Malaria cases

Clinical Correlation Cases
MS2 MICB 820 course

Hunter Ratliff, MD MPH
HunterRatliff1.com
08/04/2026

Ages, dates, and other identifying information may have been changed
I have no conflict of interest in relation to this presentation

Learning Objectives

1. Recognize the importance of considering **Malaria** in any **febrile illness** in a **returning traveler**
2. Understand **life cycle** and **pathogenesis**
3. Identify drugs used for **prevention and treatment**

In the past 5 years, where was the closest locally acquired malaria case?

Mentimeter

Case #1

Case 1: HPI

A previously healthy 22 year old male presents with **high fevers**, **chills**, and **shortness of breath**

- Was feeling normal when he returned from study abroad around a week ago
- 3 days after returning, he developed high fever, chills, **confusion**
  - Went to urgent care → diagnosed as having a viral upper respiratory infection
- Went to ED because feeling much worse

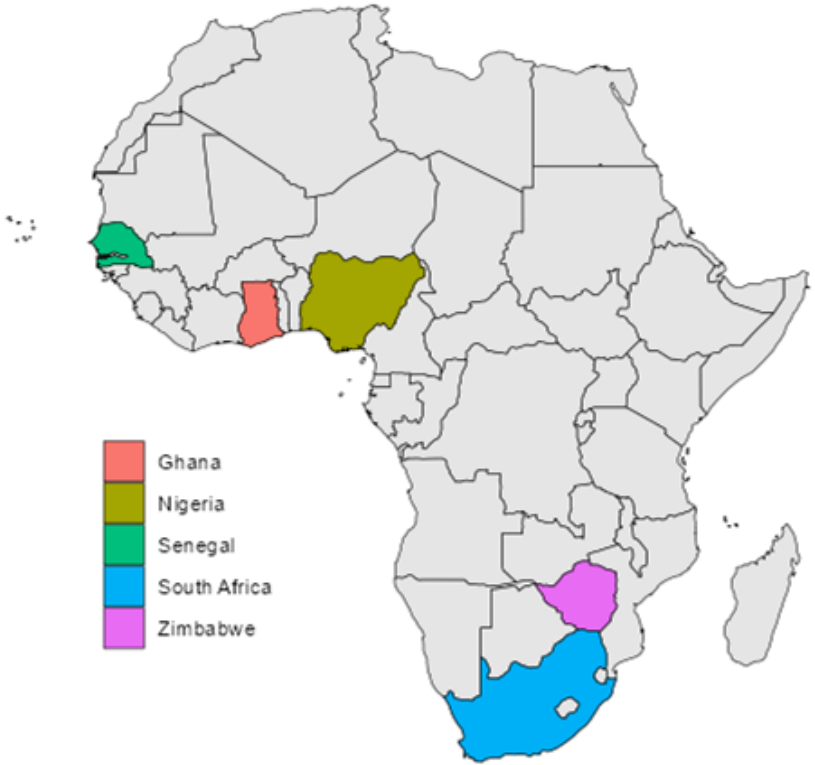
Social & exposure history

- Housing/Occupation: Engineering student, about to graduate. Lives with his family near Washington, PA
- Hobbies: Hiking & spending time outdoors
- Sex, drugs: One female partner, no condoms; occasional alcohol & THC

Case 1: Travel history

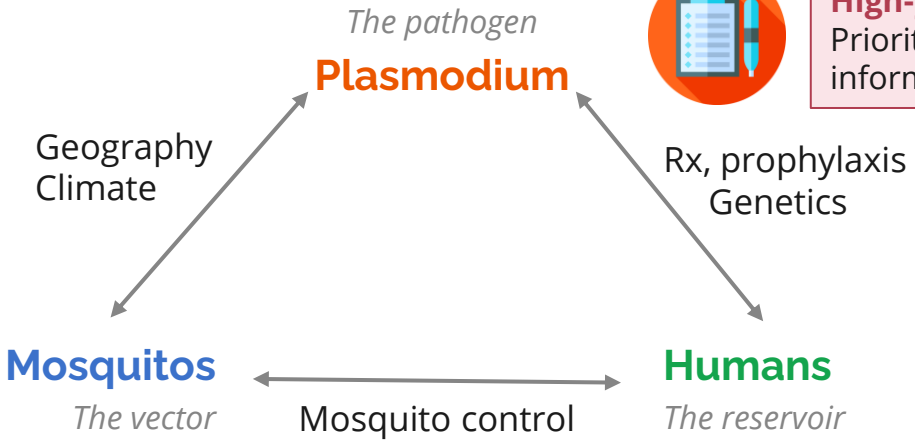
Study abroad in South Africa:

He also traveled to other countries in East, Central and West Africa



Why is travel such a strong risk factor?

How to make malaria



High-yield
Prioritize knowing this information

The more you know
This is potentially helpful information to understand, but the details are less likely to be tested

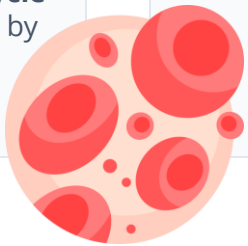
How to make malaria

To establish endemic malaria, three critical components must coexist:

01 Pathogen

Plasmodium

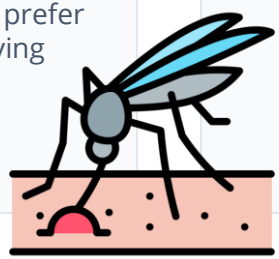
- Parasitic **single-celled eukaryotes** (protozoa)
- Unicellular nature makes them **highly adaptable** compared to multicellular organisms (like us)
- Has a **complex life cycle** that somewhat varies by species (e.g., ***P. falciparum***, ***P. vivax***)



02 Vector

Anopheles

- Transmitted exclusively by the **female** mosquito
- Found globally, represented by diverse regional species
- Key species like ***A. gambiae*** strongly prefer human hosts, driving rapid spread



03 Host

Human Reservoir

- Humans serve as the **only reservoir** for the deadliest malaria species
- Disease of the **RBCs**
- In endemic areas, **asymptomatic carriers** infect new vectors, creating a continuous cycle of transmission

Plasmodium species

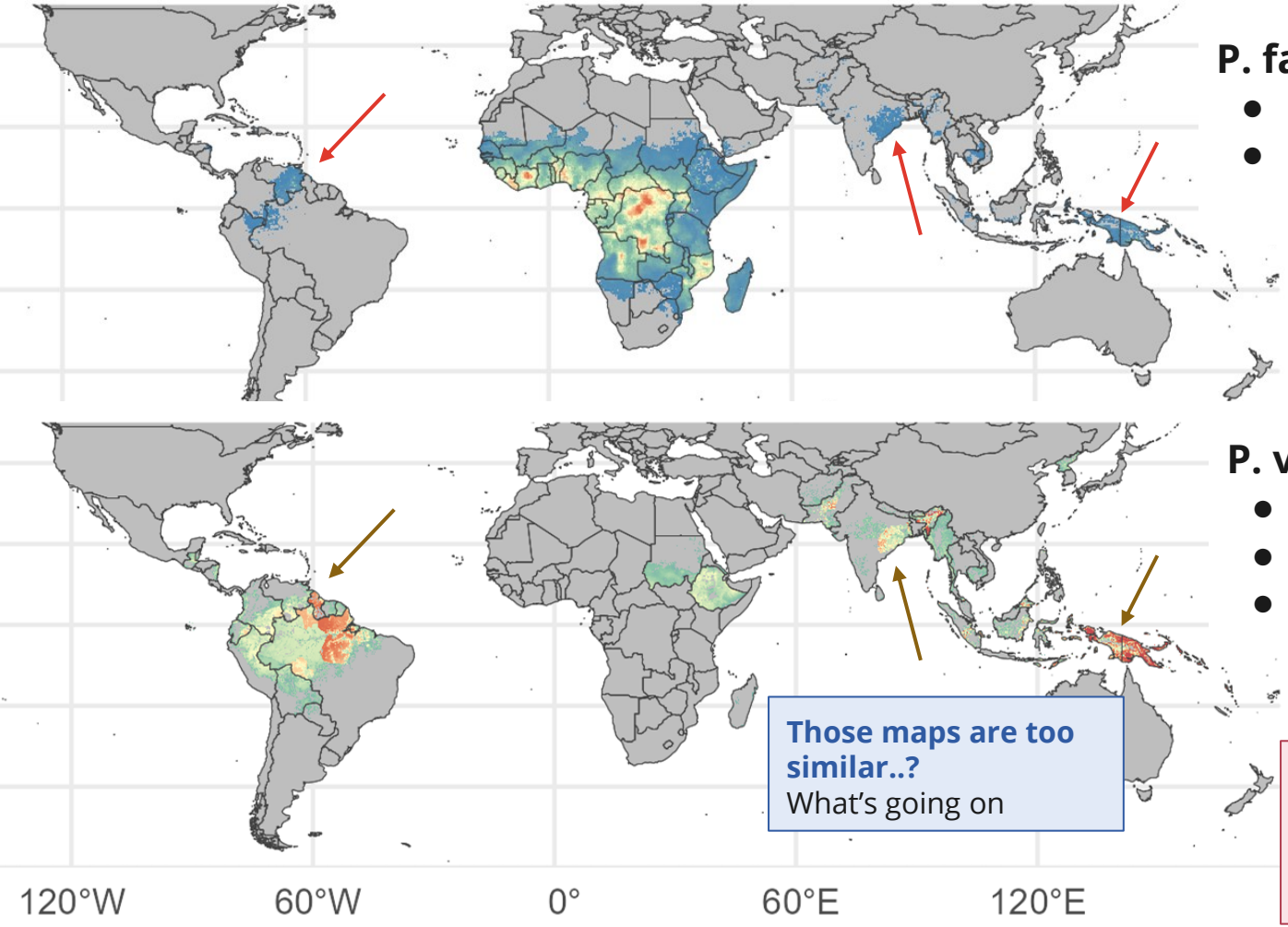
01 Pathogen

Plasmodium

Main species to know, in order of importance:

1. ***P. Falciparum*** - most **F**atal
2. ***P. vivax/ovale*** - can relapse (**hypnozoites**)
3. ***P. malariae*** - lingers (sometimes for years)

P. knowlesi - macaque-associated zoonosis (forested Southeast Asia) that has potential to cause severe disease. Not nearly as important as the ones above



P. falciparum

- **Sub-Saharan Africa**
- SE Asia & Amazon basin

P. vivax & ovale

- **Central America**
- South Asia
- Oceania

Those maps are too similar..?
What's going on

Warning
Geography can clue you in, but it does not ever rule one species out

Source: Malaria Atlas

Malaria lifecycle: Vectors & climate

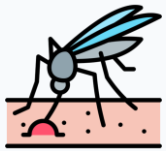
01 Pathogen

Plasmodium

Has a **complex life cycle** that somewhat varies by species (e.g., ***P. falciparum*** often **F**atal, ***P. vivax***)

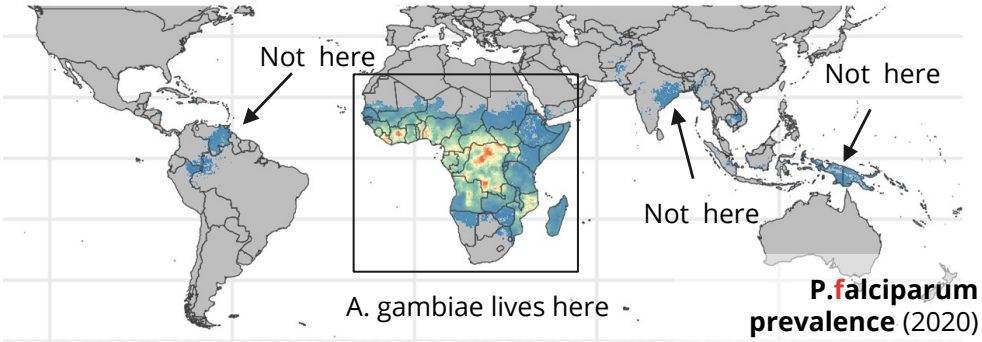
02 Vector

Anopheles



- Transmitted exclusively by the **female *Anopheles*** mosquito (men are *worthless*...)
- **A. gambiae** → malaria in large areas of Africa
  - But numerous other species of Anopheles mosquitoes also transmit malaria, including *Plasmodium falciparum*

