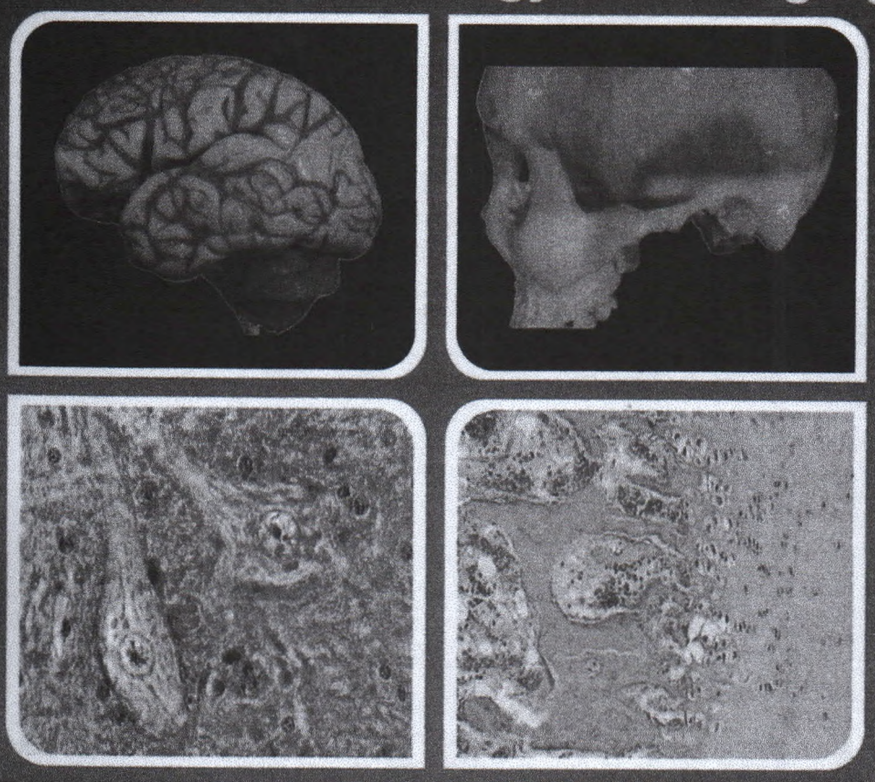
6TH ASIA PACIFIC INTERNATIONAL CONGRESS OF ANATOMY
(6TH APICA)
&
13TH NATIONAL CONGRESS OF INDONESIAN ANATOMIST ASSOCIATION
(13TH PIN-PAAI)

Proceeding Book

THE FUTURE OF ANATOMY

Clinical Anatomy
Biomolecular and Cellular Anatomy
Anatomy in Radiology and Imaging



GrahaBIK-IPTEKDOK
Faculty of Medicine of Airlangga University
Surabaya, 22nd-23rd July 2011
Indonesia

