Mathematical Approaches Towards Healing of Wounds with Underlying Sickness

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Abstract
Skin is the largest organ in the Human body which covers the body and plays vary important roles including thermoregulation, sensory and host defense. With these functions, it's very important to protects and safe guide human skin from being damage. This work explores the application of mathematical modeling on skin wound healing with underlying sickness, focusing on the significance of constant (C) in indefinite integrals result. A novel mathematical framework is developed, incorporating ordinary differential equations to describe the dynamics of wound healing. The Ordinary Differential Equations involving growth factor control of keratinocyte, epidermal and dermal wounds healing was adopted, modified, solved and analyzed with maple 18 software. The initial conditions were set to be the initial period of injury or fresh wound and as the day (time) increases the wound site reduces till it yields zero (0) for each layer of skin tissue. It was observed that the results show that constant (C) plays a crucial role in predicting wound healing outcomes and optimizing treatment strategies. Constant C plays an important role in the body system of patient with underlying sick by bringing the degenerated body system like diabetic patient back to normal with time subject to continuity and consistence's in the administrations of insulin and with proper monitoring of carbohydrate consumption. The findings are in good agreement with recent studies.

Key Words: *Constant value (C), Epidermal, Dermal, Underlying Sickness and Wound healing.*

INTRODUCTION

Wound healing is a complex and dynamic process that involves the coordinated action of Multiple cell types, growth factors, and biochemical pathways. In healthy individuals, this process is highly efficient, with wounds typically progressing through the stages of inflammation, proliferation, and remodeling to achieve complete closure. However, in individuals with underlying conditions such as diabetes, wound healing is often impaired, leading to chronic wounds that are resistant to treatment and prone to complications.

Bowden, (2015) developed a time-dependent ordinary differential equation model, and this model focusses on the main processes contributing to closure of a full thickness wound: proliferation in the epidermis and growth and contraction in the dermis. The model simulations suggest that the relative contributions of growth and contraction to healing of the dermis are altered in diabetic wounds. The work also investigates the balance between growth and contraction by developing a more detailed, spatially-resolved model using continuum mechanics. Due to the initial large

retraction of the wound edge upon injury, the adoption of a non-linear elastic framework was considered. Morpho-elasticity theory was adopted, with the total deformation of the material decomposed into an addition of mass with an elastic response. The model investigates how interactions between growth and stress influence dermal wound healing. The model reveals that contraction alone generates unrealistically high tension in the dermal tissue and, hence, volumetric growth must contribute to healing. Droplets from human body system could be transmitted and harmful (Virus Disease outbreak) if necessary, measure is not in place Bagbe et al., (2019).

Epidermis, the outermost layer of the skin, is a stratified epithelium consisting of melanocytes, Langerhans cells, Merkel cells, and the predominant cell type, keratinocytes (Abdo et al., 2020). The multilayered structure formed as a result of progressive proliferation with different of keratinocytes, which move from the lower basal layer adjacent to the surface membrane to the upper differentiating spinous, granular, and cornified layers (Lee et al., 2017). Each layer performed distinct morphology and specific gene (protein) result contributes to the development of epidermal structure and the permeability barrier. The permeability problem of the skin is primarily mediated by the outermost layer also, the stratum corneum, composed of corneocytes and inter-corneocyte lipids released from keratinocytes from lamellar bodies

Badejo et al., (2015) studied the Modeling of surface tension of micro droplets with different pressure gradients which resulted into how the surface area can be managed with the involvement of microdroplets. The researchers stressed the importance of surface area with time.

Dermis, a connective tissue layer filled with blood vessels, nerves, and with different cell types (Proksch et al., 2008). It was cleared that fibroblasts serve as the primary cells in the dermis, with myofibroblasts, mast cells, macrophages and endothelial cells having crucial cellular components within the dermal structure. Fibroblasts play key contributors to wound healing. They process of collagen and other extracellular matrix (ECM) components, provided structural support for tissue repair (Tracy et al., 2016).