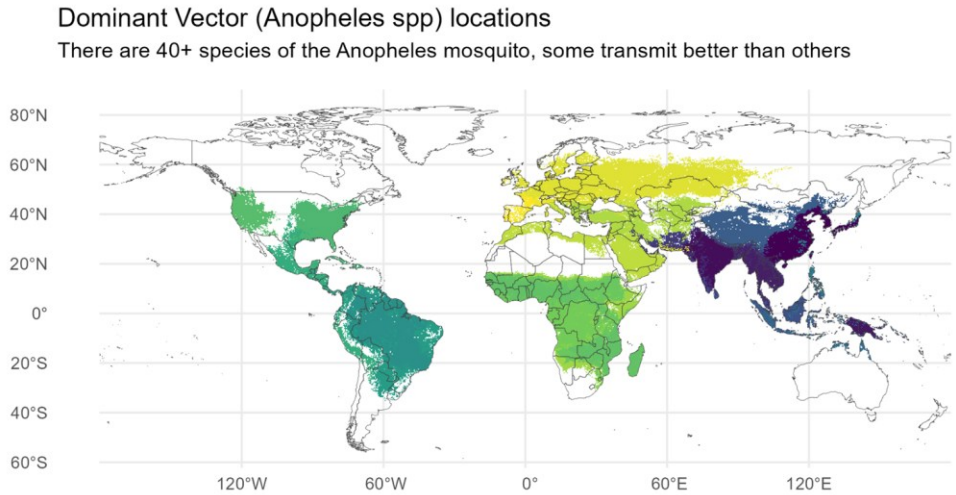
Why doesn't malaria exist everywhere there are mosquitos?

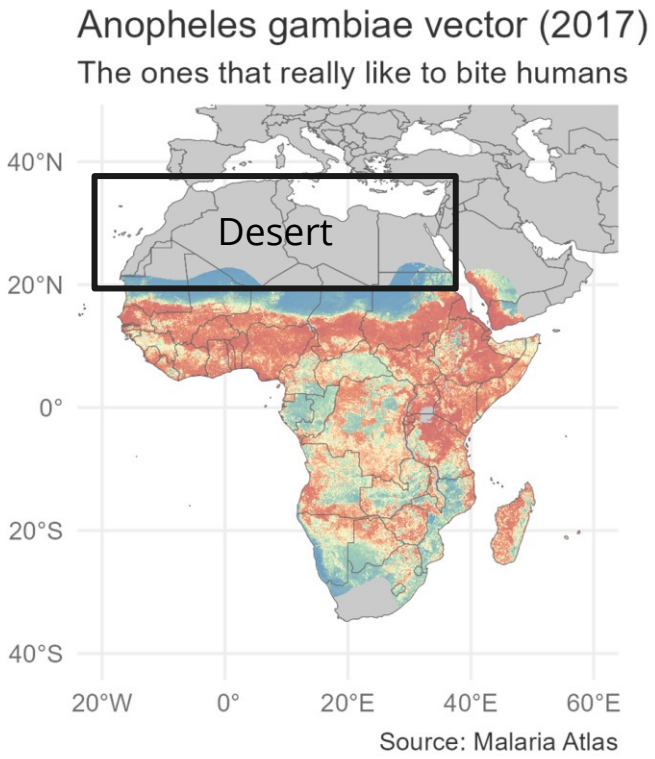


Applies to whole slide

Malaria lifecycle: Vectors & climate

- All species of Anopheles mosquitoes require **stagnant water** (fresh or brackish) to breed
  - Raining more often → more mosquitos





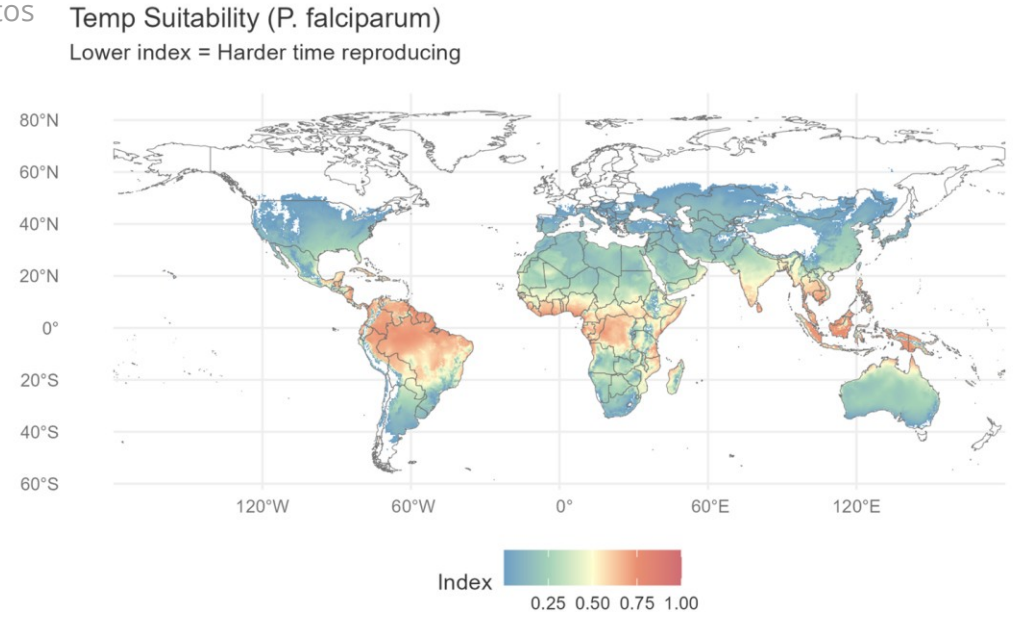
Applies to whole slide

Malaria lifecycle: Vectors & climate

- All species of Anopheles mosquitoes require **stagnant water** (fresh or brackish) to breed
  - Raining more often → more mosquitos

- Both the vector (mosquitoes) and pathogen (plasmodium) **do better when it's warmer**
  - Mosquitos thrive in humidity
  - Part of Plasmodium's life cycle depends on the heat

- Many, many other influences (but this slide **unlikely to be on the exam**)



Source: Malaria Atlas

Back to case 1

A previously healthy 22 year old male presents with 3 days of **high fevers**, chills, and **shortness of breath**. One week ago returned from a semester in **Africa**. Initial thought was viral URI (**headache**, **myalgias/arthralgias**), but now doing much worse

Case 1: Physical exam & labs

Temp	39.5 °C (103.1 °F)	Pulse	118	SpO2	90 %
General	Quite ill appearing, confused				
GI	Non-tender, splenomegaly				
Neuro	Grossly intact, no neck stiffness				

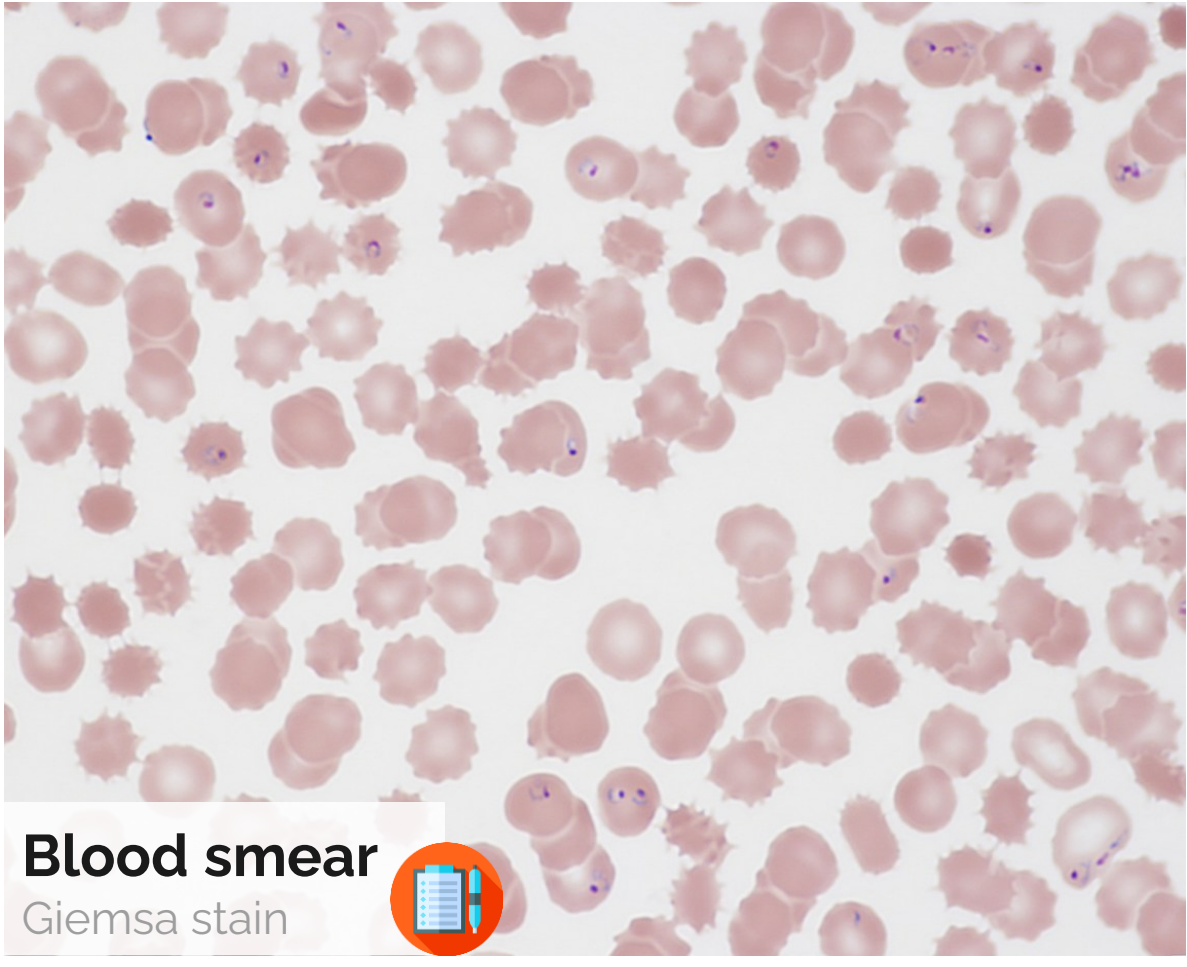
CBC	Result	Normal
WBC	5	3.7-11
Hgb	8.7→7	11-16
Platelets	17	150-400
Coags		
PT	16	25-35
PTT	39	11-14

Chem7	Result	Normal
Na	140	136-145
K	4.9	3.5-5.1
HCO3	21	23-31
BUN	47	8-25
Cr	1.5	0.6-1.1

