PREVALENCE OF INTESTINAL PARASITES IN DIABETIC PATIENTS AT THE UNIVERSITY OF BENIN TEACHING HOSPITAL (UBTH), BENIN CITY, EDO STATE

BY

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BEING A PROJECT SUBMITTED TO THE DEPARTMENT OF MEDICAL LABORATORY SCIENCE IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE AWARD OF BACHELORS DEGREE IN MEDICAL LABORATORY SCIENCE (BMLS) UNIVERSITY OF BENIN, BENIN CITY, NIGERIA.

SUPERVISED BY
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SEPTEMBER, 2025.

CERTIFICATION

This is to certify that this research work reported in this project work was carried out by IGBINEDION, UWAGBAEMEN DAVID with Matriculation Number: BMS2005034 in the Department of Medical Laboratory science, School of Basic Medical Sciences, University of Benin in partial fulfilment of the requirement for the award of Bachelor of Medical Laboratory Science (BMLS) degree.

_____ _____

DR. (MRS). I.M. MOSES-OTUTU **DATE**
(Supervisor)

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DR. (MRS) Z. OMORUYI **DATE**
(Ag. Head of Department)

DEDICATION

I dedicate this research to God Almighty, whose divine wisdom, strength, and grace have guided me through this academic journey. His unwavering presence has been my source of inspiration and perseverance. I also dedicate this work to my beloved mother, Mrs. I. J. Igbinedion, whose unconditional love, prayers, and support have been my greatest motivation. Her sacrifices and encouragement have played a vital role in shaping my academic and personal growth. My heartfelt dedication also goes to my elder sisters Dr (Mrs) A. S. Ogbebor, Dr (Mrs) O. R. Ohiro, Miss O. A. Igbinedion, Miss O. A. Igbinedion, whose love, encouragement, and constant belief in me have been a source of strength and inspiration. I also dedicate this work to my elder brother, Engr. Mr. O. D. Igbinedion, for his invaluable guidance and unwavering support throughout this journey.

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I sincerely wish to express my profound gratitude to my elder brother, Engr. Mr. O. D. Igbinedion, for his constant support, encouragement, and invaluable guidance throughout this research.

Finally, I would like to thank my beloved mother, Mrs. I. J. Igbinedion, for her unwavering love, prayers, and support throughout this academic journey.

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ABSTRACT

Intestinal parasitic infections (IPIs) remain a major public health concern, particularly among immunocompromised populations such as individuals with diabetes mellitus. Diabetic patients are at increased risk of opportunistic infections due to impaired immune response, making parasitological investigations important in this group. This study aimed to determine the prevalence of intestinal parasites among diabetic patients attending the Endocrinology Unit of the University of Benin Teaching Hospital, Benin City, Nigeria. A cross-sectional study was conducted among 156 diabetic patients who consented to participate. Data on socio-demographic characteristics, clinical history, hygiene practices, and lifestyle habits were obtained through a structured questionnaire. Stool samples were collected and examined microscopically for intestinal parasites using standard parasitological techniques. Data were analyzed using SPSS version 26, and associations between variables were assessed using chi-square tests at a significance level of ($p < 0.05$). Of the 156 respondents, the majority were married (70.5%), within the age range of 60– 75 years, and more males were represented. Most respondents had primary or secondary education, resided in urban areas, and used water closet toilets. The prevalence of intestinal parasites was 54.5%. *Entamoeba histolytica*/*dispar* and *Entamoeba coli* were the most frequently detected parasites, while *Ascaris lumbricoides* and *Strongyloides stercoralis* were least observed. Significant associations were found between infection status and variables such as educational level and sanitation practices ($p < 0.05$). This research revealed that intestinal parasites are still prevalent among diabetic patients, particularly those with lower educational attainment and poor sanitation habits. Strengthening routine parasitological screening, health education, and improved hygiene practices is recommended to reduce the burden of intestinal parasites in this vulnerable group.

CHAPTER ONE

INTRODUCTION

1.1 Background of Study

Diabetes mellitus (DM) is a chronic metabolic disorder defined by persistent hyperglycemia due to defects in insulin secretion, insulin action, or both. It is increasingly common worldwide, particularly in low- and middle-income countries, driven by factors such as urbanization, sedentary lifestyles, and dietary shifts (WHO, 2024). Chronic hyperglycemia has been shown to impair various innate immune functions neutrophil chemotaxis, phagocytosis, superoxide production, and microbial killing which in turn increase susceptibility to infections (Geerlings, 1999; Scully *et al.,* 2017; Bartlett *et al.,* 2020).

Intestinal parasitic infections (IPIs), caused by protozoa and helminths inhabiting the gastrointestinal tract, remain a major public health challenge in developing regions. Poor sanitation, contaminated water, and inadequate hygiene contribute to their high prevalence. These infections are linked to malnutrition, anemia, cognitive impairment in children, and general weakening of immunity (Hotez *et al.,* 2008; WHO, 2020).

Patients with diabetes are particularly vulnerable to IPIs because of their immunocompromised state. Evidence has grown showing that diabetic patients have a significantly higher prevalence of IPIs than non-diabetic controls. For example, a systematic review and meta-analysis of studies across Africa found a pooled prevalence of approximately 31% (95% CI: 23–38%) for intestinal parasites among diabetic patients. In Nigeria specifically, the pooled prevalence was about 33% (95% CI: 18–49%) in diabetic populations. (Debash *et al.,* 2025)

Local Nigerian studies corroborate this elevated risk. In a study in southwestern Nigeria (Remo Health Zone), 19.46% of diabetic patients had intestinal parasitic infections; risk factors included use of pit latrines, lack of hand-washing, and age (particularly 51-60 years). (Omisa-kin *et al.,* 2024) Another study (Akinbo, 2013) found a prevalence of about 18.7% in diabetic patients in Owo, Ondo State, versus controls. (Akinbo, 2013)

Despite these data, there remains a gap in published information for many regions of Nigeria, including Benin City and University of Benin Teaching Hospital (UBTH). Local prevalence, risk factor variation, and the types of parasites involved may differ due to environmental, demographic, and health service factors. Understanding these local specifics is essential to guide screening policies, clinical management, and preventive interventions among diabetic patients in these understudied areas.

1.2 Statement of Problem

Diabetes mellitus is associated with impaired immune function, rendering patients more vulnerable to infections, including intestinal parasitic infections (Casqueiro *et al.,* 2012). Despite the high prevalence of both DM and IPIs in Nigeria, there is limited data focusing on their coexistence in tertiary healthcare facilities such as the University of Benin Teaching Hospital. The absence of such data hampers the development of targeted screening protocols, treatment strategies, and preventive measures. Unrecognized and untreated parasitic infections may exacerbate glycemic dysregulation, increase complications, and negatively impact the quality of life of diabetic patients (Tesfaye *et al.,* 2025). Therefore, there is an urgent need to determine the prevalence of intestinal parasites among diabetic patients in Benin City.

1.3 Justification

Both diabetes mellitus and intestinal parasitic infections are significant public health concerns in Nigeria. Investigating their co-prevalence among diabetic patients provides crucial epidemiological data that can improve patient management and inform health policies. Early diagnosis and treatment of intestinal parasitic infections can alleviate symptoms, prevent complications, and potentially improve glycemic control in diabetic patients (Banjo *et al.,* 2022). Furthermore, diabetic patients have altered immune responses, which may increase their vulnerability to parasitic infections (Kolaczkowska, 2013). There is currently a paucity of data on the prevalence of intestinal parasitic infection among diabetic patients attending the Endocrinology Unit of the University of Benin Teaching Hospital. This study will fill this knowledge gap, providing baseline data that can guide healthcare providers in implementing routine screening, targeted treatment, and preventive interventions. Additionally, the findings will assist healthcare planners and policymakers in resource allocation and may contribute to the global understanding of co-infections in immunocompromised populations (Tesfaye *et al.,* 2025).

1.4 Aim of Study

The aim of this study was to determine the prevalence of intestinal parasites in diabetic patients at the University of Benin Teaching Hospital (UBTH), Benin city, Edo state.

1.5 Specific Objectives

The specific objectives of this study were to;

1. determine the overall prevalence of intestinal parasites among diabetic patients attending the Endocrinology Unit in the University of Benin Teaching Hospital.

2. identify the different species of intestinal parasites present in the stool samples of diabetic patients.
3. determine the association between socio-demographic factors (age, gender, educational status, occupation, etc.) and the prevalence of intestinal parasites among diabetic patients.
4. investigate the relationship between duration of diabetes, type of diabetes, and glycemic control (HbA1c levels, where available) with the prevalence of intestinal parasitic infections.
5. assess the common clinical manifestations reported by diabetic patients with intestinal parasitic infections.

1.6 Research Questions

1. What is the overall prevalence of intestinal parasites among diabetic patients attending the Endocrinology Unit in the University of Benin Teaching Hospital?

2. Which species of intestinal parasites are present in the stool samples of diabetic patients?

3. Is there an association between socio-demographic factors (age, gender, educational status, occupation, etc.) and the prevalence of intestinal parasites among diabetic patients?

4. What is the relationship between duration of diabetes and type of diabetes with the prevalence of intestinal parasitic infections?

5. What are the common clinical manifestations reported by diabetic patients with intestinal parasitic infections?

1.7 Hypothesis

Null Hypothesis (H0): There is no statistically significant prevalence of intestinal parasites among diabetic patients attending the Endocrinology Unit in the University of Benin Teaching Hospital.

Alternative Hypothesis (Ha): There is a statistically significant prevalence of intestinal parasites among diabetic patients attending the Endocrinology Unit in the University of Benin Teaching Hospital.

Null Hypothesis (H0): There is no significant association between socio-demographic factors and the prevalence of intestinal parasites among diabetic patients.

Alternative Hypothesis (Ha): There is a significant association between socio-demographic factors and the prevalence of intestinal parasites among diabetic patients.

Null Hypothesis (H0): There is no significant relationship between duration of diabetes, type of diabetes, glycemic control, and the prevalence of intestinal parasitic infections.

Alternative Hypothesis (Ha): There is a significant relationship between duration of diabetes, type of diabetes, glycemic control, and the prevalence of intestinal parasitic infections.

CHAPTER TWO

LITERATURE REVIEW

2.1 Historical Review

The link between intestinal parasitic infections (IPIs) and immunocompromised states such as Diabetes Mellitus (DM) has gained increasing research attention over the past few decades. IPIs, caused by protozoa and helminths, are endemic in developing countries, particularly among populations with poor sanitation and compromised immunity. Historically, intestinal parasitic infections were studied predominantly in children and immunosuppressed individuals, such as HIV/AIDS patients. However, with the rising global burden of diabetes—especially in low- and middle-income countries—researchers began to investigate the susceptibility of diabetic patients to these infections (Saeed *et al.,* 2020; WHO, 2023). Early work highlighted that immune compromised individual including diabetics, exhibit reduced resistance to enteric pathogens, including parasites. This susceptibility is largely due to impaired immune responses and alterations in gut microbiota associated with hyperglycemia (Chaudhry *et al.,* 2017). A landmark study in Brazil by Lima *et al.* (2010) found a significantly higher prevalence of intestinal protozoa among diabetic patients compared with non-diabetic controls, suggesting that poor glycemic control predisposes individuals to such infections. Similarly, Díaz *et al.* (2003) reported higher incidences of *Giardia lamblia* and *Blastocystis hominis* in type 2 diabetic patients, highlighting the link between metabolic disorders and parasitic colonization.

In Nigeria, a comparative cross-sectional study conducted by Akinbo *et al.* (2013) in Benin City reported that diabetic patients were 2.5 times more likely to be infected with intestinal parasites than non-diabetic controls. This was attributed to impaired immunity, reduced gastric acidity,

and exposure to contaminated food or water. Helminthic infections such as *Ascaris lumbricoides*, Hookworm, and *Strongyloides stercoralis* were also prevalent. More recently, Ghenghesh *et al.* (2020) in Libya emphasized that protozoan infections were the most common, but helminthic infections were also observed, especially in diabetics with gastrointestinal complications. They also noted that asymptomatic diabetics may act as reservoirs for community transmission. Standard medical microbiology textbooks also support this connection. According to Brooks *et al.* (2022) in Jawetz, Melnick and Adelberg's Medical Microbiology, the immune dysfunction in diabetics increases vulnerability to opportunistic infections, including parasitic pathogens, particularly in the presence of poor glycemic control and comorbidities. The association between parasites and human diseases dates back centuries. Ancient civilizations documented symptoms consistent with parasitic infections, and early physicians such as Hippocrates described intestinal worms (Roberts and Janovy, 2018). The advent of microscopy in the 17th century revolutionized parasitology, with Leeuwenhoek's discovery of *Giardia lamblia* in his stool marking a milestone (Cox, 2012). The 19th and 20th centuries witnessed significant advancements in understanding parasite life cycles, transmission, and diagnostic methods (Peters and Gilles, 2007). Meanwhile, the recognition of diabetes as a distinct clinical condition also evolved, with its earliest mentions traced to ancient Egypt and India, but modern understanding was shaped by the discovery of insulin in the 20th century (Ritzmann, 2019). Overall, historical and contemporary evidence strongly suggests a higher prevalence of intestinal parasites in diabetic populations, particularly in endemic regions. This underscores the need for continued surveillance, routine stool analysis, hygiene promotion, and prompt antiparasitic interventions among diabetic patients (WHO, 2023).