The subcutaneous tissue, also referred to as hypodermis, is located under the dermis and is primarily composed of adipocytes and connective tissue. Adipocytes provide thermal insulation and energy storage, and their presence do influence wound healing indirectly by affecting overall skin nature. The subcutaneous tissue also contains nerves and blood vessels that contribute to supplying nutrients and oxygen to the surrounding tissues, indirectly supporting the healing process in the skin (Bailey et al., 2014). Adipose tissue releases cytokines and growth factors that modulate the immune response and influence the overall healing process. These intricate skin layers form the foundation for the remarkable regenerative capacities of this organ during the wound healing process.

Badejo et al., (2015) studied the modeling of surface tension of microdroplets. The researchers varied the Pressure gradient in order to observed different area or site with microdroplet. The results obtained shows that the presence of surfactant at interface reduces the surface tension at the site also, reducing interfacial tension improves the stability of a droplet which is in line with improving the contact between the drug and skin which will leads to enhanced permiability of the drug through skin layers.

Dasari et al., (2021) studied wound healing as a complex process that proceeds through phases of inflammation, proliferation, and remodelling. It was also observed that diabetes results in several pathological changes that impair almost all of these healing processes. It was clearly noted that diabetic wounds are often characterized by excessive inflammation therefore, reduced

angiogenesis. The reseach resulted that due to these changes, diabetic patients are at a higher risk for postoperative wound healing complications.

Kolimi et al., (2022) studied Innovative Treatment Strategies to Accelerate Wound Healing and it was noted that Chronic wounds affect patients' quality of life along with increased morbidity and mortality with huge financial burden to healthcare systems worldwide thus, the required specialized biomedical intensive treatment management will always be involved. The researchers also noted that clinical assessment and management of chronic wounds remains challenging despite the development of various therapeutic regimens owing to its long-term treatment with pains it required not to talk of its complex wound healing mechanism.

Growth factors are endogenous signaling proteins that promote wound healing by stimulating angiogenesis, mitogenesis, granulation, remodeling, and reepithelization (Martí-Carvajal et al, 2015, Yamakawa et al., 2019). As mentioned previously, diabetic wound healing is hindered by deregulated growth factors. Notable growth factors that have successfully aided wound healing in nonhuman models are platelet-derived growth factors (PDGF), vascular endothelial growth factors (VEGF), and fibroblast growth factors (FGF). The only growth factor therapy that is currently approved for clinical use by the Food and Drug Administration (FDA) is PDGF in the treatment of DFUs (FDA, 2021)

Adebayo *et al*., (2023), studied Mathematical investigation of normal and abnormal wound healing dynamics, adopting local and non-local models. The investigation covered the movement of cells during wound healing, the result of biomechanical interactions that combine cell responses with growth factors as well as cell-cell and cell-matrix interactions. The researchers observed that cells can communicate and interact locally and non-locally with other cells inside the tissues through mechanical forces that act locally and at a distance, as well as through long non-conventional cell protrusions. The model later investigated both normal and abnormal wound healing also, how the wound margins come together from the at the wound site, and when the wound heals from the bottom of the wound site.

Takeo et al., (2015) Studied remarkable examples of scarless healing in fetal skin and appendage regeneration in adult skin following the infliction of large wounds. The models used in the studies have offered a new platform for investigations of the cellular and molecular mechanisms concerning underlying wound healing and skin regeneration in mammals. The article focuses on the contribution of skin appendages to wound healing and, conversely, skin appendage regeneration following injuries.

Diabetic foot ulcers (DFUs) are a common and debilitating complication of diabetes, affecting an estimated 15% of patients worldwide. These wounds are characterized by impaired blood flow, neuropathy, and altered biochemical pathways, which collectively disrupt the normal wound healing process. As a result, DFUs often require prolonged treatment, leading to increased healthcare costs, reduced quality of life, and heightened risk of amputation.