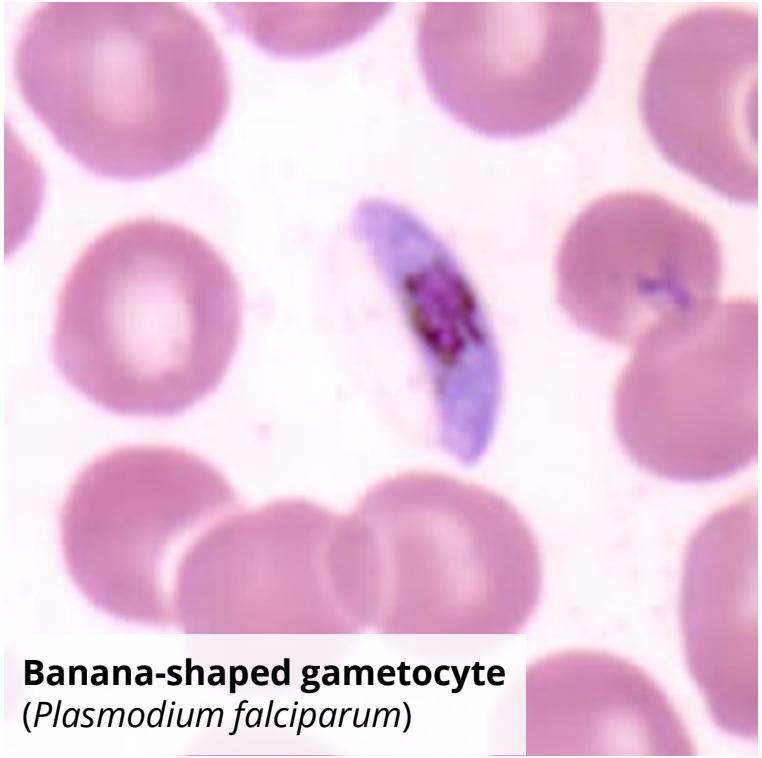
LFTs	Result	Normal
AST	135	11-34
ALT	279	<31
Alk Phos	140	55-145
Bili	6.0	0.3-1.3
(Unconj)	5.4	

Case 1

- Develops respiratory distress
  - Requires **intubation**
- **Blood smear** reveals his diagnosis
- Started on treatment
  - **Gradually improves** in coming days

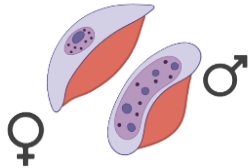


Ring form trophozoites
(*Plasmodium falciparum*)



Banana-shaped gametocyte
(*Plasmodium falciparum*)

Giemsa stains



Clinical
&
Laboratory manifestations

Malaria: Clinical & laboratory manifestations

Typical presentation

- **Fever and chills**
  - Often accompanied by headache, myalgias, arthralgias, fatigue/weakness
  - "Flu like illness"
- Variable: nausea, vomiting, and diarrhea

Severe malaria (often *P falciparum*)

- Altered mental status, seizures, or coma
- Hypoglycemia
- Acute kidney injury
- Pulmonary edema or acute respiratory distress
- Severe anemia, jaundice
- Shock, or other multiorgan dysfunction

Physical exam

- Pallor (from anemia) →
- Splenomegaly →
- Jaundice (when severe) →
- No lymphadenopathy

Labs

- Anemia
- Thrombocytopenia
- ↑ bilirubin
- Normal/low WBC

Malaria: Diagnosis

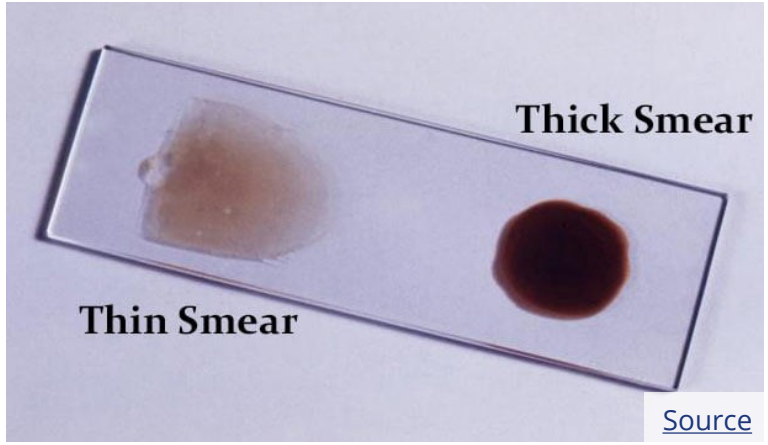
Gold standard: **Giemsa-stained** thick and thin **peripheral blood smears**

- Thick smear: More **sensitive** for parasite detection
- Thin smear: Identifies ***Plasmodium* species** and quantifies parasitemia

If the initial smear is negative but suspicion remains high, **repeat every 12–24 hours** for a total of three sets

Additional diagnostic methods

- Rapid antigen-detection tests
- Acridine orange fluorescence staining
- PCR or other nucleic acid amplification tests