PROGRAM DAY-2

July 23rd, 2011

Time	Activity	
	VENUE: GRAHABIK-IPTEKDOK (GRABIK)	
07.00-08.00 am	Re-registration	
	Venue: Grabik 2nd floor	
	Moderator: Prof. H. Bambang Rahino S, dr.	
08.00-08.20 am	**Keynote speaker I**: Prof. H. Ari Gunawan, dr., MS., PhD (Indonesia). (KS II.I) "The Role of Anatomy in Supporting Medical Sciences"	
08.20-08.40 am	**Keynote speaker II**: Prof Madya. Dr Srijit Das (Malaysia) (KS II.II) "Active Research in Anatomy : Do We Really Care for the Needy"	
	Venue: GRABIK 2nd floor	
	Moderator: Prof. Jeanne A Pawitan, dr., PhD	
08.40-09.00 am	**Keynote Speaker III**: Dr. Parker B. Antin (AAA,USA)(KS II.III)	
09.00-09.20 am	**Keynote Speaker IV**: Assc/ Prof. Christopher Briggs, PhD (ANZACA,Australia)(KS II.IV)	
09.20-09.40 am	**Keynote Speaker V**: Djoko Santoso, dr., PhD., Sp.PD, K-GH (Indonesia) (KS II.V)	
09.40-10.00 am	**Keynote Speaker VI** : Prof. Dr. Teddy Ontoseno, dr., Sp.A (K)., Sp.JP (Indonesia) (KS II.VI)	
10.00-10.30 am	Discussion	
10.30-10.50 am	Coffee break	
	Venue: GRABIK 2nd floor	**Venue: RK. Anatomi**
	Plenary session IV: Cellular & Biomolecular Anatomy(Moderator: Prof. Dr. Yanwirasti, dr)	**Plenary session V:** Clinical anatomy (Moderator: Prof. Dr. Nancy M. Rehatta, dr, SpAn K.IC)
10.50-11.10 am	Speaker XIII: Prof. In-Sun Park (Chairman of KAS) (PSIV.I)	Speaker XVII: Prof. Dr. Doddy M. Soebadi, dr, Sp.U (Indonesia) (PSV.III)
11.10-11.30 am	Speaker XIV: M.H. Nasr-Esfahani, B.Sc, PhD (Iran) (PSIV.II)	SpeakerXVIII:Prof.PasukMahakkanukrauh (Thailand) (PSV.I)
11.30-11.50 am	Speaker XV: Assc/ Prof. Heidari M. Hassan, PhD (Iran) (PSIV.III)	Speaker XIX: Prof. Chang-Seok Oh, MD, Ph (Korea) (PSV.IV)
11.50-12.10 am	Speaker XVI: Prof. Dr. Gayatri Rath (India) (PSIV.IV)	Discussion
12.10-12.40 am	Discussion	
12.40-02.00 pm	**Venue: Grabik 2nd floor** **Lunch & poster presentation** (even numbers) Jury : Teddy H. Wardhana, dr., Sp.OT, dr. Ni Wajan T, dr., MS, PA, Prof. Dr.Nasronuddin, dr, Sp.PD., K-PTI., FINASIM, Prof. Pasuk Mahakkanukrauh, Assc/ Prof. Heidari M. Hassan, PhD, Visiting Prof. Yoshiyuki Tohno. Prof. Purnomo S, dr, MS, Dr., Dr. Kumkum Rana Note: Presenter for poster competition must stand beside his/her poster from 01.00-01.45 pm for jury to be able to mark (Q&A) **Small warm meeting** Moderator : Prof. Dr. Nancy M. Rehatta, dr., SpAn K.IC Agenda : Discussion of potential networking in Reasearch and Educational Training Participant : University representative, Dean and Vice Dean III of Medical Faculty Airlangga University, Head of Department of Anatomy and Histology, Senior Lecturers of Department of Anatomy and Histology FMAU, Head of UPPM FMAU, Invited guests, APICA, and ANZAC Representative members Venue : VIP Room GRABIK Lt 2.	