Despite the clinical significance of DFUs, current treatment strategies are often ineffective, highlighting the need for innovative approaches to wound healing. Mathematical modeling has emerged as a powerful tool for understanding complex biological systems, including wound healing. By integrating mathematical techniques with experimental data, researchers can develop predictive models that simulate the dynamics of wound healing and identify key factors that influence treatment outcomes.

This research aims to adopt and apply mathematical approaches to investigate wound healing in diabetic patients either with ordinary or partial differential equation as found in Usman *et al*.,

(2015). Specifically, we will employ ordinary differential equations (ODEs), by considering it solutions of indefinite, and agent-based modeling to simulate the spatiotemporal dynamics of wound healing in the presence of diabetes-related complications. Our goal is to identify novel therapeutic targets constant (C) and strategies for enhancing wound healing in diabetic patients by adopting the constant (C) as the approachable solution, with the ultimate aim of improving treatment outcomes and reducing the burden of DFUs on healthcare systems worldwide.

Olowu et al., (2024) studied analytical Investigation of the Impact of Failed Treatments on the Transmission Dynamics of Onchocerciasis using a model that revealed the proportion of affected human being who were able to complete their treatment as well as the relative infectiousness of those that failed to complete their treatment have significant influence on the movement of onchocerciasis in the whole number of human inhabitants domiciled in an environment.

It is clear that diabetes can affect wound healing in several ways, like Blood flow which can make it difficult for blood to reach the wound healing site. Inflammation which can prolong the inflammatory phase of wound healing site. Collagen which can also delay the formation of granulation site tissue. Keratinocytes which reduce epithelialization of the wound site. Fibroblasts which can prevent collagen deposition. Immunity which can make it harder to fight infection. Neuropathy which can make it hard to detect wounds site. Hypoxia which can contribute to poor wound healing. Angiogenesis which can impair wound healing. pH which can affect the microbiome. Insulin this can decrease the expression of insulin-like growth factors (IGFs), which can affect wound healing site. Akanbi et al., (2024) examined the influence of socio-demographic factors on care types received by elderly people.

Choudhary *et al*., (2024) explores the diverse array of skin wound healing models utilized in research, ranging from rodent excisional wounds to advanced tissue engineering constructs and microfluidic platforms. the influence of lipids on the wound healing process was examined, by emphasizing their role in enhancing barrier function in restoration, modulating inflammation, promoting cell proliferation, and promoting remodeling of the wound site. Free flow of fluid in the system reduces the body temperature Badejo et al., (2024). The work emphasized on Lipids, such as phospholipids, sphingolipids, and ceramides, effects that play crucial roles in membrane structure, cell signaling, and tissue repair. considering the interplay between lipids and the wound microenvironment provides valuable insights into the development of novel therapeutic strategies for promoting efficient wound healing and tissue regeneration. The work highlights the significance of investigating skin wound healing models and elucidating the intricate involvement of lipids in the healing process, importance of pressure gradient (Badejo *et al*., 2019 & Usman *et al*., 2019) which will offer potential avenues for improving clinical outcomes in wound management.

MATHEMATICAL PARAMETERS

These are ordinary differential equation model for epidermal and dermal wound healing. An existing model of wound healing that focus on either epidermal or dermal healing was adopted and it will be solved and analyzed base on the focus of this wok. Full thickness wounds injure the epidermis and the dermis, and therefore require both layers to heal simultaneously because in healthy skin these layers interact both chemically and mechanically. Usman et al., (2020) concluded that the methods of Klein-Gordon equations is capable to converge to exact solutions with least number of iterations. Mathematical models involving growth factor control of epidermal wound healing have focused on epidermal growth factor (EGF), which is produced in the

epidermis. However, the activity of keratinocyte growth factor (KGF), which is produced by fibroblast cells, is restricted to epidermal cells and has been shown to be a stronger mitogen than EGF [Marchese *et al*., 1990]

$$A_e^* = \frac{A_e}{AR}, A_s^* = \frac{A_s}{AR}, A_d^* = \frac{A_d}{A_R}, t^* = \frac{t}{T} \quad (1)$$

where T is an appropriately chosen timescale. Dimensionless parameters are defined as follows:

$$\lambda = rT, \beta_o = k_o T, \beta_1 = K_1 T, \quad \alpha = \frac{c}{E}, \quad \epsilon = \frac{\mu}{ET}, \quad t_c^* = \frac{t_c}{T}, \quad \theta^* = \frac{\theta}{T} \quad (2)$$