Source

Malaria: Clinical & laboratory manifestations

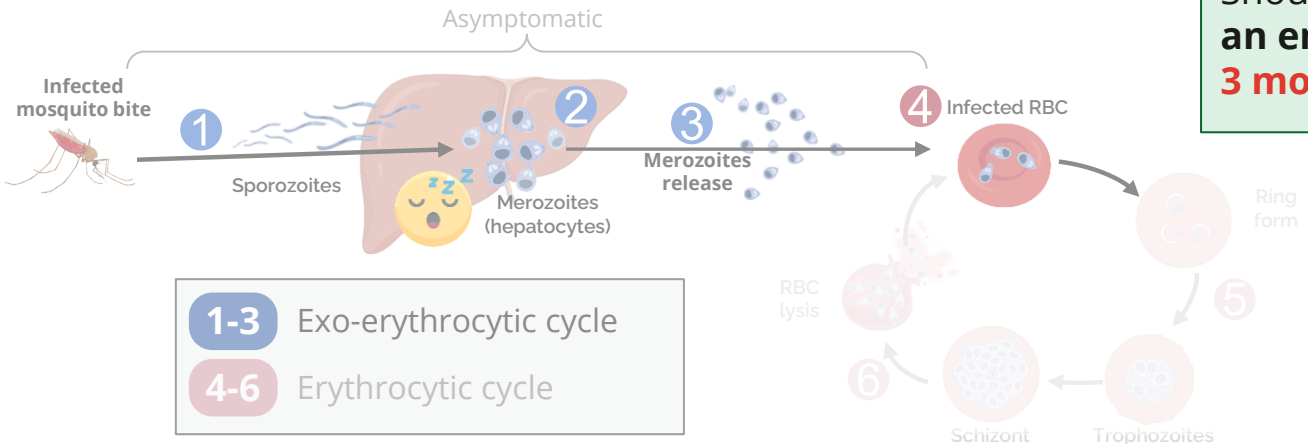
Incubation period

Asymptomatic until the erythrocytic stage

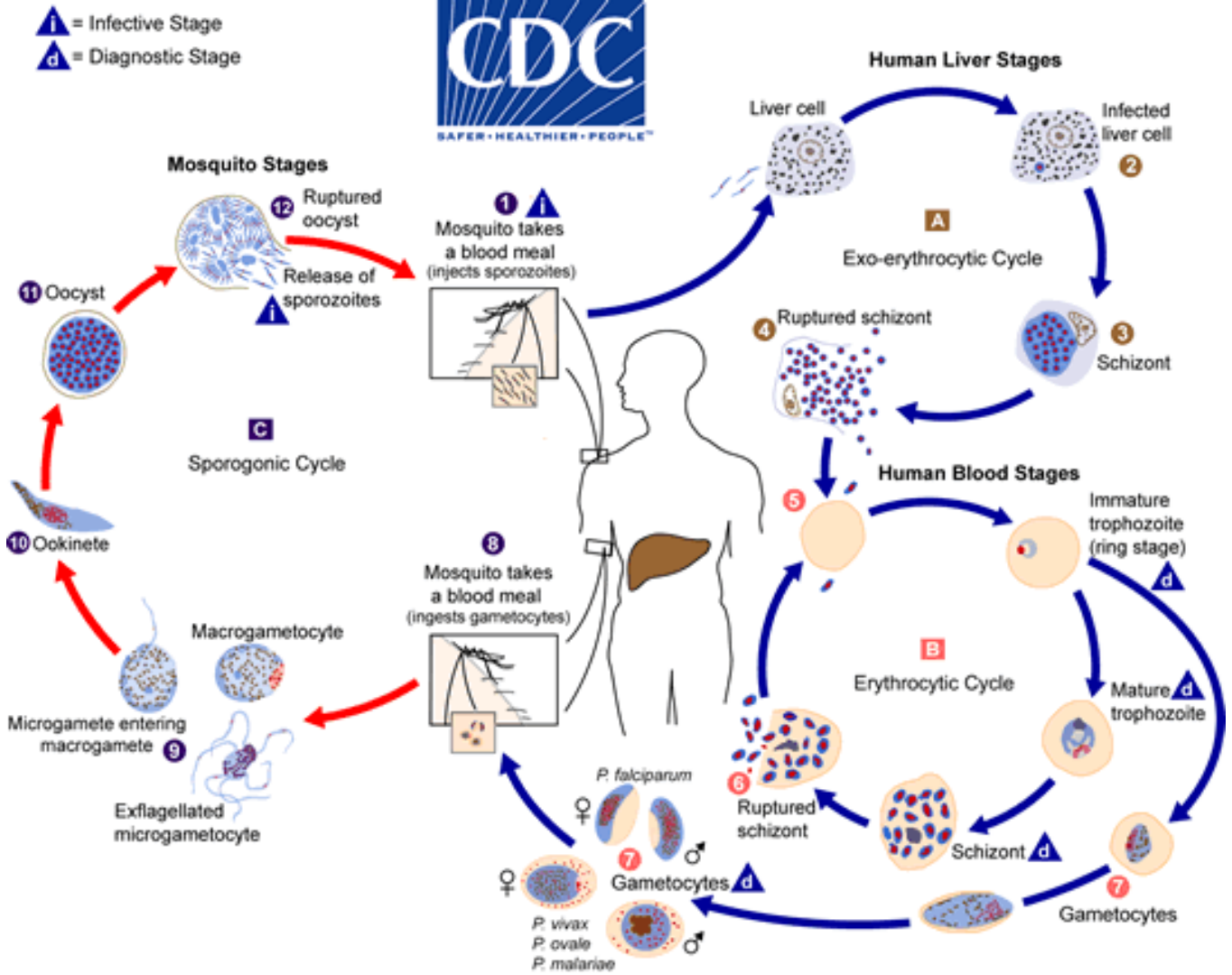
Incubation period typically 1-4 weeks

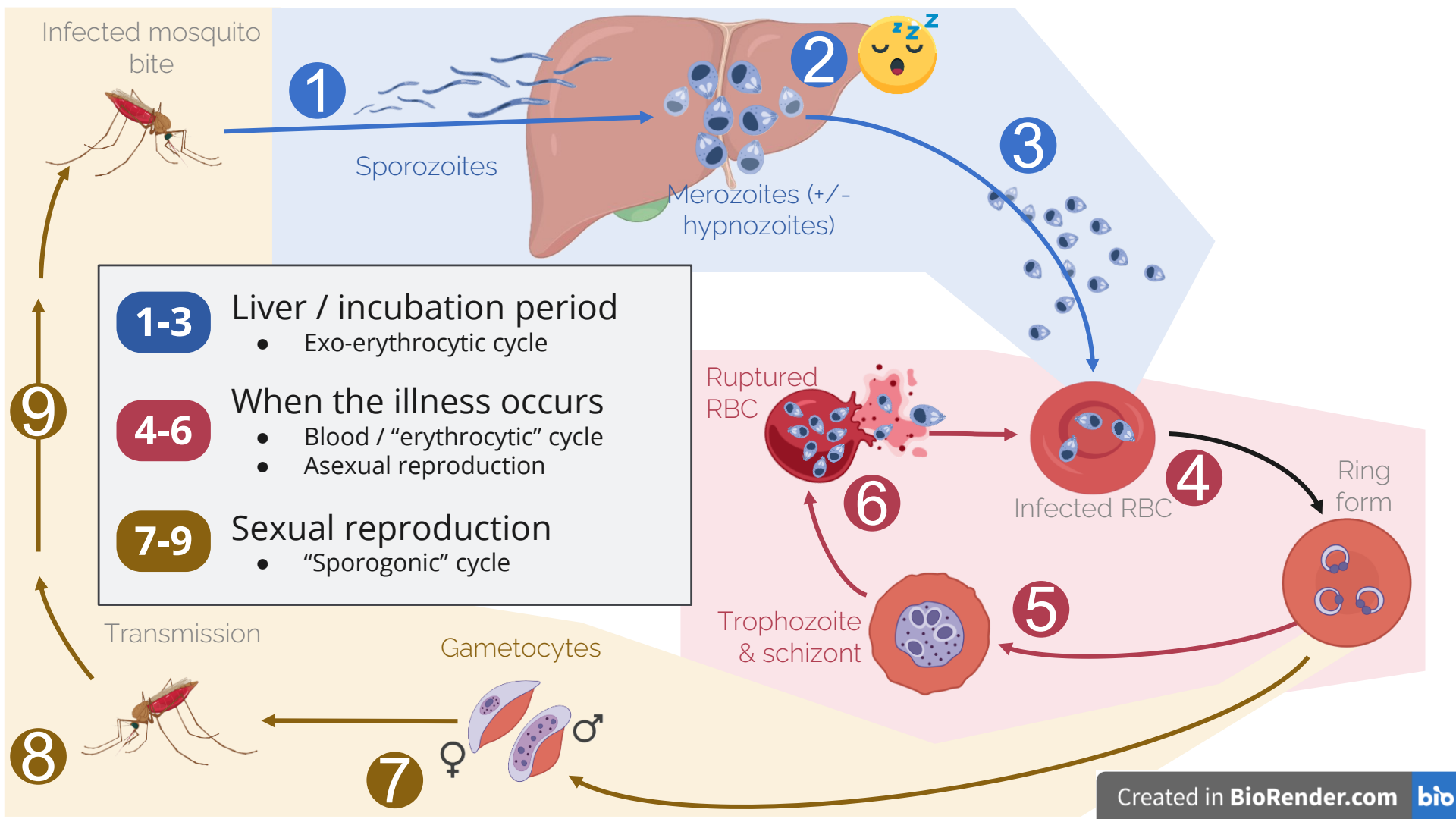
- Variable by species, climate, genetics
- Many with **vivax/ovale** got ill 90+ days from return (majority before 3 years)

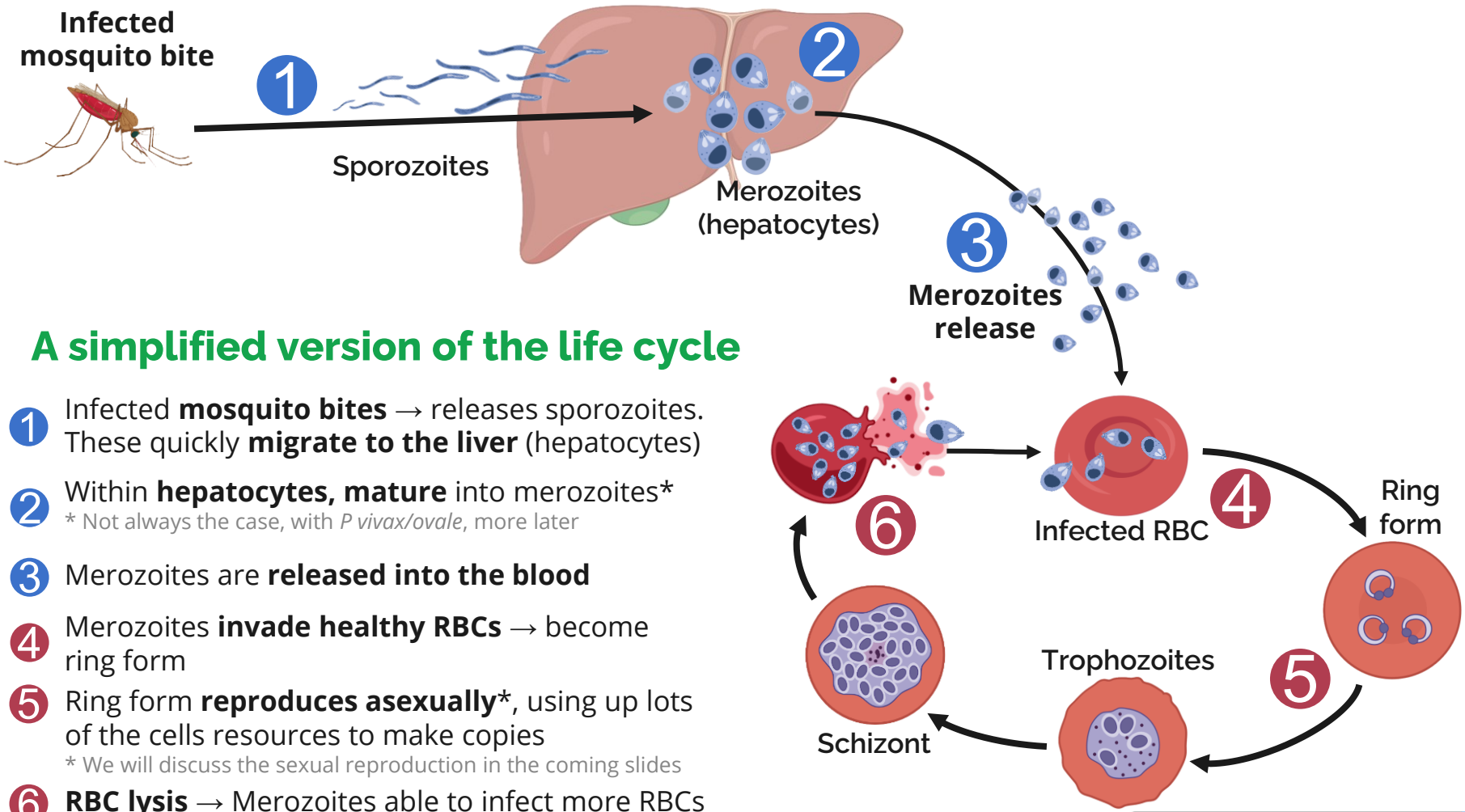
Should be on DDx if **been in an endemic area within past 3 months** (esp for falciparum)



Pathogenesis
&
Life cycle


i = Infective Stage
d = Diagnostic Stage
CDC
SAFER·HEALTHIER·PEOPLE™
Human Liver Stages
Liver cell
Infected liver cell
2
A
Exo-erythrocytic Cycle
Ruptured schizont
4
3
Schizont
Human Blood Stages
5
Immature trophozoite (ring stage)
d
B
Erythrocytic Cycle
Mature trophozoite
d
6
Ruptured schizont
Schizont
d
7
Gametocytes
P. falciparum
7
Gametocytes
d
P. vivax
P. ovale
P. malariae
Mosquito Stages
12
Ruptured oocyst
Release of sporozoites
i
11
Oocyst
1
i
Mosquito takes a blood meal
(injects sporozoites)
C
Sporogonic Cycle
10
Ookinete
8
Mosquito takes a blood meal
(ingests gametocytes)
Macrogametocyte
Microgamete entering macrogamete
9
Exflagellated microgametocyte


Infected mosquito bite
1
Sporozoites
2
Merozoites (+/- hypnozoites)
3
Ruptured RBC
6
Infected RBC
4
Ring form
Trophozoite & schizont
5
Gametocytes
♀
♂
7
8
Transmission
9
1-3
Liver / incubation period
Exo-erythrocytic cycle
4-6
When the illness occurs
Blood / "erythrocytic" cycle
Asexual reproduction
7-9
Sexual reproduction
"Sporogonic" cycle
Created in BioRender.com
bio



A simplified version of the life cycle

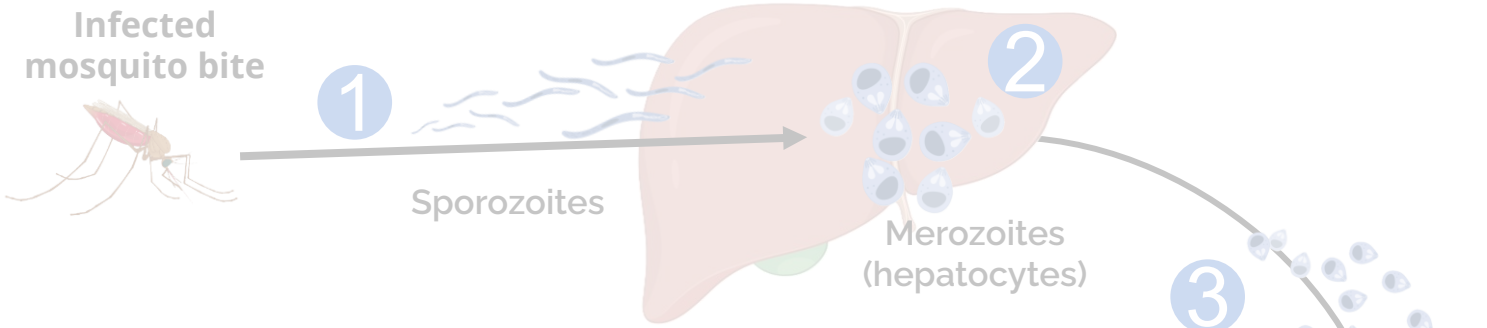
1. Infected **mosquito bites** → releases sporozoites. These quickly **migrate to the liver** (hepatocytes)
2. Within **hepatocytes, mature** into merozoites*
   * Not always the case, with *P vivax/ovale*, more later
3. Merozoites are **released into the blood**
4. Merozoites **invade healthy RBCs** → become ring form
5. Ring form **reproduces asexually***, using up lots of the cells resources to make copies
   * We will discuss the sexual reproduction in the coming slides
6. **RBC lysis** → Merozoites able to infect more RBCs

Pop quiz:
What resources are y'all using for micro?
(besides this lecture)

Bonus points if someone sends me an updated Sketchy video (:

Mentimeter

1. Sketchy Microbiology
2. Anki: AnKing deck
3. Anki: Pepper deck
4. Amboss
5. Boards & Beyond
6. Something else

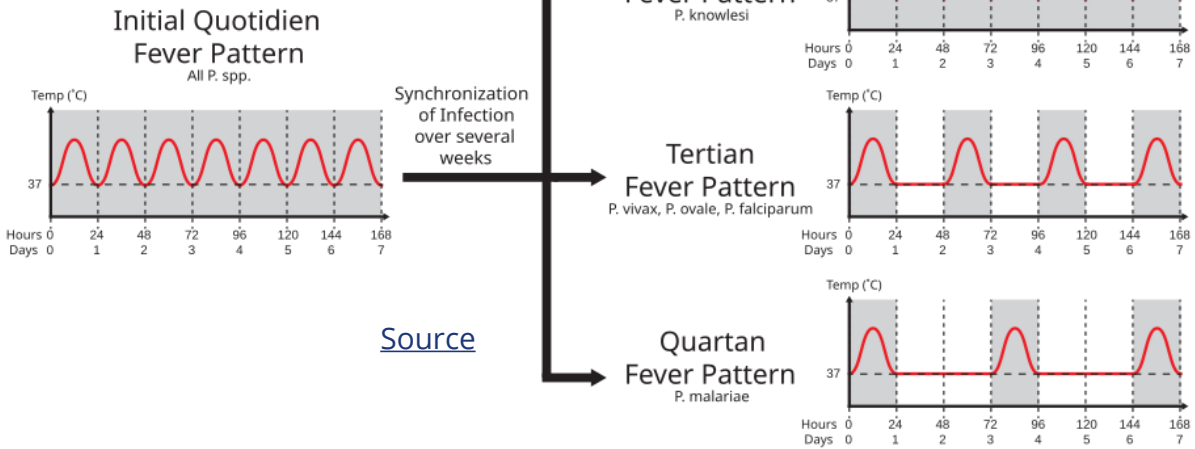


In the blood ("erythrocytic" cycle)

