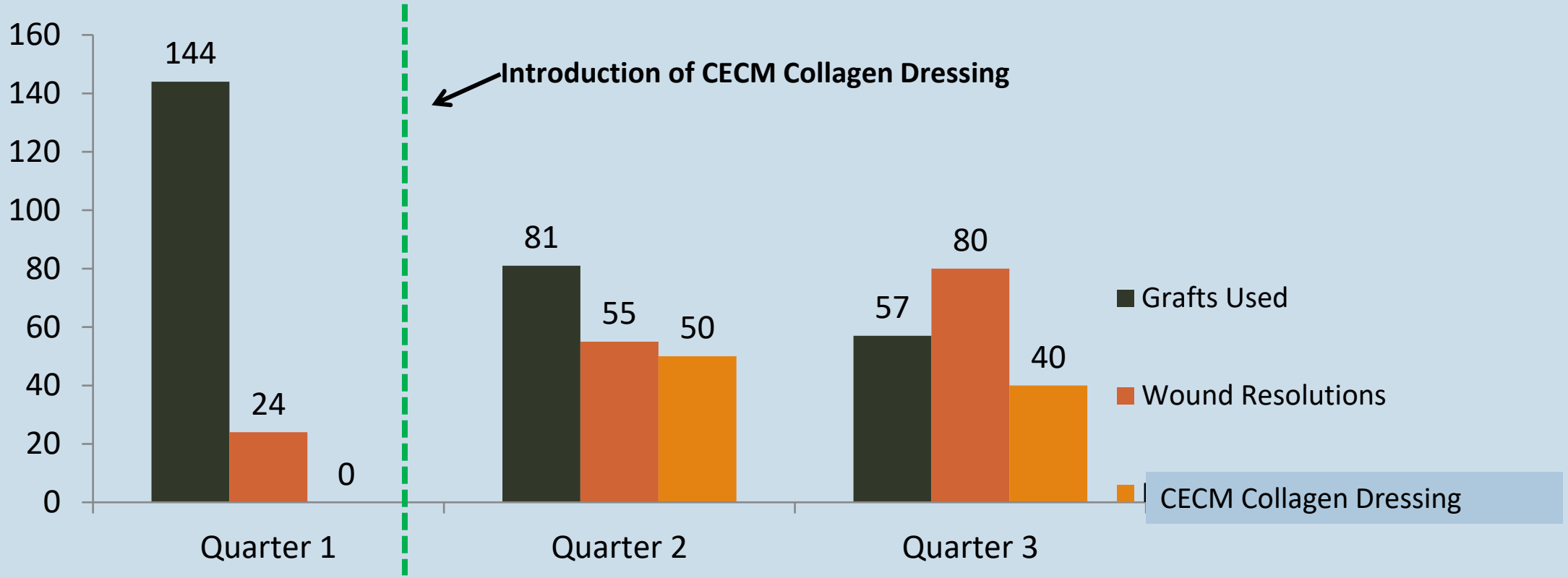
- Retrospective review of advanced graft expenditures and wound resolutions in a VA wound center
- Showed standardization of assessment, treatment and management of wounds to promote wound closure
- Established a dual protocol algorithm:
  - Decision and Treatment arms
  - Utilized CECM Collagen dermal as the first line collagen of treatment
  - Clinical decision for treatment was based on whether

there was a 30% - 50% wound size reduction over 4 weeks

- > 30% WSR - continue with CECM Collagen
- <30% WSR – advance to biologic

Ferreras D., Craig S., Malcomb R., Utilization of an ovine collagen dressing with an intact extracellular matrix (CECM) within a dual-protocol algorithm to improve wound closure times and reduce expenditures in a VA Hospital poster presentation at SAWC Fall 2015