Change in epidermis with time

$$\frac{dA_e}{dt} = \lambda(i + v - A_e)\left(\frac{A_e - \gamma A_d}{1 - \gamma A_d}\right) \quad (3)$$

Change in the area of the relaxed dermal wound with time

$$\frac{dA_s}{dt} = -\left(\omega + \Psi\left(\frac{A_s - A_d}{1 + v - A_d}\right)\right)(1 + v - A_d)A_d \quad (4)$$

Change in epidermis with time

$$\epsilon\frac{dA_d}{dt} = A_s - A_d - \alpha\mathcal{H}(t - t_c)A_d \quad (5)$$

Expanding equation (3) yields

$$\frac{dA_e}{dt} = \frac{\lambda\left(iA_e - i\gamma A_d + vA_e - v\gamma A_d - {A_e}^2 + A_e\gamma A_d\right)}{1 - \gamma A_d} \quad (6)$$

Factor out $(1 - \gamma A_d)$ from equation (6) yields

$$\frac{dA_e}{dt} = \frac{\lambda}{1 - \gamma A_d}\left(iA_e - i\gamma A_d + vA_e - v\gamma A_d - {A_e}^2 + A_e\gamma A_d\right) \quad (7)$$

Integrating equation (7) results into

$$A_e = \frac{\lambda}{1 - \gamma A_d}\left(iA_e - i\gamma A_d + vA_e - v\gamma A_d - {A_e}^2 + A_e\gamma A_d\right)t + C_1 \quad (8)$$

Further expansion of (8) results into

$$A_e = \frac{\lambda}{1 - \gamma A_d}\left(iA_e t - i\gamma A_d t + vA_e t - v\gamma A_d t - {A_e}^2 t + A_e\gamma A_d t\right) + C_1 \quad (9)$$

here is the solution of equation (3)

From equation (4) there is changes in the area of the relaxed dermal wound with time

$$\frac{dA_s}{dt} = -\left(\omega + \Psi\left(\frac{A_s - A_d}{1 + v - A_d}\right)\right)(1 + v - A_d)A_d$$

$$\frac{dA_s}{dt} = -\left(\omega + \frac{A_s\Psi - A_d\Psi}{1 + v - A_d}\right)\left(A_d + A_d v - {A_d}^2\right) \quad (10)$$

$$\frac{dA_s}{dt} = \frac{\left(\omega A_d + \omega v A_d - \omega {A_d}^2 + \Psi A_s A_d + \Psi A_s v A_d - \Psi A_s {A_d}^2 - \Psi {A_d}^2 - \Psi {A_d}^2 v + \Psi {A_d}^3\right)}{1 + v - A_d} \quad (11)$$

$$\frac{dA_s}{dt} = \frac{1}{1+v-A_d}\left(\omega A_d + \omega v A_d - \omega {A_d}^2 + \Psi A_s A_d + \Psi A_s v A_d - \Psi A_s {A_d}^2 - \Psi {A_d}^2 - \Psi {A_d}^2 v + \Psi {A_d}^3\right) \quad (12)$$

Integrating equation (12) results into

$$A_s = \frac{1}{1+v-A_d}\left(\omega A_d + \omega v A_d - \omega {A_d}^2 + \Psi A_s A_d + \Psi A_s v A_d - \Psi A_s {A_d}^2 - \Psi {A_d}^2 - \Psi {A_d}^2 v + \Psi {A_d}^3\right)t + C_2 \quad (13)$$