Rupture of RBCs (6) accounts for much of symptoms

- Can have a paroxysmal, **cyclical fever** pattern

Cyclical fevers take weeks to establish, no not a reliable clue

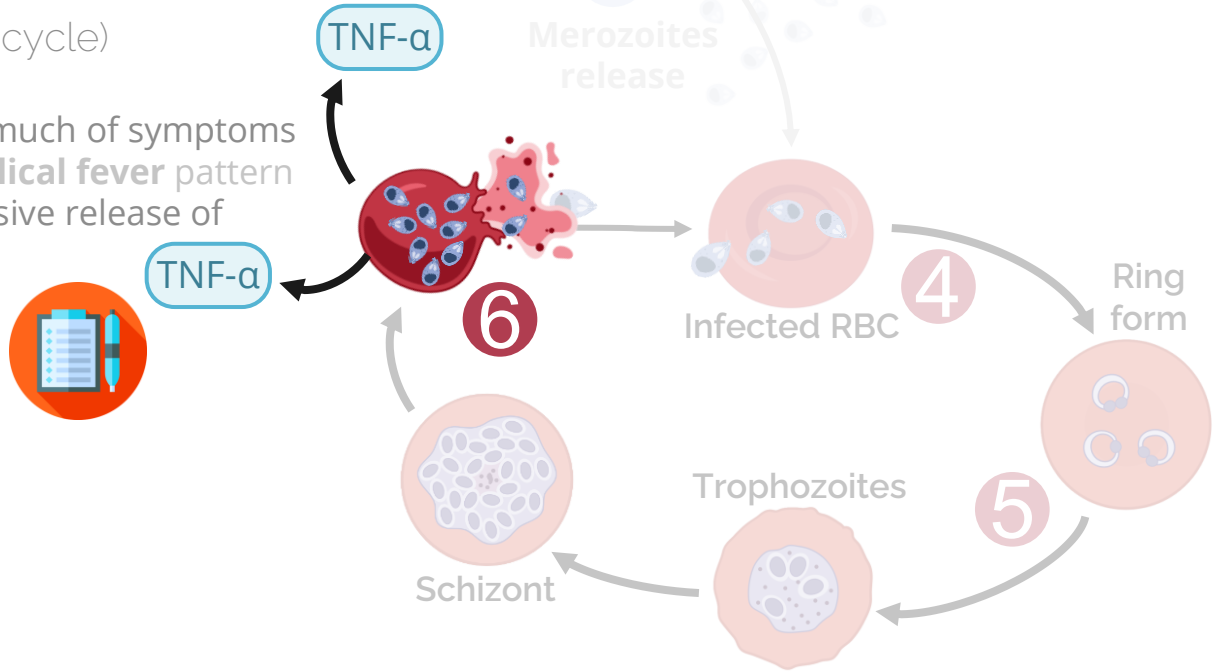


Source

In the blood ("erythrocytic" cycle)

Rupture of RBCs 6 accounts for much of symptoms

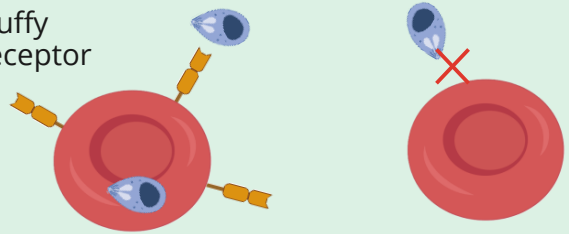
- Can have a paroxysmal, **cyclical fever** pattern
  - **Fevers** driven by massive release of **cytokines like TNF-α**
- Hemolysis → **anemia**, ↑ **bili**



Pathogenesis: In the blood ("erythrocytic" cycle)

Rupture of RBCs 6
- Fevers driven by massive release of cytokines like TNF-α
- Hemolysis → **anemia**, ↑ **bili**

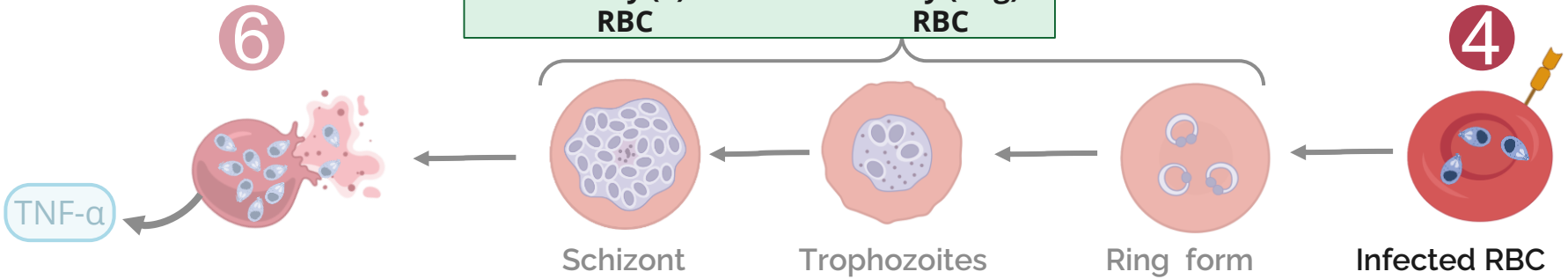
Duffy antigen & P vivax



Infected RBCs 4 go to spleen → splenic sequestration
- Splenomegaly
- ↓ Platelets
- Splenic rupture

P vivax uses the **duffy antigen** to **enter reticulocytes**

Duffy neg = ↓↓ rates of P vivax



Pathogenesis: In the blood ("erythrocytic" cycle)

Rupture of RBCs 6
- Fevers driven by massive release of cytokines like TNF-α
- Hemolysis → **anemia**, ↑ **bili**

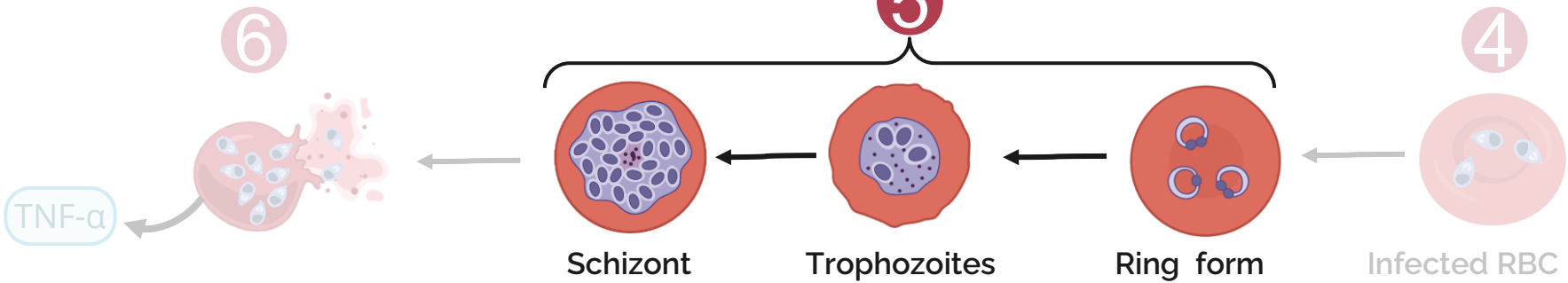
Severe malaria isn't from hemolysis *alone* 6, it can can also be from the **asexual replication** 5

Anaerobic glycolysis by the parasite:
- ↓ **glucose**
- **Lactic acidosis**

P falciparum avoids splenic sequestration by **cytoadherence** to the **microvasculature**

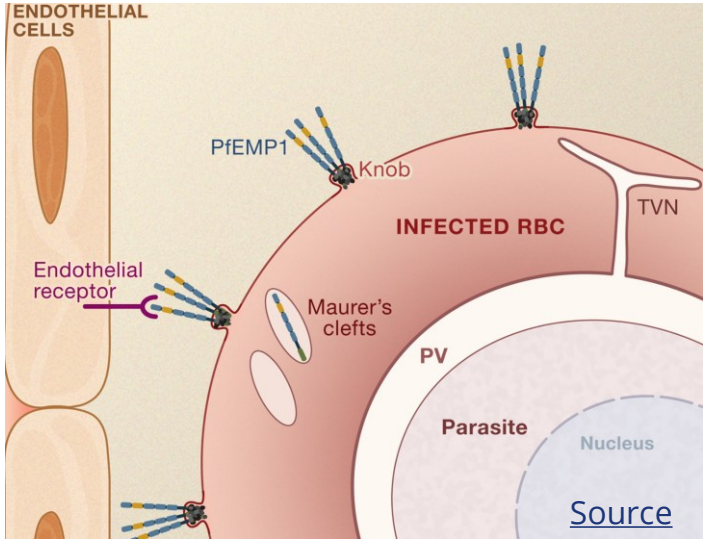
Infected RBCs 4 go to spleen → **splenic sequestration**
- **Splenomegaly**
- ↓ **Platelets**
- Splenic rupture

Duffy neg = ↓↓ rates of P vivax



P falciparum: **Microvascular cytoadherence**

P falciparum evades immune system (splenic sequestration) by **cytoadherence** to the **microvasculature**

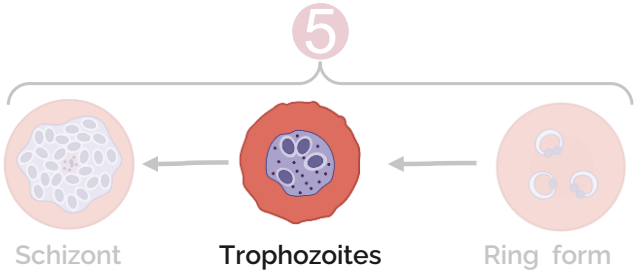


Source

P. falciparum's **trophozoites** produce a **polymorphic protein** that is expressed on infected RBCs membranes

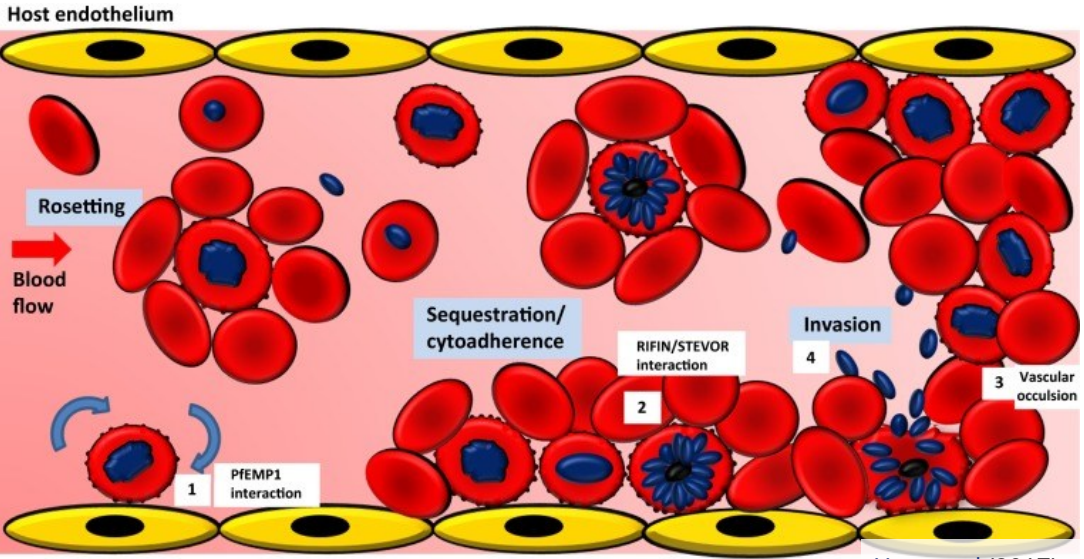
- **PfEMP1**: Plasmodium falciparum erythrocyte membrane protein 1
- **Anchors RBCs in the periphery** by binding to endothelial receptors in the microvasculature (e.g. ICAM-1)
  - Almost mimics leukocyte adhesion (but with RBCs)
- These PfEMP1 **antigens change** substantially **each cycle** 4-6
  - **Immune system evaded** by **antigenic variation**