	Parallel session II Oral presentation for competition (@ 10 mins presentation, 5 mins Q&A)				**Venue: Ruang Praktikum Anatomi**	**Venue: Ruang Sidang Anatomi**
	Venue: GRABIK 2nd floor	**Venue: RK Anatomi**	**Venue: RK Histologi**	**Venue: RK Khusus**		
00-13.00 pm	Moderator: dr. Ni Wajan T, dr., MS, PA Jury : Prof.H. Bambang Rahino S, dr., Prof. Jong Eun Lee	Moderator: Teddy H. Wardhana, dr., Sp.OT Jury : Prof. Dr. Teddy Ontoseno, dr., Sp.A (K)., Sp.JP, Prof. Changman Zhou	Moderator: Prof. Dr. Bambang Sektiari L, drh., DEA Jury:Dr., Dra. Toetiek Koesbardiati, Prof. Fedik A. Rantam, drh., M.Kes, PhD	Moderator: Ferdiansyah, dr., SP.OT Jury : Prof. Sudjono Aswin, dr., PhD, Prof. Dr. Abdul Hafid Bajamal, dr., Sp.BS	Moderator: Susy, drg., MS, Prof. Anita Tuli **Forensic Anthropology** Maciej Henneberg, MSc (summa cum laude), PhD, DSc., FAIBiol (Australia) **Facial Reconnstruction Demonstration** Myrtati DA, Dra.,MA., PhD (Indonesia)	PIC: Prof. Rio Sofwanhadi, dr., PA(K) APICA organization meeting Moderator: Prof. Kyung Ah Park Prof Joghataei, Prof Yun-Qing Li, Prof. Park (representative member from KAA), Dr Abdurrachman, APICA members
II.IV)	Participant OB17-OB20	Participant OB21,OC1-OC3	Participant OC4-OC7	Participant OC8-OC11		
KS II.VI)						
my Rehatta, dr						
I. Soebadi						
anukrauh						
Dh, MD, Ph	**Parallel session III** Oral presentation for competition(@ 10 mins presentation, 5 mins Q&A)					
	Venue: GRABIK 2nd floor	**Venue: RK Anatomi**	**Venue: RK Histologi**	**Venue: RK Khusus**		
30 pm	Moderator : Yan Efrata S, dr.,Sp.BTKV Jury: Prof P. Gopalakhrisnakone MBBS, PhD., FAMS., DSc, Prof. Dr. Yanwirasti, dr	Moderator : Tomy Lesmana, dr., SpB. KBD Jury : Paulus Rahardjo, dr., Sp.Rad (K) , Myrtati DA,Dra.,MA.,Ph.D	Moderator : Mustofa Helmi Effendi, drh., MS Jury: Prof.Dr. Gayatri Rath, Djoko Santoso, dr., PhD., Sp.PD, K-GH	Moderator : Dr. H. Bambang Purnomo, drh., MS Jury : Prof. Eryati Darwin, dr., Dr.Parker B. Antin		
ronuddin, d Hassan, Ph						
01.45 pm f	Participant OC12-OC17	Particpant OC18-OC23	Participant OC24-OC26, OB22	Participant OC27-OC29, OB23		
lty Airlang Department nd ANZAC	Coffee Break & Social time				**Venue: RK Khusus/ RS Anatomi** Jury meeting for oral & poster award-winning	
5.30-finish pm	**Venue: Grabik 2nd floor** **Award winning ceremony, PA & PA(K) Brevet award** **Closure**					

Biomolecular and Cellular Anatomy (PB23)

VIRGIN COCONUT OIL (VCO) AND POVIDONE IODINE ON THE DENSITY OF AND WOUND CONTRACTION IN WOUND HEALING PROCESS IN RATS

Darwin, E[1], Jamil M[2]

Histology, [2]School of Nursing, Faculty of Medicine, University of Andalas, Padang, Indonesia
Email : eryatidarwin@fk.unand.ac.id

ABSTRACT

nduction: Indonesia is an archipelago of biodiversity with around 30,000 plant species, and more species have been used for treatment. Virgin Coconut Oil (VCO) derived from palm that grows along Indonesian contains lauric, that induce wound healing process. **Objective:** To know the effect Coconut Oil (VCO) on wound healing on male and healthy Wistar rast. **Methods and material:** divided into three groups, there are group I (control), group II smeared with a VCO, and in group with povidone iodine for 10 days. After day 10, wound contraction was measured, and examination of wound tissue to determine the density of collagen. **Results:** The result shows significant differences in wound contraction between group II and II compared with control group histopstologic appearance, dense collagen density seen in the group who get the VCO, medium collagen density in the group of rats who received povidone iodine, and the density of collagen was abscess in the control group. **Conclusion:** The results of this study show that the VCO play a role in process such as povidone iodine.

VCO, wound, collagen

CTION

The skin is a greater total mass than any other organ in the body. Its made up of outer layer, the is composed of stratified squamous keratinizing epithelium. The deeper layer, the dermis is of vascularized and irregularly arranged fibroelastic connective tissue. The epidermis contains 5 From bottom to top the layers are named: stratum basale, stratum spinosum,stratum granulosum,stratum and stratum corneum. The bottom layer, the stratum basale, has cells that are shaped like columns. In the cells divide and push already formed cells into higher layers. As the cells move into the higher they flatten and eventually die. The top layer of the epidermis, the stratum corneum, is made of dead, flat that shed about every 2 weeks. There are three types of specialized cells in the epidermis, the produces pigment (melanin), the Langerhans' cell is the frontline defense of the immune system in and the Merkel's cell's function is not clearly known

The dermis also varies in thickness depending on the location of the skin. It is 0.3 mm on the eyelid and the back. The dermis is composed of three types of tissue that are present throughout - not in layers. of tissue are: collagen, elastic tissue, and reticular fibers. The two layers of the dermis are the and reticular layers: the upper, papillary layer, contains a thin arrangement of collagen fibers, and the ticular layer, is thicker and made of thick collagen fibers that are arranged parallel to the surface of the

The skin has many important functions, such as aesthetics and communication, sensation, regulation, storage and synthesis, contlor of evaporation and primarily for protection: an anatomical barrier from and damage between the internal and external environment in bodily defense. Once the protective is broken, the normal (physiologic) process of wound healing is immediately set in motion and repair after injury. Wound healing, which requires the concerted effort of numerous cell types, involves cell proliferation, differentiation, and apoptosis[3].

