



TRASDERMAL PATCH OF NATURAL ORIGIN AND IDENTIFICATION OF PHYTOCHEMICAL

Arpita R. Pawar, Aman S.Nadaf, Saurabh A.Madane,
Prathmesh P. Deshmukh, *Devraj A. Patil.*

Rajarambapu Collage Of Pharmacy, Kasegoan, Tal:-Walwa, Dist:-Sangli, Maharashtra- 415409

ABSTRACT

Current research is interested in advanced therapeutic dressings that participate actively in wound healing to achieve quick and complete healing of chronic wounds. Due to the massive financial load experienced by the world as a whole, fresh strategies to promote rapid wound healing are desired. This article examines the state of wound healing and wound care products today, focusing on the need for more sophisticated forms of wound therapy as well as some of the current issues and causes that are influencing this need. The paper mostly reviews data from peer-reviewed literature and other readily accessible sources, such as the FDA. Treatment of chronic wounds, including amputations, diabetes and leg ulcers, pressure sores, surgical wounds, and traumatic wounds (such as burns and accidents), when patient immunity is poor and the risk of infections and consequences is high, is a key area of focus. The major types of dressings include medicated moist dressings, tissue-engineered alternatives, biological dressings based on biomaterials, biological dressings obtained from both biological and natural sources, medicated sutures, and various combinations of the aforementioned groups. The review concludes with a brief discussion of the prospects for advanced wound healing in routine clinical care, including some of the newly developed techniques such as hyperbaric oxygen, negative pressure wound therapy, and laser wound healing.

KEY WORD:-Transdermal, Adhesive, Corneum, Appendageal

Introduction:

Nowadays, 74% of medications are consumed orally and are not as valuable as the most sought-after. A transversal drug delivery method was created to advance such characters. Transdermal drug delivery system (TDDS) emerged as a significant component of innovative drug delivery systems with the development of today's pharmaceutical dosage forms. Because of their unique advantages, transdermal dosage forms—while still more expensive than traditional formulations—are growing in popularity. enhanced bioavailability, controlled absorption, more equal plasma levels, painless administration, decreased side effects Transdermal

medication delivery may have several benefits, such as the ability to stop drug administration by simply removing the skin patch [1]. Oral The most popular medication delivery systems are conventional dosage forms like tablets and capsules, although these dosage forms have issues with first pass gastric drug/enzyme metabolism. Additional issues with the oral route include bad taste, aroma, and colour. Numerous extra issues

are developing when using medications, which causes issues during treatment. Patients occasionally stop complying. Transdermal patches are a non-invasive and irritant-free delivery system for TDDS medications, ensuring that they have a precise duration of action. It is a desirable alternative to traditional procedures for systemic medication administration [2]. Transdermal drug delivery is a type of topical drug delivery that involves delivering a substance via the skin to have a systemic impact.