Severe malaria isn't from hemolysis *alone* 6, it can can also be from the asexual replication 5



P falciparum: **Pathophys of severe malaria**

P falciparum evades immune system (splenic sequestration) by **cytoadherence** to the **microvasculature**

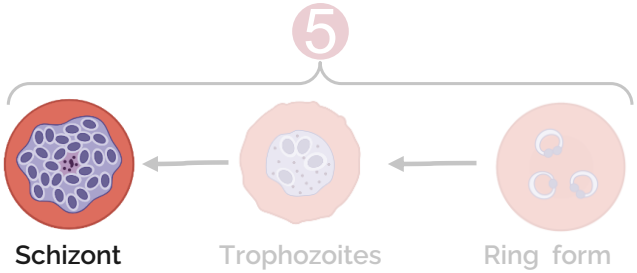


Yam et al (2017) Trends in Parasitology

Infected schizonts **bind to uninfected RBCs** causing **rosetting** (big clumps of RBCs)

Clinical manifestations vary based on the site (of the microvasculature):

- Brain - **neuro Sx**, **cerebral malaria**
- Kidneys - **Renal failure**
- Bone marrow - **cytopenias**
- Lungs - **respiratory failure**
- Liver, heart - end organ failure



Revisiting: Clinical & laboratory manifestations

Typical presentation

- **Fever and chills**
  - Often accompanied by headache, myalgias, arthralgias, fatigue/weakness
  - "Flu-like illness"
- Variable nausea, vomiting, and diarrhea

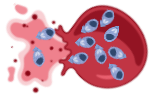
TNF-α

Physical exam

- Pallor (from anemia)
- Jaundice (when severe)
- No lymphadenopathy
- Splenomegaly

Labs

- Anemia
- ↑ bilirubin
- Normal/low WBC
- Thrombocytopenia



RBC lysis

Severe malaria (often *P falciparum*)

- Altered mental status, seizures, or coma
- Hypoglycemia
- Acute kidney injury
- Pulmonary edema or acute respiratory distress
- Severe anemia, jaundice
- Shock, or other multiorgan dysfunction

Microvasculature cytoadherence (*P falciparum*)

Splenic sequestration of infected RBCs

Host genetics

Sickle cell

An **evolutionary trade off** from dying of malaria as a kid -vs- dying from sickle cell

- HbS **"sickles" in periphery** → ↓ parasite growth in peripheral vessels
- Also **disrupts PfEMP-1** → ↓ cytoadherence

Similar dynamic with thalassemias

The pathogen

Plasmodium

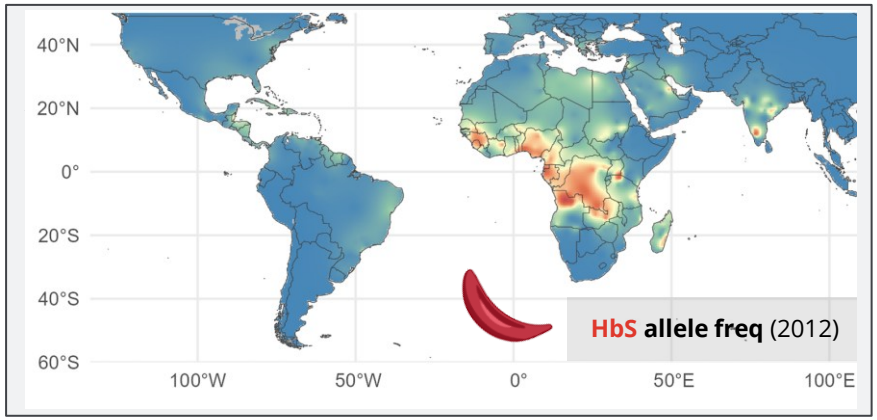
Coevolution

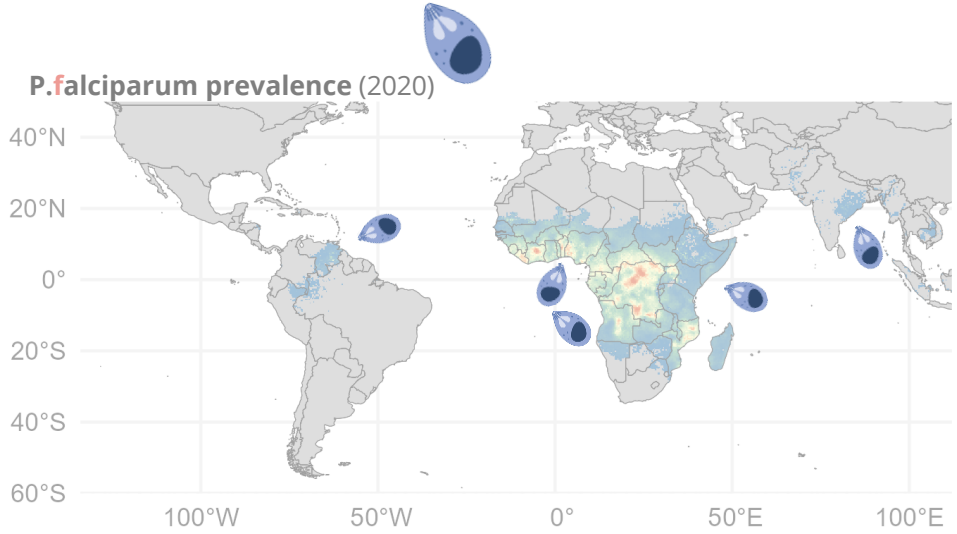
- Malaria developed **PfEMP1** to evade immune system
- What did humans do?

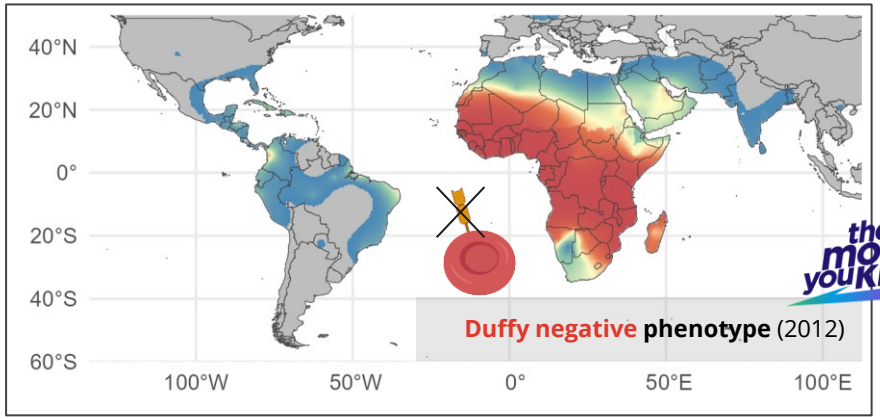
Humans

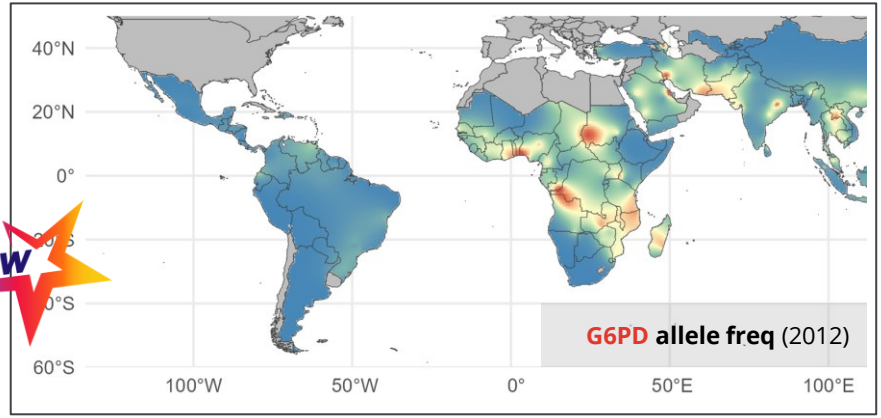
The reservoir

Host genetics: evolutionary trade offs









Case #2

Case 2

A 25M WVU student traveled **home to India** for holidays after staying in US for 3 years

Within **4 months of his return** to Morgantown, he developed **fever** & **chills**

He was seen in ED, where he was found to have:

- Low **WBC** of **3.5** (lower limit normal: 3.7)
- Low **platelet** count of **90** (LLN: 150)
- **Hemoglobin** had **dropped to 11g**
- **Splenomegaly**

He was told he could have leukemia

1. What **additional history** would you like?
2. What **additional laboratory** data would you obtain?

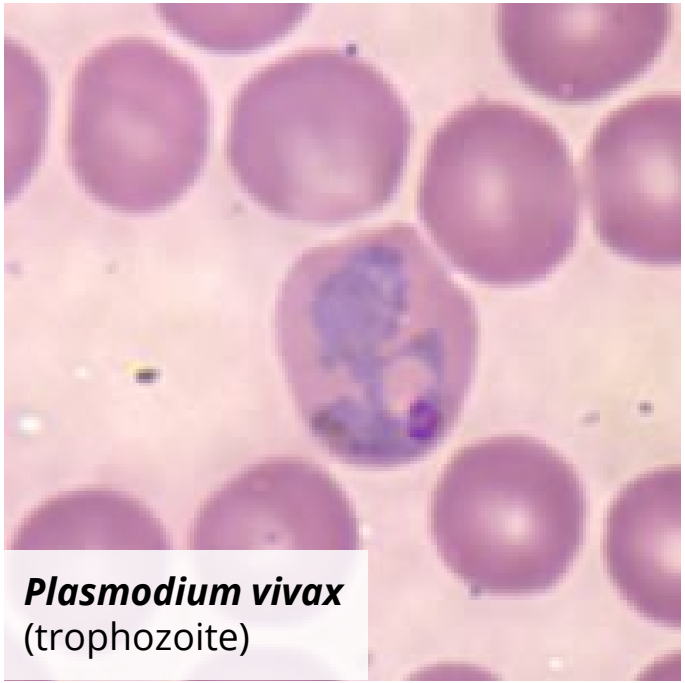
Most likely way this patient acquired his infection?