Results of a VAMC Dual Protocol

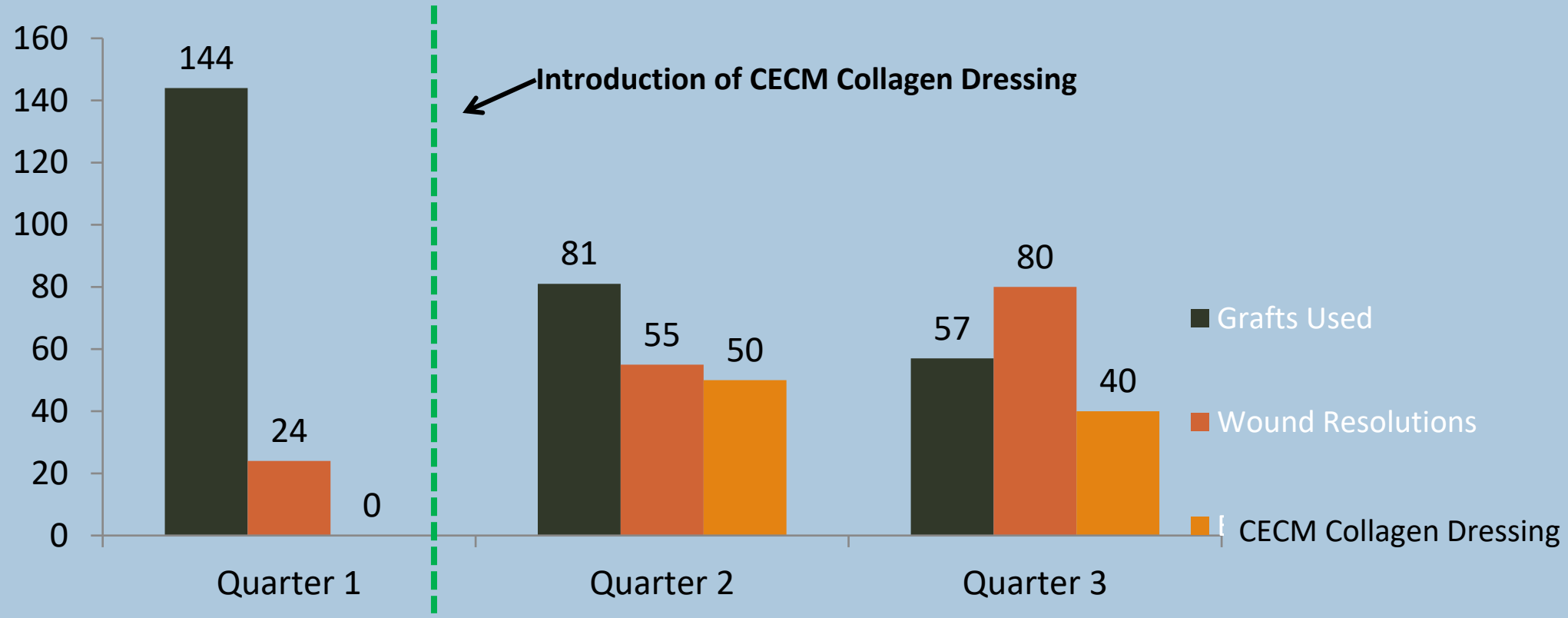


After the introduction of the CECM Collagen in this VA hospital

- Number of wound resolutions were increased by 70%
- Advanced graft expenditures were reduced by 71.6%

Ferreras D., Craig S., Malcomb R., Utilization of an ovine collagen dressing with an intact extracellular matrix (CECM) within a dual-protocol algorithm to improve wound closure times and reduce expenditures in a VA Hospital poster presentation at SAWC Fall 2015

Results of a VAMC Dual Protocol



After the introduction of the CECM Collagen in this VA hospital

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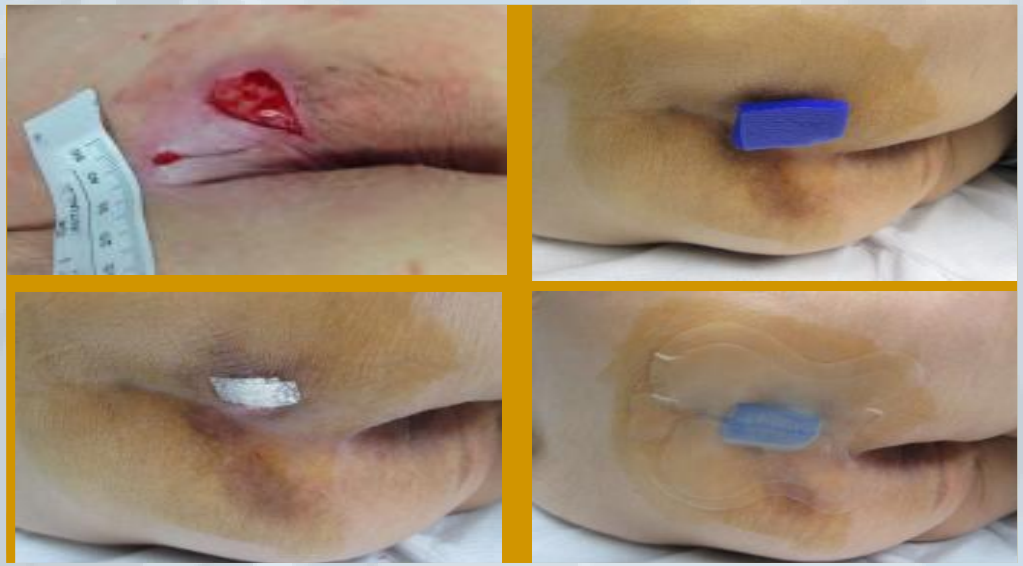
Ferreras D., Craig S., Malcomb R., Utilization of an ovine collagen dressing with an intact extracellular matrix (CECM) within a dual-protocol algorithm to improve wound closure times and reduce expenditures in a VA Hospital, poster presentation at SAWC Fall 2015

