وزارة التعليم العالي والبحث العلمي
الجامعة التقنية الشمالية
المعهد التقني الطبي كركوك

الحقيبة التعليمية

القسم العلمي: تقنيات التمريض

اسم المقرر:
علم الأحياء المجهريه
Microbiology

المرحلة / المستوى:الأول

الفصل الدراسي: الاول

السنة الدراسية:2024-2025

معلومات عامه

اسم المقرر:	**علم الأحياء المجهريه**						
القسم:	**قسم تقنيات صحة المجتمع**						
المعهد:	**المعهد التقني الطبي كركوك**						
المرحلة / المستوى	**الاول**						
الفصل الدراسي:	**الثاني**						
عدد الساعات الاسبوعية:	نظري	2	عملي	2			
عدد الوحدات الدراسية:	4						
الرمز:	CHT115						
نوع المادة	نظري		عملي		كلهما		نعم
هل يتوفر نظير للمقرر في الاقسام الاخرى	**لايوجد**						
اسم المقرر النظير	**لايوجد**						
القسم	**لايوجد**						
رمز المقرر النظير							

معلومات تدريسي المادة

اسم مدرس (مدرسي) المقرر:	**ا.م.د. سوزان عادل رشيد**
اللقب العلمي:	**استاذ مساعد**
سنة الحصول على اللقب	2021
الشهادة :	**دكتوراه**
سنة الحصول على الشهادة	**2017**
عدد سنوات الخبرة (تدريس)	**25 سنه**

الوصف العام للمقرر

يشمل مقرر الأحياء المجهريه تقسيم الكائنات الحية المجهرية اعتمادا على تركيبها الخلوي الى البكتريا، الطحالب ،الفيروسات، الفطريات والطفيليات وطرق تغذيتها و تصبيغها لتشخيصها ودورة حياتها و يتيح المقرر للطالب التعرف على عوامل الضراوة التي تمكن الكائن المجهري من احداث المرض والأمراض التي تسببها.

الاهداف العامة

- سيتعلم الطالب ماهي الأحياء المجهريه وتقسيمها والتركيب الخلوي لكل منها .
- سيتعلم الطالب الأنواع البكتيريه الممرضه والمقارنه بين الايجابيه لصبغة كرام والسالبه للصبغة ومعنى الذوائف الزنجاريه والعقديه القيحيه وغيرها .
- سيتعلم الطالب تعريف الفايروسات وطرق تضاعفه وانتشاره وامراضيته وأنواعها وتركيبها .
- سيتعلم الطالب معنى الطفيليات و أنواعها وطرق انتقالها والأمراض التي تسببها.

الأهداف الخاصة

يهدف هذا المقرر الى اكساب الطلبة المعلومات المرتبطة بعلم الأحياء المجهريه وطرق تقسيمها والتعرف على الامراض التي تسببها وطرق انتقالها والتي تشمل :

- سيكون الطالب قادر على تعريف البكتريا وتركيبها وتشخيصها والمقارنة بين الأنواع الموجبة والسالبة لصبغة كرام وعوامل الضراوة ولإمراضيه لبعض الأنواع البكتيرية.
- سيكون الطالب قادر على تشخيص بعض الأنواع البكتيرية والفطريات الطبية من خلال تنميتها على الأوساط الزرعيه المختلفة .
- سيكون الطالب قادرا على القيام بالفحوصات المختلفة لتشخيص الفايروسات وتحديد أنواعها والسيطرة على انتشارها.
- سيكون الطالب قادر على تشخيص الطفيليات والديدان ودورة حياتها .

أمثلة الأهداف التعليمية .

إكساب المتعلم مهارات القراءة.

إلمام المتعلم بأنواع الأحياء المجهرية وتقسيمها وطرق تشخيصها وإمراضيتها.

الأهداف السلوكية او نواتج التعلم

بعد الانتهاء من الدرس (المحاضرة) سيكون الطالب قادرا على ان:

- يعرف تركيب البكتريا الموجبة والسالبة لصبغه كرام.
- يعرف بعض الأمراض الناتجة عن الاصابات البكتيرية
- يعرف تشخيص البكتريا .
- يعرف المقارنة بين بدائية النواة وحقيقية النواة.

المتطلبات السابقة

- يجب ان يسجل في مقررات الفصل الدراسي الأول .

الأهداف السلوكية او مخرجات التعليم الأساسية		
ت	تفصيل الهدف السلوكي او مخرج التعليم	آلية التقييم
1	أن يعرف الطالب تقسيم الأحياء المجهريهوتركيب البكتريا والمقارنه بين بدائية النواة وحقيقية النواة.	الاختبارات والامتحانات ،التقارير ،النقاشات، الملاحظة.
2	أن يتعرف الطالب على الأنواع البكتيريه ومقاومتها للظروف البيئيه والظروف التي تؤثر على النمو البكتيري و أطوار النمو.	الاختبارات والامتحانات ،التقارير ،النقاشات، الملاحظة.
3	أن يستعرض الطالب طرق تثبيط النمو البكتيري والتعقيم وماهي طرق التعقيم المتبعه مختبريا	الاختبارات والامتحانات ،التقارير ،النقاشات، الملاحظة.
4	ان يتعرف الطالب على أنواع البكتريا الطبيه و امراضيتها وطرق معالجتها.	الاختبارات والامتحانات ،التقارير ،النقاشات، الملاحظة.
		الاختبارات والامتحانات ،التقارير ،النقاشات، الملاحظة.
5	أن يستعرض الطالب أنواع بكتريا النايسيريا والكلوستريديا وخصائصها العامهوامراضيتها والوقايه منها.	الاختبارات والامتحانات ،التقارير ،النقاشات، الملاحظة.
6	ان يتعلم الطالب دراسة الأمراض التي تسببها مجموعه من البكتريا الطبيه والممرضه والأمراض التي تسببها ومعالجتها والوقايه منها	الاختبارات والامتحانات ،التقارير ،النقاشات، الملاحظة.
7	ان يتعرف الطالب على أنواع البكتريا الطبيه الاخرى وعلاقتها في التسمم الغذاءي وتشخيصها ووصفها ليكون من السهل دراستها كما في بكتريا الشيكلا.	الاختبارات والامتحانات ،التقارير ،النقاشات، الملاحظة.
8	أن يتعرف الطالب على ضمات الكوليرا والتريبونيما وخصاءصها والأمراض التي تسببها والاختبارات التشخيصيه والمعالجه	الاختبارات والامتحانات ،التقارير ،النقاشات، الملاحظة.