Mentimeter

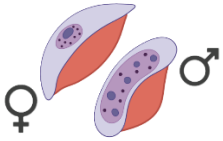
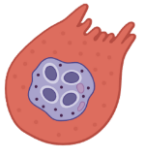
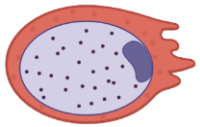
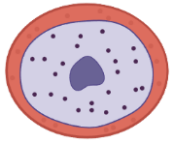
1. Bite of an infected female Anopheles mosquito
2. Tick bite
3. Blood transfusion with infected RBCs
4. Reactivation of dormant hepatic P falciparum
5. Relapse from dormant hepatic P vivax or P ovale

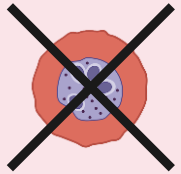
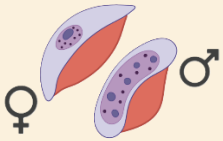
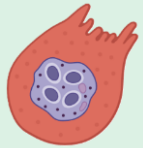
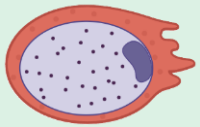
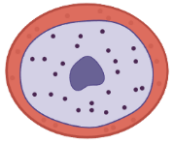
Giemsa stains

Plasmodium ovale
(trophozoite)

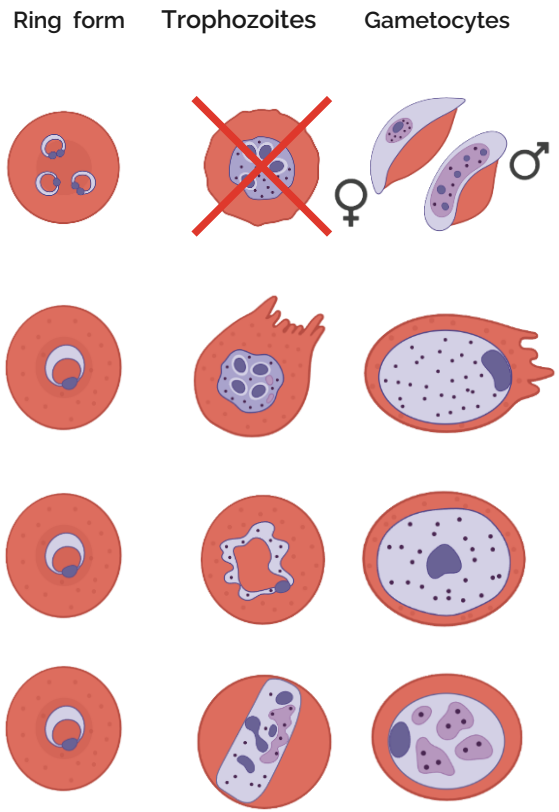


Plasmodium vivax
(trophozoite)

	RBC size	***Ring form*** *Rings per RBC*	***Trophozoite***	***Gametocyte***
P falciparum	Normal			
P ovale	Enlarged			
P vivax	Enlarged			
P malariae	Normal			

	RBC size	***Ring form*** *Rings per RBC*	***Trophozoite***	***Gametocyte***
P falciparum	Normal			
P ovale	Enlarged			
P vivax	Enlarged			
P malariae	Normal			

	RBC size	***Ring form*** *Rings per RBC*	***Trophozoite***	***Gametocyte***
P falciparum	Normal	Can be **multiple**	Frequently **not seen on smear** (sequestered in the periphery)	**Elongated** “banana shaped”
P ovale	Enlarged	Single ring	**Ragged edges** More oval shaped*	**Ragged edges** More oval shaped*
P vivax	Enlarged	Single ring	Ameboid shaped * Can also be oval shaped	* Can also be oval shaped
P malariae	Normal	Single ring	Band shaped	



What do I need to know about each species?

	P Falciparum	*P vivax & ovale*	*P Malariae*
Which RBCs?	Infect RBC's of all ages	Only young RBC's (reticulocytes)	Only **M**ature RBC's
Severity			
Peripheral sequestration?	Yes (part of why is **F**atal in up to 30% of cases)	No (less deadly)	
Immune factors	Severe disease **if no immunity** (children, pregnancy, travel)	**Hypnozoites** → Late relapses 	**Low grade** infection (can **persist >40 years**) → immune complex → glomerulonephritis
Geography	All of them **are in the tropics** (with great overlap) so <u>do not rule out</u> based on geography alone		
Slight preferences	Sub-Saharan Africa	Everywhere else	

What if he didn't travel anywhere internationally?

What other ways can malaria (and its domestic mimics) be transmitted?

Mentimeter

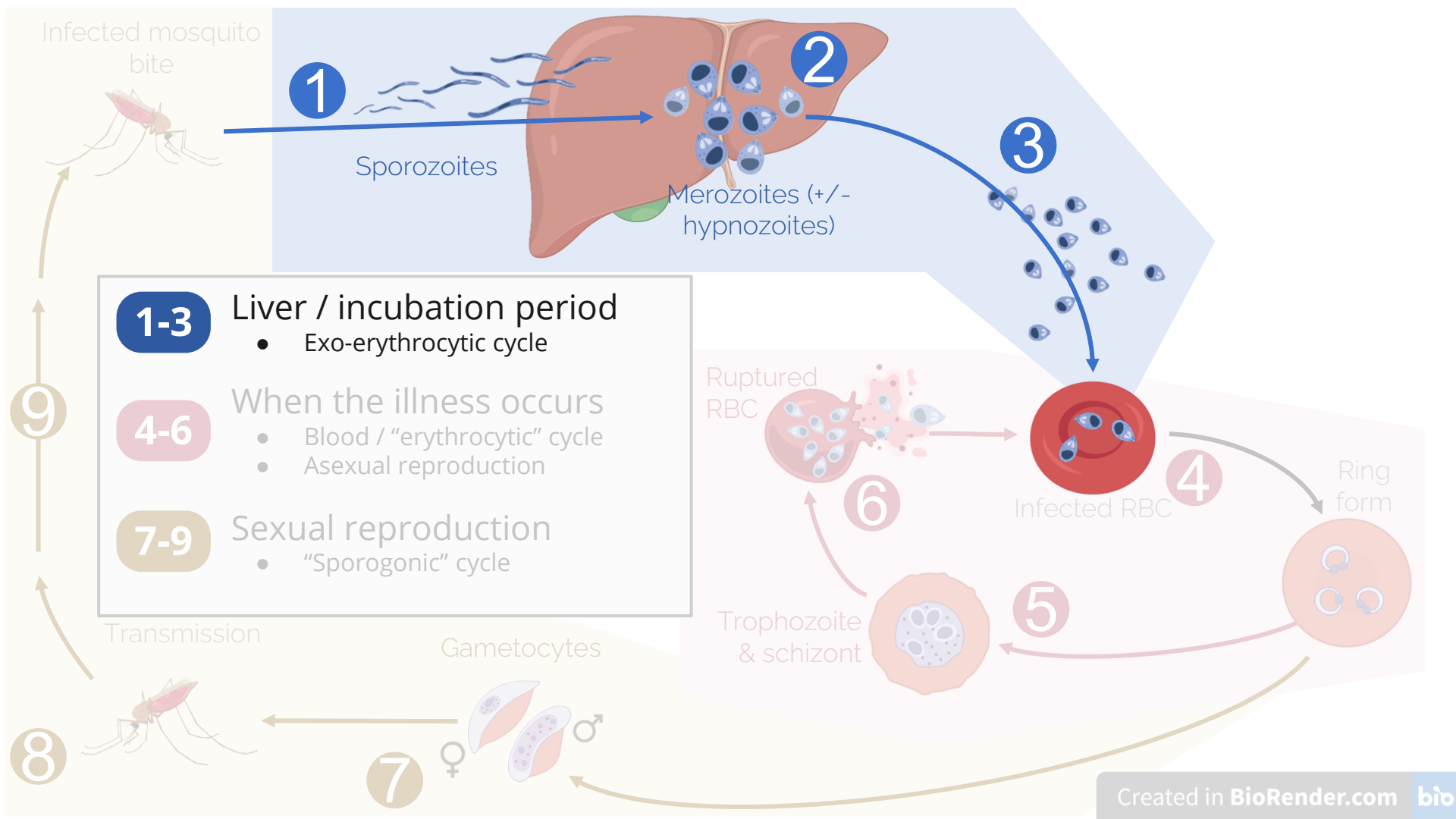
1. Bite from Anopheles mosquito in Florida
2. Ixodes tick bite
3. Drinking unpurified water from a river
4. Blood transfusion with infected RBCs
5. Sexual exposure
6. Domestic air travel

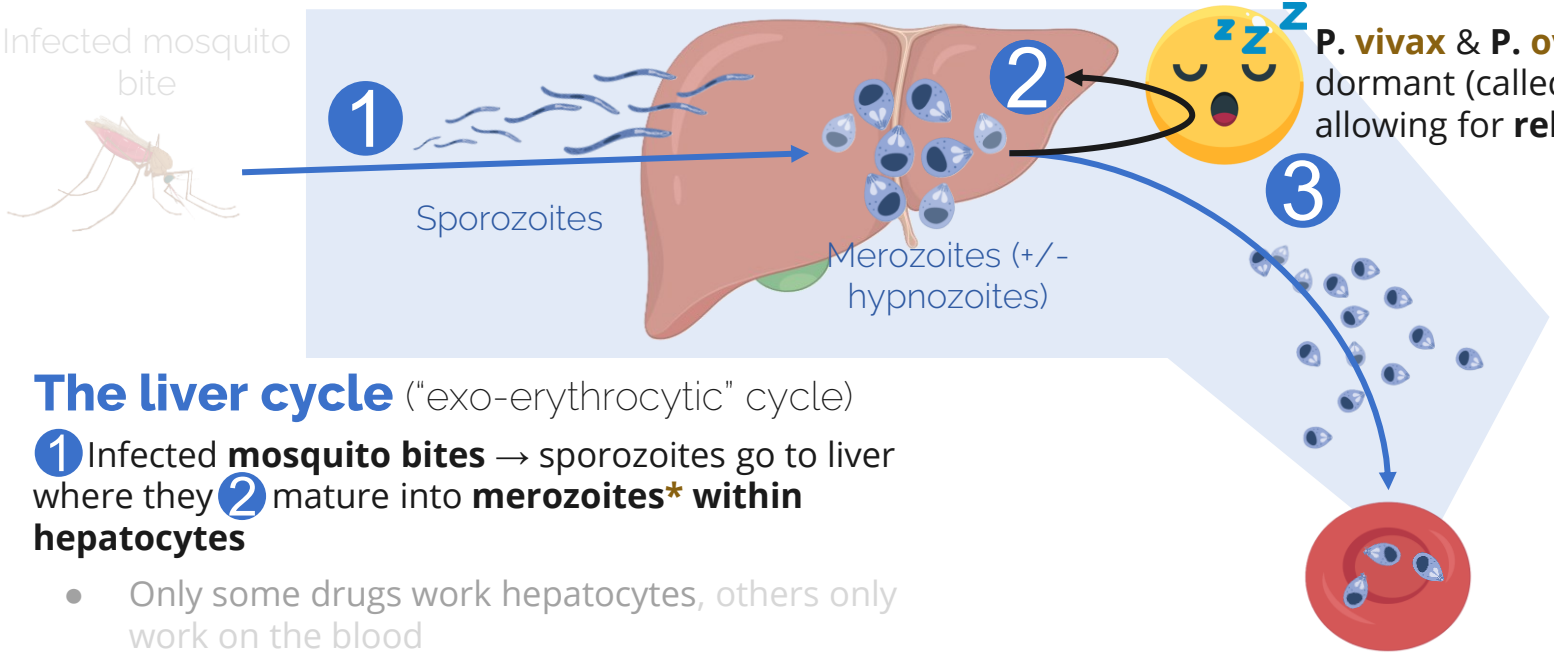
Aside: Malaria & Babesia are very similar

	Malaria (*Plasmodium* spp)	**Babesiosis** (*Babesia microti*)
Pathophys	Infected RBC → hemolysis	
Transmitted by	**Anopheles mosquito** spp	**Ixodes tick** (just like Lyme)
Geography	**Tropics**: Sub-Saharan Africa, Oceana, Central America, S/SE Asia	**USA:** Northeast (inc **WV**), west coast, great lakes
Diagnosis	Blood smear (thick & thin) showing pathogens in RBCs	
Clinical presentation	Mild: flu-like illness (fevers/chills/myalgia), can be asymptomatic Moderate: Hemolytic anemia (↓ Hgb, ↑ bili), ↓ Plts, splenomegaly Severe: end organ damage (kidney, lungs, brain)	
Who is at greatest risk?	1. Children 2. Pregnancy 3. Travelers	1. Asplenia 2. Immunocompromise

How life cycle informs treatment

Not a joke about pharmacology


Infected mosquito bite
1
Sporozoites
2
Merozoites (+/- hypnozoites)
3
1-3
Liver / incubation period
Exo-erythrocytic cycle
4-6
When the illness occurs
Blood / "erythrocytic" cycle
Asexual reproduction
7-9
Sexual reproduction
"Sporogonic" cycle
Ruptured RBC
6
Infected RBC
4
Ring form
Trophozoite & schizont
5
Gametocytes
♀
♂
7
8
Transmission
9
Created in BioRender.com



The liver cycle ("exo-erythrocytic" cycle)

1 Infected **mosquito bites** → sporozoites go to liver where they 2 mature into **merozoites* within hepatocytes**

- Only some drugs work hepatocytes, others only work on the blood

* *P vivax* & *P ovale* can **hibernate** in hepatocytes (**hypnozoites** = "sleeper cell")

- Can cause **relapse** weeks (sometimes years) later
- Must use specific drugs to kill hypnozoites (primaquine)

3 Merozoites are **released into the blood**

- It is only at this point that symptoms develop (incubation period: 1-4 wk)

Treatment algorithm

Severe disease?