Mechanisms of Wound Healing

The goal of wound-healing process is to repair the damaged skin. The classic model of wound healing is into three or four sequential, yet overlapping phases: considered a (1) inflammatory, (2) proliferative (3) maturation and remodeling. When tissue is first wounded, blood comes in contact with collagen, blood platelets to begin secreting inflammatory factors. Platelets also express glycoproteins on their membranes that allow them to stick to one another and to aggregate, forming a mass. Fibrin and fibronectin link together and form a plug that traps proteins and particles and prevents further blood loss. This fibrin-nectin plug is also the main structural support for the wound until collagen is deposited. Migratory cells use plug as a matrix to crawl across, and platelets adhere to it and secrete factors. The clot is eventually lysed replaced with granulation tissue and then later with collagen. Platelets, the cells present in the highest

numbers shortly after a wound occurs, release a number of things into the blood, including ECM cytokines, including growth factors. Growth factors stimulate cells to speed their rate of division. release other proinflammatory factors like serotonin, bradykinin, prostaglandins, prostacyclins, and histamine, which serve a number of purposes, including to increase cell proliferation and area and to cause blood vessels to become dilated and porous[4].

In the inflammatory phase, bacteria and debris are phagocytosed and removed, and factors that cause the migration and division of cells involved in the proliferative phase. The proliferative characterized by angiogenesis, collagen deposition, granulation tissue formation, epithelialization, contraction. In angiogenesis, new blood vessels are formed by vascular endothelial cells. In granulation tissue formation, fibroblasts grow and form a new, provisional extracellular matrix excreting collagen and fibronectin. Concurrently, re-epithelialization of the epidermis occurs, in cells proliferate and 'crawl' atop the wound bed, providing cover for the new tissue[5].

In contraction, the wound is made smaller by the action of myofibroblasts, which establish wound edges and contract themselves using a mechanism similar to that in smooth muscle cells. maturation and remodeling phase, collagen is remodeled and realigned along tension lines and cells longer needed are removed by apoptosis.

The final stage of wound healing is maturation and remodeling, in which the granulation fibroplasia recede. In this phase, collagen is remodeled and realigned along tension lines and cells longer needed are removed by apoptosis. During this stage, the epidermis regenerates by undergoing of transient hypertrophy, while the provisional matrix is replaced by a dermal matrix of collagen and low cellularity scar. Degradation of the collagen matrix is mediated by matrix metalloproteinases, secreted by the epidermal cells, fibroblasts, endothelial cells, and macrophages. Eventually, the replaced by a new functional tissue. However, this process is not only complex but fragile, and interruption or failure leading to the formation of chronic non-healing wounds. Factors which may this include diabetes, venous or arterial disease, old age, and infection[6,7].

The process of wound healing consists of integrated cellular and biochemical events reestablishment of structural and functional integrity with regain of strength of injured tissue. Clinically often encounters non-healing, under-healing or over healing. Therefore the aim of treating a wound is shorten the time required for healing or to minimize the undesired consequences. Plants and their immense potential for the management and treatment of wounds. Various herbal products have been many countries in management and treatment of wounds such as *Aloe vera, Azardica indica, Lantana* Linn, *Tridax procumbens, Hydnocarpus wightiana, Chromolaena odorata, Helianthus annus* Linn, *auriculatum, Ginkgo biloba, Cedrus deodara, Centella asiatica, VCO* etc [8,9].

Virgin Coconut Oil (VCO)

As a tropical country Indonesia is rich in various species of flora, from about forty thousand flora that grows in the world, there are thirty thousand species have grown in Indonesia and 26% have cultivated and the rest still grows wild. About 940 species used as traditional medicine. One of that coconut tree, it has the world's most extensive coconut tree field (3712 million ha), and there are is the island of Java, Sumatra and Sulawesi[10].