9	التعرف على تركيب الفطريات وانواعها وطرق تشخيصها والأمراض الناتجه عنها ومقومتها للمضادات الفطريات وعلاقة الاصابه الفطريه بالحاله المناعيه للأفراد.	الاختبارات والامتحانات ،التقارير ،النقاشات، الملاحظة.
10	التعرف على الطفيليات وانواع العلاقات التطفلي وعلاقة الطفيلي بالمضيف ودورات الحياة والأمراض الناشئة عن الطفيليات وضرورة التعرف على المسبب المرضي الحقيقي سواء بكتريا أم طفيلي .	الاختبارات والامتحانات ،التقارير ،النقاشات، الملاحظة.
11	التعرف على الجياردیا المعويه وتشخيصها وعلاجها وتشخيص مرض البلانتديوم كولاي ودورة الحياة.	الاختبارات والامتحانات ،التقارير ،النقاشات، الملاحظة.
12	يستعرض الطاالب أنواع الديدان المعويه(الدبوسيه، دودة الاسكارس والتعرف على أشكالها سواء كانت بيضه أو ذكر أو انثى) وخطورتها والأمراض الناشئه عنها ودورة حياتها ومعالجتها .	الاختبارات والامتحانات ،التقارير ،النقاشات، الملاحظة.
13	التعرف على الديدان الشريطيه ودورة حياتها ومعالجتها	الاختبارات والامتحانات ،التقارير ،النقاشات، الملاحظة.
14	التعرف على شكل البلهارزيا وشكلها (الذكر ، الانثى، البيضه) ودورة حياتها والتشخيص والعلاج.	الاختبارات والامتحانات ،التقارير ،النقاشات، الملاحظة.
15	التعرف على الأمراض الناشئه عن الاصابه بالليشمانيا المداريه والأنواع الأخرى وشكلها وتشخيصها وعلاجها.	الاختبارات والامتحانات ،التقارير ،النقاشات، الملاحظة.

أساليب التدريس (حدد مجموعة متنوعة من أساليب التدريس لتناسب احتياجات الطلاب ومحتوى المقرر)

الاسلوب او الطريقة	مبررات الاختيار
1.التنظيم التقليدي(المحاضرات مباشرا)	وذلك لتنظيم الوقت وسهولة التنفيذ والوضوح وللسيطرة على الفصل الدراسي والحفاظ على النظام والتركيز
2.التعليم التعاوني	وذلك لتعزيز مهارات التواصل وتنمية مهارات العمل التعاوني وزيادة الدافعية والتحفيز
3.الاستقصاء	وذلك لتنمية مهارات البحث والتحليل وتطوير التعلم الذاتي والاستقلاليه
4.المناقشة والحوار	وذلك لتطوير مهارات التفكير النقدي وتعميق الفهم ويعزز الابتكار والابداع

الفصل الاول من المحتوى العلمي							
عنوان الفصل	الوقت						
التوزيع الزمني	النظري	العملي	العنوان الرئيسي	العنوان الفرعي	طريقة التدريس	التقنيات	طرق القياس
الأسبوع الأول	2ساعه	2ساعه	تقسيم الأحياء المجهريه	البكتريا	محاضرة	عرض تقديمي، شرح، أسئلة وأجوبة, مناقشة	الاختبارات القصيره ، الاسئلة والمناقشة وتحليل الردود
				الفطريات			
الأسبوع الثاني	2ساعه	2ساعه		الطحالب			
				الفيروسات			
				الديدان			
الاسبوع الثالث	2ساعه	2ساعه	الأبواغ البكتيريه	أنواع الأبواغ	محاضرة	عرض تقديمي، شرح، أسئلة وأجوبة, مناقشة	الاختبارات القصيره ، الاسئلة والمناقشة وتحليل الردود
				المقاومة للظروف البيئيه			
				أطوار النمو			
			تثبيط النمو البكتيري	طرق التعقيم	محاضرة	عرض تقديمي، شرح، أسئلة وأجوبة, مناقشة	لاختبارات القصيره ، الاسئلة والمناقشة وتحليل الردود
				أنواع المعقمات والمطهرات			
				التعقيم بالأشعه			
				التعقيم بالترشيح			

الفصل الثاني							
عنوان الفصل	الوقت						
التوزيع الزمني	النظري	العملي	العنوان الفرعي		طريقة التدريس	التقنيات	طرق القياس
	2 ساعة	2 ساعه	2 ساعة	العناوين الفرعية	محاضرة	عرض تقديمي، شرح، أسئلة وأجوبة, مناقشة	الاختبارات القصيره ، الاسئلة والمناقشة وتحليل الردود
الأسبوع الرابع	2 ساعه	2 ساعه	االبكتريا الطبيه		محاضرة	عرض تقديمي، شرح، أسئلة وأجوبة, مناقشة	الاختبارات القصيره ، الاسئلة والمناقشة وتحليل الردود
				الذائفه الزنجاريه			
				السموم			
				المكورات العنقوديه			
				الامراضيه والمعالجه			
الأسبوع الخامس	2 ساعه	2 ساعه	مجموعة النايسيريا	الخصائص العامه	محاضرة	عرض تقديمي، شرح، أسئلة وأجوبة, مناقشة	الاختبارات القصيره ، الاسئلة والمناقشة وتحليل الردود
				التشخيص والمعالجه			
				البكتريا اللاهوائيه			
الاسبوع السادس	2ساعه	2ساعه	الكلوستريديا الغازيه	الامراضيه ووصف العلاج	محاضرة	عرض تقديمي، شرح، أسئلة وأجوبة, مناقشة	الاختبارات القصيره ، الاسئلة والمناقشة وتحليل الردود
				السموم البكتيريه			
				التسمم الغذائي			
				الأعراض			
				العلاج			

الفصل الثالث							
عنوان الفصل	الوقت						
التوزيع الزمني	نظري	عملي	العنوان الرئيسي	العنوان الفرعي	طريقة التدريس	التقنيات	طرق القياس
الأسبوع السابع	2ساعه	2ساعه	أنواع البكتريا المعوية	الأهميه الطبيه	محاضرة	عرض تقديمي، شرح، أسئلة وأجوبة, مناقشة	الاختبارات القصيره ، الاسئلة والمناقشة وتحليل الردود
				تحديد الأنواع البكتريا المعوية			
				مجموعة السالمونيلا والشكيلا			
				التسمم الغذائي			
				التشخيص والعلاج			
				التشخيص والعلاج			
الاسبوع الثامن	ساعه2	2ساعه	الكوليرا	الخصائص العامة للمرض الناتج عنها	محاضرة	عرض تقديمي، شرح، أسئلة وأجوبة, مناقشة	الاختبارات القصيره ، الاسئلة والمناقشة وتحليل الردود
				التريبونيما			
				التشخيص والعلاج			
الاسبوع التاسع	2ساعه	2ساعه	علم الفطريات	تشخيص الفطريات	محاضرة	عرض تقديمي، شرح، أسئلة وأجوبة, مناقشة	الاختبارات القصيره ، الاسئلة والمناقشة وتحليل الردود
				تصنيف الفطريات			
				الأمراض التي تسببها الفطريات			
				العلاقة بين الاصابات الفطريه والحاله المناعية			