Collagen and Pressure Ulcers



Graumlich; No difference between ORC collagen and hydrocolloid 65 patients

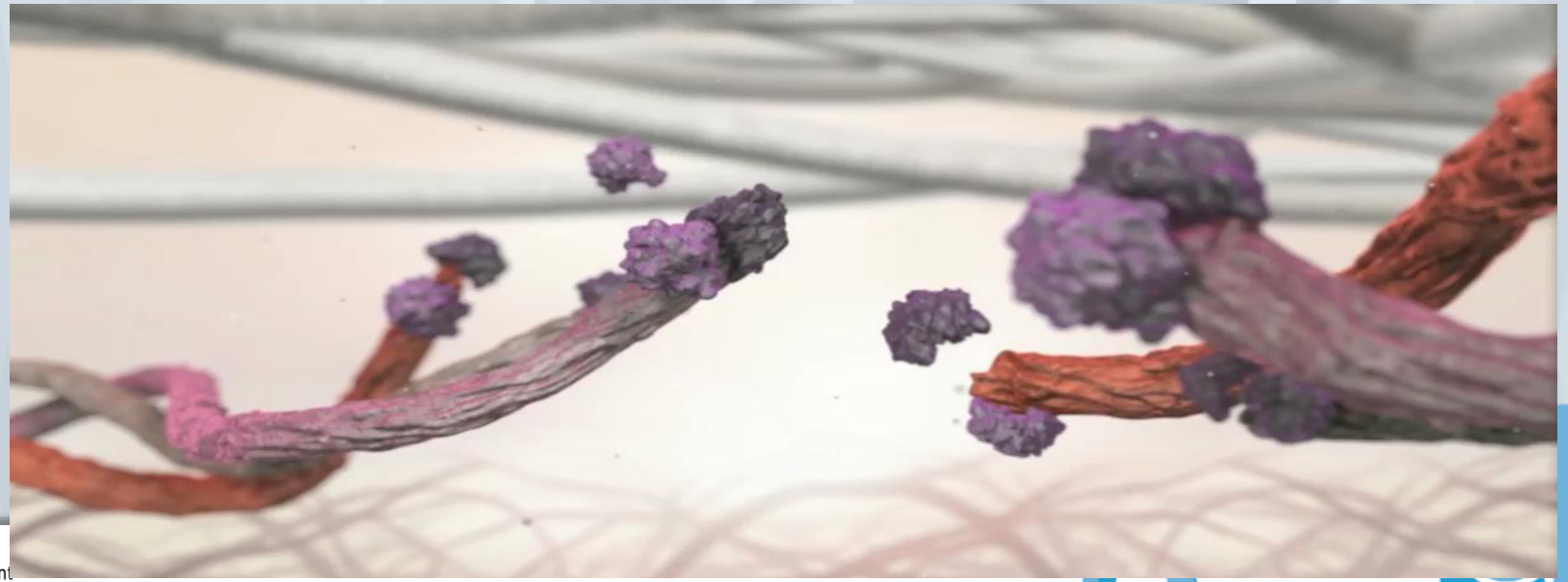
No Stage 3 Healers at 4 weeks

CECM Collagen with Hydrocolloid 20 patients

61% healed Stage 3 at 4 weeks

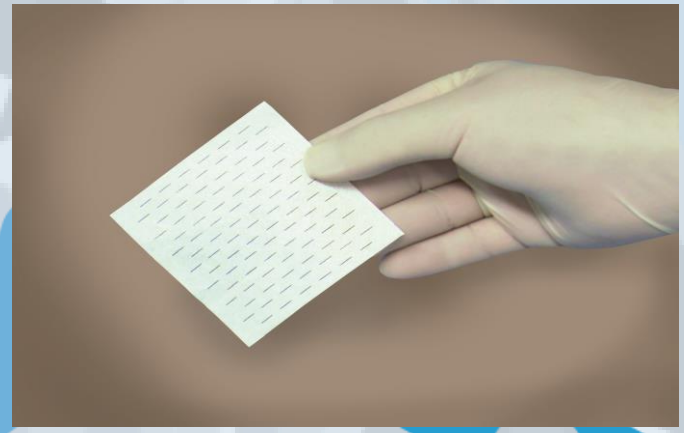
Managing MMPs: Collagen

- Acts as a sacrificial substrate
- MMPs attack the collagen fibers within the dressings instead of the body's ECM
- Reduces excess MMP activity



Collagen Extra Cellular Matrix

- To reduce excess MMP activity, collagen dressings act as a sacrificial substrate[1]
- Intact, native extracellular matrix promotes tissue granulation[2] and epithelialization for final wound closure[3]
- Extracellular Matrix regulates cellular function and next phenotype expression[4].



1.Schultz, G., Ladwig, G., & Wysocki, A. (2005). Extracellular matrix: Review of its roles in acute and chronic wounds. *World Wide Wounds*. Retrieved from http://www.worldwidewounds.com/2005/august/Schultz/Extrace-Matric-Acute-Chronic-Wounds.html
2.Tonnesen MG et al. Angiogenesis in Wound Healing. The Society for Investigative Dermatology, Inc. Vol 5, 1; 2000.
3. Pastar I et al. Epithelialization in Wound Healing: A Comprehensive Review. Adv in Skin and Wound Care, Vol 3, 7; 2014.
4.) Schultz, Davidson, Krisner et al. Dynamic Reciprocity in the Wound Microenvironment Wound Repair Regeneration 2011 Mar 19 (2) 134-148