Further expansion of equation (13)

$$A_s = \frac{1}{1+v-A_d}\left(\omega A_d t + \omega v A_d t - \omega {A_d}^2 t + \Psi A_s A_d t + \Psi A_s v A_d t - \Psi A_s {A_d}^2 t - \Psi {A_d}^2 t - \Psi {A_d}^2 v\, t + \Psi {A_d}^3 t\right) + C_2 \quad (14)$$

Here is the solution of equation (4)

from equation (5)

$$\epsilon \frac{dA_d}{dt} = A_s - A_d - \alpha \mathcal{H}(t - t_c)A_d$$

Rearranging equation (5) yields

$$\frac{dA_d}{dt} = \frac{1}{\epsilon}(A_s - A_d - \alpha \mathcal{H}(t - t_c)A_d) \quad (15)$$

Integrating equation (15) to yields

$$A_d = \frac{1}{\epsilon}(A_s - A_d - \alpha \mathcal{H}(t - t_c)A_d)t + C_3 \quad (16)$$

Further expansion of equation (16) results into

$$A_d = \frac{1}{\epsilon}\left(A_s t - A_d t - \alpha \mathcal{H} t A_d (t - t_c)\right) + C_3 \quad (17)$$

Here is the solution of equation (5)

The following equations (9), (14) and (17) will be computed together along with the initial condition with the help of maple software to determine the final results and analysis.

With the initial conditions

$$A_e(0) = 1, \qquad A_s(0) = 1, \quad A_d(0) = \frac{A_\circ}{A_r} = K_d. \quad (18)$$

RESULTS AND DISCUSSION

Figure 1 Present the wound site profile of Dermal tissue, Epidermal tissue and the subset or relax tissue. The high values of $A_d(t)$, $A_e(t)$ and $A_s(t)$ was as a result of inflammation at the early period of the injury and shows that the response amplitude increases as the force of the load increases Usman *et al*., (2020). It was observed that wound site reduces with time as the value of Dermal tissue $A_d(t)$, Epidermal tissue $A_e(t)$ and other essential part of the wound site communicate well like allowing the flow of fluids (blood) to visit the site gradually. $A_s(t)$ of both tissues reduces with increase in time till the size of the wound reduced to zero (0), indicating the total closure of the wound site.

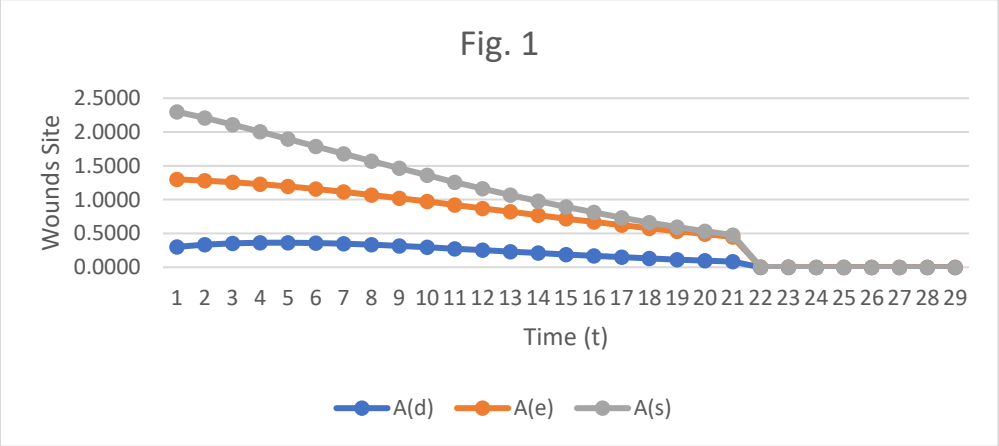

Figure 2 Present the wound site profile with the effect of time on the skin tissues. It was observed that the wound site was building up with time as the value of Dermal tissue $A_d(t)$, Epidermal tissue $A_e(t)$ and the presence of fluids flow or what we assumed to be the subset $A_s(t)$ of both tissues. The parallel level of the three tissues representing the wound site, is clearly seen that with time all the layers (Tissues) at the wound site that were damage will align gradually indicating the total closure of the wound site. But it is obvious that the profile moved from down left to the top right-hand side. This also shows that new tissue filled up the wound site with time.