الفصل الرابع (من المحتوى العلمي)							
عنوان الفصل	الوقت						
التوزيع الزمني	نظري	عملي	العناوين الرئيسية	العناوين الفرعية	طريقة التدريس	التقنيات	طرق القياس
الأسبوع العاشر	2ساعه	2ساعه			محاضرة	عرض تقديمي، شرح، أسئلة وأجوبة, مناقشة	الاختبارات القصيره ، الاسئلة والمناقشة وتحليل الردود
	2ساعه	2 ساعه	الطفليات	تعريف الطفيليات	محاضرة	عرض تقديمي، شرح، أسئلة وأجوبة, مناقشة	الاختبارات القصيره ، الاسئلة والمناقشة وتحليل الردود
				العلاقات الطفيليه			
				العلاقه مع المضيف دورة حياتها			
				الزحار الأميبي			
				الأميبا القولونيه			
الاسبوع الحادي عشر	2 ساعه	2 ساعه	الأسواط المعويه والأهداب المعويه	شكل الأسواط	محاضرة	عرض تقديمي، شرح، أسئلة وأجوبة, مناقشة	الاختبارات القصيره ، الاسئلة والمناقشة وتحليل الردود

			تشخيص الأسواط				
			دورة الحياة				
			دورة حياة الدوده الدبوسيه والاسكارس	الديدان المعويه	محاضرة	عرض تقديمي، شرح، أسئلة وأجوبة, مناقشة	الاختبارات القصيره ، الاسئلة والمناقشة وتحليل الردود
			المقارنه بين الذكر والانثى		محاضرة	عرض تقديمي، شرح، أسئلة وأجوبة, مناقشة	الاختبارات القصيره ، الاسئلة والمناقشة وتحليل الردود
			دورة الحياة				
الاسبوع الثاني عشر	2 ساعه	2 ساعه	الأمراض والعلاج				

الفصل الرابع (من المحتوى العلمي)						
عنوان الفصل	الوقت					
التوزيع الزمني	نظري	عملي			طريقة التدريس	التقنيات	طرق القياس
الأسبوع الثالث عشر	2	2	العناوين الرئيسية	العناوين الفرعية	محاضرة		
			الدوده الشريطيه	دورة الحياة	محاضرة	عرض تقديمي، شرح، أسئلة وأجوبة, مناقشة	الاختبارات القصيره ،
				الامراضيه			

			التشخيص العلاج				الاسئلة والمناقشة وتحليل الردود
الاسبوع الرابع عشر	2	2	دورة الحياة الامراضيه التشخيص العلاج	شيستوسوما البلازموديوم	محاضرة	عرض تقديمي، شرح، أسئلة وأجوبة, مناقشة	الاختبارات القصيره ، الاسئلة والمناقشة وتحليل الردود
	2	2	دورة الحياة	الليشمانيا	محاضرة	عرض تقديمي، شرح، أسئلة وأجوبة, مناقشة	الاختبارات القصيره ، الاسئلة والمناقشة وتحليل الردود
الاسبوع الخامس عشر			الامراضيه التشخيص العلاج				

المحتوى العلمي

خارطة القياس المعتمدة

المحتوى التعليمي	عناوين الفصول	الأهمية النسبية	الأهداف السلوكية					عدد الفقرات
			المعرفة		الفهم	التطبيق	التحليل	التقييم	
			النسبة	%20	%25	%20	%15	%20	
الفصل الاول	تقسيم الأحياء المهريه	%13	1	1	1	1	1	1	5
الفصل الثاني	نمو الأحياء المجهريه	%20	2		3	2	1	2	10
الفصل الثالث	البكتريا الطبيه	%20	2		2	2	1	2	5
الفصل الرابع	علم الفطريات	%20	2		2	2	1	2	10
الفصل الخامس	الديدان المعويه	%20	1		2	1	1	1	10
الفصل السادس	الليشمانيا	%17	2		2	2	2	2	10
المجموع		%100							**50**

المحتويات (لكل فصل في المقرر)

رقم المحاضرة: الأولى والثانيه	
عنوان المحاضرة:	علم الأحياء المجهريه
اســـم المدرس:	أ.م. د. سوزان عادل
الفئة المستهدفة :	طلاب المستوى الثاني
الهدف العام من المحاضرة :	
الأهداف السلوكية او مخرجات التعلم:	1- التعرف على الخلية البكتيريه 2- التعرف على الاختلافات بين البكتريا بدائية النواة وحقيقية النواة 3- التعرف على الظروف الملائمة لتنمية البكتريا
استراتيجيات التيسير المستخدمة	عرض تقديمي ، شرح ، الصبوره
المهارات المكتسبة	مهارات التعرف على تركيب البكتريا و أنواعها
طرق القياس المعتمدة	الاختبارات التحريرية ،االأسئلة والمناقشة ، تحليل الردود، المشاركه

الاسئلة القبلية

Q1: define: Prokaryote, cell membrane, microbiology.

Microbiology

Medical Microbiology deals with microorganisms, such as bacteria, viruses, fungi and parasites, which are of medical importance and are capable of causing diseases in human beings. It deals with etiology, pathogenesis, laboratory diagnosis, treatment, epidemiology and control of infection.

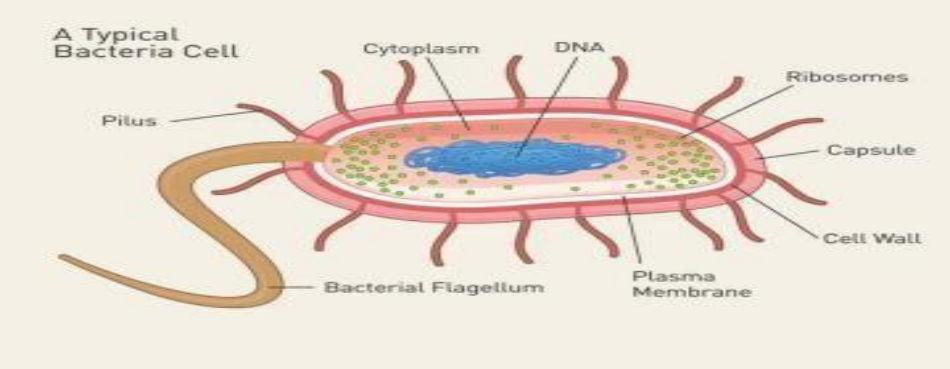
Medical Microbiology includes six sciences:-

1. Parasitology: deals with parasites causing diseases in human.
2. Mycology: deals with fungus causing diseases in human.
3. Bacteriology: deals with bacteria.