OBJECTIVES

To determine the effect of VCO on the wound healing process, we conducted the research on were wounded. The wound were given with VCO and Povidone iodine. The wound contraction and of collagen in the wound were obvserved, and were compared with self healing wound.

METHODS AND MATERIAL

Three sets of experiments with three groups of male Wistar rats each consisting of 7 animals for studying wound healing. This research was recomended by Research Ethic Comitee of Medicine, Andalas University. Health Wistar rats, aged 8-12 weeks and 150-250 gram weight, and evaluated for 10 days . Group 1 treated with NaCl 0.9% was the control, group 2 was treated with group 3 was treated with Povidone iodine.

After the experimental period, the healing property of VCO was evaluated by monitoring the for contraction of the wound, collagen solubility pattern and histopathology of the tissue were also

Data were taken based on the comparison between two experimental groups. Data analysis for was calculated by ANOVA test. As for non-parametric data using Kruskal-Wallis test. The approach is experimental research designs.

evaluation of wound contraction, it was showed there were statistically significant difference ontrol group (group I) and treated group (group II and III) with p value <0.05. While the comparison ound contraction between group II and group III showed no statistically significant difference (p>0.05)

esentation of wound contraction on control group rats which reciefed NaCl (group I) and group II with VCO and group III treated with povidone iodine in day 10 after wounded

No	Group I (NaCl)	Group II	Group III
1	92,85	97,07	98,76
2	91,76	99,33	99,84
3	97,50	100,00	97,66
4	98,82	99,73	98,30
5	96,47	100,00	98,30
6	92,50	99,69	99,00
7	92,50	100,00	98,42
Mean	94,62	99,40	98,67
SD	2,87	1,05	0,68

p (II) = 0,02. p (I) : p (III) =0,011. p (II) : p (III) = 0,073

e 1: Comparison of wound healing in control group rats given (a), group II which treated with VCO (b) roup III which treated with povidone iodine (c) at day-10.

Histolpathological appearance of wound healing process on control group 10 days after wounded, ed incomplete epithelization, abcess with many capillaries. The density of collagen were moderate to e (table 2: figure 2).

On the group I which treated with VCO , the histopathological treatment showed complete elization, there are few capillaries and few inflammatory cells. The density of collagen in all of animal nd preparat are dens. While in group III which treated with povidone iodine the density of collagen are dens ost animal wound preparat (figure 3).

e 2: The density of colagen of wound on control group rats which reciefed NaCl (group I) and group II ed with VCO and group III treated with povidine iodine in day 10 after wounded

No	Group I	Group II	Group III
1	1	2	2
2	1	2	2
3	2	2	2
4	2	2	2
5	2	2	2
6	2	2	1
7	2	2	2

sity of collagen on wound
0 : few collagen fiber predominantly with loss conective tissue
1 : moderat collagen fiber with moderate loss conective tissue
2 : dens collagen fiber with a few connective tissue

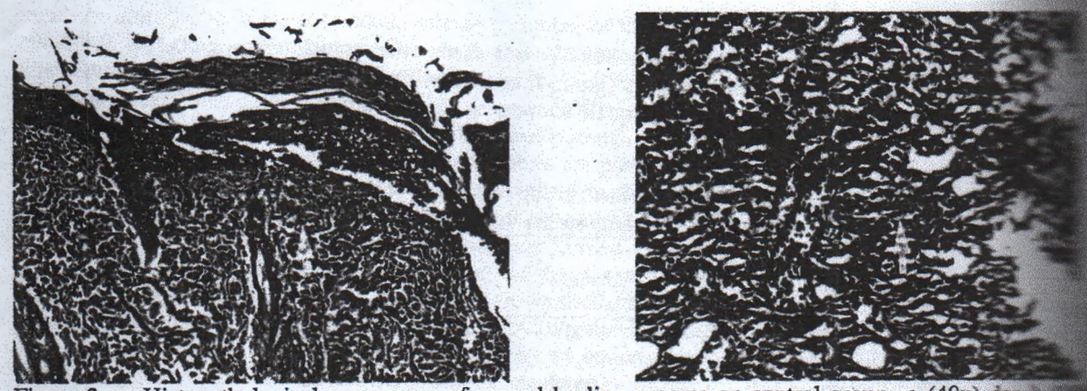


Figure 2 : Histopathological appearance of wound healing process on control group a (40x) dan
Collagen fiber and inflammatory cell (arrow)