2.2 Historical Background of Parasitology and Diabetes

Parasitology: The study of parasites dates back to ancient observations, where symptoms of intestinal worm infections were recorded in Egyptian papyri and Indian Ayurvedic texts (Roberts and Janovy, 2018). The development of microscopy by Robert Hooke in the 17th century and Antonie van Leeuwenhoek's discovery of protozoa, including *Giardia lamblia*, revolutionized parasitology (Cox, 2012). Major contributions such as Schaudinn's work on *Entamoeba histolytica* and Leuckart's elucidation of helminth life cycles further advanced the field (Peters, 2007). Over time, diagnostic methods evolved from direct stool microscopy to concentration techniques and molecular methods, improving accuracy in parasite detection (Garcia, 2019). Improved sanitation and hygiene measures in the 20th century significantly reduced the burden of parasitic diseases in developed countries, though they remain endemic in many low- and middle-income regions (WHO, 2023).

Diabetes: Historical descriptions of diabetes mellitus can be traced to ancient India and Egypt, where physicians described a condition characterized by "sweet urine" (Ritzmann, 2019). In the 17th century, Thomas Willis confirmed the sweetness of urine in diabetic patients through direct taste. The role of the pancreas in diabetes was clarified in the 19th century, with Minkowski and von Mering's experiments demonstrating that removal of the pancreas induced diabetes in dogs (Bliss, 1982). The landmark discovery of insulin by Banting and Best in 1921 transformed diabetes from a fatal disease into a manageable chronic condition (Bliss, 1982). Modern understanding now distinguishes between type 1 and type 2 diabetes, with ongoing research highlighting links between metabolic disorders, immunity, and increased susceptibility to infections, including parasitic diseases (Brooks *et al.,* 2022).

Intestinal parasites are a diverse group of organisms inhabiting the human gastrointestinal tract. They are broadly classified into protozoa and helminths (Garcia and Bruckner, 2019).

Protozoa: Single-celled eukaryotes such as *Entamoeba histolytica* (amoebiasis), *Giardia lamblia* (giardiasis), *Cryptosporidium parvum* (cryptosporidiosis), *Cyclospora cayetanensis* cyclosporiasis), and *Blastocystis hominis* (though its pathogenicity is debated) (Heyworth and Shrimpton, 2017).

Helminths: Multicellular organisms including nematodes (roundworms) such as *Ascaris lumbricoides, Trichuris trichiura, Ancylostoma duodenale, Necator americanus, Strongyloides stercoralis,* and *Enterobius vermicularis* (Ash and Orihel, 2015); Trematodes (flukes) such as *Fasciolopsis buski, Heterophyes heterophyes*, and *Metagonimus yokogawai* (Smyth, 1994); and cestodes (Tapeworms) including *Taenia saginata, Taenia solium*, and *Hymenolepis nana* (Pawlowski *et al.,* 1991).

2.3 Morphological Characteristics of Intestinal Parasites in Stool

1. Protozoan Parasites

Entamoeba histolytica:

Trophozoite: 10–60 µm, with central karyosome, fine peripheral chromatin, and cytoplasm that may contain ingested red blood cells.

Cyst: 10–20 µm, round, with 1–4 nuclei and cigar-shaped chromatoid bodies (Cheesbrough, 2009; Garcia, 2007).

Giardia lamblia:

Trophozoite: Pear-shaped, 10–20 µm, with two nuclei, four pairs of flagella, and a ventral sucking disc.

Cyst: Oval, 8–14 µm, with 4 nuclei and internal fibrils (Garcia, 2007; CDC, 2020).

2. Helminths (Worms)

Ascaris lumbricoides:

Ova: Fertilized eggs are oval, 45–75 µm, with thick mammillated shell; unfertilized eggs are longer and thinner (Cheesbrough, 2009; WHO, 2013).

Trichuris trichiura:

Ova: Barrel-shaped with bipolar plugs, measuring 50–54 µm (Garcia, 2007).

Hookworms (*Ancylostoma duodenale* and *Necator americanus*):

Ova: Oval, thin-shelled, 60–75 µm, with clear space between shell and embryo (Garcia, 2007; CDC, 2020).

Strongyloides stercoralis:

Larvae: Rhabditiform larvae in stool: 180–380 µm, short buccal cavity, prominent genital primordium (Cheesbrough, 2009).

Cestodes

Taenia spp.:

Ova: Round, 30–40 µm, thick striated shell containing an oncosphere with six hooklets (Garcia, 2007).

2.4 Taxonomy and Nomenclature

The classification (taxonomy) and naming (nomenclature) of intestinal parasites are based on morphological, developmental, and increasingly molecular characteristics. Parasites are broadly

divided into protozoa and helminths (nematodes, cestodes, and trematodes), and are named using a standardized binomial system (Roberts and Janovy, 2018; Garcia and Bruckner, 2019).

1. Protozoan Parasites

Entamoeba histolytica

Taxonomy:

Kingdom: Protista

Phylum: Amoebozoa

Class: Archamoebae

Order: Amoebida

Family: Entamoebidae

Genus: Entamoeba

Species: *E. histolytica*

Clinical significance: Causes amoebic dysentery and liver abscesses. The species name histolytica reflects its tissue-lysing ability (Clark and Diamond, 2002).

Giardia lamblia (also known as *G. intestinalis* or *G. duodenalis*)

Taxonomy:

Kingdom: Protista

Phylum: Metamonada

Class: Trepomonadea

Order: Diplomonadida

Family: Hexamitidae

Genus: Giardia

Species: *G. lamblia*

Clinical significance: A flagellated protozoan that causes giardiasis, particularly prevalent among children and immunocompromised individuals (Adam, 2001).

Cryptosporidium parvum

Taxonomy:

Kingdom: Protista

Phylum: Apicomplexa

Class: Conoidasida

Order: Eucoccidiorida

Family: Cryptosporidiidae

Genus: Cryptosporidium

Species: *C. parvum*

Clinical significance: Causes cryptosporidiosis; notable for resistance to chlorine in water supplies (Fayer, Morgan, and Upton, 2000).

2. Nematodes (Roundworms)

Ascaris lumbricoides

Taxonomy:

Kingdom: Animalia

Phylum: Nematoda

Class: Secernentea

Order: Ascaridida

Family: Ascarididae

Genus: Ascaris

Species: *A. lumbricoides*

Clinical significance: One of the most common soil-transmitted helminths worldwide (Bethony *et al.,* 2006).

Trichuris trichiura

Taxonomy:

Kingdom: Animalia

Phylum: Nematoda

Class: Enoplea

Order: Trichocephalida

Family: Trichuridae

Genus: Trichuris

Species: *T. trichiura*

Clinical significance: Known as whipworm; inhabits the large intestine and causes trichuriasis, often associated with anemia and growth retardation (Bundy and Cooper, 1989).

Ancylostoma duodenale and *Necator americanus* (Hookworms)

Taxonomy:

Kingdom: Animalia

Phylum: Nematoda

Class: Secernentea

Order: Strongylida

Family: Ancylostomatidae

Genus: Ancylostoma, Necator

Species: *A. duodenale, N. americanus*

Clinical significance: Major cause of iron-deficiency anemia through intestinal blood loss (Hotez *et al.,* 2004).

Strongyloides stercoralis

Taxonomy:

Kingdom: Animalia

Phylum: Nematoda

Class: Secernentea

Order: Rhabditida

Family: Strongyloididae

Genus: Strongyloides

Species: *S. stercoralis*

Clinical significance: Unique for autoinfection; can cause hyperinfection syndrome in immunocompromised individuals (Grove, 1996).

3. Cestodes (Tapeworms): *Taenia saginata* and *Taenia solium*

Taxonomy:

Kingdom: Animalia

Phylum: Platyhelminthes

Class: Cestoda

Order: Cyclophyllidea

Family: Taeniidae

Genus: Taenia

Species: *T. saginata, T. solium*

Clinical significance: *T. saginata* is beef tapeworm; *T. solium* is pork tapeworm and may cause neurocysticercosis (Garcia, 2003).

Hymenolepis nana

Taxonomy:

Kingdom: Animalia

Phylum: Platyhelminthes

Class: Cestoda

Order: Cyclophyllidea

Family: Hymenolepididae

Genus: Hymenolepis

Species: *H. nana*

Clinical significance: Unique for being directly infectious to humans without requiring an intermediate host (Roberts and Janovy, 2009).

4. Trematodes (Flukes) *Fasciolopsis buski*

Taxonomy:

Kingdom: Animalia

Phylum: Platyhelminthes

Class: Trematoda

Order: Echinostomida

Family: Fasciolopsidae

Genus: Fasciolopsis

Species: *F. buski*

Clinical significance: Acquired by eating contaminated aquatic plants; causes fasciolopsiasis (Toledo and Fried, 2005).

Schistosoma mansoni

Taxonomy:

Kingdom: Animalia

Phylum: Platyhelminthes

Class: Trematoda

Order: Digenea

Family: Schistosomatidae

Genus: Schistosoma

Species: *S. mansoni*

Clinical significance: Causes intestinal schistosomiasis; endemic in Africa and South America (Colley, 2014).

2.5 Mode of Transmission and Pathogenesis

1. Fecal-Oral Route: The most prevalent route is fecal-oral transmission, where infective stages such as cysts, eggs, or larvae are ingested via contaminated food, water, or unwashed hands. Protozoa such as *Giardia duodenalis* and *Entamoeba histolytica*, and helminths like *Ascaris lumbricoides*, follow this route (WHO, 2020; Kotloff *et al.,* 2019).

2. Skin Penetration: Helminths such as *Strongyloides stercoralis* and hookworms penetrate intact human skin, typically through bare feet exposed to contaminated soil. The infective larvae actively invade the skin and migrate through the bloodstream (Hotez *et al.,* 2004; CDC, 2022).

3. Autoinfection: Autoinfection occurs when parasites complete part of their lifecycle within the same host, leading to reinfection without external exposure. This is characteristic of *Enterobius vermicularis* and *Strongyloides stercoralis* (Viney and Lok, 2007; CDC, 2022).

4. Person-to-Person Transmission: Direct hand-to-mouth transfer of infective forms is common in crowded households, schools, and institutional settings. This is particularly observed with *Giardia* and *Enterobius* infections (Traub *et al.,* 2004; Fletcher *et al.,* 2012).

5. Vector and Fomite Transmission: Mechanical transmission by flies and cockroaches, as well as contamination of fomites (toys, utensils, surfaces), can facilitate the spread of infective eggs or cysts (Roberts and Janovy, 2013; WHO, 2020).

2.6 Pathogenesis

1. Mechanical Damage: *Entamoeba histolytica* causes direct tissue destruction by lysing colonic epithelial cells, producing flask-shaped ulcers, dysentery, and possible perforation (Stanley, 2003; Shirley *et al.,* 2018). Heavy *Ascaris lumbricoides* infections may cause intestinal obstruction (Jourdan *et al.,* 2018).

2. Nutrient Competition and Malabsorption: Hookworms (*Ancylostoma duodenale, Necator americanus*) consume host blood, leading to chronic blood loss and iron-deficiency anemia. *Giardia duodenalis* disrupts intestinal absorption of fats and carbohydrates, leading to malabsorption (Al-Mekhlafi *et al.,* 2005; WHO, 2020).

3. Toxin Production: Some protozoa produce cytotoxins and proteases. For example, *E. histolytica* secretes amoebapores and cysteine proteases that degrade host tissues and trigger inflammatory responses (Bansal *et al.,* 2005; Lejeune *et al.,* 2009).

4. Immune-Mediated Damage: Host immune responses may exacerbate tissue pathology through inflammation or hypersensitivity. Chronic helminth infections often induce granulomatous inflammation and fibrosis (Maizels *et al.,* 2018).

5. Tissue Migration: Some parasites migrate through host tissues before localizing in the intestines. *A. lumbricoides* larvae migrate via the lungs, producing eosinophilic pneumonitis (Löffler's syndrome) (Crompton and Nesheim, 2002; Jourdan *et al.,* 2018).

2.7 Lifecycle

Intestinal parasites, including protozoa and helminths, have diverse lifecycles, often involving infective and diagnostic stages. Understanding these cycles is critical for diagnosis, treatment, and control (Garcia, 2016; CDC, 2022).

Major Groups:

Protozoa (*Giardia duodenalis, Entamoeba histolytica, Cryptosporidium* spp.)