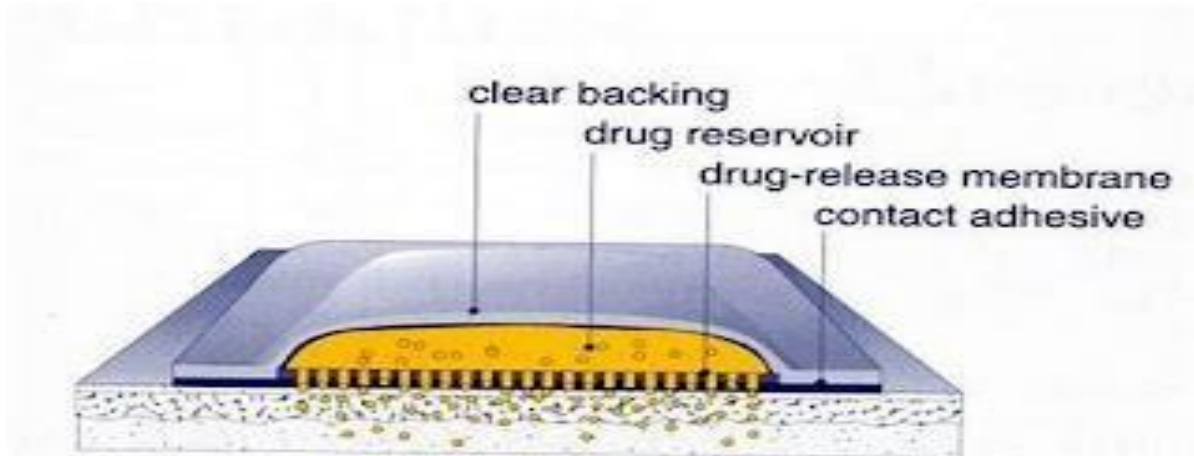


Figure 1: Transdermal patch

Types of transdermal patch:

1. Single layer drug-in-Adhesive Patches: In this system, the drug remains in contact with the adhesive layer which is attached to the skin. In the layer of adhesive helps to releasing the drug and also serve to adhere to the various layers together along with the skin.



Figure 2: Single layer drug-in-Adhesive

2. Multi-layer drug-in-Adhesive Patches: The single layer drug in adhesive, which includes introducing the medicine directly into the adhesive layer, is comparable to the multi layer drug in adhesive. In this technique, the medication is immediately released from the reservoir by one of the layers. This patch features both a long-lasting backing and a short-term liner.

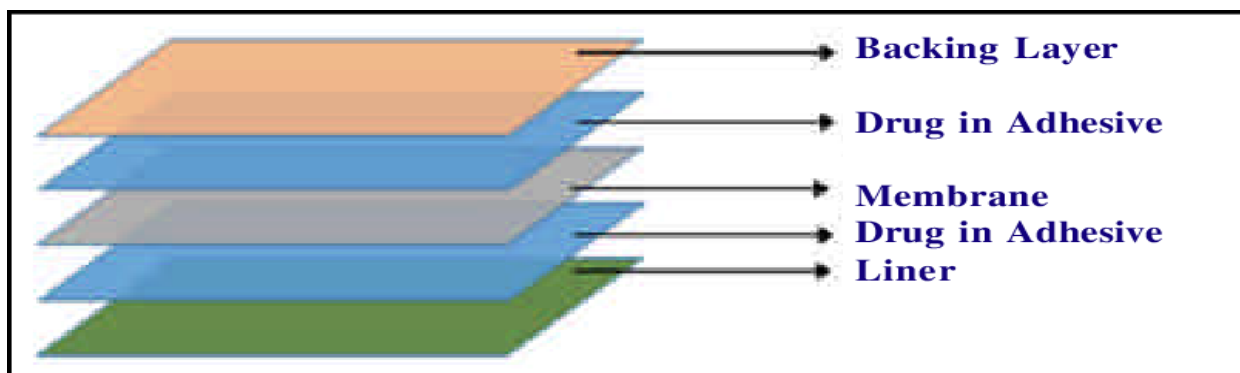


Figure 3: Multi-layer drug-in adhesive patch

3. Reservoir type patches: The single layer drug-in-adhesive and multilayer drug-in-adhesive transdermal systems do not have a separate drug layer like the reservoir system. This system has a compartment for fluids containing a medicine solution or suspension that is kept apart from the liner by an adhesive and membrane. The backing layer also supports this patch scheme. The rate of release in the reservoir system is zero order.