CECM Collagen/ORC

Intact Collagen
- Type I
- Type III
- Type IV

Fibronectin

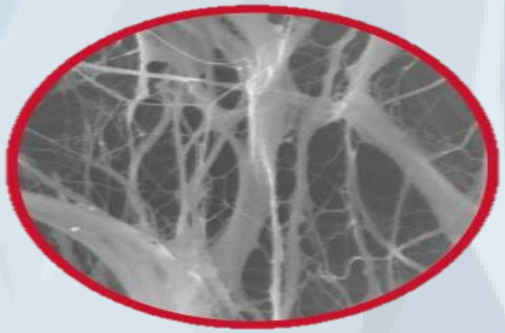
Laminin

Hylauronic acid

Elastin

Heparan Sulfate

Glycosaminegylcans



Denatured type I collagen

ORC Oxidized Reduced Cellulose

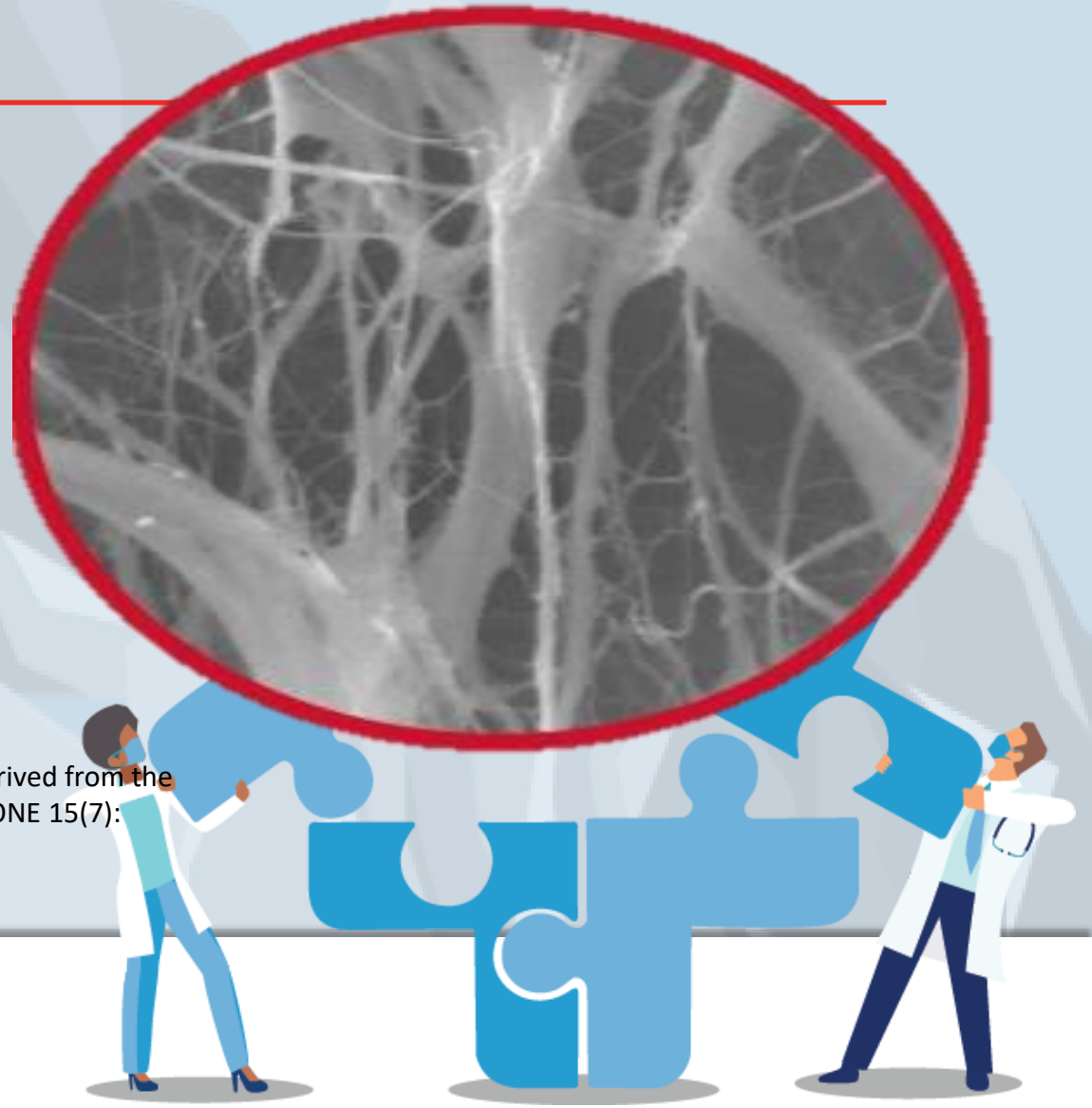
Next Generation Collagen

Functional Role of ECM

Microarchitecture to support cell function

Cofactors to orchestrate cellular interaction