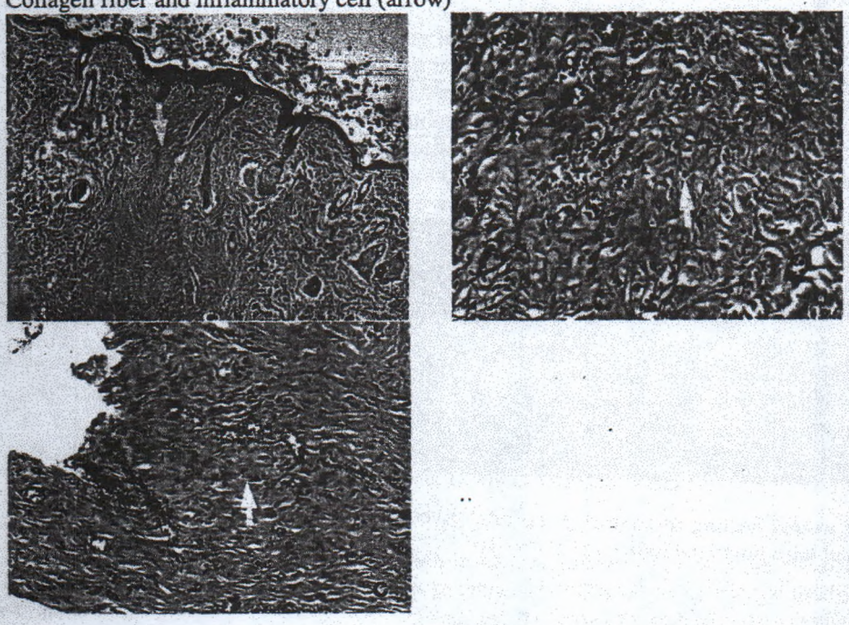


Figure 3 : Histopathological appearance of wound healing process in treated group. Group II treated
(a) and (b) and group III treated with povidone iodine (c)

DISCUSSION

Wound healing is a complex process that involves the organization of cells, chemical
extracellular matrix to repair the tissue. In turn, the treatment of wounds tries to quickly close the
obtain a functionally and esthetically satisfactory scar. To that end, it is indispensable to
understanding of the biological process involved in the healing of wounds and tissue regeneration[11].

One of fibroblasts' most important duties is the production of collagen. Collagen deposition
because it increases the strength of the wound; before it is laid down, the only thing holding the
the fibrin-fibronectin clot, which does not provide much resistance to traumatic injury. Also, cells
inflammation, angiogenesis, and connective tissue construction attach to, grow and differentiate on
matrix laid down by fibroblasts. Type III collagen and fibronectin are generally beginning to be
appreciable amounts at somewhere between approximately 10 hours and 3 days, depending mainly
size. Their deposition peaks at one to three weeks. They are the predominating tensile substances
phase of maturation, in which they are replaced by the stronger type I collagen[12].

This study shows that day 10 after wounded is the the final stage of wound healing , there
remodeling phase. During this phase, collagen is deposited by fibroblasts and formed into an organized
Initially, the collagen strands laid down in the wound are thin and run parallel to the wound surface.
remodeling, however, collagen production increases. At the same time, some destruction of the original

making room for the formation of new collagen, which is thicker and tends to be oriented along the lines within the wound.

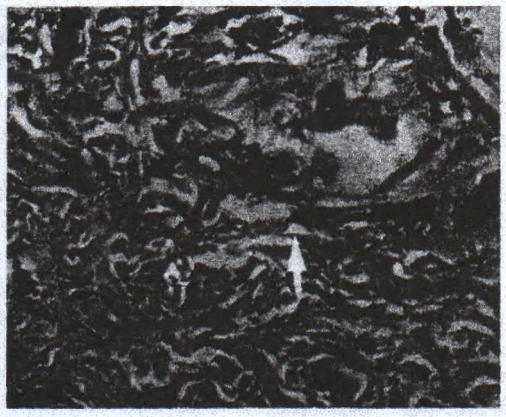


Figure 4: Histopathologis appearant of collagen fiber on wound process healing in rat, which treated with VCO, days after wounded