4. Immunology: includes mechanism involved in the development of resistance by body to infectious diseases.
5. Genetics: is the study of heredity and variations.
6. Virology: is the study of viruses.

Definition of Bacteria:

Bacteria are unicellular free living organisms, without chlorophyll, having both RNA and DNA, capable of performing all essential processes of life growth, metabolism and reproduction. They have rigid cell wall containing muramic acid.



The Typical bacterial cell

Comparison between Eukaryotic and Prokaryotic cells

Characters	Prokaryotes	Eukaryotes
Examples	Bacteria, green, algae	Fungi, protozoa, Slime moulds
Membrane-bound organelles	Absent	Present
Nucleus Nuclear membrane Nucleolus Chromosome Mitotic division	 Absent Absent One Absent	 Present Present More than one Present
Cytoplasm Mitochondria Golgi apparatus Endoplasmic reticulum	 Absent Absent Absent	 Present Present Present

Chemical composition Steroil Muramic acid	Absent present	Present Absent

Examples of bacteria:- According to the shape, bacteria are classified as:-

1. Cocci (spherical shape) due to arrangement:
   a. Cocci in cluster ---------- *Staphylococci*
   b. Cocci in chain ---------- *Streptococci*
   c. Cocci in pair ---------- *Diplococci*
2. Bacilli (rod or cylindrical shape)
3. Vibrio (comma shaped)
4. Spirochetes (several coils and flexible)
5. Actinomycetes (branching filamentous bacteria)
6. Mycoplasma (Do not possess a stable morphology, due to lack cell wall).

Appears rounded or oval bodies with interlacing filaments.

General structure of bacteria :-The general structure of bacteria includes

Bacteria (singular-bacterium) are prokaryotes, and have a simple cell structure consisting of:
1- containing the bacterial chromosome, ribosomes, stored energy inclusions, and often plasmids.
2-Cytoplasmic membrane and mesosomes
3-Cell wall (except bacteria with deficient cell walls such as Mycoplasma)
4-External structures such as capsule, fimbriae (pili), and flagella, depending on the species.

Flagella: These are long, sinuous, contractile filamentous appendages known as flagella. They are organ of locomotion. They are antigenic and composed of protein.

-Pili (fimbriae): Are filamentous, short, thin, straight, hair like.
(0.5 *M* long less than 10nm. thick).

Function:

1. Organ of adhesion.
2. Conjugation tube through which genetic material is transmitted from donor to recipient cell.

3. They are antigenic.

5-Some bacteria such as Bacillus and Clostridium also produce endospores in unfavorable environmental conditions.

6- Ribosome: These are ribonucleoprotein granules. They are sites of protein synthesis, measuring (100-200) Angstrom unit.

7-Mesosome: Sites of respiratory enzymes in bacteria.

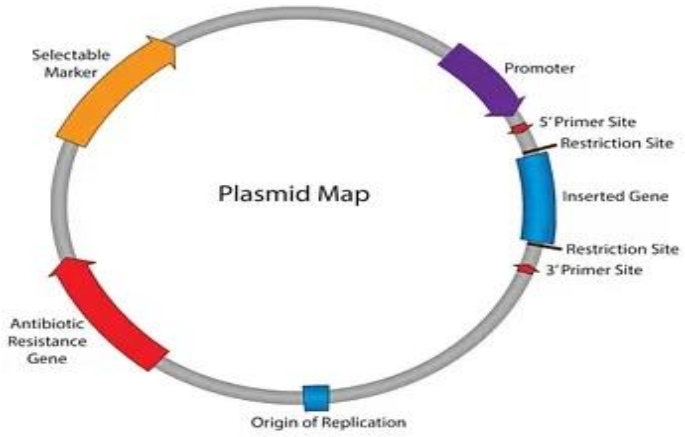
8-Cytoplasmic membrane: It is thin semi permeable membrane which lies just beneath the cell wall.

Function:1- Controls in flow and out flow of metabolites to and from protoplast.

2-Presence specific enzyme (permease) plays important role in passage through membrane.

Although most prokaryotes cannot survive in nature without their cell walls, some do so naturally. These include the mycoplasmas, a group of pathogenic bacteria that causes several infectious diseases of humans and other animals, and the Thermoplasma group, species of Archaea that naturally lack cell walls. These bacteria are able to survive without cell walls because they either contain unusually tough cytoplasmic membranes or because they live in osmotically protected habitats such as the animal body.

Plasmids are extra-chromosomal genetic elements that replicate independently of the host chromosome. They are small, circular (some are linear), double-stranded DNA molecules (mostly) that exist in bacterial cells and in some eukaryotes. The sizes of plasmids range from roughly one to more than 1000 kilobase pairs.