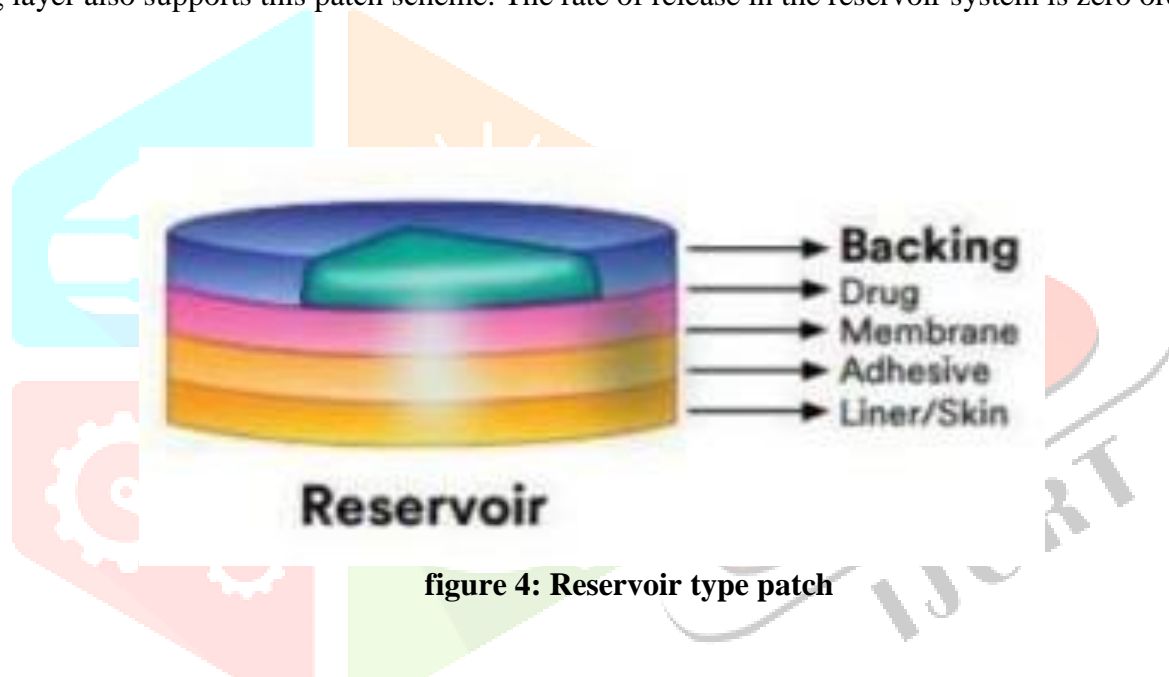


figure 4: Reservoir type patch

4. Matrix type patches: The matrix system consists of a medicament layer of a semisolid matrix that is in direct contact with the liner layer and includes a medication in the form of a solution or suspension. In this device, the drug layer is partially covered by the adhesive layer, which surrounds it.



Figure 5: Matrix type Patch

5. Vapour patches: The adhesive layer in this kind of patch system not only holds the various layers together but also lets out vapour. These brand-new patches are widely used to release essential oils for up to 6 hours. These patches are mostly utilised in cases of decongestion and release essential oils. There are many different kinds of vapour patches on the market that are used to increase sleep quality and decrease cigarette smoking.

ADVANTAGES OF TRANSDERMAL DRUG DELIVERY SYSTEM:

Transdermal drug delivery systems are safe, effective, and easy to remove as needed by the patient, which are all significant advantages over more conventional methods. Transdermal drug delivery (TDD) offers a number of important benefits, including the restriction of hepatic first pass metabolism, enhancement of therapeutic effectiveness, and preservation of a constant plasma level of the drug. The following are the main benefits of TDDS:

- The hepatic first pass metabolism is avoided.
- Sustained and controlled delivery over a prolonged period of time.
- Direct access to target or diseased site.
- Provide relatively steady and sustained drug concentration in plasma in contrast to conventional systems where peaks and troughs are a common feature.
- Provides utilization of drugs with short biological half-lives.
- Ease of dose termination in any adverse reactions either systemic or local.
- Inter and intra-patient variations.
- Termination of therapy is easy at any point of time.
- Variability due to factors such as pH, intestinal motility, food intake, etc., which make vast differences in the bioavailability of the drugs given through oral route, are not existent.
- Painless and suitable administration.
- Expected and unlimited duration of activity.
- A stable rate of absorption is possible in a huge variety of diverse patient population.
- Maintain plasma concentration of potent drugs.
- Greater patient compliance due to exclusion of multiple dosing profiles.
- Ability to deliver drug more selectively to a particular site.
- Provide appropriateness for self-administration.
- Avoidance of gastro-intestinal incompatibility.
- Ease of dose termination in any adverse reactions either systemic or local.
- Drugs that cause gastro-intestinal upset can be good candidates for transdermal delivery because this method avoids direct effects on stomach and intestine.
- Avoiding the fluctuation in drug levels.
- At some stage in application of gel amount and area of application are not specified but in patch both are specific.
- Condensed side effects and improved therapy due to preservation of plasma levels up to the end of the dosing interval.
- Ease of dose termination in any adverse reactions either systemic or local.
- Inter and intra-patient variations.
- Termination of therapy is easy at any point of time.
- Variability due to factors such as pH, intestinal motility, food intake, etc., which make vast differences in the bioavailability of the drugs given through oral route, are not existent.
- Painless and suitable administration.

- Expected and unlimited duration of activity.
- A stable rate of absorption is possible in a huge variety of adverse patient population. Improving physiological and pharmacological comeback.
- Maintain plasma concentration of potent drugs.
- Greater patient compliance due to exclusion of multiple dosing profiles.
- Ability to deliver drug more selectively to a particular site.
- Provide appropriateness for self administration.
- Avoidance of gastro intestinal incompatibility.
- Ease of dose termination in any adverse reactions either systemic or local.
- Drugs that cause gastro intestinal upset can be good candidates for Transdermal delivery because this method avoids direct effects on stomach and intestine.
- Avoiding the fluctuation in drug levels.
- At some stage in application of gel amount and area of application are not specific but in patch both are specific.
- Condensed side effects and improved therapy due to preservation of plasma levels up to the end of the dosing interval.

Elasticity of terminating the drug administration by simply removing the patch from the skin.

DISADVANTAGES OF TRANSDERMAL DRUG DELIVERY SYSTEM:

- Only relatively potent drugs are suitable candidates for TDDS.
- Local irritation and arrhythmia are possible. Enzymes in epidermis or derived from micro organisms present on the skin may denature the drugs.
- Can be used only for drugs, which require very small plasma concentrations for action.
- The drug must have some desirable physicochemical properties for penetration through stratum corneum.
- The barrier function of the skin changes from one site to another on the same person, from person to person and with age.
- Variation in absorption efficiency at different sites of skin.
- Difficulty of adhesion to certain skin types like excess oily skin.
- Difficulty in duration of time for which a patch can be left on any area due to permeability changes (usually not more than 7 to 10 days).
- The transdermal delivery will be very difficult, if the drug dose required is more than 10 mg/day for their therapeutic application.
- Another significant disadvantage of Transdermal drug delivery is that skin is less permeable because it serves as protective barrier for the entry of foreign particles.
- In order to maintain constant release states, transdermal patches must contain surplus of active drug

IDEAL PROPERTIES OF TRANSDERMAL DRUG DELIVERY SYSTEM:

- Ø Shelf life up to 2 years
- Ø Small size patch (i.e., less than 40 cm²)
- Ø Convenient dose frequency (i.e., once a day to once a week)
- Ø Cosmetically acceptable (i.e., clear, white colour)
- Ø Simple packaging (i.e., minimum number of pouches and steps required to apply the system)
- Ø Easy removal of the release liner (i.e., for children and elderly patients)
- Ø Adequate skin adhesion (i.e., no fall off during the dosing interval and easy removal without skin trauma)
- Ø No residue i.e., —cold flow (around the edge of the patch in storage or after application to skin or beneath the patch after removal)

MECHANISM OF ACTION OF TRANSDERMAL PATCHES:

Different processes are used for the transdermal patch's operation and the movement of the active medicinal ingredient from the patch via the skin to the circulatory system. A systemically active medicine must possess certain physicochemical characteristics that make it simple for the drug to enter the microcirculation and be absorbed via the skin.