Nematodes (*Ascaris lumbricoides*, *Trichuris trichiura, Strongyloides stercoralis*)

Cestodes (*Taenia* spp., *Hymenolepis nana*)

Trematodes (rare intestinal species such as *Fasciolopsis buski*)

Examples:

1. *Giardia duodenalis*

Stages: Trophozoite (active) and cyst (infective). Transmission occurs through ingestion of cysts in contaminated food or water. In the small intestine, excystation releases trophozoites that attach

to the mucosa, multiply, and encyst before being shed in feces. The simple direct cycle enables rapid spread in poor sanitation settings (Adam, 2021; Lane and Lloyd, 2022).

2. *Entamoeba histolytica*

Stages: Trophozoite (invasive) and cyst (infective). Transmission via ingestion of mature cysts in contaminated food/water. Excystation occurs in the small intestine; trophozoites migrate to the colon, invade tissue, and may disseminate to the liver (Shirley *et al.,* 2018; CDC, 2022).

3. *Ascaris lumbricoides*: Eggs passed in stool become embryonated in soil. Ingestion leads to larval hatching in the intestine, followed by migration through the liver, lungs, and back to the intestine to mature into adults (Jourdan *et al.,* 2018; WHO, 2020).

4. Hookworms (*A. duodenale, N. americanus*): Infective filariform larvae penetrate the skin. They migrate via the bloodstream to the lungs, ascend the trachea, are swallowed, and mature in the intestine where they attach and feed on blood (Hotez *et al.,* 2004).

5. *Strongyloides stercoralis*

Unique for autoinfection: rhabditiform larvae in stool can develop into infective filariform larvae either in the environment or within the same host, causing hyperinfection in immunocompromised individuals (Viney and Lok, 2007; CDC, 2022).

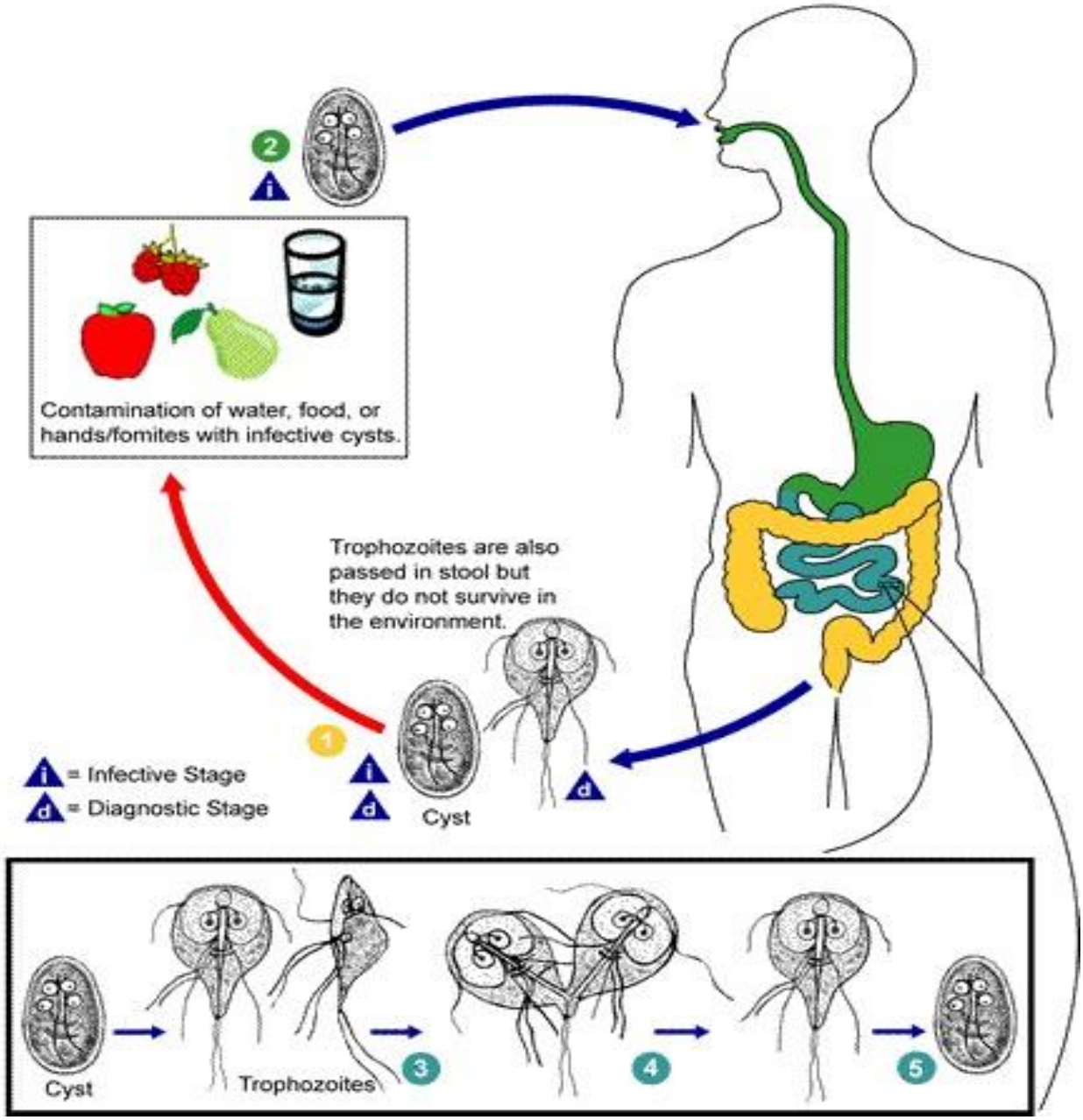

Figure 2.1: Lifecycle of *Giardia duodenalis* (Centre for Disease Control and Prevention, 2020).

b. *Entamoeba histolytica*: The infective stage is the cyst. After ingestion, cysts pass through the stomach and excyst in the small intestine, releasing trophozoites. These trophozoites colonize the large intestine, where they may remain non-invasive, invade the mucosa causing amoebic dysentery, or disseminate to extraintestinal sites such as the liver, leading to abscess formation. Encystation occurs in the colon, and mature cysts are excreted in feces, facilitating transmission (Shirley *et al.,* 2018; Watanabe and Petri, 2020).

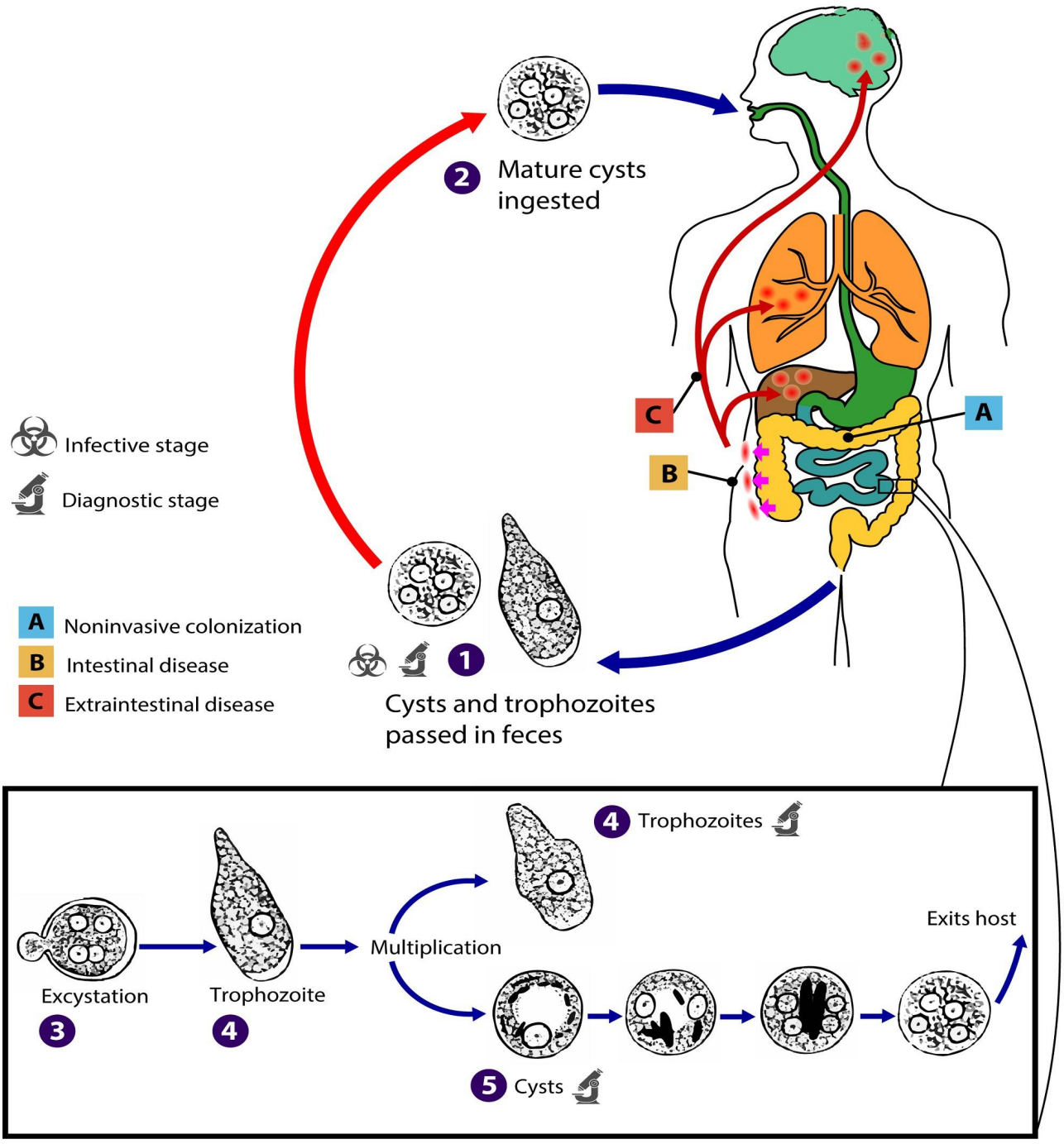

Figure 2.2: Lifecycle of *Entamoeba histolytica* (Centre for Disease Control and Prevention, 2020).

c. *Cryptosporidium* spp.: The infective and diagnostic stage is the oocyst. Following ingestion, excystation in the small intestine releases sporozoites that invade epithelial cells. Intracellular development produces type I and II meronts, gametes, and new oocysts. These oocysts are shed in feces and may cause new infections or autoinfection within the same host. Cryptosporidium is unique in completing its entire life cycle within a single host, enabling sustained transmission and persistence (Checkley *et al.,* 2015; Ryan *et al.,* 2021).

2. Helminths (Intestinal Worms)

Ascaris lumbricoides: The infective stage is the embryonated egg. After ingestion, eggs hatch in the small intestine, releasing larvae that penetrate the intestinal wall, migrate through the bloodstream to the liver and lungs, and then return to the intestine after being coughed up and swallowed. Adult worms develop in the small intestine, where females produce eggs excreted in feces. This hepatopulmonary migration contributes significantly to the pathogenesis of Ascaris infections (Jourdan *et al.,* 2018; Pullan *et al.,* 2019).