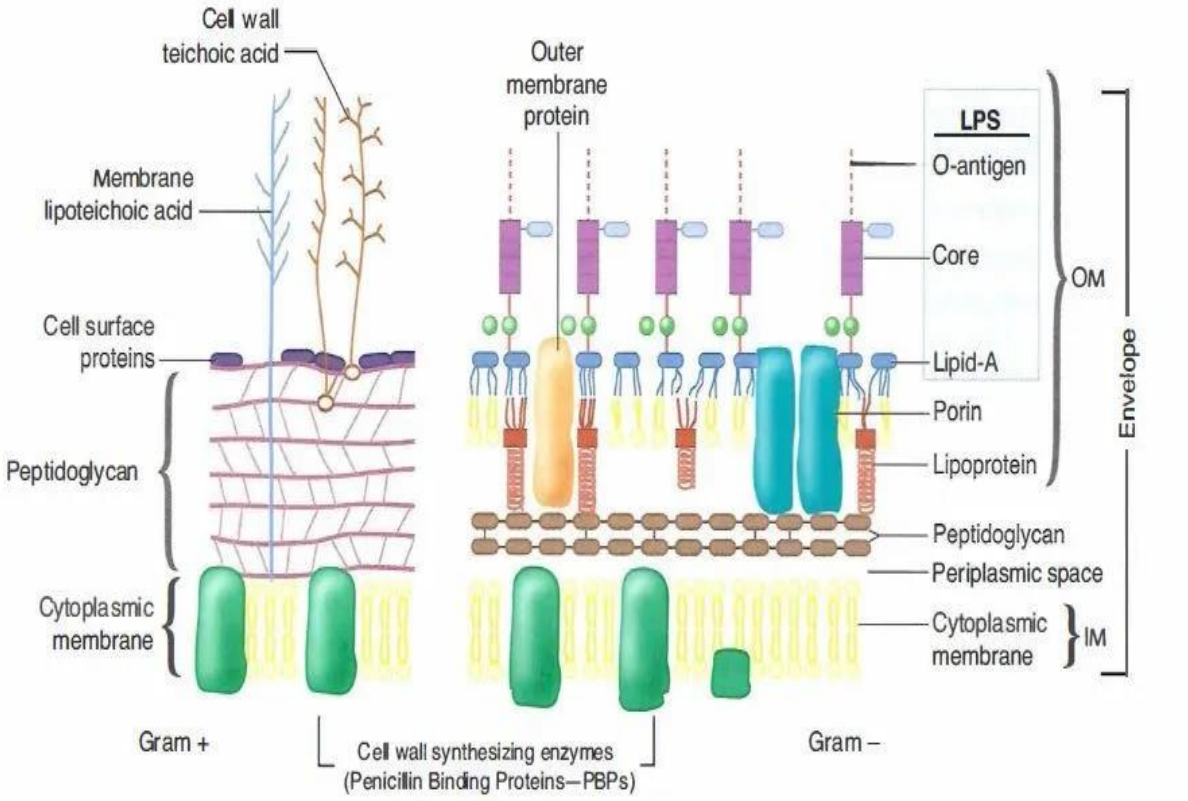


The number of plasmids may vary from none to several per bacterial cell. Different plasmids are present in a cell in a particular number called the copy number. Some plasmids are present in the bacterial cell in only 1-3 copies, whereas others may present in as many as

100 copies. This feature is controlled by the genes present on the plasmid and by interactions between the host and the plasmid.
Some important components of plasmids are

Gram-Positive vs. Gram-Negative Bacteria

The cell wall of Gram-negative bacteria is more complex than those of Gram-positive bacteria. Gram-negative bacteria contain an extra layer of cells called outer membrane or lipopolysaccharide (LPS) layer which surrounds the thin peptidoglycan layer. LPS layer is absent in Gram-positive bacteria.



Major differences between Gram-positive and Gram-negative bacteria

Properties	Gram Positive Bacteria	Gram Negative Bacteria
Thickness of cellwall	Thicker than Gram negative bacteria. around 20 to 25 nm	Generally thinner, 11 to 15 nm
Gram reaction	Gram positive bacteria stain a deep blue color (violet/purple) in Gram staining technique	Gram negative bacteria stain pink to red color in Gram staining technique.
Lipopolysaccharide (LPS) layer	Absent	Only present in Gram negative bacteria.
Peptidoglycan layer	Thick (multilayered) peptidoglycan layer is present in Gram positive bacteria.	Thin (single-layered).
Teichoic acids	Cell wall of gram positive bacteria bacteria contains teichoic acids.	Absent
Periplasmic space	Periplasmic space is single and smaller in Gram positive bacteria	There are two periplasmic space in Gram negative bacteria;
Flagellar structure	2 rings in basal body	4 rings in basal body
Toxins produced	Primarily exotoxins	Primarily endotoxins, LPS layer has a endotoxic property.
Examples	*Clostridium spp*, *Bacillus* spp, ***Listeria monocytogenes***, *Staphylococcus*, *Streptococcus*, etc.	***Escherichia coli, Klebsiella pneumoniae, Pseudomonas aeruginosa***, ***Neisseria gonorrhoeae***, ***Vibrio cholerae.***

Environmental conditions of bacterial growth

In order to have a sufficient bacterial growth, the following environmental conditions are required:

1. Temperature: divided into 3 groups:
   a.Psychrophilic: (0–25˚C) mostly soil and water bacteria.

   a- Mesophilic: (20–45˚C) includes bacteria producing disease.

   b- Thermophilic: (50–60˚C) Bacillus, Algae.

2. Oxygen: Bacteria are classified into groups due to oxygen requirement:
   a. Aerobes: Grow only in presence of oxygen, e.g. *Bacillus*, *Pseudomonas*.
   b. Facultative anaerobes: Can live with or without oxygen .e.g. *E. coli*, *Salmonella*, *Staphylococcus*, *Shigella*.
   c. Obligate anaerobes: Multiply only in the absence of oxygen . e.g. *Clostridium*, *Bacteroides*.

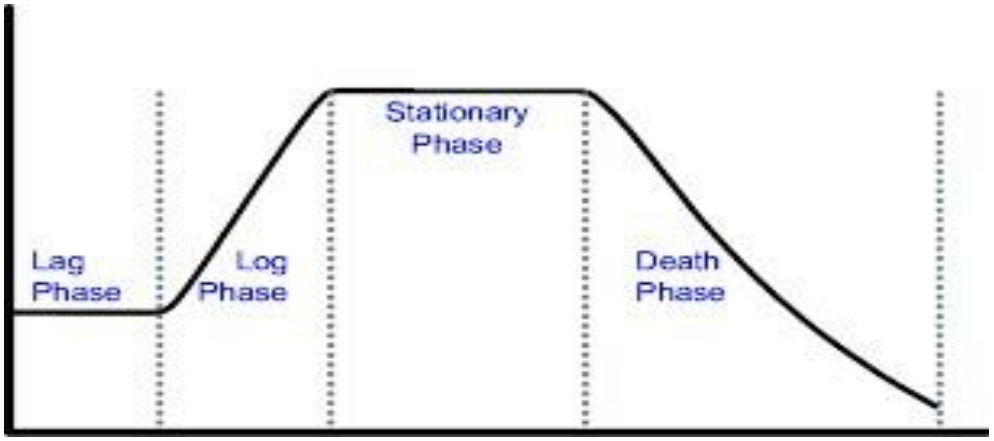
1. Carbon dioxide: Some types of bacteria need CO_2 for metabolic activities, e.g. *Neisseria gonorrhoeae*.
2. Moisture: Bacteria requires water for growth, desiccation may kill most of bacteria.
3. pH: Optimum pH for the bacterial growth ranged between,
   e.g. Vibrio grow at alkaline pH, but Lactobacilli grow at acidic pH.