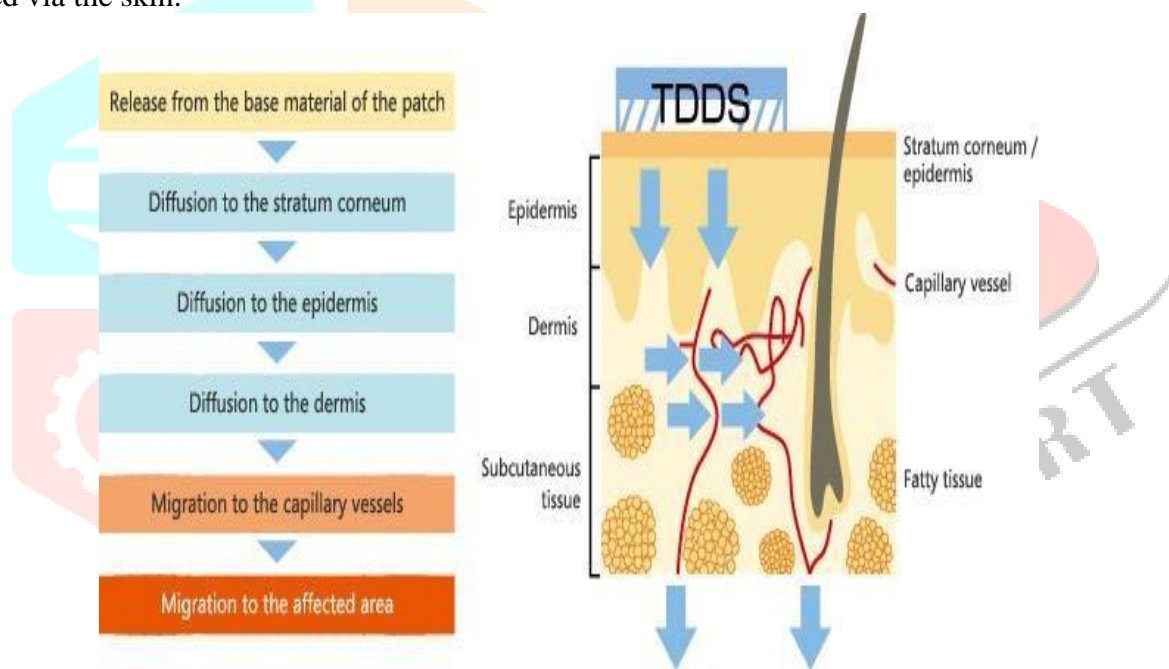


Figure 6: mechanism of action of transdermal patches

- When a TDDS is applied to the skin, medications enter the skin by various skin channels, including the appendageal route and the epidermal route, and then go to the systemic circulation. The appendageal route involves passing through sweat glands and hair follicles with the sebaceous glands that are connected to them. These paths are referred to as "shunt" routes because they avoid penetrating the stratum corneum. Due to its tiny size—roughly 0.1% of the overall skin area—this pathway is regarded as being of secondary relevance. In the epidermal route, the transcellular pathway and the intercellular pathway may be used to penetrate the medication into the stratum corneum. Transcellular route Drugs that penetrate the skin transcellularly pass via corneocytes on their way in. Highly hydrated keratin-containing corneocytes offer an aqueous environment through which hydrophilic medicines can pass. A drug's transcellular diffusion pathway involves a number of partitioning and diffusion phases.