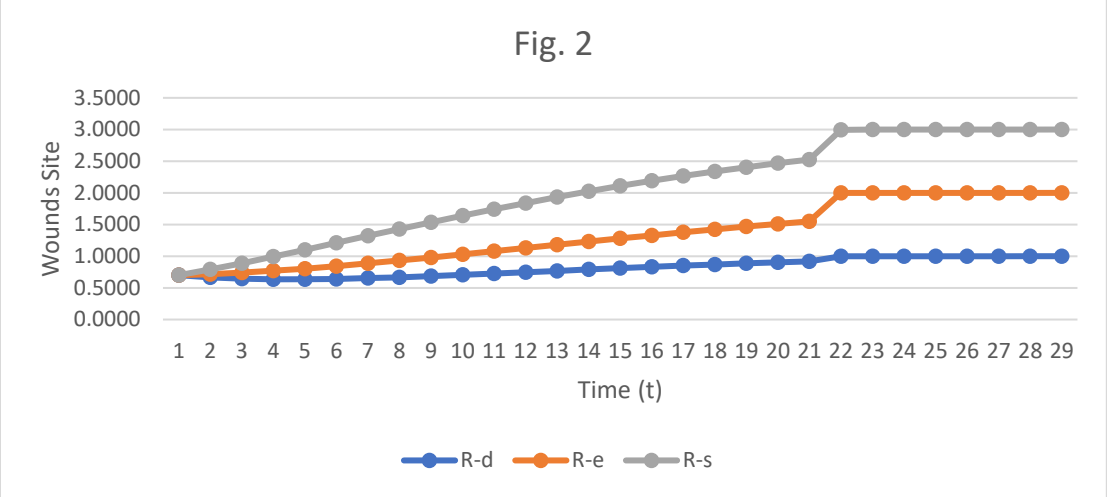

Figure 3 Present the wound site profile for both Fig. 4.1 and Fig. 4.2 which we can also call super impose of the two figures in a particular profile. It was observed that the wound site close up within the proximity of 0.0000 to 1.0000 while the time remain as it were in both Figures above.