Stages of bacterial growth

The bacterial growth curve represents the number of live cells in a bacterial population over a period of time. There are four distinct phases of the bacterial growth, i.e., lag, exponential, stationary and death or decline phase. The initial phase is the lag phase, where bacteria are metabolically active but not dividing. After that, the exponential phase begins, which is time of exponential growth.

In the stationary phase, growth reaches a plateau, as the number of dying cells equals the number of dividing cells. Finally, the death phase is characterized by an exponential

decrease in the number of living cells, as the population growth begins to decline, due to depletion of nutrients and accumulation of waste products.



The bacterial growth curves

Generation time

The time required for bacterium to produce two daughter cells under optimum condition is called generation time. For example, the generation time of *E. coli* is 20 minutes.

Bacterial growth inhibition by sterilization and disinfection

Sterilization: Is the process by which all forms of life (bacteria, fungi, viruses….etc) killed or eliminated.

Various agents used in sterilization are:

A: Physical

1- Sun light. 2- Drying. 3- Filtration 4- Radiation

5- Heat. This divided into dry heat and moist heat.

6- Ultrasonic vibration.

B: Chemical .

1- Acid 2- Alkalis. 3- Salts. 4- Halogens. 5- Oxidizing agent.

6- Reducing agents. 7- Formaldehyde. 8- Phenol.

9- Soap. 10- Dyes. 11- Aerosol

Physical methods

1- Sun light: This is one of the natural methods of sterilization in case of water in tanks, river and lakes, due to ultraviolet rays.

2- Drying: Drying in air has a serious effect on many spores

3- Heat: The factors influencing sterilization by heat are

1) Type of heat: a- Dry. b- Moist.

2) Temperature and time.

3) Number of organisms present.

4) Whether organism has sporing capacity.

Types of heat :-

Dry heat

a. Red heat: It is used to sterilize metallic object by holding them in flame till they are red hot e.g. inoculating wires, needles, scalpels, and forceps.

b. Flaming: The subject is passed over flame without allowing it to become red hot, e.g. cotton wool plugs and glass slides.

c. Incineration: It is an excellent method for rapidly destroying material

e.g. soiled dressing, animal's carcasses, bedding and pathological material.

d. Hot air oven: Sterilization by hot air oven requires temperature of 160° C for one hour for sterilizing glass, syringes, Petri dishes, test tubes.

Moist heat

A) Temperature blow 100° C

Pasteurization of milk: Temperature employed is either 63˚C for 30 minutes (Holder method) or 72˚ C for 15-20 seconds (Flash method) to kill organisms like *Mycobacterium*, *Salmonella* and *Brucella*.

B) Temperature at 100˚C

Tynddallization: This is the process by which medium is exposed to steam at 100˚C for 30 minutes each on 3 successive days.

Boiling: Most of vegetative form of bacteria fungi and viruses are killed at 50-70˚C in short time.

C) Steam at atmosphere pressure (100˚C): Free steam is used to sterilize culture media which may decompose if subjected to higher temperature. Steam under pressure: For bacteriological and surgical work boiling is not sufficient because spore can survive hence high pressure sterilizer or autoclave is used.

4- Filtration: This method of sterilization is useful for antibiotic solutions and serum.

5- Radiation: Ultraviolet radiation is a chief bactericidal factor present in sun light, as it causes following changes in the bacterial cell:

1- Denaturation of protein.

2- Damage of DNA.

Chemical methods

1- Acids and alkalis: Are inhibitory agents for the bacterial growth,
e.g., *Mycobacteria* are more resistant to acid than alkalis.

2- Distilled water: causes loss of viability, which could be due to traces of metal in distilled water.

3- Oxidizing agents: Are weak antiseptic e.g H_2O_2 and potassium permanganate.

4- Halogens: Iodine is used for skin, chlorine combines with water to form hypochloric acid, which is bactericidal.

5- Formaldehyde: 5-10% solution in water kills many bacteria. It is bactericidal sporicidal and lethal to viruses also.

6- Phenol: It is used for sterilizing surgical instruments and for killing culture accidentally spread in the laboratory.

7- Soap and detergents: Are bactericidal and bacteriostatic for gram positive organisms.

8- Alcohol: Ethyl alcohol is most effective in 70% solution than 100% alcohol fort killing spores.

Disinfection: Refers to the elimination of all pathogenic organisms that cause infection. There are two major methods:

1. Chemical method: by using chemical agents such as, chlorine, ozone, halogens bromide, iodine, phenol, ethanol, formaldehyde and hydrogen peroxide.
2. Physical method: includes, UV (ultraviolet) light, gamma-ray irradiation, sonification and heat.

الأسئلة البعديه :

Q1: Compare between :

1-prokaryote and eukaryote.

2-sterilization and disinfection

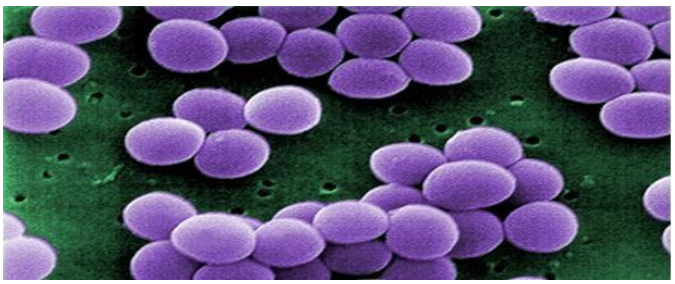
رقم المحاضرة: الثالثة والرابعة و الخامسة	
عنوان المحاضرة:	البكتريا الطبية
اســـم المدرس:	ا.م.د. سوزان عادل
الفئة المستهدفة :	طلاب المستوى الثاني
الهدف العام من المحاضرة :	
الأهداف السلوكية او مخرجات التعلم:	1-التعرف على الخصائص العامه للبكتريا الطبيه والأجناس البكتيريه 2-التعرف على الامراضيه وعوامل الضراوه 3- التعرف على دور السموم البكتيريه في احداث الاصابات
استراتيجيات التيسير المستخدمة	عرض تقديمي، الشرح ، الصبوره
المهارات المكتسبة	مهارات التعلم على تشخيص الأنواع البكتيريه الممرضه، مهارات الوقايه ،العرض والتقديم
طرق القياس المعتمدة	الاختبارات التجريبيه ، الأسئلة والمناقشة، تحليل الردود

الأسئلـه القبليه :

Q1: whats the meaning of pathogenesis.
Q2:whats the general characters of *Clostridium botulinium.*

Staphylococcus aureus

General characters: Bacteria are oval or spherical (0.8 to 0.9m) non motile, non-capsulated, non-spore forming, Gram positive, arranged in cluster (grape like). Cluster formation occurs by active aggregation of multiple cells into one location.