- **Intercellular pathway:** The medication diffuses across the continuous lipid matrix in the intercellular route. This approach is a major challenge for two reasons:
- The interdigitating nature of the corneocytes produces a complex road for intercellular drug absorption, in contrast to the comparatively direct path of the transcellular route. This is reminiscent of the "bricks and mortar" model of the stratum corneum. In the intercellular domain, bilayers with different structures alternate.
- Therefore, a medication needs to repeatedly diffuse across and partition into aqueous and lipid domains. It is widely acknowledged that this pathway is the most typical way for tiny, uncharged compounds to penetrate skin.

REFERENCES

1. Boateng JS, Matthews KH, Stevens HN, Eccleston GM 2008.

Wound healing dressings

and drug delivery systems: a review. *J Pharm Sci* 97(8):2892-2923.

2. Enoch S, Leaper DJ 2008. Basic science of wound healing. *Surgery (Oxford)* 26(2):31-37.

3. Guo S, Di Pietro LA 2010. Factors Affecting Wound Healing. *J Dental Res* 89(3):219-229.

4. Gurtner GC, Callaghan MJ, Longaker MT 2007. Progress and potential for regenerative medicine. *Annu Rev Med* 58:299-312.

5. Gurtner GC, Werner S, Barrandon Y, Longaker MT 2008.

Wound repair and

regeneration. *Nature* 453(7193):314-321.

6. Martin P 1997. Wound healing - Aiming for perfect skin regeneration. *Sci* 276(5309):75-81.

7. Nawaz Z, Bentley G 2011. Surgical incisions and principles of wound healing. *Surg* 29(2):59-62.

8. Reinke JM, Sorg H 2012. Wound repair and regeneration. *Eur Surg Res* 49(1):35-43.

9. Velnar T, Bailey T, Smrkolj V 2009. The wound healing process: an overview of the cellular and molecular mechanisms. *J Int Med Res* 37(5):1528-1542.

Thu HE, Zulfakar MH, Ng SF 2012. Alginate based bilayer hydrocolloid films as potential slow-release modern wound dressing. *Int J Pharm* 434(1-2):375-383.

10. Percival J 2002. Classification of wounds and their management. *Surg* 20:114-117.

11. Moore K, McCallion R, Searle RJ, Stacey MC, Harding KG 2006. Prediction and monitoring the therapeutic response of chronic dermal wounds. *Int Wound J* 3(2):89-96.

12. Broderick N 2009. Understanding chronic wound healing. *The Nurse Practitioner* 34(10).

13. Trent JT, Kirsner RS 2003. Wounds and malignancy. *Adv Skin Wound Care* 16(1):31-34.

52

14. Cutting KF, White RJ 2002. Maceration of the skin and wound bed. 1: Its nature and causes. *J Wound Care* 11(7):275-278.

15. Krasner D, Kennedy KL, Rolstad BS, Roma AW 1993. The ABCs of wound care dressings. *Ostomy Wound Manage* 39(8):66, 68-69.

16. Ferreira MC, Tuma P, Jr., Carvalho VF, Kamamoto F 2006. Complex wounds. *Clinics (Sao Paulo)* 61(6):571-578.

17. Kirketerp-Møller K, Zulkowski K, James G. 2011. Chronic Wound Colonization, Infection, and Biofilms. *Biofilm Infections*, ed.: Springer New York. p 11-24.

