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### Maggot Therapy for Treating Diabetic Foot Ulcers

Amber Nikolaus

Otterbein University, [amber.nikolaus@otterbein.edu](mailto:amber.nikolaus@otterbein.edu)

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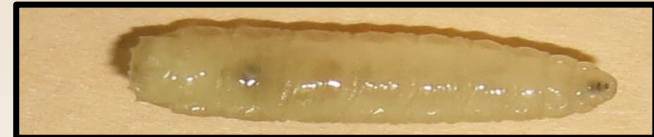
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# Maggot Therapy for Treating Diabetic Foot Ulcers

Amber Nikolaus, BSN, RN

Otterbein University, Westerville, Ohio



## Introduction

Diabetes mellitus, Type 1 and Type 2, are associated with numerous debilitating consequences. One of the most common and serious complication is diabetic foot ulcers (DFU) (Baltzis, Eleftheriadou, & Veves, 2014, p.817).

- DFUs are “caused by loss of glycemic control, peripheral neuropathy, peripheral vascular disease, and immunosuppression” and account for greater than 80,000 amputations per year in the United States (Aumiller & Dollahite, 2015, p.28).
- Approximately 15% of people with diabetes have diabetic foot ulcers (Aumiller & Dollahite, 2015, p.28) and studies demonstrate that 45% to 55% of patients presenting with neuropathic and ischemic diabetic foot ulcers will die within 5 years (Snyder, 2010, p.191).

To facilitate healing and establish a foundation for other treatments, these wounds must be debrided (Spear, 2010, p.56).

- Debridement** is the “removal of sloughy, necrotic, or damaged tissue from a wound until the surrounding healthy tissue is exposed...cornerstones of good wound practice and is vital for reducing the bacterial burden within the wound” (Mudge, Price, Neal, & Harding, 2014, p. 43).
- Wound debridement plays a crucial role in healing by:
  - facilitating the removal of damaged tissue and biofilms
  - restoring normal bacterial flora,
  - improving growth factors
  - repairing defective remodeling of extracellular matrix proteins (Opletalova et al., 2012, p. 432).

There are copious types of wound debridement such as: autolytic (moisture), surgical (surgery), biosurgical (larval therapy), and mechanical (wet-to-dry) (Brown, 2013, pp. 17-18). However, the focus of this poster will be diabetic foot ulcers and the use of maggot debridement therapy (MDT).

Sadly, by 2025 the number of people diagnosed with diabetes will have increased to at least 300 million (Snyder, 2010, p. 191); therefore, recognition and knowledge of treatments for DFUs will be life-saving skills that all health care providers must possess.

## Presentation of Process/Signs and Symptoms

An important factor to be aware of is that 60% of patients with diabetic foot ulcers present with diabetic neuropathy (Woo, Santos, & Gamba, 2013, p. 36). According to the American Podiatric Medical Association website (2015), secondary to the neuropathy, pain will not be a common symptom voiced by patients; discharge from the area will typically be the first aspect they will notice.

On inspection, there will be an open wound with:

- Redness
- Swelling
- Foul odor (if ulcer has progressed)

Due to the nature of these wounds the risk of infection is high, so understanding the signs and symptoms of an infected DFU is vital. The signs and symptoms of an infected diabetic foot ulcer are:

- abnormal discoloration of tissue
- increase in pain
- increased exudate
- increased wound size
- increased periwound temperature
- mild to moderate edema (Woo, Santos, & Gamba, 2013, p. 40)
- fever
- tachycardia
- tachypnea (Aumiller & Dollahite, 2015, p. 29).

## Underlying Pathophysiology

Diabetic foot ulcers form as a consequence to some of the negative mechanisms associated with diabetes: neuropathy, peripheral vascular disease, and reduced resistance to infection (Dinh et al., 2012, p.2937).

- Neuropathy**, a result of the oxidative stress on nerve cells secondary to hyperglycemia, has a key role in the development of diabetic ulcers.
  - Sensory nerve dysfunction leads to decrease ability to feel trauma or notice wounds on foot that may progress to an ulcer.
  - Motor nerve dysfunction to the muscle of the foot can lead to deformities, which may cause areas of increased pressure and ultimately ulcerations.
  - Autonomic nerve dysfunction impairs sweat glands, decreasing foot’s ability to moisturize, leading to cracks and skin breakdown (Aumiller & Dollahite, 2015, p. 29).
- Hyperglycemia also has a hand in vascular changes by provoking alterations of endothelial cells in the peripheral arteries:
  - Hyperglycemia
  - Reactive oxygen species(ROS)
  - Decreased nitric oxide(NO) bioavailability
  - Endothelial dysfunctions
- The endothelial dysfunctions include: impairment of barrier functions, disturbances in proliferative capacities, angiogenic properties, and attenuation of synthetic functions (Kolluru, G.K., Bir, S.C. & Kevil, C.G., 2012, pp. 1-2).
- Clinical factors regarding the delayed healing of diabetic foot ulcers were linked to microbiome colonizing.
  - Microbial burden or “bioburden” has three dimensions: microbial load (total quantity of microbes); microbial diversity (number of different bacterial populations present); and pathogenicity (Staphylococcus, Streptococcus, Proteobacteria, and anaerobic bacteria) (Gardner, Hillis, Heilmann, Segre, & Grice, 2013, p. 923).
  - A correlation was found between ulcer depth and duration and microbial diversity and pathogenicity, with deep ulcers with longer durations having more diverse microbiota and higher levels of anaerobes and Proteobacteria.
  - Individuals with poor glycemic control had a higher abundance of Staphylococcus and Streptococcus (Gardner, Hillis, Heilmann, Segre, & Grice, 2013, pp. 927-928)