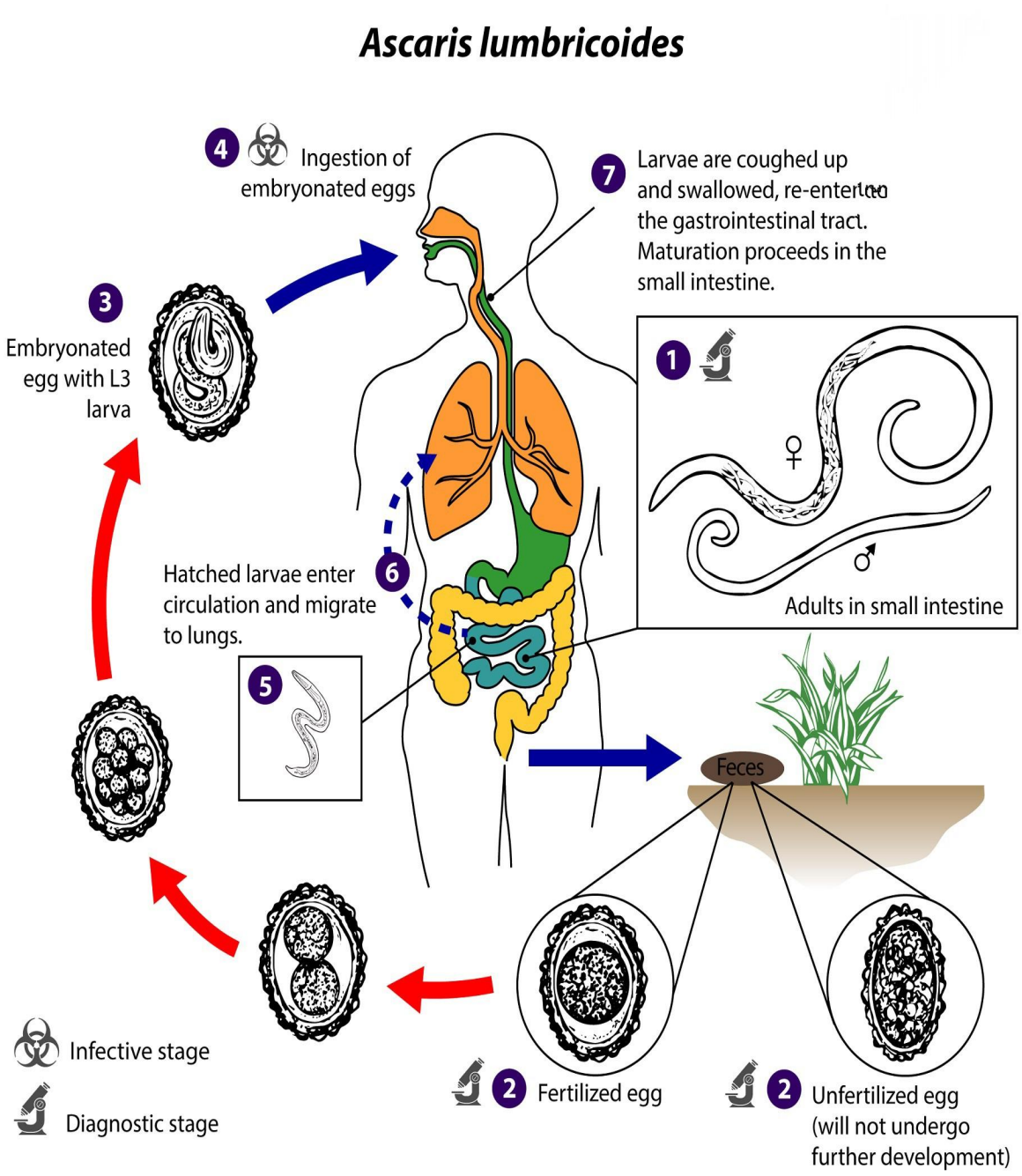

Figure 2.3: Lifecycle of *Ascaris lumbricoides* (Centre for Disease Control and Prevention, 2020)

b. *Trichuris trichiura* (Whipworm): The infective stage is the embryonated egg. After ingestion, eggs hatch in the small intestine, releasing larvae that migrate to the colon where they mature into adults. Female worms deposit unembryonated eggs that are excreted in feces and develop into infective embryonated eggs in soil. The cycle is sustained in areas with poor sanitation (Hotez *et al.,* 2014; Brooker *et al.,* 2015).

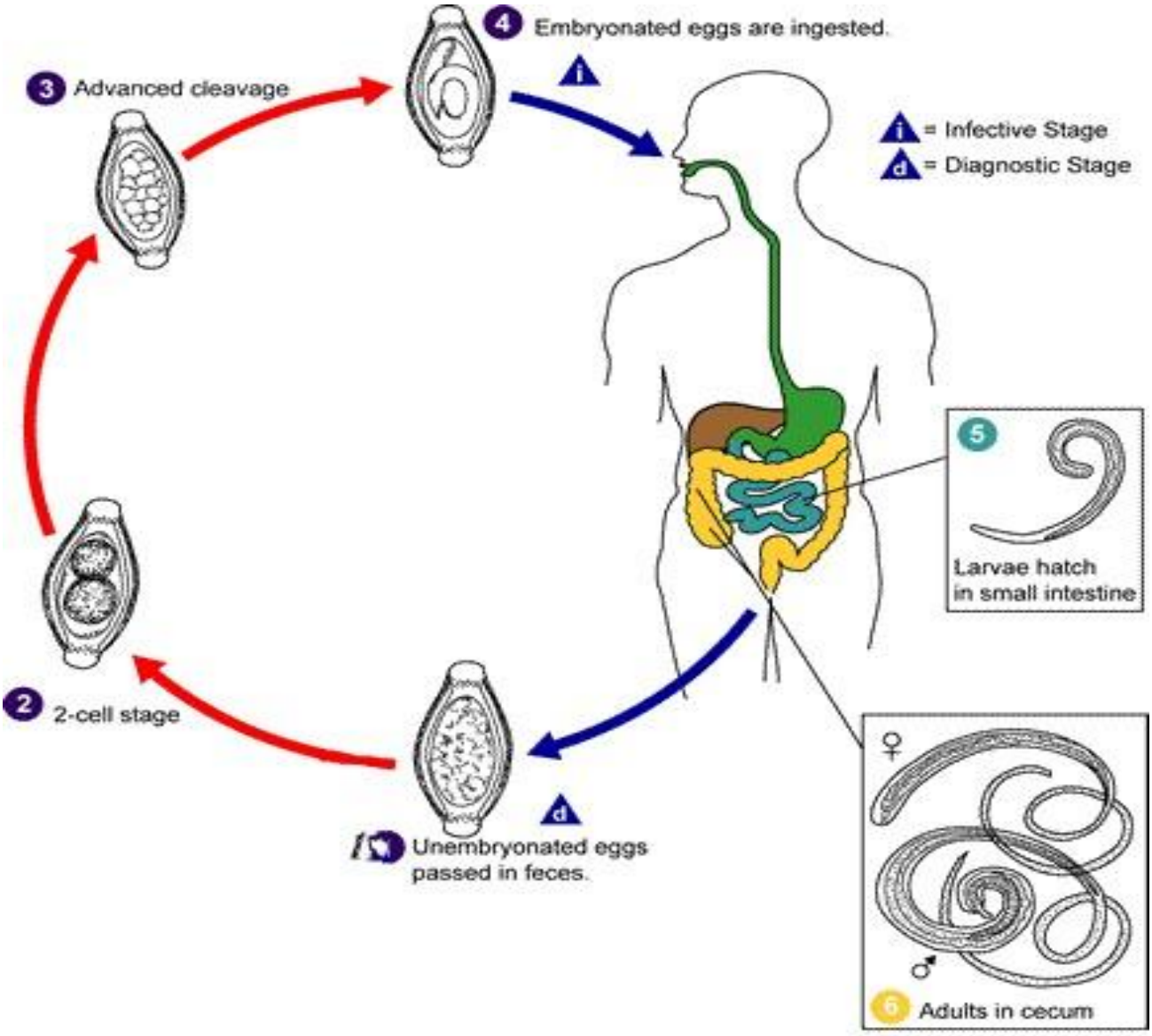

Figure 2.4: Lifecycle of *Trichuris trichiura* (Centre for Disease Control and Prevention, 2020).

c. *Strongyloides stercoralis:* The infective stage is the filariform larva. Once in contact with the skin (often bare feet), larvae penetrate, enter the bloodstream, migrate through the lungs and trachea, and are swallowed into the intestine. Parthenogenetic females reproduce in the intestinal mucosa, and rhabditiform larvae may either be excreted in stool or transform into filariform larvae within the host, causing autoinfection. This unique ability allows lifelong infection in untreated individuals (Page *et al.,* 2018; Buonfrate *et al.,* 2020).

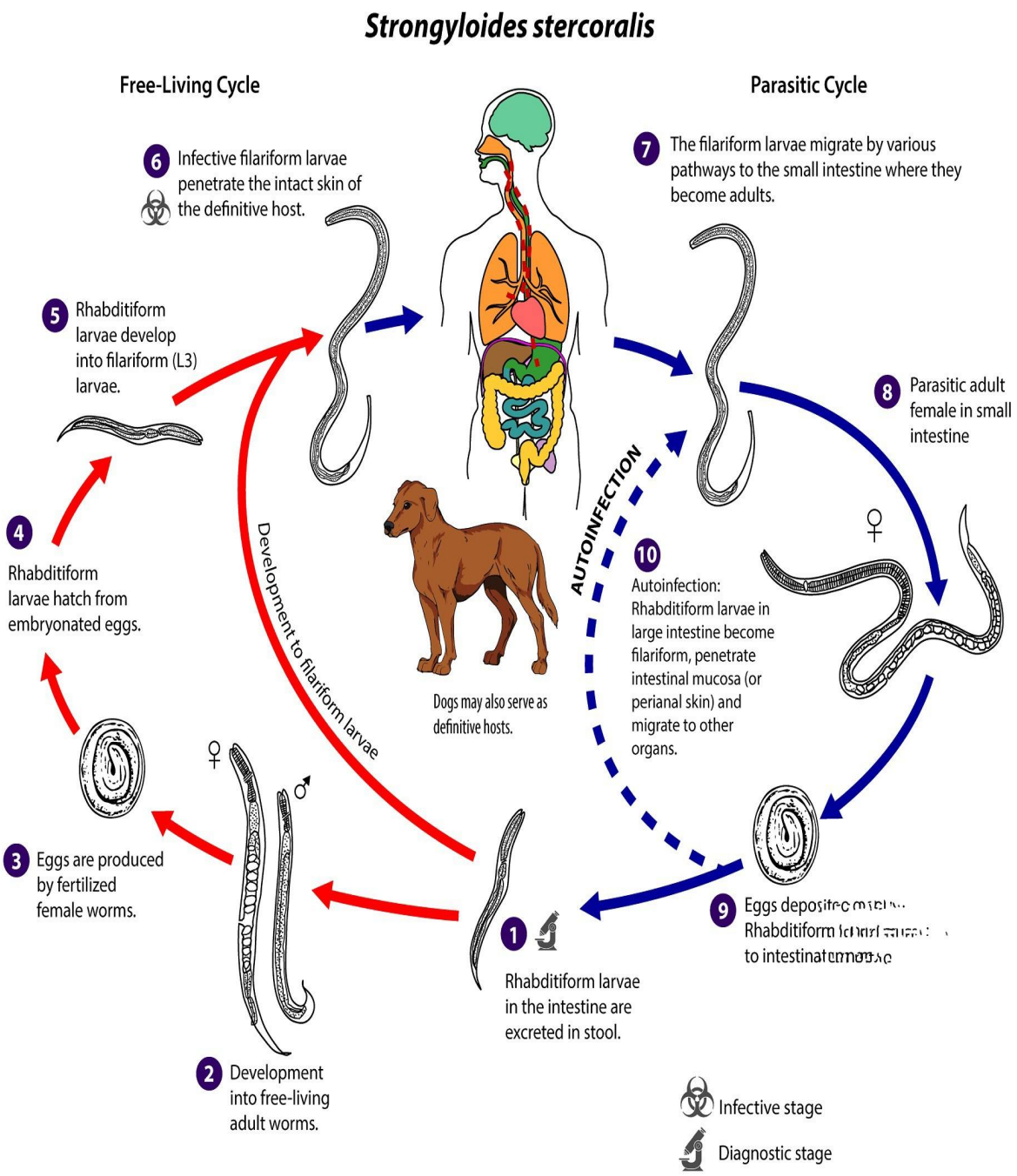

Figure 2.5: Lifecycle of *Strongyloides stercoralis* (Centre for Disease Control and Prevention, 2020).

3. Cestodes (Tapeworms)

Taenia saginata and *Taenia solium*: The infective stage is the cysticercus in undercooked beef (*T. saginata*) or pork (*T. solium*). After ingestion, cysticerci develop into adult tapeworms in the intestine, producing eggs and proglottids excreted in feces. In the case of *T. solium*, ingestion of eggs by humans can lead to cysticercosis, including neurocysticercosis, a major cause of epilepsy in endemic areas (White and Coyle 2018; Garcia *et al.,* 2020).