Yes → **IV ARTesunate**

No → **Uncomplicated malaria**
generally use combination therapy

Improves

Treat the blood first
ARTemether–lumefantrine
- Prefered due to ↓ resistance
- "Broad spectrum" including for *P. Falciparum*

Dormant liver hypnozoites?
Only Vivax/Ovale?

Yes → **G6PD testing first!!**

→ Finish with **primaquine** to kill hypnozoites

Nope (e.g. *P. falciparum*)

Nothing else is needed!

Alternate agents
- Other **ATCs**
- **Atovaquone-Proguanil**
- Chloroquine only if from a chloroquine sensitive area (or *P vivax*)

ATC (artemisinin combo therapy)
- Combination meds that start with "**ART**"
- **ART**emether + lumefantrine
- **ART**esunate + mefloquine

Prophylaxis choices

Medication	Pro	Con	Pregnancy?
Atovaquone-Proguanil (Malarone)	Tolerated well	More expensive (for long trips)	✓
Doxycycline	Cheap	Photosensitivity (sunscreen)	✗
Mefloquine	Weekly	Neuropsychiatric issues Need to start ≥2 weeks before	✓
Chloroquine	Weekly	Only useful in chloroquine-sensitive areas	✓

Recap

Learning Objectives

1. Recognize the importance of considering **Malaria** in any **febrile illness** in a **returning traveler**
2. Understand **life cycle** and **pathogenesis**
3. Identify drugs used for **prevention and treatment**

Clinical & laboratory manifestations

TNF-α

Typical presentation

- **Fever and chills**
  - Often accompanied by headache, myalgias, arthralgias, fatigue/weakness
  - "Flu like illness"
- Variable: nausea, vomiting, and diarrhea

Severe malaria (often *P falciparum*)

- Altered mental status, seizures, or coma
- Hypoglycemia
- Acute kidney injury
- Pulmonary edema or acute respiratory distress
- Severe anemia, jaundice
- Shock, or other multiorgan dysfunction

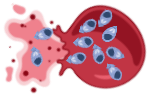
Physical exam

- Pallor (from anemia)
- Jaundice (when severe)
- No lymphadenopathy
- Splenomegaly

Labs

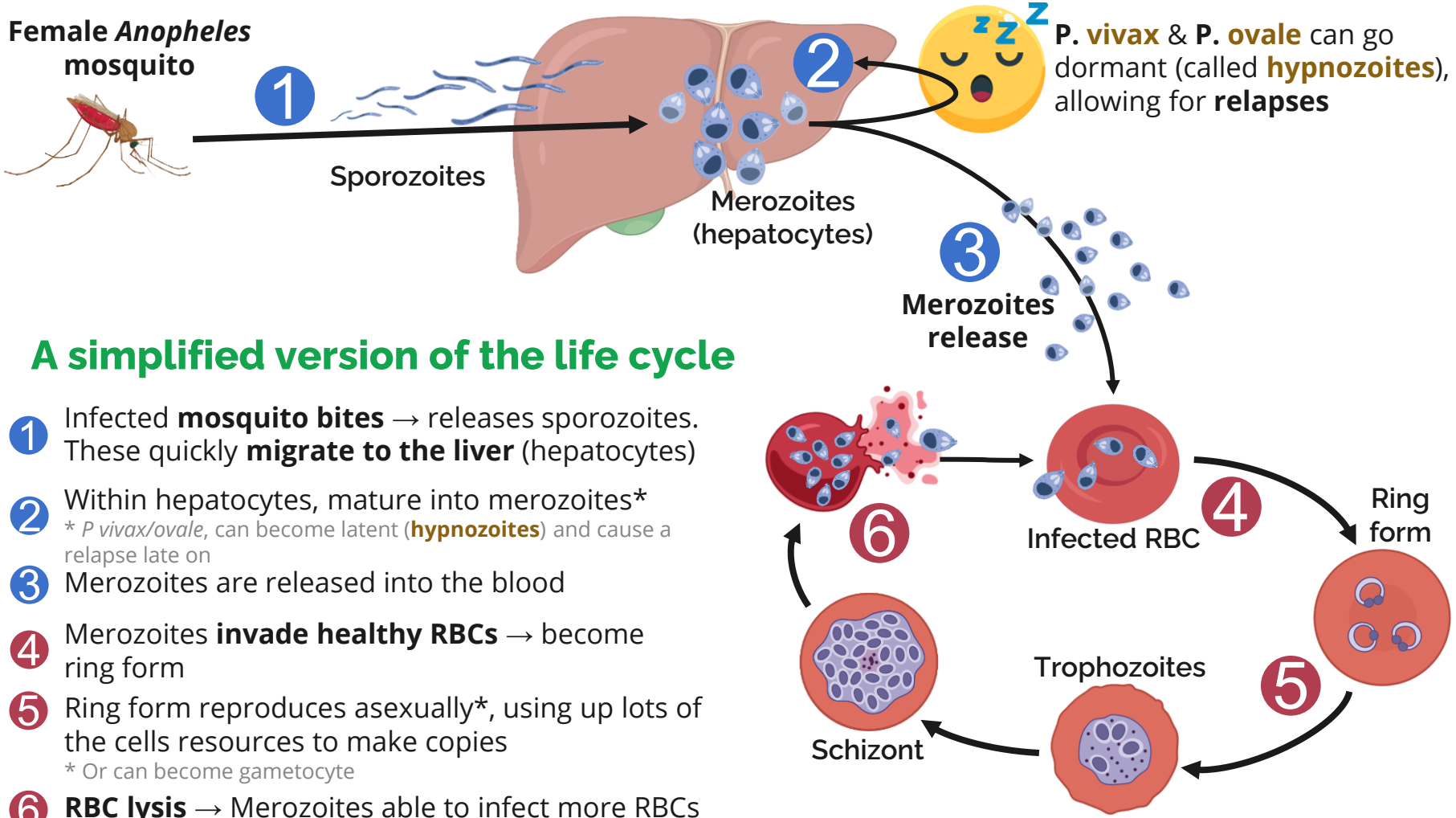
- Anemia
- ↑ bilirubin
- Normal/low WBC
- Thrombocytopenia

RBC lysis



Microvasculature cytoadherence (*P falciparum*)

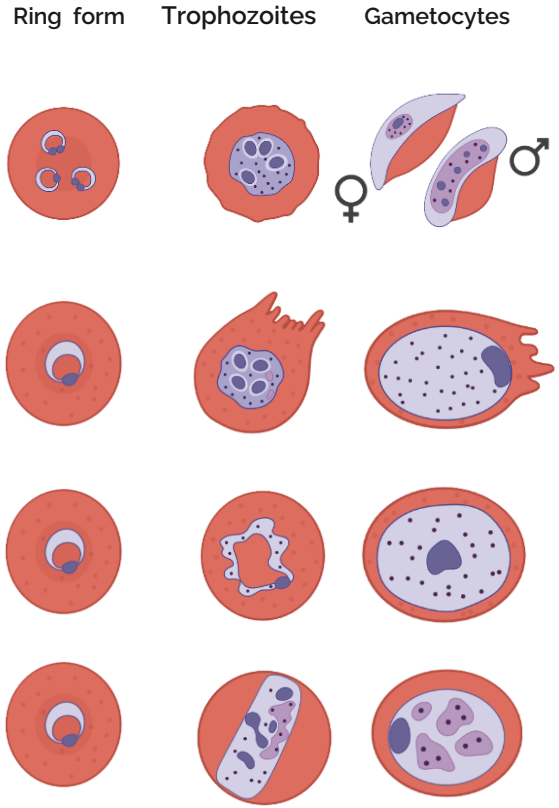
Splenic sequestration of infected RBCs



A simplified version of the life cycle

1. Infected **mosquito bites** → releases sporozoites. These quickly **migrate to the liver** (hepatocytes)
2. Within hepatocytes, mature into merozoites*
   * *P vivax/ovale*, can become latent (**hypnozoites**) and cause a relapse late on
3. Merozoites are released into the blood
4. Merozoites **invade healthy RBCs** → become ring form
5. Ring form reproduces asexually*, using up lots of the cells resources to make copies
   * Or can become gametocyte
6. **RBC lysis** → Merozoites able to infect more RBCs

	RBC size	Ring form *Rings per RBC*	Trophozoite	Gametocyte
P falciparum	Normal	Can be **multiple**	Frequently **not seen on smear** (sequestered in the periphery)	**Elongated** "banana shaped"
P ovale	Enlarged	Single ring	**Ragged edges** More oval shaped*	**Ragged edges** More oval shaped*
P vivax	Enlarged	Single ring	Ameboid shaped * Can also be oval shaped	* Can also be oval shaped
P malariae	Normal	Single ring	Band shaped	



What do I need to know about each species?

	P Falciparum	*P vivax & ovale*	*P Malariae*
Which RBCs?	Infect RBC's of all ages	Only young RBC's (reticulocytes)	Only **M**ature RBC's
Severity			
Peripheral sequestration?	Yes (part of why is **F**atal in up to 30% of cases)	No (less deadly)	
Immune factors	Severe disease **if no immunity** (children, pregnancy, travel)	**Hypnozoites** → Late relapses 	**Low grade** infection (can **persist >40 years**) → immune complex → glomerulonephritis
Geography	All of them **are in the tropics** (with great overlap) so do not rule out based on geography alone		
Slight preferences	Sub-Saharan Africa	Everywhere else	

Treatment algorithm

Severe disease?

Yes → **IV ARTesunate**

No → **Uncomplicated malaria**
generally use combination therapy

Improves

Treat the blood first
ARTemether–lumefantrine
- Prefered due to ↓ resistance
- "Broad spectrum" including for *P. Falciparum*

Dormant liver hypnozoites?
Only Vivax/Ovale?

Yes → **G6PD testing first!!**

→ Finish with **primaquine** to kill hypnozoites

Nope (e.g. *P. falciparum*)

Nothing else is needed!

Alternate agents
- Other **ATCs**
- **Atovaquone-Proguanil**
- Chloroquine only if from a chloroquine sensitive area (or *P vivax*)

ATC (artemisinin combo therapy)
- Combination meds that start with "**ART**"
- **ART**emether + lumefantrine
- **ART**esunate + mefloquine

Prophylaxis choices

Medication	Pro	Con	Pregnancy?
Atovaquone-Proguanil (Malarone)	Tolerated well	More expensive (for long trips)	✓
Doxycycline	Cheap	Photosensitivity (sunscreen)	✗
Mefloquine	Weekly	Neuropsychiatric issues Need to start ≥2 weeks before	✓
Chloroquine	Weekly	Only useful in chloroquine-sensitive areas	✓