## Maggot Debridement Therapy (MDT)

Maggot therapy is “an application of live fly larvae to wounds in order to aid in wound debridement (cleaning), disinfection, and/or healing (Sherman, 2014, p. 1). MDT is used to treat “severe, infected acute, and chronic wounds” (Cazander et al., 2012, p. 879), and is used as a combination with conventional treatments including systemic antibiotics and chemotherapy (Peck & Kirkup, 2012, p. 1137).

- MDT was approved by the US Food and Drug Administration in 2004 for physicians to prescribe for wound debridement (Opletalova et al., 2012, p. 432).
- Implemented for centuries to treat chronic wounds (Harris, Nigam, Sawyer, Mack, & Pritchard, 2013, p. 1393).
- Introduced into clinical practice in the 1930’s by Dr. William S. Baer. Baer, an orthopedic surgeon at John Hopkins Hospital, used MDT to heal osteomyelitis in children with great success (Cazander et al., 2012, p. 879).
- Research on maggot therapy has shown that there are numerous mechanisms involved beyond necrotic tissue debridement:
  - reduction in wound organisms (ingestion and antibacterial secretions)
  - wound healing (excretions or secretions promote fibroblast motility through remodeling and stimulating cellular responses)
  - preventing biofilm development (excretions and secretions) (Campbell, N. & Campbell, D., 2014, pp. 17-18).
- MDT has also been shown to increase the hepatocyte growth factor (HGF), which is an important factor involved in cutaneous wound healing (Honda, et al., 2011, c1423).
- The most common species used is the *Lucilia sericata* secondary to the fact that it only consumes necrotic tissue (Campbell, N., & Campbell, D., 2014, p.17).
- The maggots secrete proteolytic enzymes which transform necrotic tissue into a semi-liquid and debridement occurs when they consume the digestible liquid of organisms and wound debris (Campbell, N., & Campbell, D., 2014, p.17).
- The excretions and secretions of the maggots stimulate fibroblast motility through remodeling the extracellular matrix and stimulating cellular responses (Campbell, N., & Campbell, D., 2014, p.18).
- They promote wound healing by “mechanical stimulation of the wound surface, changes in wound surface pH, and contributions from larval metabolic byproduct (Campbell, N., & Campbell, D., 2014, p.18).

## Significance of Pathophysiology

- The pathophysiology of the development of diabetic foot ulcers provides insight and a direction for critical thinking and planning.
- Understanding the underlying pathophysiology will allow for more accurate screening and education for diabetic patients.
- Hyperglycemia, particularly chronic, uncontrolled glucose, has detrimental effects on numerous systems of the body. The significance of knowing what those effects are and how they originated gives the medical professional a knowledge base regarding the needs of the patient.
- Knowing what facilitated a particular ulcer may possibly warrant different types of treatment, education, prevention measures, and/or future considerations

## Implications for Nursing Care

Diabetic foot ulcers not only impede the patient’s quality of life, they can be an omen for death. Combating DFUs requires “multidisciplinary management, patient education, glucose control, debridement, offloading, infection control, and adequate perfusion (Braun, Fisk, Lev-Tov, Kirsner, & Isseroff, 2014, p. 267). Therefore, health care providers must be cognizant of the following:

- The importance of doing a thorough assessment on all patients, but particularly known diabetics and their feet.
- The likelihood of *being the one* to discover a diabetic foot ulcer, or the early signs of one, are very good.
- That neuropathy, vascular changes, and immunity impairment are only a few of the ramifications of hyperglycemia.
- The primary goal would be the prevention or reversal of diabetes.
- The patient, unable or unwilling to meet that goal, must be educated on the importance of glucose control and good foot care for the prevention of diabetic foot ulcers.

## Conclusions

- Diabetes is a devastating disease with numerous complications such as: high blood pressure, heart disease, blindness, and kidney disease (CDC, 2014).
- In the United States, diabetes is the 7<sup>th</sup> leading cause of death, although the Centers for Disease Control and Prevention documents that deaths related to diabetes may be underreported, and cost an estimated \$245 billion per year (CDC, 2014).
- The estimated cost per diabetic foot ulcer, secondary to how difficult they are to treat, is between \$7,000- \$10,000; amputation of a limb can cost up to \$65,000 (Marineau, Herrington, Swenor, & Eron, 2011, p. 121).

These statistics show how imperative, physically and financially, prevention and education are to patients. Diabetes can be not only prevented, but reversed with education, resources, and willpower. We may not be able to provide the willpower, but our job is to provide the education and resources.

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## Additional Resources

Before treatment: 34 year old with diabetic gangrene



During treatment: 34 year old with diabetic gangrene



After treatment: 34 year old with diabetic gangrene



<http://www.btmcl.com/eng/cases2.html>, 2012