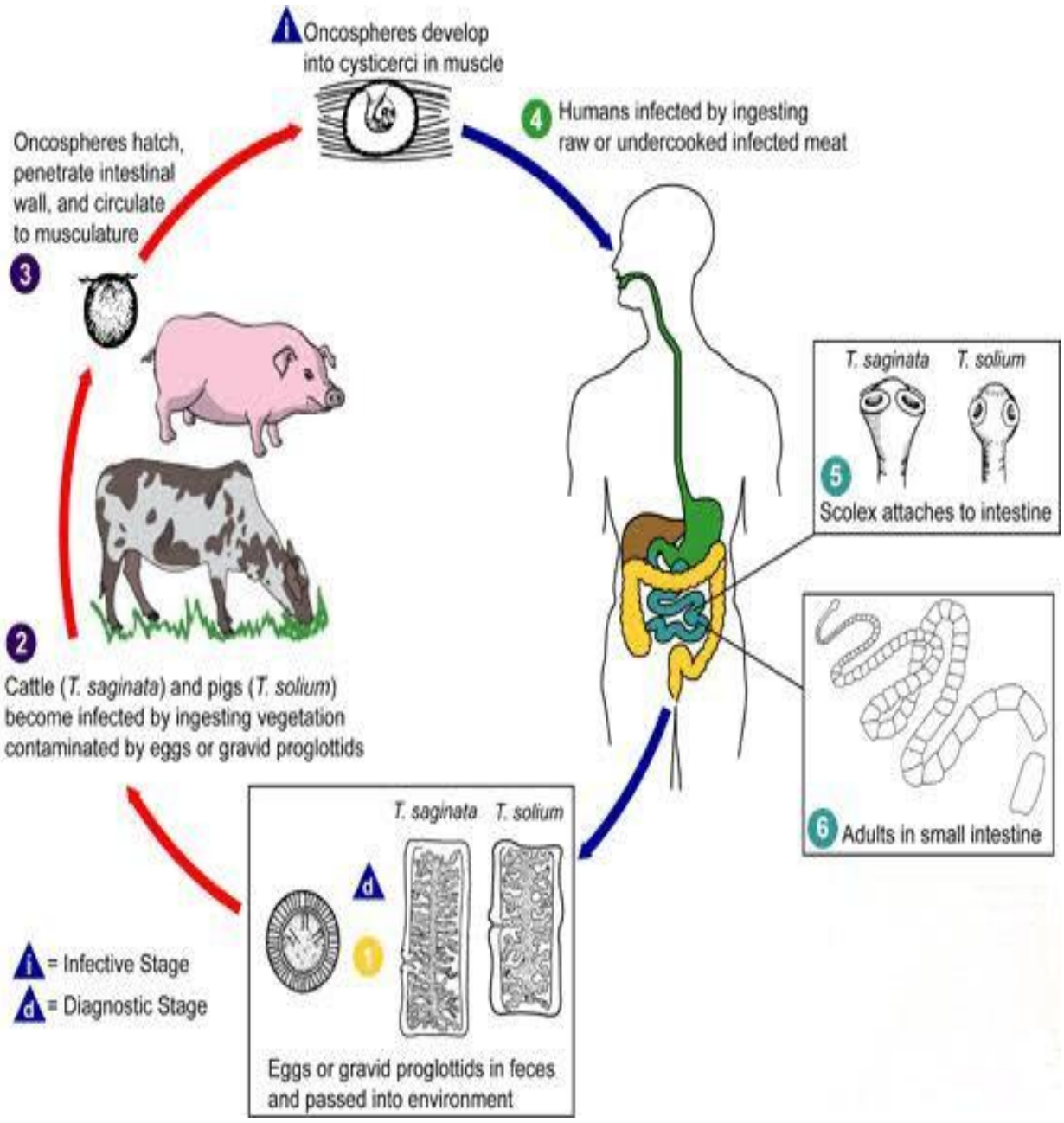

Figure 2.6: Lifecycle of *Taenia* species (Centre for Disease Control and Prevention, 2020).

b. *Hymenolepis nana:* Unique among cestodes, *H. nana* can complete its life cycle entirely within a single human host. The infective stage is the egg. After ingestion, eggs hatch in the intestine, and larvae penetrate villi, developing into cysticercoid larvae. These then re-enter the intestinal lumen as adult worms. Autoinfection is possible, contributing to chronic infections (Soriano-Arandes *et al.,* 2019; CDC, 2022).

2.8 Immune Response to Intestinal Parasites

The human immune system responds to intestinal parasites through a combination of innate and adaptive mechanisms. These responses vary depending on the type of parasite whether protozoa (e.g., *Giardia lamblia, Entamoeba histolytica*) or helminths (e.g., *Ascaris lumbricoides*, *Strongyloides stercoralis*). Intestinal parasites have evolved strategies to evade or modulate host immune responses, leading to chronic infections or asymptomatic carriage. The balance between host defense and parasite evasion determines the outcome of infection.

1. Innate Immune Response:

a. Physical and Chemical Barriers: Mucus and peristalsis help expel parasites, while gastric acid and digestive enzymes destroy ingested pathogens. Antimicrobial peptides such as defensins disrupt parasite membranes (Bevins and Salzman, 2011).

b. Cellular Response: Epithelial cells detect parasites through pattern recognition receptors (PRRs), including Toll-like receptors (TLRs), which trigger cytokine production (Kumar *et al.,* 2009). Macrophages, neutrophils, and dendritic cells engulf parasites and initiate inflammation (Allen and Maizels, 2011).

c. Eosinophils and Mast Cells: Helminth infections stimulate eosinophils to release cytotoxic granules that damage parasite cuticles, while mast cells increase intestinal permeability, promoting parasite expulsion (Anthony *et al.,* 2007).

2. Adaptive Immune Response:

a. Humoral Immunity: Secretory IgA neutralizes protozoa such as Giardia lamblia (Singer and Nash, 2000). Elevated IgE in helminth infections promotes mast cell degranulation and eosinophil recruitment (Maizels and Yazdanbakhsh, 2003).

b. Cell-Mediated Immunity: A Th1 response (IFN-γ, IL-12) is essential for intracellular parasites such as *Entamoeba histolytica* and *Cryptosporidium parvum* (Haque *et al*., 2007), while a Th2 response (IL-4, IL-5, IL-13) dominates in helminth infections, driving IgE production, eosinophilia, and mucus secretion (Allen and Maizels, 2011). Regulatory T cells (Tregs) limit tissue damage but may also facilitate chronic parasite survival (Belkaid and Rouse, 2005).

3. Modulation and Evasion by Parasites: Parasites evade immunity through antigenic variation (e.g., Giardia, Entamoeba) (Touz *et al.,* 2008), immunosuppression via Treg induction or cytokine mimicry (Maizels *et al.,* 2004), and shielding within host tissues (e.g., *Trichinella spiralis*) (Anthony *et al.,* 2007). Helminths often create tolerogenic environments that promote persistence while reducing autoimmune responses (Yazdanbakhsh *et al.,* 2002).

4. Memory and Long-Term Immunity: Immunity to protozoa such as Giardia and Cryptosporidium is often strain-specific and short-lived (Ehigiator *et al.,* 2007), whereas protective immunity to helminths is weak, requiring repeated exposures for partial resistance (Anthony *et al.,* 2007).

2.9 Epidemiology

1. Epidemiology of Intestinal Parasites

a. Global Burden: Intestinal parasites affect more than 1.5 billion people worldwide, especially in sub-Saharan Africa, South Asia, and Latin America (WHO, 2020). Risk factors include poor sanitation, unsafe water, malnutrition, and overcrowding (Jourdan *et al.,* 2018). A meta-analysis reported soil-transmitted helminths (STHs) alone account for ~450 million infections, mainly among children and immunocompromised individuals (Pullan *et al.,* 2014).

b. Nigeria and Sub-Saharan Africa: In Nigeria, intestinal parasitic infections (IPIs) remain endemic, with a pooled prevalence of 61.5% in rural settings (Ojurongbe *et al.,* 2021). Disparities exist between rural and urban areas due to unequal access to healthcare and safe water.

2. Epidemiology of Diabetes Mellitus

a. Global Trends: Diabetes mellitus affects over 537 million adults globally, with projections estimating 643 million by 2030 (IDF, 2021). Low- and middle-income countries (LMICs) account for 80% of cases due to urbanization, lifestyle changes, and limited healthcare access.

b. Nigeria and Africa: In Nigeria, DM prevalence among adults is ~4–5%, with higher rates in urban centers (Chinenye and Ogbera, 2018). Undiagnosed cases remain high due to poor screening coverage.

3. Intersection Between IPIs and Diabetes

a. Altered Immunity in Diabetes: Diabetes impairs both innate and adaptive immunity, making patients more susceptible to IPIs (Geerlings and Hoepelman, 2017). Hyperglycemia disrupts neutrophil activity and increases intestinal permeability (Liu *et al.,* 2022).

b. Higher Prevalence of IPIs Among Diabetic Patients: Studies report higher IPI prevalence among diabetics: 32.6% in Egypt (Mohamed *et al.,* 2021), significantly higher protozoa in Iran (Kiani *et al.,* 2016), and 28% in Sudan (Ibrahim *et al.,* 2018).

2.10 Risk Factors for Intestinal Parasites in Diabetic Patients

Risk factors include:

1. Immunosuppression: Hyperglycemia-induced immune dysfunction impairs parasite clearance (Geerlings and Hoepelman, 2017; Liu *et al.,* 2022).

2. Poor Sanitation: Unsafe water and contaminated food increase exposure to protozoa such as Giardia lamblia (Kiani *et al.,* 2016).

3. Low Health Literacy: Poor hygiene practices and lack of awareness increase transmission risk (Njinga *et al.,* 2022).

4. Rural Residence: Diabetics in rural areas face greater exposure to helminths via contaminated soil and water (Pullan *et al.,* 2014; Ibrahim *et al.,* 2018).

5. Frequent Antibiotic Use: Antibiotic-induced dysbiosis predisposes to IPIs (Tilg *et al.,* 2020).

6. Nutritional Deficiencies: Malnutrition worsens susceptibility to parasitic infections (Singh *et al.,* 2020; Ranjan *et al.,* 2021).

7. Advanced Age and Co-morbidities: Elderly diabetics are at higher risk (Chinenye, 2018; Njinga *et al.,* 2022).

2.11 Correlation between Intestinal Parasite and Diabetes

1. Immune Dysregulation: Diabetes-induced immune impairment increases susceptibility to IPIs (Geerlings and Hoepelman, 2017; Wang *et al.,* 2022).

2. Epidemiological Evidence: Higher IPI prevalence in diabetics has been reported in Egypt (Mohamed *et al.,* 2021), Iran (Kiani *et al.,* 2016), Sudan (Ibrahim *et al.,* 2018), and Ethiopia (Kure *et al.,* 2019).

3. Gut Microbiome Interactions: IPIs disrupt gut microbiota, influencing glucose metabolism. Helminths may paradoxically improve insulin sensitivity via immunomodulation (Tilg *et al.,* 2020; Maizels, 2021).

4. Nutritional and Clinical Implications: IPIs worsen malnutrition, impair glycemic control, and may mask diabetes complications (Ranjan *et al.,* 2021; Mohamed *et al.,* 2021).

5. Bidirectional Influence: Chronic parasitic infections may contribute to insulin resistance through inflammation (Chen *et al.,* 2016; Wang *et al.,* 2022).

6. Public Health Implications: Routine IPI screening in diabetics is recommended, especially in endemic areas (Njinga *et al.,* 2022).

2.12 Clinical Manifestation

Intestinal parasitic infections are among the most common globally, especially in low- and middle-income countries where poor sanitation, unsafe water, and inadequate healthcare access persist. Clinical manifestations vary depending on the parasite type (protozoa vs. helminths),

host immunity, parasite burden, infection site, and duration. Some individuals remain asymptomatic, while others experience acute or chronic disease, ranging from mild gastrointestinal upset to life-threatening complications (Jourdan *et al.,* 2018; Ojurongbe *et al.,* 2021).

1. Protozoan Infections

Giardia lamblia: Presents with watery, foul-smelling diarrhea, bloating, flatulence, abdominal cramps, weight loss, and malabsorption. Disease is more severe in children, malnourished individuals, and immunocompromised patients. Chronic giardiasis can impair child growth and development (Ajjampur and Kang, 2015; Lane and Lloyd, 2022).

Entamoeba histolytica: Causes amoebic dysentery, characterized by bloody/mucoid diarrhea, tenesmus, abdominal pain, and fever. Severe cases progress to amoebic liver abscess, with right upper quadrant pain, hepatomegaly, and fever (Haque *et al.,* 2020).

Cryptosporidium parvum: A major cause of persistent diarrhea in immunocompromised hosts (e.g., HIV/AIDS patients). Symptoms include profuse watery diarrhea lasting >14 days, nausea, vomiting, abdominal cramps, dehydration, and electrolyte imbalance (Fayer *et al.,* 2021).

2. Helminthic Infections

Ascaris lumbricoides: Often asymptomatic but may cause intestinal obstruction, abdominal pain, vomiting, and bloating. Pulmonary symptoms (cough, wheeze, eosinophilia) occur during larval migration (Löffler's syndrome) (Ngui *et al.,* 2015).

Trichuris trichiura: Mild cases are asymptomatic, while heavy infection may cause chronic diarrhea, rectal prolapse, iron-deficiency anemia, and impaired growth in children (Jourdan *et al.,* 2018).

Hookworms (*Ancylostoma duodenale, Necator americanus*): Feed on blood from intestinal mucosa, leading to iron-deficiency anemia, fatigue, pallor, and "ground itch" at penetration sites. Larval migration may cause cough and wheezing. Hookworm anemia is especially severe in schoolchildren and pregnant women (Brooker *et al.,* 2015; Keiser and Utzinger, 2019).

Strongyloides stercoralis: May remain asymptomatic or cause abdominal pain, diarrhea, and weight loss. In immunosuppressed patients, disseminated hyperinfection syndrome can occur, affecting lungs, brain, and liver (Moser *et al.,* 2021).

3. Non-Gastrointestinal Manifestations

Neurocysticercosis (*Taenia solium* eggs): Seizures, headaches, and neurological deficits (White, 2018).

Strongyloides: Migratory skin rashes and respiratory symptoms.

Ascaris migration: Transient eosinophilia and pneumonitis (Ngui *et al.,* 2015).

4. Special Populations

Children: Increased risk of growth retardation, anemia, and cognitive impairment (Ajjampur and Kang, 2015).

Immunocompromised: Severe and persistent infections with Cryptosporidium, Strongyloides, and Isospora, often life-threatening (Haque *et al.,* 2020).