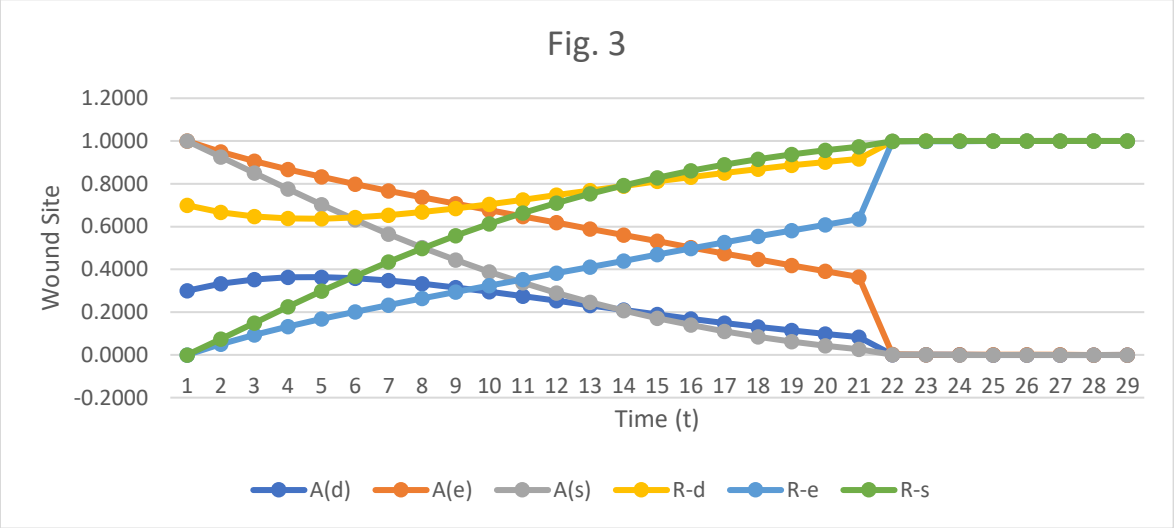

Figure 4 Presents a profile with the space each tissue occupied in the wound site. The result shows that the wound site was covered after some time but all the tissues did not gain the exalt shape of the site before the wound or injury occurred. It was also observed that all the tissues represented did not again it shape back as at the time the wound site was covered up. Suggesting that total wound healing does not take place immediately when the wound site cover up, research shown that it will takes some period for total healing to take place at the site. The constant (C) has been identified to be the recovery value that will determine the exact time of total recovery of the wound site.

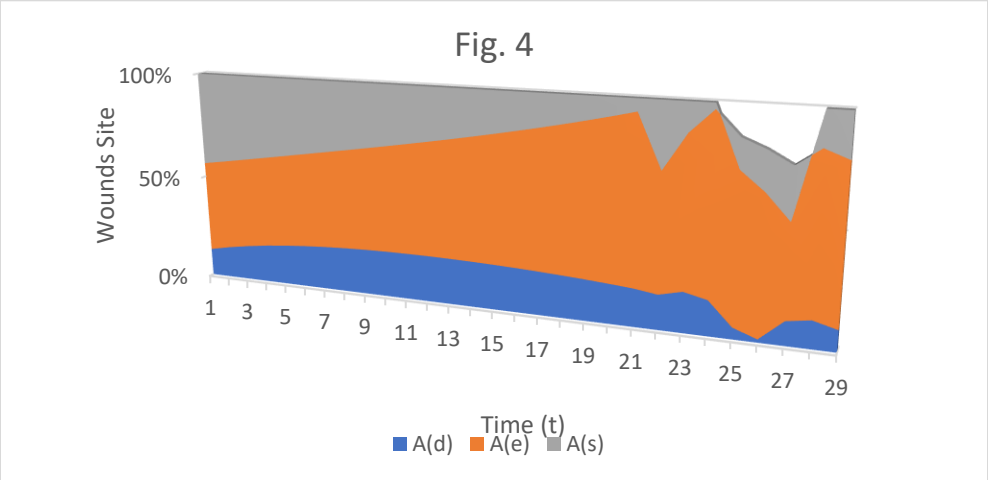

Figure 5 Presents the wound site profile with the space each tissue occupied with time. This shows that constant (C) was adopted with time and the results recorded that all the tissues regained their shape back as they were before the wound. This will enable the proper fluids flow at the wound site.