Attracts Stem Cells to wound Site



Dempsey SG, Miller CH, Schueler J, Veale RWF, Day DJ, et al. (2020) A novel chemotactic factor derived from the extracellular matrix protein decorin recruits mesenchymal stromal cells in vitro and in vivo. PLOS ONE 15(7): e0235784. https://doi.org/10.1371/journal.pone.0235784

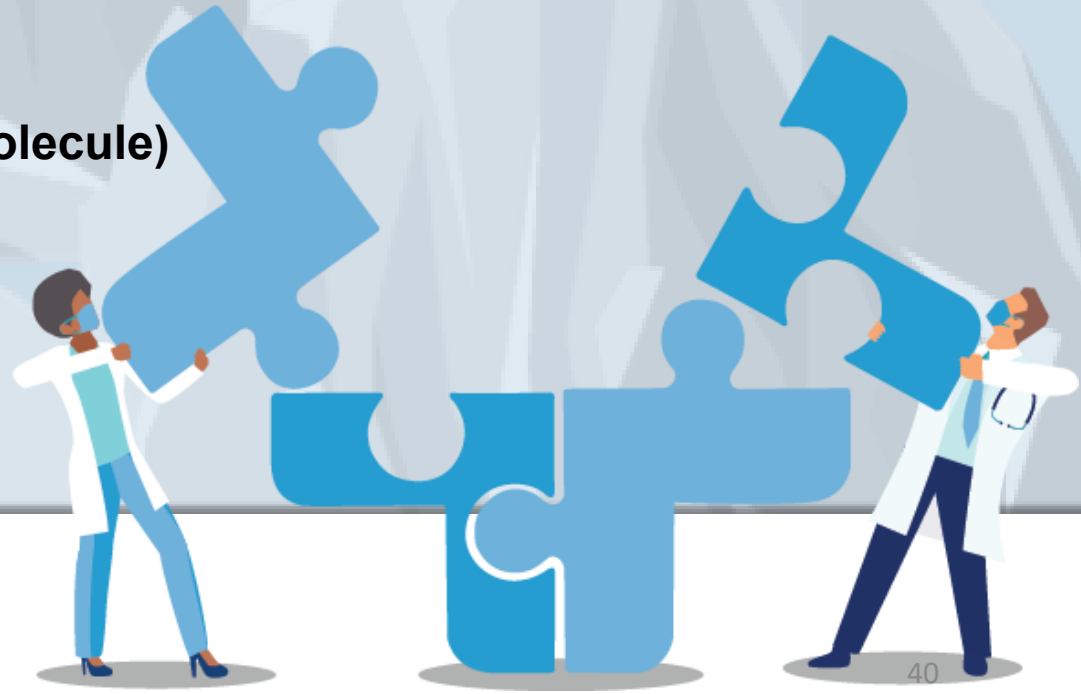
MMPs and the Next Generation of Collagen Dressings