The anti-inflammatory property and the presence of *Lauric acid* of VCO is in the early synthesis of collagen fibers by mimicking. Farmacological activity of *Lauric acid* in VCO increase in blood quality,antioxidant, membrane stabilizing, improvement in cognition and pro-healing. Its can also promote epithelization without altering wound contraction. The other activity of *lauric acid* is to lysis of microbial membrane that inactivate various microba

The use of VCO as phyto-medicines for wound healing are not only cheap and affordable but are also reportedly safe as hyper sensitive reactions are rarely encountered with the use of these agents. These natural agents induce healing and regeneration of the lost tissue by multiple mechanisms. However, there is a need for scientific validation, standardization and safety evaluation of plants of the traditional medicine before these could be recommended for healing of the wounds

CONCLUSION

Wound is defined simply as the disruption of the cellular and anatomic continuity of a tissue. Wound can be produced by physical, chemical, thermal, microbial or immunological insult to the tissue. VCO is cheap, safe and natural active agent that can induce wound healing by stimulate cellular and colagen fiber. There is need advance research to use VCO for treatment of wound healing.

REFERENCES

1. Cormack DH, Essential Histology. 2nd ed. LW &W, 2001
2. Junqueira LC. Basic Histology.9th ed. Appleton and Lange, 2007
3. Raina R, Prawez S, Verma PK Pankaj NK. Medicinal Plants andtheir Role in Wound Healing. VetScan, 2008.3(1): 1-7.
4. Fuller BJ et al. Transforming Growth factor-B and wound healing. Focus on Basic Science. Perspectives in vascular surgery and endovascular therapy :2006 : 55-56.
5. Gillitzer R and Goebeler M.. Chemokines in cuteneus wound healing. Journal of Leukocyte Biology. 2003; 513-519.
6. Bello YM and Phillips TJ.. Recent Advances in Wound Healing. *JAMA*. 2000 ; 283 : 716-718.
7. Baines SR , Dang C, Soo C and Ting K. The phases of cutaneous wound healing. Expert Reviews in Molecular Medicine: 2003.
8. Suguna L. Efficacy of Butea monosperma on dermal wound healing in rats. Int J Biochem Cell Biol.1998; 37: 566–573
9. Molan P.C.The role of honey in the management of wounds. Journal of Wound Care. 1999: 8 (8):415-419
10. Setiaji B dan Prayogo S Membuat VCO Berkualitas Tinggi. Penebar Swadaya :2006.
11. Diegelmenn RF and Evans MC. Wound Healing : An Overview of Acute, Fibrotic and Delayed Healing. Frontiers in Bioscience 9 : 2004, 283-289.
12. Broughton G 2nd, Janis JE, Attinger CE . The basic science of wound healing: retraction. Surg Clin North Am 2006; 77:509–528.

•THE EFFECT OF VIRGIN COCONUT OIL (VCO) AND POVIDONE IODINE ON THE DENSITY OF COLLAGEN AND WOUND CONTRACTION IN WOUND HEALING PROCESS IN RATS

Darwin Eryati * and Mohammad Jamil **. * Departement Histology and **School of Nursing, Faculty of Medicine, University of Andalas. Padang, Indonesia

y

Background

The skin has many important functions, such as aesthetics and communication, sensation, regulation, excretion, storage and synthesis, control of evaporation and primarily for protection. Once the protective barrier is broken, the physiologic process of wound healing is immediately set in motion and repair itself after injury, which requires the concerted effort of numerous cell types, involves cell migration, proliferation, differentiation, and apoptosis. Clinically, one often encounters non-healing, under-healing or over healing. Therefore the aim of treating a wound is to either shorten the time required for healing or to minimize the undesired consequences.Plants and their extracts have immense potential for the management and treatment of wounds. Various herbal products have been used by many countries in management and treatment of wounds such as *Aloe vera, Azardica indica, Lantana camara* Linn, *Tridax procumbens, Jasminum auriculatum, Ginkgo biloba, VCO* etc. To determine the effect of VCO on the wound healing process, the wound contraction and the density of collagen in the experimental animal wound were obvserved.