Virulence factors: Virulence is defined as the capacity of a pathogen (usually a micro-organism) to cause disease.

The broad range of infections caused by *Staph. aureus*, is related to a number of virulence factors that allow it to adhere to surface, invade or avoid the immune system and cause harmful toxic effects to the host.
These Factors are:

1. Adherence factor: One major class of *Staph. aureus* adhesions comprise protein that attached to the cell peptidoglycan, which in turn, attached to the plasma or extracellular matrix components.
2. Exoprotein: Nearly all strains of *Staph. aureus* secret a group of exoprotein such as, exotoxins and enzymes, including nucleases, proteases, lipases… etc. The main function of these proteins may be to convert host tissues into nutrients required for the bacterial growth.

Pathogenesis: *Staphylococci* are one of the most important causative agents of hospital-acquired infection, especially post-operative wound infection. It causes the majority of acute pyogenic lesions in human. Staphylococcal diseases are classified as:

a. Cutaneous lesions: boils, abscess, impetigo, eye infection in new born, (*pemphigus neonatorum*).
b. Deep infection: acute osteomyelitis, tonsillitis, pharyngitis, abscess breast (mastitis), *Staphylococcal septicemia*.
c. Staphylococcal food poisoning. This occurs when food (meat, fish, milk products) contaminated with enterotoxin B, which produced by staphylococci after 6 hours of consuming, leading to diarrhea and vomiting.

Toxins:

1- Haemolysin: *Staph. aureus* produces at least 3 types of hemolysin known as alfa, beta, and gamma.
2- Leukotoxins lyse white blood cells.
3- Staphylococcal enterotoxins (SEs) cause vomiting and diarrhea and the toxins are one of the most common causes of food-borne diseases.
4- Fibrinolysin: *Staph. aureus* produces staphylo-kinase during the later stage of growth, which causes lysis of fibrin.

Other toxins: (a) Nucleases (b) Lipases (c) Proteases
(d) Scarlatina toxin.

Streptococcus

spherical or oval cells about 1*M* in diameter, arranged in chains, gram positive, non-motile, non-spore forming and sometimes capsulated, *Streptococcus* can be classified into the following groups due to their hemolytic activity:

1. Beta- hemolytic: Breaks down the red blood cells and hemoglobin completely. This leaves a clear zone around the bacterial growth or colony, e.g. *Streptococcus pyogenes*.

2. Alfa- hemolytic: Partially breaks down the red blood cells and leaves a greenish color behind on blood agar plates due to oxidizing hemoglobin, e.g. *Streptococcus viridans*.
3. Gamma-hemolytic: (non-haemolytic) many streptococci do not produce any kind of haemolysis, generally commensal, e.g. *Streptococcus faecalis*.

Streptococcus includes the following species:

1- Streptococcus pyogenes *2- Streptococcus viridans*
3- Streptococcus faecalis

***Streptococcus pyogenes*:**

Virulence factors include:

1. M protein and lipoteichonic acid for attachment.
2. Hyaluronic acid capsule that inhibits phagocytosis.
3. Other extracellular products such as, pyrogenic toxin, which causes the rash of scarlet fever.

Pathogenesis

1. Respiratory infection like throat infection, tonsillitis, pharyngitis.
2. Skin infection like suppurative infection of skin e.g. wound and burn.

3. Scarlet fever is caused by a strain producing erthrogenic toxin. It is characterized by a bright red rash on the body, usually accompanied by a high fever and sore throat.
4. Genital tract infection causing puerperal sepsis.

Toxins: Erythrogenic toxins also referred to as *Strep. Pyogenic* toxins, which induces inflammation.

Streptococcus viridans:

Pathogenesis: Causes sub-Acute bacterial endocarditis, which is an infection of the heart involving damaged valves or endothelium.

Streptococcus faecalis:

Virulence factors include: The contribution of the surface protein to colonization and persistence of in urinary tract infections has been shown in an animal model.

Pathogenesis: It may cause disease in human being when introduced into the blood stream or urinary tract accidentally.

Pneumococcus

General characters: Gram positive, lancet shaped, which arranged in pairs or short chains. They are capsulated, surrounded by poly- saccharide capsule. They are non-motile and non-sporing.

Virulence factor: one virulence factor is a polysaccharide capsule that releases *pneumococci* from the host by preventing phagocytosis. Another factor is pneumolysin, which inhibits antibody synthesis.

Pathogenesis: *Diplococcus pneumoniae* cause lobar pneumonia, broncho-pneumonia, pneumococcal meningitis, otitis media, sinusitis, conjunctivitis.

Corynebacterium diphtheria

General characters: Is a Gram-positive, non-motile, aerobic, rod-shaped bacterium. Strains grow in tissues or old cultures, causing diphtheria, can be characterized as toxigenic or non-toxigenic, or those causing diphtheria and those that do not, respectively.

Virulence factors: *C. diphtheriae* has two main virulence factors, i.e., pilli and toxin that contribute to its survival in the host. They help the process of adherence in the host and the colonization of the respiratory tract to cause infection.

Pili

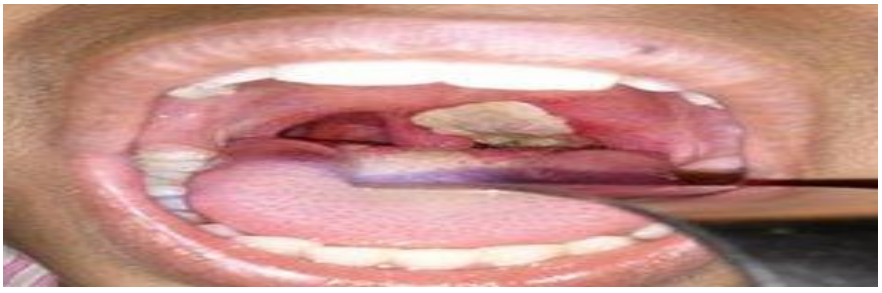
The pilli found on the surface of *C. diphtheriae* are beneficial in the adherence to host cells (pharyngeal epithelial cells, lung and laryngeal epithelial cells)

Toxin

The main virulence factor of *C. diphtheriae* is diphtheria toxin (DT), an exotoxin, released by the bacteria after entering the human body. The major function of the toxin is to enter the cytoplasm and inhibit protein synthesis in host cells.