2.13 Laboratory Diagnosis

Accurate laboratory diagnosis is essential for effective treatment, epidemiology, and disease control. Diagnostic methods depend on parasite type, patient presentation, and laboratory capacity. While stool microscopy remains the cornerstone, antigen-based and molecular tests provide greater sensitivity and specificity (Verweij and Stensvold, 2014; Garcia, 2016).

1. Direct Microscopy

Wet Mount (Saline and Iodine Preparation): Identifies motile protozoa (e.g., Giardia trophozoites) and cysts. Low cost and rapid but limited sensitivity in light infections (Garcia, 2016; WHO, 2019).

Concentration Techniques: Formol-ether sedimentation and zinc sulfate flotation improve parasite recovery, especially in light infections (Cheesbrough, 2015; Barda *et al.,* 2020).

2. Staining Techniques

Trichrome Stain: Differentiates pathogenic protozoa (*E. histolytica*) from nonpathogenic species (E. coli) (Garcia, 2016).

Modified Acid-Fast Stain: Detects coccidian parasites (*Cryptosporidium, Cyclospora, Isospora*). Ziehl–Neelsen variants are widely used in HIV settings (Chacín-Bonilla, 2018; Zheng *et al.,* 2022).

3. Antigen Detection: ELISA and Rapid Diagnostic Tests (RDTs) detect parasite antigens in stool or serum. High sensitivity and specificity for *Giardia, E. histolytica*, and *Cryptosporidium* (Verweij and Stensvold, 2014).

4. Molecular Techniques: PCR and real-time PCR detect parasite DNA/RNA with >95% sensitivity and specificity. Distinguishes morphologically similar species (e.g., *E. histolytica* vs. *E. dispar*). Valuable for mixed infections and epidemiological studies (Stark *et al.,* 2016; Wang *et al.,* 2022).

5. Culture Methods: Used mainly for research and drug testing in protozoa like *E. histolytica* and *Blastocystis hominis*. Rare in clinical practice (Ajjampur *et al.,* 2016; Tan, 2021).

6. Emerging Tools: Digital microscopy and AI-based recognition are being developed for rapid, remote diagnosis (Gómez *et al.,* 2023).

7. Sensitivity and Specificity of Common Diagnostic Methods

Method	Sensitivity (%)	Specificity (%)	Notes
Wet mount microscopy	40–60%	~80%	Rapid, low-cost, low sensitivity
Concentration methods	60–90%	~90%	Enhances detection in low parasite loads
Antigen detection (ELISA)	85–95%	>95%	Good for *Giardia* and *E. histolytica*
PCR	>95%	>98%	High precision; detects mixed infections
Acid-fast stain	70–85%	>95%	Used for coccidian parasites

A combination of diagnostic methods is often needed for accurate identification of intestinal parasites. While microscopy remains the cornerstone in low-resource settings, antigen-based and molecular methods offer higher accuracy and should be used when available, particularly in

vulnerable populations like immunocompromised or diabetic patients. The choice of test should depend on clinical suspicion, epidemiology, and laboratory capacity.

2.14 Prevention and Control

Strategies to reduce the burden of intestinal parasitic infections:

Improved Sanitation: Access to clean water, proper disposal of human faeces, latrine construction.

Hygiene Education: Handwashing with soap and water, especially before meals and after defecation.

Food Safety: Thorough cooking of meat, washing fruits and vegetables, avoiding raw or undercooked food.

Health Education: Awareness campaigns on transmission routes and preventive measures.

Mass Drug Administration (MDA): Deworming programs in endemic areas, especially for school-aged children (WHO recommendations).

Vector Control: Less relevant for direct intestinal parasites.

Specific measures for diabetic patients: Emphasis on personal hygiene due to increased susceptibility, prompt treatment of infections.

CHAPTER THREE

MATERIALS AND METHOD

3.1 Study Area

The research was carried out at the University of Benin Teaching Hospital (UBTH), located in Benin City, Edo State, Nigeria, between July and August 2025. Benin City is the capital of Edo State and lies within latitude 6.34°N and longitude 5.61°E (Akinbode, 2018). The city has an estimated population of over 1.2 million people, according to the National Population Commission (NPC) and United Nations estimates (NPC, 2006; United Nations, 2022). Benin City is situated in the South-South geopolitical zone of Nigeria. It is bounded to the north by Kogi State, to the west by Ondo State, and to the south by Delta State (Eboh and Oduaran, 2020).

The University of Benin Teaching Hospital (UBTH) is a premier and multi-specialty healthcare service provider in West Africa. It was officially commissioned on May 12, 1973, following the enactment of Edict No. 12 of the then Bendel State Government (UBTH, 2023). UBTH serves as a tertiary healthcare institution, catering to a large population with diverse health needs, including a significant number of diabetic patients attending its Endocrinology Unit. The hospital is located in Ugbowo, Benin City, Edo State, Nigeria.

3.2 Study Population

Diabetic patients attending the endocrinology clinic at UBTH during the study period.

3.3 Sampling Design

A convenience sampling technique was used to recruit participants who met the inclusion criteria and consented to participate. The convenience approach was selected because the study

population comprised clinic attendees during the study period. The limitation of this method (potential selection bias and reduced generalizability) is acknowledged and discussed in the study limitations.

3.4 Inclusion Criteria

Participants included in this research were:

known diabetic patients (Type 1 or Type 2)

diabetic patients who consented to participate in the study

ages 18 years and above

3.5 Exclusion Criteria

Excluded from this research were:

non-diabetic patients

diabetic patients who recently received antiparasitic treatment

known diabetic patients who decline consent to participate in this study

3.6 Sample Size Determination

The sample size was calculated using the formula for a single population proportion (Cochran, 1977; Charan and Biswas, 2013)

$$n = \frac{Z^2 p(1-p)}{d^2}$$

Where:

n = Required sample size

Z = Standard normal deviate for desired confidence level at 95% (standard value of 1.96)

p = Prevalence of IPIs among diabetic patients (Banjo *et al.,* 2022)

d = Margin of error at 5% (standard value = 0.05)

$$n = \frac{(1.96)^2 \times 0.219 \times (1-0.219)}{(0.05)^2}$$

$$n = \frac{3.842 \times 0.171}{0.0025}$$

$$n = \frac{0.657}{0.0025}$$

$$n = 262.4$$

Adding 10% to account for non-response:

$$n_{final} = 262.4 + 0.1 \times 262.4 = 262.4 + 262.4 = 288.64$$

Sample size = 289 diabetic patients

3.7 Ethical Approval

Ethical clearance was obtained from the Ethics and Research Committee of the University of Benin Teaching Hospital with Protocol number: **ADM/E 22/A/VOL. VII/1486549125534.** Participants were informed of the study objectives and written consents were obtained from each patient before sample collection. Study objectives were explained to prospective participants; written informed consent was obtained from each participant prior to enrolment. Confidentiality

was maintained by assigning unique identification numbers to each participant and storing data in password-protected files. Participants who tested positive for medically treatable intestinal parasites were referred to the attending clinician and treated according to hospital guidelines.

3.8 Specimen Collection and Transport

Container: Stool samples were collected in clean, dry, wide-mouthed, screw-capped containers labeled with the participant unique ID, date and time of collection, age and sex.

Quantity: Participants were instructed to provide approximately 5–10g of fresh stool (about a thumb-size portion) avoiding contamination with urine or water.

Instructions: Participants were given verbal and written instructions for collection and were asked to return samples as soon as possible.

Preservation: For immediate examination, specimen was transported to the laboratory within 2 hours and kept at room temperature. If delay was expected (>2 hours) or for concentration procedures, an aliquot was preserved in 10% formalin (or SAF) at a ratio of approximately 1 part stool to 3–10 parts preservative depending on the protocol used.

Biosafety: Laboratory personnel used appropriate personal protective equipment (gloves, lab coat, face shield as required). All stools and consumables were handled as potentially infectious and disposed of by autoclaving and incineration according to hospital biomedical waste policy.

3.9 Reagents and Equipment

Reagents

Normal saline (0.85% NaCl)

Lugol’s iodine (iodine solution)

10% formalin (formaldehyde 10% v/v)

Ethyl acetate (preferred defatting agent) or diethyl ether (if ethyl acetate unavailable) for the sedimentation step

Distilled water

Equipment

Microscope (light microscope with 10× and 40× objectives)

Centrifuge capable of achieving 500 × g

Gauze or disposable filter funnels

15 mL conical centrifuge tubes

Wooden applicator sticks, pasteur pipettes, cotton tipped applicators

3.10 Specimen Processing / Sample Analysis

The stool examination consisted of a sequence of macroscopic and microscopic tests.

A. Macroscopic Examination

Each stool sample was examined visually for:

Colour: brown (normal), pale (steatorrhea), black/tarry (upper GI bleeding), red (lower GI bleeding)

Consistency: formed, soft, loose or watery (diarrhoea)

Presence of visible blood, mucus, adult worms or proglottids

Presence of gross abnormal material

Macroscopic findings were recorded on the laboratory form and used to guide immediate microscopic processing (for example, diarrhoeal specimens prioritized for wet mount to observe trophozoites).

B. Microscopic Examination

1. Direct wet mount (saline and iodine)

A small portion of stool (pea-sized) was emulsified in a drop of normal saline on a glass slide, covered with a cover slip and examined immediately under 10× (to locate items) and 40× (to identify structures) objectives to look for motile trophozoites, cysts, ova and larvae.

A parallel mount using Lugol's iodine was prepared to demonstrate cyst morphology and internal nuclei/chromatoid bodies.

Interpretation: Motile trophozoites seen on a fresh saline wet mount indicate active infection and viable organisms. Iodine helps visualize cyst wall and nuclei but kills trophozoites.

2. Concentration — Sedimentation (Formalin-Ethyl Acetate) Procedure

A sedimentation concentration step was included for each specimen to increase detection sensitivity for ova, cysts and larvae. The formalin-ethyl acetate sedimentation technique (a diphasic sedimentation) was used as the main concentration method. The procedure below follows standard laboratory practice for formalin-ethyl acetate concentration. (Volumes and centrifugation parameters are consistent with standard protocols.)

Formalin-Ethyl Acetate Sedimentation Concentration (step-wise)

1. Mix the stool specimen thoroughly. Prepare a fecal suspension by emulsifying a representative portion of stool in 10% formalin to make about 15 mL of suspension.

2. Strain approximately 5 mL of the fecal suspension through wetted gauze placed over a disposable funnel into a 15 mL conical centrifuge tube. Adjust the volume to 15 mL with 0.85% saline or 10% formalin.

3. Centrifuge the tube at 500 × g for 10 minutes and decant the supernatant.

4. Add about 10 mL of 10% formalin to the sediment and mix thoroughly with an applicator stick to resuspend.

5. Add 4 mL of ethyl acetate to the tube, cap it and shake vigorously in an inverted position for about 30 seconds. Remove the cap carefully.

6. Centrifuge again at 500 × g for 10 minutes. Four layers will form: a top layer of ethyl acetate, a plug of debris, the formalin layer, and a sediment at the bottom that contains concentrated parasite stages.

7. Loosen the debris plug from the tube walls with an applicator stick and decant the top layers carefully into a sink or appropriate waste container (handling flammable solvents carefully according to safety policy).

8. Use a cotton-tipped applicator to remove residual debris from the tube walls, then resuspend the sediment in several drops of 10% formalin.

9. Place a drop of the sediment on a clean slide, cover with a coverslip and examine under 10× and 40× objectives for ova, cysts, larvae and trophozoites.

Ethyl acetate is commonly used as a safer substitute for diethyl ether in the Ritchie (formalin-ether) method and gives comparable or improved recovery of some organisms. If diethyl ether is used, ensure appropriate fume extraction and fire-safety measures. Commercial

fecal concentrators and prefilled tubes are alternative options to streamline the procedure and reduce handling of solvents.

3. Staining and Permanent Mounts (if required)

Selected positive and uncertain specimens were made into permanent smears (e.g., trichrome stain) for clearer morphological evaluation and archival records. Special stains or immunoassays (antigen detection) or molecular methods (PCR) were considered in cases where species-level identification was required to distinguish Entamoeba histolytica from morphologically identical non-pathogenic species.

3.11 Quality Control and Validation

Each slide was examined independently by two trained laboratory scientists; discordant results were resolved by a senior parasitologist.

Positive control slides (known parasite slides) and negative controls were read regularly to ensure staining and microscopy quality.

Equipment calibration (microscope optics, centrifuge rotor balance and speed) and reagent expiry checks were performed routinely.

3.12 Result Interpretation

Interpretation of stool microscopy results was performed using morphology and the context of clinical and macroscopic findings.

3.13 General Approach

A sample was reported as positive when ova, cysts, trophozoites or larvae with clear morphologic features of intestinal parasites were identified.

A sample was reported as negative if repeated microscopy (direct mount and concentrated sediment) failed to reveal parasite stages.