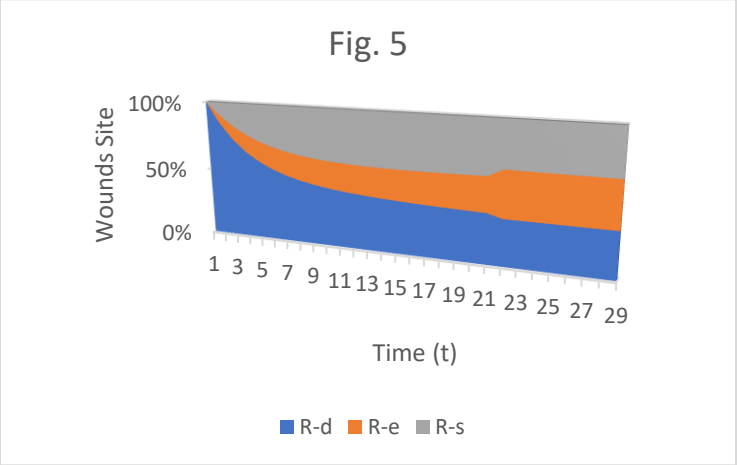

Table 1 shows the observations and the results of the Dermal wound site $(A_d(t))$, Epidermal wound site $(A_e(t))$, healing site factor $(A_s(t))$ or subset of Dermal and Epidermal wound site. Also, the complement of the wound site. Complement of the wounds healing site $(A_R - A_d)(t)$ *and* $(A_R - A_e)(t)$ Dermal and epidermal respectively. The initial conditions were set and used as the initial point of the wound state and it was observed that the site of wound was coverup or filled up gradually with time. Dermal wound site $(A_d(t))$ was observed to increasing from $0.3000mm^2$ to $0.3636mm^2$ initially, indicating swell up of the wound site while other tissues reduce gradually with time. As the time increases it shows that the wound site reduced in size with the cooperation of all tissue. As time increases to 8.40 the changes in the tissue level have reduced to zero (0), indicating the closure of the wound site.

Time (t)	$A_d(t)$	$A_e(t)$	$A_s(t)$	$(A_R - A_d)(t)$	$(A_R - A_e)(t)$	$(A_R - A_s)(t)$
0.00	0.3000	1.0000	1.0000	0.7000	0.0000	0.0000
0.10	0.3328	0.9501	0.9261	0.6672	0.0499	0.0739
0.20	0.3529	0.9067	0.8505	0.6471	0.0933	0.1495
0.30	0.3625	0.8678	0.7753	0.6375	0.1322	0.2247
0.40	0.3636	0.8321	0.7022	0.6364	0.1679	0.2978
0.50	0.3580	0.7988	0.6320	0.6420	0.2012	0.3680
0.60	0.3472	0.7670	0.5651	0.6528	0.2330	0.4349
0.70	0.3325	0.7363	0.5021	0.6675	0.2637	0.4979
0.80	0.3150	0.7063	0.4430	0.6850	0.2937	0.5570
0.90	0.2954	0.6767	0.3879	0.7046	0.3233	0.6121
1.00	0.2746	0.6474	0.3368	0.7254	0.3526	0.6632
1.10	0.2531	0.6182	0.2897	0.7469	0.3818	0.7103
1.20	0.2314	0.5891	0.2465	0.7686	0.4109	0.7535
1.30	0.2100	0.5601	0.2071	0.7900	0.4399	0.7929
1.40	0.1890	0.5312	0.1714	0.8110	0.4688	0.8286
1.50	0.1687	0.5026	0.1393	0.8313	0.4974	0.8607
1.60	0.1494	0.4741	0.1106	0.8506	0.5259	0.8894

1.70	0.1311	0.4461	0.0851	0.8689	0.5539	0.9149
1.80	0.1140	0.4185	0.0627	0.8860	0.5815	0.9373
1.90	0.0981	0.3915	0.0432	0.9019	0.6085	0.9568
2.00	0.0834	0.3651	0.0264	0.9166	0.6349	0.9736
7.00	0.0005	0.0017	0.0009	0.9995	0.9983	0.9991
8.00	0.0002	0.0009	0.0002	0.9998	0.9991	0.9998
8.40	0.0001	0.0006	0.0000	0.9999	0.9994	1.0000
9.00	0.0000	0.0004	-0.0001	1.0000	0.9996	1.0001
9.20	0.0000	0.0003	-0.0001	1.0000	0.9997	1.0001
10.00	0.0000	0.0001	0.0000	1.0000	0.9999	1.0000
12.00	0.0000	0.0000	0.0000	1.0000	1.0000	1.0000
12.10	0.0000	0.0000	0.0000	1.0000	1.0000	1.0000

Table 1

CONCLUSION

Firstly, this work addressed how the wound site can recover to its original state with time for body system that does not have an underlying sickness like diabetics. Natural the wound site closure will take place with time provided there is no infection.

It also considered the cases of system with underlying sicknesses like diabetics' body system but the closure of the wound and the total healing of the site will only take place by adopting the idea and applications of constant (C) which plays a crucial role in predicting wound healing outcomes and optimizing treatment strategies. Constant C plays an important role in the body system of patient with underlying sick by bringing the degenerated body system like diabetic patient back to normal with time subject to continuity and consistence's in the administrations of insulin and with proper monitoring of carbohydrate consumption.

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