18. Skorkowska-Telichowska K, Czemplik M, Kulma A, Szopa J 2013. The local treatment
19. potential slow-release modern wound dressing. *Int J Pharm* 434(1-2):375-383. Percival J 2002. Classification of wounds and their management. *Surg* 20:114-117.
20. Moore K, McCallion R, Searle RJ, Stacey MC, Harding KG 2006. Prediction and monitoring the therapeutic response of chronic dermal wounds. *Int Wound J* 3(2):89-96.
21. Broderick N 2009. Understanding chronic wound healing. *The Nurse Practitioner* 34(10).
22. Trent JT, Kirsner RS 2003. Wounds and malignancy. *Adv Skin Wound Care* 16(1):31-34.
23. Cutting KF, White RJ 2002. Maceration of the skin and wound bed. 1: Its nature and causes. *J Wound Care* 11(7):275-278.
24. Krasner D, Kennedy KL, Rolstad BS, Roma AW 1993. The ABCs of wound care dressings. *Ostomy Wound Manage* 39(8):66, 68-69.
25. Ferreira MC, Tuma P, Jr., Carvalho VF, Kamamoto F 2006. Complex wounds. *Clinics (Sao Paulo)* 61(6):571-578.
26. Kirketerp-Møller K, Zulkowski K, James G. 2011. Chronic Wound Colonization, Infection, and Biofilms. *Biofilm Infections*, ed.: Springer New York. p 11-24.
27. Skorkowska-Telichowska K, Czemplik M, Kulma A, Szopa J 2013. The local treatment and available dressings designed for chronic wounds. *J Am Acad Dermatol* 68(4):e117-126.
28. Peh K, Khan T, Ch'ng H 2000. Mechanical, bioadhesive strength and biological evaluations of chitosan films for wound dressing. *J Pharm Pharm Sci* 3(3):303-311 potential slow-release modern wound dressing. *Int J Pharm* 434(1-2):375-383.
29. Percival J 2002. Classification of wounds and their management. *Surg* 20:114-117.
30. Moore K, McCallion R, Searle RJ, Stacey MC, Harding KG 2006. Prediction and monitoring the therapeutic response of chronic dermal wounds. *Int Wound J* 3(2):89-96.
31. Broderick N 2009. Understanding chronic wound healing. *The Nurse Practitioner* 34(10).
32. Trent JT, Kirsner RS 2003. Wounds and malignancy. *Adv Skin Wound Care* 16(1):31-34.
33. Cutting KF, White RJ 2002. Maceration of the skin and wound bed. 1: Its nature and causes. *J Wound Care* 11(7):275-278.
34. Krasner D, Kennedy KL, Rolstad BS, Roma AW 1993. The ABCs of wound care dressings. *Ostomy Wound Manage* 39(8):66, 68-69.
35. Ferreira MC, Tuma P, Jr., Carvalho VF, Kamamoto F 2006. Complex wounds. *Clinics (Sao Paulo)* 61(6):571-578.
36. Kirketerp-Møller K, Zulkowski K, James G. 2011. Chronic Wound Colonization, Infection, and Biofilms. *Biofilm Infections*, ed.: Springer New York. p 11-24.
37. Skorkowska-Telichowska K, Czemplik M, Kulma A, Szopa J 2013. The local treatment and available dressings designed for chronic wounds. *J Am Acad Dermatol* 68(4):e117-126.
38. Peh K, Khan T, Ch'ng H 2000. Mechanical, bioadhesive strength and biological evaluations of chitosan films for wound dressing. *J Pharm Pharm Sci* 3(3):303-311 potential slow-release modern wound dressing. *Int J Pharm* 434(1-2):375-383.
39. Percival J 2002. Classification of wounds and their management. *Surg* 20:114-117.
40. Moore K, McCallion R, Searle RJ, Stacey MC, Harding KG 2006. Prediction and monitoring the therapeutic response of chronic dermal wounds. *Int Wound J* 3(2):89-

96.

41. Broderick N 2009. Understanding chronic wound healing. *The Nurse Practitioner*

34(10).

42. Trent JT, Kirsner RS 2003. Wounds and malignancy. *AdvSkin Wound Care* 16(1):31-34.

43. Cutting KF, White RJ 2002. Maceration of the skin and wound bed. 1: Its nature and causes. *J Wound Care* 11(7):275-278.

44. Krasner D, Kennedy KL, Rolstad BS, Roma AW 1993. The ABCs of wound care dressings. *Ostomy Wound Manage* 39(8):66, 68-69.

45. Ferreira MC, Tuma P, Jr., Carvalho VF, Kamamoto F 2006. Complex wounds. *Clinics (Sao Paulo)* 61(6):571-578.