Dermal TemplateCollagen Provides Multiple Components Of The Extracellular Matrix

- ✓ Intact type I collagen – major structural protein on dermis
- ✓ Intact type III collage – an important fibrillar collagen
- ✓ Intact type IV collagen – basement membrane component
- ✓ Intact elastin – major protein responsible for skin elasticity
- ✓ Intact fibronectin – multidomain cell adhesion protein
- ✓ Intact laminin – basement membrane component
- ✓ Intact FGF2 (bFGF)
- ✓ Intact hyaluronic acid (HA – major water holding molecule)

ORC Collagen Contains No Intact Collagens Or Elastin

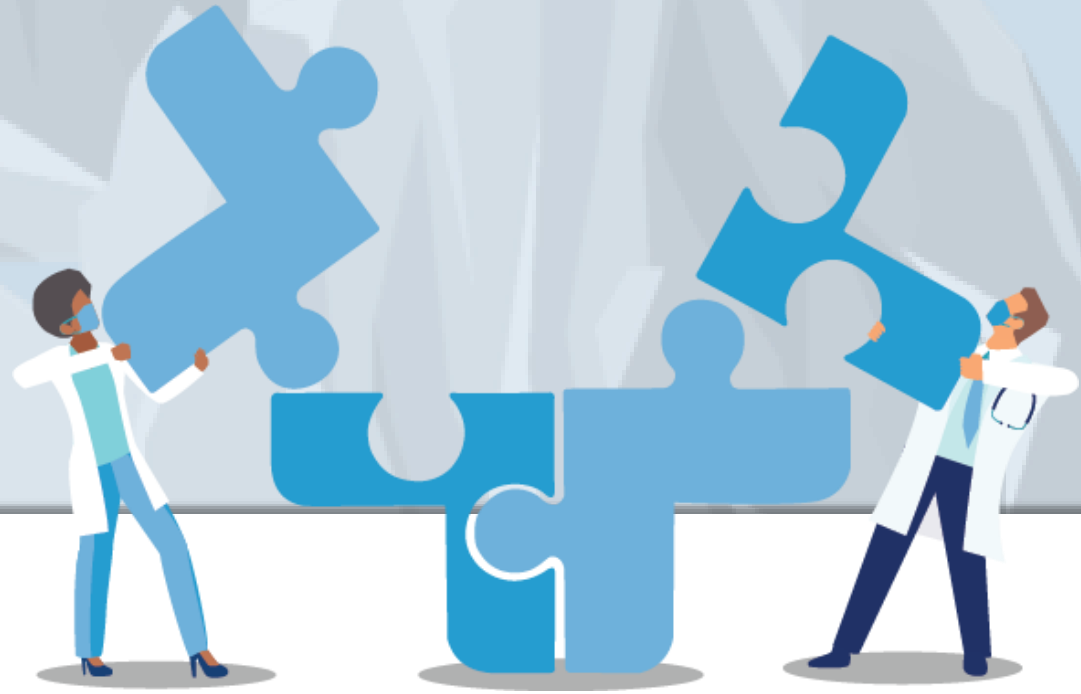
- ➢ 50% gelatin (denatured type I collagen) and
- ➢ 50% oxidized regenerated cellulose



Biofilm Management Strategy

Biofilms regenerate in as little as 24 hours

Non-Cytotoxic management to retard or slow reformation to elevate MMP production or infection