3.14 Specific Considerations and Diagnostic Implications

Protozoa (e.g., *Giardia, Entamoeba* spp.): Cysts and trophozoites of protozoa are identified by size, shape and internal features. The presence of motile trophozoites (on fresh wet mounts) often indicates active infection. Some protozoa (notably *Entamoeba histolytica, E. dispar* and *E. moshkovskii*) are morphologically indistinguishable on light microscopy; only trophozoites that contain ingested erythrocytes can be taken as suggestive of invasive *E. histolytica*. Species-level differentiation typically requires antigen detection or molecular methods. Repeated sampling or concentration methods increase detection sensitivity for protozoa.

Helminths (eggs and larvae): Eggs of soil-transmitted helminths (*Ascaris lumbricoides*, *Trichuris trichiura*, hookworms) have characteristic sizes and morphologies which allow identification to genus/species in many cases (e.g., barrel shape with polar plugs for *Trichuris*, thick mammillated shell for *Ascaris*). Some eggs (e.g., hookworm eggs) cannot be reliably differentiated to species by light microscopy and are reported generically (e.g., "hookworm eggs") unless further testing/culture is performed.

Intensity/semi-quantitative grading: The study used a qualitative (presence/absence) approach as the main outcome. Where parasite loads were clearly abundant (many eggs or cysts visible in numerous fields), these samples were noted as heavy infections in the data collection form. For accurate quantitative intensity (eggs per gram), methods such as Kato-Katz or McMaster should be used; these were not implemented in this research.

Clinical correlation: Detection of parasites should be interpreted in the clinical context: not all intestinal protozoa are pathogenic (some are commensals) and detection of cysts alone does not always imply symptomatic disease. Patients with diarrhoea and parasite stages consistent with pathogenic organisms (and/or trophozoites showing invasive features) were flagged for clinical follow up.

3.15 Limitations of Interpretation

A single stool specimen has limited sensitivity for intermittent shedding parasites; multiple samples on consecutive days increase detection rates but were not obtained due to logistic constraints. Direct microscopy has lower sensitivity for low-intensity infections; concentration methods improve sensitivity but do not replace antigen or molecular assays where higher sensitivity or species differentiation is required.

3.16 Data Management and Statistical Analysis

Data were entered into Microsoft Excel and analysed using Statistical Package for Social Sciences (SPSS) version 26.

Descriptive statistics: prevalence (%) of intestinal parasites, mean and standard deviation for continuous variables.

Inferential statistics: Chi-square ($\chi 2$) test was used to assess associations between categorical variables (e.g., parasitic status and demographic/clinical variables). Where appropriate, logistic regression analysis (binary logistic regression) was performed to identify independent predictors of infection, adjusting for potential confounders. Odds ratios (OR) and 95% confidence intervals (CI) were reported; statistical significance was set at $p < 0.05$.

CHAPTER FOUR

RESULTS

Table 4.1: Socio-Demographic Characteristics of Respondents with Diabetes Mellitus

Table 4.1 shows the socio-demographic characteristics of respondents with diabetes mellitus. The study recorded a high response rate of 95.5%, reflecting reliable participation. More males (57.1%) than females (42.9%) took part, and the majority were elderly (56.4% aged 73–78 years). Most respondents were married (70.5%) and had low to moderate educational attainment, with 55.8% having primary or secondary education and 6.4% having no formal education.

In terms of occupation, retirees made up the largest group (32.1%), followed by civil servants (28.2%) and the self-employed (25.6%). Most participants lived in urban areas (51.3%), while a smaller proportion (12.8%) resided in rural settings. Regarding sanitation, 70.5% used water closets, 28.8% used pit latrines, and 0.6% practiced open defecation.

Overall, the table highlights an elderly, urban-based population with moderate education and generally good sanitation practices, though the continued use of pit latrines and open defecation points to potential risks for intestinal parasite transmission.

Table 4.1: Socio-Demographic Characteristics of Respondents with Diabetes Mellitus

Variable	Frequency(n=156)	Percentage
Respondents		
Completed	149	95.5
Not completed	7	4.5
Gender distribution		
Male	89	57.1
Female	67	42.9
Age distribution		
60–66	20	12.8
67-72	48	30.8
73-78	88	56.4
Marital status		
Single	40	25.6
Married	110	70.5
Divorced	6	3.8
Educational status		
Primary	10	6.4
Secondary	87	55.8
Tertiary	59	37.8
Occupation		
Trader	22	14.1
Self employed	40	25.6
Civil servant	44	28.2
Retired	50	32.1
Residence		
Urban	80	51.3
Semi-urban	56	35.9
Rural	20	12.8
Hygiene and sanitation		
Water closet	110	70.5
Pit latrine	45	28.8
Open defecation	1	0.6

Table 4.2: Medical and Lifestyle History Among Respondents with Diabetes Mellitus

Table 4.2 illustrates the medical and lifestyle history of respondents with diabetes mellitus. A striking finding is that 58.3% of respondents were unsure of their type of diabetes, indicating possible gaps in patient education and record-keeping. Among those who were certain, 28.8% reported Type 2 diabetes—the more common form—while 12.8% reported Type 1. This suggests that although Type 2 diabetes predominates, awareness of disease classification among patients remains poor.

With respect to treatment, the majority of respondents (76.9%) reported being on medication or insulin therapy, demonstrating some level of adherence to diabetes management. However, 23.1% were not on any form of treatment, a concern since untreated diabetes may compromise immunity and increase susceptibility to infections, including intestinal parasites.

Overall, the findings reveal limited knowledge of diabetes type among respondents, which may reflect deficiencies in healthcare counseling and patient follow-up. Such knowledge gaps could affect broader health awareness and hinder the adoption of preventive practices essential for reducing infection risks.

Table 4.2: Medical and Lifestyle History Among Respondents with Diabetes Mellitus

Variable	Frequency (n=156)	Percentage
Type of diabetes		
Type 1	20	12.8
Type 2	45	28.8
Not sure	91	58.3
Medication/insulin use		
Yes	120	76.9
No	36	23.1

Table 4.3: Environmental and Hygiene Practices Among Respondents with Diabetes Mellitus

Table 4.3 highlights the environmental and hygiene practices of respondents with diabetes mellitus. A majority of the participants (83.3%) reported always practicing handwashing, indicating generally good personal hygiene within the study population. However, 12.8% admitted to washing hands only sometimes, while 3.8% rarely did so.

Although the proportion with poor hand hygiene is relatively small (16.6%), such lapses remain significant, as even a minority with inadequate hygiene can contribute to the persistence and transmission of intestinal parasites. This finding underscores the importance of consistent hand hygiene practices, particularly among vulnerable groups such as individuals with diabetes, whose immune systems may already be compromised.

Table 4.3: Environmental and Hygiene Practices Among Respondents with Diabetes Mellitus

Variable	Frequency(n=156)	Percentage
Handwashing habit		
Always	130	83.3
Sometimes	20	12.8
Rarely	6	3.8

Table 4.4: Clinical and Gastrointestinal Symptoms Among Respondents with Diabetes Mellitus

Table 4.4 summarizes the clinical and gastrointestinal symptoms reported by respondents with diabetes mellitus. The most common complaints were abdominal pain (38.5%), weight loss (35.3%), and fatigue (32.1%). These symptoms, though nonspecific, may reflect both diabetes-related complications and possible underlying parasitic infections.

Gastrointestinal manifestations were also noted, with 12.8% of respondents experiencing diarrhea and 9.6% reporting blood or mucus in stool. These symptoms are more directly suggestive of gastrointestinal infections, including intestinal parasitosis.

The overlap between typical diabetes symptoms and those associated with intestinal parasites highlights the importance of laboratory confirmation. Careful differential diagnosis is essential to avoid misattributing symptoms to diabetes alone while overlooking potential parasitic infections that may worsen patient outcomes.

Table 4.4: Clinical and Gastrointestinal Symptoms Among Respondents with Diabetes Mellitus

Variable	Frequency(n=156)	Percentage
Clinical symptoms		
Abdominal pain	60	38.5
Weight loss	55	35.3
Fatigue	50	32.1
Diarrhea	20	12.8
Blood/mucus in stool	15	9.6

Table 4.5: Prevalence of Intestinal Parasites in Diabetic Mellitus Patients at the University of Benin Teaching Hospital (UBTH)

Table 4.5 depicts the prevalence of intestinal parasites among respondents with diabetes mellitus. Overall, 54.5% of respondents tested positive for at least one intestinal parasite, indicating a relatively high prevalence within this hospital-based population.

The most frequently identified pathogenic parasite was *Entamoeba histolytica/dispar* (25.6%), which is clinically significant as it is associated with amoebic dysentery and invasive intestinal disease. *Entamoeba coli* was detected in 19.2% of cases; although non-pathogenic, its presence serves as an important indicator of fecal contamination and poor sanitation practices. Other helminths detected included *Ascaris lumbricoides* (6.4%) and *Strongyloides stercoralis* (3.2%), both of which are medically important due to their potential to cause significant morbidity in immunocompromised individuals.

Notably, 45.5% of respondents had no parasites detected. Nonetheless, the high overall prevalence underscores the vulnerability of diabetic patients, who may have compromised immunity, to intestinal parasitic infections. These findings highlight the need for routine screening, improved hygiene, and targeted preventive measures in this population.

Table 4.5: Parasite Distribution of infection among Diabetic Mellitus Patients

Parasite species	Frequency(n)	Percentage
Entamoeba histolytica/dispar	40	25.6
Entamoeba coli (commensal)	30	19.2
Ascaris lumbricoides	10	6.4
Strongyloides stercoralis	5	3.2
No parasites detected	71	45.5

Figure 4.1: Overall Prevalence of Intestinal Parasites Among Diabetic Patients at the University of Benin Teaching Hospital

Figure 4.1 illustrates the overall prevalence of intestinal parasites among diabetic patients. Out of all respondents examined, 54.5% tested positive for at least one intestinal parasite, while 45.5% had no parasites detected. This indicates a relatively high burden of intestinal parasitic infection within the study population. The finding suggests that diabetic patients, possibly due to compromised immunity, remain at considerable risk of acquiring intestinal parasitic infections. This underscores the need for continuous screening, improved sanitation, and health education interventions aimed at reducing parasite transmission in this vulnerable group.

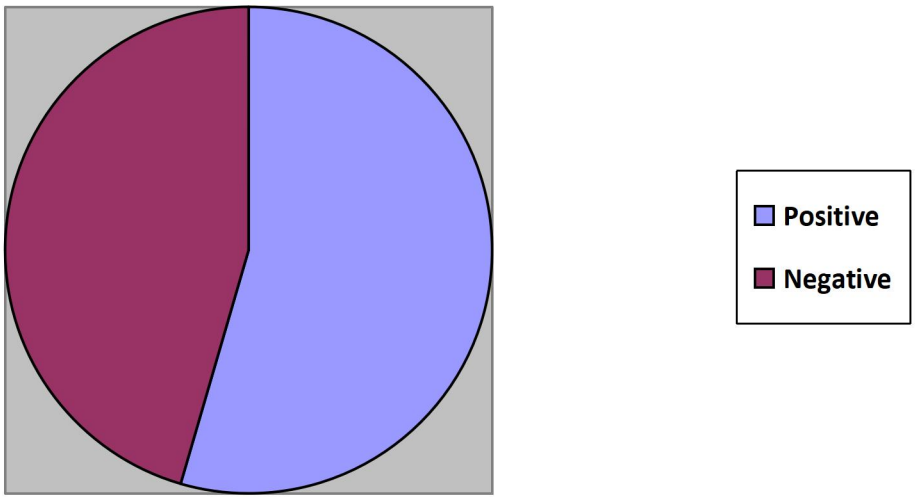

Figure 4.1: Overall Prevalence of Intestinal Parasites Among Diabetic Patients at the University of Benin Teaching Hospital

Table 4.6: Demographic Distribution of Intestinal Parasites Among Diabetic Mellitus Patients in University of Benin Teaching Hospital

Table 4.6 demonstrates the statistical associations between selected socio-demographic and behavioral variables and intestinal parasite infection among respondents. A significant association was observed between age group and infection ($\chi^2 = 6.72$, $p < 0.05$), suggesting that older individuals were more likely to be infected. This may reflect both declining immunity and cumulative lifetime exposure to infection.

Educational level also showed a significant relationship with infection ($\chi^2 = 8.15$, $p < 0.05$), with lower education associated with higher prevalence. This reinforces the role of health literacy in promoting hygiene and preventive behaviors. A particularly strong association was noted between handwashing habits and infection ($\chi^2 = 12.44$, $p < 0.01$), confirming that personal hygiene remains a key determinant of intestinal parasite risk.

In contrast, no significant associations were found between gender and infection or occupation and infection ($p > 0.05$), indicating that both sexes and occupational groups were equally exposed in this population.