Methods

Three groups of male Wistar rats each consisting of 7 health Wistar rats, aged 8-12 weeks and 150-250 gram weight, were wounded and evaluated for 10 days . Group 1 treated with NaCl 0.9% was the control, group 2 was treated with VCO, and group 3 was treated with Povidone iodine. After the experimental period, the healing property of VCO was evaluated by monitoring the time taken for contraction of the wound, collagen solubility pattern and histopathology of the tissue were also analyzed.

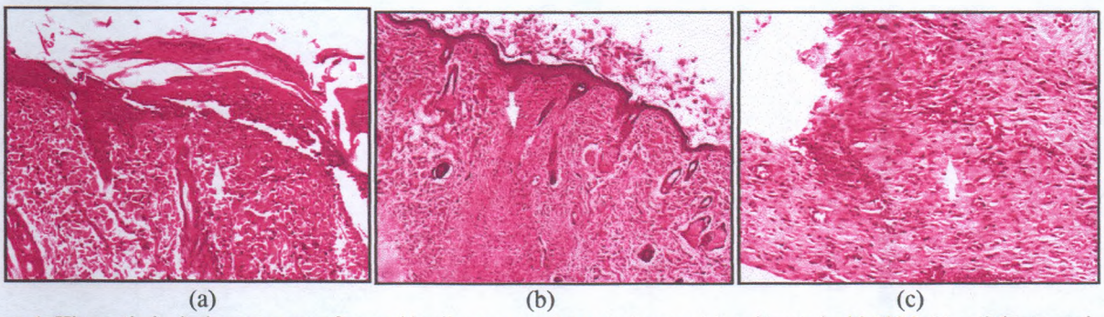
Results:

In evaluation of wound contraction, it was showed there were statistically significant difference between control group (group I) and treated group (group II and III) with p value <0.05. While the comparison of wound contraction between group II and group III showed no statistically significant difference (p>0.05) (table 1).On the group I which treated with VCO , the histopathological treatment showed complete epithelization, there are few capillaries and few inflammatory cells. The density of collagen in all of animal wound preparat are dens. While in group III which treated with povidone iodine the density of collagen are dens in almost animal wound preparat (Figure:1)

This study shows that during the skin-remodeling phase collagen is deposited by fibroblasts and formed into an organized network. Initially, the collagen strands laid down in the wound are thin and run parallel to the wound surface. At the same time, some destruction of the original collagen occurs, making room for the formation of new collagen, which is thicker and tends to be oriented along the lines of stress within the wound.

Table 1: Percentage of wound cintraction on control group(Group I), treated group (group II) and treated with povidine iodine (group III) 10 days after wounded

No.	Group I (NaCl)	Group II	Group III
1	92,85	97,07	98,76
2	91,76	99,33	99,84
3	97,50	100,00	97,66
4	98,82	99,73	98,30
5	96,47	100,00	98,30
6	92,50	99,69	99,00
7	92,50	100,00	98,42
Mean	94,62±2,87	99,40±1,05	98,67±0,68



(a) (b) (c)

Figure 1: Histopathological appearance of wound healing process in control group (a) and treated with VCO (a) and (b) treated with povidone iodine (c).Arrow: collagen fiber

Conclusion

Wound is defined simply as the disruption of the cellular and anatomic continuity of a tissue. Wound may be produced by physical, chemical, thermal, microbial or immunological insult to the tissue. VCO is cheap, safe, and natural active agent that can induce wound healing by stimulate cellular and colagen fiber. There is need advance research to use VCO for treatment of wound healing

ASIA-PACIFIC INTERNATIONAL CONGRESS OF ANATOMY (APICA)
INDONESIAN ANATOMIST ASSOCIATION
(PERHIMPUNAN AHLI ANATOMI INDONESIA-PAAI)

Certificate

This certificate is presented to:

Prof. Eryati Darwin, dr

As Speaker/~~Participant/Moderator/Jury/Editor boards/Committee~~

in

6th Asia-Pacific International Congress of Anatomy

13th National Congress of Indonesian Anatomist Association

(6th APICA & 13th PIN-PAAI)

22nd-23rd of July, 2011

Faculty of Medicine Airlangga University

Surabaya, Indonesia

Prof. Yun Qing Li

Dr. Abdurachman, dr., M.Kes, PA(K)