NON Cytotoxic Bioburden Management Strategy

Hypochlorous Acid ($HOCL^-$)

- **Generated by myeloperoxidative burst by neutrophils, monocytes and macrophages**
- **significant activity against aerobic, anaerobic, fungal and viral pathogens.**
- ***HOCL generated inhibits human MMP-7 limiting proteolytic activity[5].**

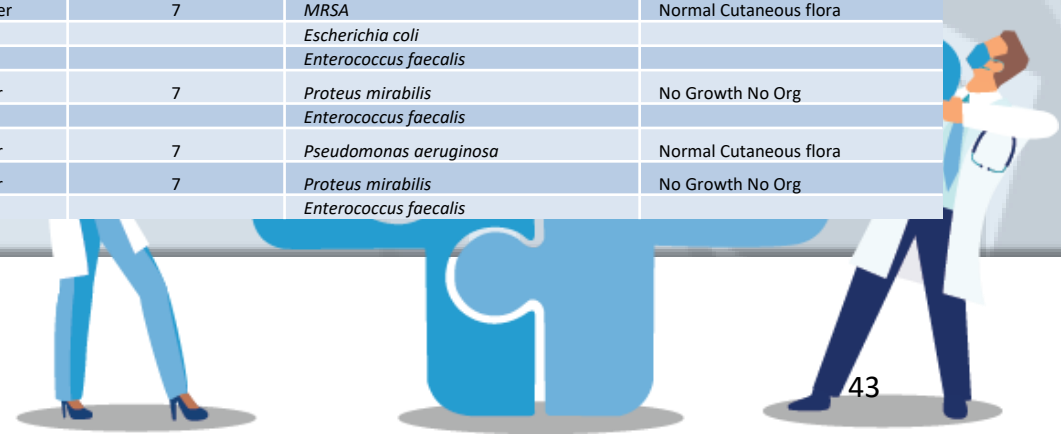
Hypochlorous Acid / Collagen

Used to treat 18 tissue cultured wounds positive for pathogens

17 / 18 wound tissue culture negative at 2 weeks

Wound Type	Application/Week	Initial Culture	Week 2 Culture
DFU	3	*Escherichia coli*	Normal Cutaneous flora
		MRSA	
		Pseudomonas aeruginosa	
DFU	3	*Pseudomonas aeruginosa*	Normal Cutaneous flora
		MRSA	
		Enterococcus faecalis	
DFU	3	*MRSA*	*MRSA*
DFU	3	*Enterobacter cloacae*	No Growth No Org
DLE	3	*Klebsiella pneumoniae*	Normal Cutaneous flora
		Enterobacter cloacae	
		Enterococcus faecalis	
		MRSA	
		Proteus mirabilis	
DLE	5	*Pseudomonas aeruginosa*	Normal Cutaneous flora
		Staphylococcus aureus	
DLE	5	*Escherichia coli*	No Growth No Org
		Staphylococcus aureus	
DLE	5	*Klebsiella pneumoniae*	No Growth No Org
		Streptococcus pneumoniae	
		Strep Group B	
DLE	3	*MRSA*	No Growth No Org
VLU	3	*Staphylococcus aureus*	No Growth No Org
VLU	3	*Enterobacter cloacae*	Normal Cutaneous flora
		Enterococcus faecalis	
VLU	3	*MRSA*	Normal Cutaneous flora
VLU	3	*MRSA*	No Growth No Org
Pressure ulcer	7	*Pseudomonas aeruginosa*	Normal Cutaneous flora
		Escherichia coli	
Presssure Ulcer	7	*MRSA*	Normal Cutaneous flora
		Escherichia coli	
		Enterococcus faecalis	
Pressure ulcer	7	*Proteus mirabilis*	No Growth No Org
		Enterococcus faecalis	
Pressure ulcer	7	*Pseudomonas aeruginosa*	Normal Cutaneous flora
Pressure ulcer	7	*Proteus mirabilis*	No Growth No Org
		Enterococcus faecalis	

Bohn GA, et al, Can The Use of *Hypochlorous Acid Change Your Dressing Selection in Treating Chronic Wounds? CSASWC 2014 poster presentation



Summary

Model of Chronicity involves biofilm and host response to that biofilm

Host Inflammation Destructive to ECM and healing

Era of Diagnostics in Wound Care will help identify opportunity to intervene.

Early and Aggressive therapy preferred approach, Step down when plausible

Thank You
Questions?

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