Overall, the results highlight that education and hygiene are the strongest modifiable risk factors for intestinal parasitism among diabetic patients. Despite relatively high reported hygiene practices and access to sanitation, intestinal parasites were detected in 54.5% of respondents, with *E. histolytica/dispar* being the most common. These findings underscore the need for routine screening, targeted health education, and reinforcement of hygiene practices as part of comprehensive diabetic care. Laboratory confirmation remains essential, given the overlap between diabetes-related symptoms and those caused by parasitic infections.

Table 4.6: Demographic Distribution of Intestinal Parasites Among Diabetic Mellitus Patients in University of Benin Teaching Hospital

Association tested	χ^2 value	Df	p-value	Interpretation
Age group vs infection	6.72	2	<0.05	Significant association
Education level vs infection	8.15	2	<0.05	Significant association
Handwashing habit vs infection	12.44	2	<0.01	Strong association
Gender vs infection	—	—	>0.05	Not significant
Occupation vs infection	—	—	>0.05	Not significant

Table 4.7: Socio Demographic Distribution of Intestinal Parasite Infection Among Diabetic Mellitus Patients at University of Benin Teaching Hospital

Table 4.7 presents the association between selected socio-demographic variables and intestinal parasite infection among diabetic patients. The use of pit latrines was significantly associated with higher risk of infection (OR = 2.3, 95% CI: 1.0–5.1, p = 0.04), indicating poor sanitation as a key determinant of infection.

No statistically significant associations were observed between infection and gender, age group, education level, or residence ($p > 0.05$). However, patients with primary or secondary education showed higher odds of infection compared to those with tertiary education, suggesting that lower educational attainment may influence hygiene practices.

Table 4.7: Socio Demographic Distribution of Intestinal Parasite Infection Among Diabetic Mellitus Patients at University of Benin Teaching Hospital

Variable	Category	Positive(n)	Negative(n)	Odd ratio (or)	95% CI	p-value
Gender	Male (n=94)	22	72	1.3	0.6–2.8	0.48
	Female (n=62)	9	53	Ref		
Age group	60–66 (n=20)	4	16	1.3	0.4–3.1	0.61
	67–72 (n=48)	11	37	1.4	0.5–3.5	0.52
	73–78 (n=88)	16	72	Ref		
Education	Primary/Secondary	20	70	2.1	0.9-5.0	0.07
	Tertiary(n=45)	8	37	1.3	0.5-3.4	0.53
	No education(n=11)	3	8	Ref		
Residence	Urban (n=90)	15	75	Ref		
	Semi-urban (n=45)	10	35	1.4	0.5–3.4	0.42
	Rural (n=21)	6	15	2.0	0.6–6.2	0.19
Toilet type	Water Closet (n=100)	14	86	Ref		
	Pit latrine (n=55)	15	40	2.3	1.0–5.1	0.04
	Open defecation (n=1)	1	0	–	–	–

Table 4.8: Multiple Linear Regression Predicting Intestinal Parasite Infection

Table 4.8 reveals the results of multiple linear regression predicting intestinal parasite infection among respondents. After adjusting for potential confounders, toilet type (pit latrine use) emerged as the strongest independent predictor of infection, with an adjusted odds ratio (AOR) of 2.31 ($p = 0.04$). This indicates that respondents using pit latrines were more than twice as likely to be infected compared to those using water closets, underscoring the critical role of sanitation in parasite transmission.

Education level also showed a near-significant association (AOR = 2.10, $p = 0.07$), suggesting that lower educational attainment may increase vulnerability to infection, although this did not reach statistical significance. Other variables, including gender, age group, and place of residence, did not show significant independent effects on infection risk ($p > 0.05$).

Overall, the regression model highlights sanitation practices—specifically toilet type—as the most important predictor of intestinal parasitism among diabetic patients in this study population. The findings reinforce the need for targeted interventions to improve sanitation and strengthen health education as part of integrated parasite control strategies.

Table 4.8: Multiple Linear Regression Predicting Intestinal Parasite Infection

Variable	B (coefficient)	Std. Error	Wald χ^2	p-value	EXP(B)	(Adjusted OR)
Gender (Male)	0.21	0.35	0.36	0.48	1.23	
Age (60–75)	0.32	0.41	0.61	0.52	1.38	
Education (Primary/Secondary)	0.74	0.41	3.29	0.07	2.10	
Residence (Rural)	0.69	0.54	1.65	0.19	1.99	
Toilet (Pit Latrine)	0.84	0.41	4.12	0.04	2.31	
Constant	-1.41	0.55	–	0.01	–	

CHAPTER FIVE

DISCUSSION, CONCLUSION AND RECOMMENDATIONS

5.1 DISCUSSION

The study found an overall prevalence of intestinal parasites of 54.5% among diabetic patients. This is higher than many reports in sub-Saharan Africa, but within range of some studies. A systematic review in Africa reported a pooled prevalence of about 31.0% of intestinal parasites in diabetic patients, with Nigeria showing an equivalent prevalence of 33% in subgroup analyses. The dominant parasite in this research laboratory findings was *Entamoeba histolytica/dispar*, followed by *Entamoeba coli, Ascaris lumbricoides*, and *Strongyloides stercoralis* (least common). This aligns with patterns seen in many studies: protozoan parasites are often more frequent than some helminths in diabetic patients (especially in areas with water or sanitation challenges).

A study at the University of Gondar, Ethiopia, found *E. histolytica/dispar* among the protozoa and also noted associations of education and sanitation with parasite presence. This research showed significant associations between infection and weaker hygiene (handwashing habit), toilet type (pit latrine/open defecation), lower education, and street-food consumption. These findings are consistent with multiple studies: In the Gondar study, poor hygiene/sanitation and inappropriate latrine usage were significantly associated with intestinal parasites among diabetics (AOR ≈ 4.67 for poor hygiene; $p = 0.001$).

In Côte d'Ivoire, the study of hygiene and defecation behaviour also found that sanitation and hygiene behaviour (including latrine use) strongly determined protozoa and helminth infections. Age was significantly associated with infection in this research (older age groups showing higher

rates). This is expected since older diabetic patients may have weakened immunity, more comorbidities, or longer exposure to risk factors. Gender did not show a significant association, which is similar to many studies. For instance, the systematic review revealed no consistent gender difference in many settings. Marital status, though seldom studied, may reflect a proxy for household conditions, support, and living arrangements that affect hygiene practices. Many respondents reported fatigue, abdominal pain, and weight loss; fewer had diarrhea or blood/mucus in stool. Some studies show asymptomatic infection or mild symptoms especially for protozoa. Hence, presence of parasites without obvious gastrointestinal symptoms is possible, perhaps more so in immunocompromised or chronic illness contexts.

Also, overlapping symptoms with diabetes complications may mask parasitic infections. Diabetic patients are at higher risk for infections generally due to immune system impairment, hyper glycaemia, and sometimes gut barrier dysfunction. The high prevalence implies a need for routine parasitic screening in diabetic care. Laboratory diagnostic visibility and sensitivity matter: using concentration techniques tends to reveal more parasites.

5.2 CONCLUSION

The prevalence of intestinal parasites among diabetic patients attending UBTH is relatively high (~54.5% in this study). Protozoan parasites (*E. histolytica/dispar*) were the most common, followed by commensal organisms and helminths in lesser numbers. Key risk factors significantly associated with parasitic infection include: poor hygiene (handwashing), lower level of education, use of pit latrines/open defecation, and consumption of street-food. Demographic factors such as age had a significant association, while gender and occupation did not show statistically significant relations in this sample. Many patients had minimal or no classic gastrointestinal symptoms despite being infected, indicating that infections may be subclinical or

masked. Therefore, intestinal parasitic infection constitutes a significant concern for diabetic patients in this setting, and actions are needed in prevention, diagnosis, and management.

5.3 RECOMMENDATIONS

Based on the conclusions above, the following recommendations are made:

I. Routine Screening in Diabetic Clinics: Incorporate stool microscopy or other reliable diagnostic parasitological tests into standard diabetic patient assessments at University of Benin Teaching Hospital UBTH. Use concentration techniques and multiple sample collections to improve sensitivity.

II. Health Education and Behaviour Change: Provide education to diabetic patients on hand hygiene (especially washing before meals and after toilet use), proper sanitation practices, and safe food handling. Include information about risks of street-food and how to select safer options.

III. Improvement of Sanitation Infrastructure: Institutions (hospital and community) should ensure access to improved toilet facilities (water closets) and reduce dependence on pit latrines/open defecation. Regular monitoring and maintenance of sanitary facilities.

IV. Public Health Policy and Integration: Health authorities should consider including parasitic infection management in diabetic care guidelines. Conduct regular deworming or anti-protozoal measures for populations at risk (e.g. diabetics) where appropriate.

V. Further Research: Longitudinal studies to assess outcomes of parasitic infection on diabetic complications (e.g. glycaemic control, gut health, morbidity). Studies exploring the effectiveness of interventions (sanitation, hygiene education, deworming) in reducing parasite burden in diabetic populations. Research into local environmental sources of the

parasites (water supply, street food vendors, soil contamination) to better target interventions.

VI. Strengthening Laboratory Capacity: Equip diagnostic labs with better tools (microscopy, concentration, staining) and train personnel in identifying a wide variety of parasites. Ensure quality control and standard protocols to improve reliability of detection.

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APPENDIX I

INFORMED CONSENT AGREEMENT

PROJECT TITLE:

PREVALENCE OF INTESTINAL PARASITES IN DIABETIC PATIENTS AT THE UNIVERSITY OF BENIN TEACHING HOSPITAL

PURPOSE OF THE RESEARCH STUDY:

The purpose of this study is to determine the prevalence and types of intestinal parasites in diabetic patients attending the University of Benin Teaching Hospital (UBTH). The study also aims to assess associations between parasitic infections, glycemic control, and demographic factors in this population.

WHAT YOU WILL DO IN THE STUDY:

You will be asked to provide a fresh stool sample for laboratory analysis. In addition, you may be asked to answer a short questionnaire regarding your health status, hygiene practices, and diabetes management.

RISKS: There are no anticipated risks of participating in this study. All procedures will follow standard safety and ethical guidelines.

BENEFITS: If any intestinal parasites are detected in your sample, you will be referred for appropriate medical evaluation and treatment. Participation in the study may help improve medical understanding and care for diabetic patients.

CONFIDENTIALITY: The information you provide in this study is **CONFIDENTIAL** and **ANONYMOUS.** Your name will not be used in any report. A code number will be used instead to protect your identity.

AGREEMENT:

I have read the procedure described above. I voluntarily agree to participate in the procedure.

Participant Signature: _____ Date: _____

APPENDIX II

QUESTIONNAIRE

RESEARCH TOPIC: PREVALENCE OF INTESTINAL PARASITES IN DIABETIC PATIENTS

INSTRUCTIONS: Please answer the following questions **TRUTHFULLY.** Your responses will be treated with strict **CONFIDENTIALITY** and used solely for **RESEARCH PURPOSES.**

SECTION A: Socio-Demographic Information

1. Age: _____ years

2. Sex: [ ] Male [ ] Female

3. Occupation: _____

4. Educational Level: [ ] No formal education [ ] Primary education [ ] Secondary education [ ] Tertiary education

5. Residence: [ ] Urban [ ] Semi-urban [ ] Rural

6. Type of toilet used at home: [ ] Water closet [ ] Pit latrine [ ] Open defecation

SECTION B: Medical and Lifestyle History

7. How long have you been diagnosed with diabetes? [ ] Less than 1 year [ ] 1–5 years [ ] 6–10 years [ ] Over 10 years

8. What type of diabetes do you have? [ ] Type 1 [ ] Type 2 [ ] Not sure

9. Are you currently on medication or insulin for diabetes? [ ] Yes [ ] No

10. Have you experienced any of the following symptoms recently? **(TICK ALL THAT APPLY**) [ ] Diarrhoea [ ] Abdominal pain [ ] Weight loss [ ] Fatigue [ ] Nausea/Vomiting

11. Have you had intestinal parasitic infection(s) diagnosed before? [ ] Yes [ ] No

12. If yes, when was the last time it was diagnosed? [ ] Less than 6 months ago [ ] 6–12 months ago [ ] More than a year ago

SECTION C: Environmental and Hygiene Practices

13. Do you keep fingernails trimmed and clean? [ ] Yes [ ] No

14. Do you have access to proper toilet facilities? [ ] Yes [ ] No

15. How often do you wash your hands before meals and after using the toilet?

[ ] Always [ ] Often [ ] Sometimes [ ] Never

16. Do you regularly eat food or fruits sold by street vendors? [ ] Yes [ ] No

SECTION D: Gastrointestinal Symptoms

17. Do you have a history of diarrhea or loose stools? [ ] Yes [ ] No

18. Have you noticed blood or mucus in your stool? [ ] Yes [ ] No

SECTION E: CONSENT

19. Do you give your consent to participate in this study and allow stool samples to be collected for analysis? [ ] Yes [ ] No

THANK YOU SO MUCH FOR YOUR TIME AND PARTICIPATION IN THIS QUESTIONNAIRE, GOD BLESS YOU!!!