Pathogenesis: Diphtheria can cause a thick gray coating to build up in throat or nose making it difficult to breathe and swallow. Diseased individuals may experience a sore throat, overall weakness, fever, and swollen glands. Respiratory involvement typically begins with sore throat and mild pharyngeal inflammation.

Development of a localized or coalescing pseudomembrane can occur in any portion of the respiratory tract. The pseudomembrane is characterized by the formation of a dense, gray debris layer composed of a mixture of dead cells, fibrin, RBCs, WBCs, and organisms.

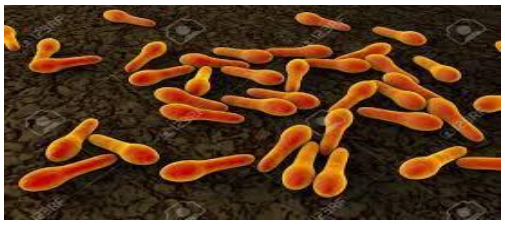


Vaccination: There are four vaccines that have been developed to treat diphtheria: are given to children under the age of seven, and are administered during adulthood. The latter two vaccines are used as boosters and are not given at the same time.

Clostridium

The genus *Clostridium* is Gram positive, anaerobic, (4-6) micron in length, spore forming, pleomorphic bacilli and spindle shape.

1- *Clostridium tetani*: Spore is terminal, oval and allocated outside the bacilli, this appearance is called drum stick.



Virulence factors: *C. tetani* grows at the wound site, it releases the toxins tetanolysin and tetanospasmin. The function of tetanolysin is unclear, although it may help *C. tetani* to establish infection within a wound. Tetanospasmin ("tetanus toxin") is responsible for the symptoms of tetanus. Tetanospasmin spreads via the lymphatic system and bloodstream throughout the body, where it is taken up into various parts of the nervous system.

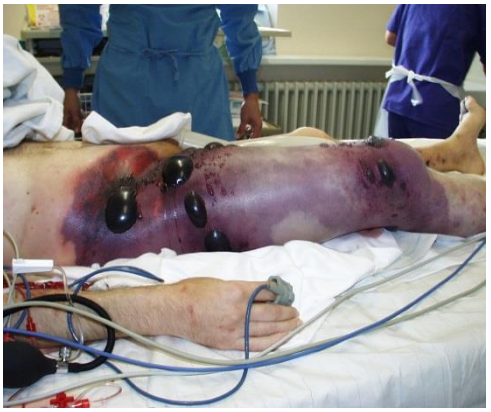
Toxins: It is an obligate anaerobic bacterium whose spores produce two distinct toxins, i.e., tetanolysin, which causes local tissue destruction and tetanospasmin that leads to causes clinical tetanus.

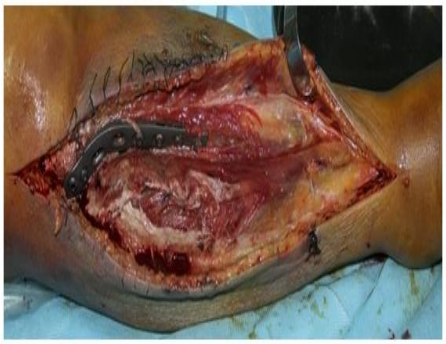
2. *Clostridium perfringens*:

General characters: Gram-positive, anaerobic bacterium that is widely distributed in the environment; it is found in soil, however, commonly inhabits are the gastrointestinal tract of humans and animals. This bacterium is a major cause of histotoxic and enteric diseases.

Toxins: Toxins are the main cause of lesions and symptoms associated with diseases caused by its infection. There are two main groups of toxins; major and minor. The major toxins are alpha, beta, epsilon and iota toxins. Theses toxins are lethal and necrotizing agents. Minor toxins such as, eta, theta, kappa and enterotoxin. Recently there are newly discovered toxins such as enterotoxin and a cytotoxic beta -2 toxin.

Pathogenesis: Toxins produced by the bacterium results in a broad range of diseases including gas gangrene, various enterotoxaemias, food poisoning and necrotic enteritis.





3. *Clostridium botulinum*:

General characters: Gram positive, obligate anaerobic, spore-forming, rod-shaped bacterium. *C. botulinum* organisms are commonly found in soils and marine sediments throughout the world. It also colonizes the gastro-intestinal tract of fishes, birds and mammals.

Virulence factors: The only virulence factor of *C. botulinum* has is its neurotoxin. It targets the peripheral nervous system and has similar functions to that of the tetanus toxin. It is secreted and absorbed into the blood stream.

Pathogenesis: includes the following:

1. Transmission: *C. botulinum* can be transmitted through home canned foods, poorly packaged preserved foods, open wounds, injections and honey products. Since spores can be dormant for several years canned foods are highly susceptible for contamination.

2. Infectious dose: incubation and colonization: One reason Botulism is so toxic is due to its low infectious dose. It only takes 75 mg of toxin to kill a 75 kg person or 1 mg/1 kg weight of an individual. Colonization can occur at the site of infection like a wound. Since the toxin is produced in the tissues the bacteria grow near blood vessels so the toxins can be absorbed easily into the blood.

Disease:

1) Foodborne botulism.
2) Wound botulism.
3) Infant Botulism.

Bacillus anthracis

General characters: It is a Gram-positive, rod-shaped bacterium, spore forming. they tend to form long chains. Most *B. anthracis* strains produce a capsule that gives colonies a slimy mucus-like appearance.

Virulence factors and toxins: Virulent forms of *B. anthracis* are two large pathogenicity related plasmids which carries the genes responsible for capsule synthesis and degradation. and the production of a toxic complex consisting of three proteins known as protective antigen (PA), lethal factor (LF), and edema factor (EF).

Pathogenesis: Transmitted through airborne exposure, direct contact and contaminated food or water. is spread by contact with the bacterium's spores, which often appear in infectious animal products. Contact is by breathing or eating or through an area of broken skin. It does not typically spread directly between people. Risk factors include people who work with animals or animal products, travelers and military personnel.

Disease:-

1- Cutaneous anthrax, also known as hide-porter's disease, is when anthrax occurs on the skin. Cutaneous anthrax presents as a boil-like skin lesion that eventually forms an ulcer with a black center (eschar).

2- Inhalation anthrax usually develops within a week after exposure, but may take up to 2 months. During the first few days of illness, most people have fever, chills, and fatigue. These symptoms may be accompanied by cough, shortness of breath, chest pain, and nausea or vomiting, making inhalation anthrax difficult to distinguish from influenza and community-acquired pneumonia.

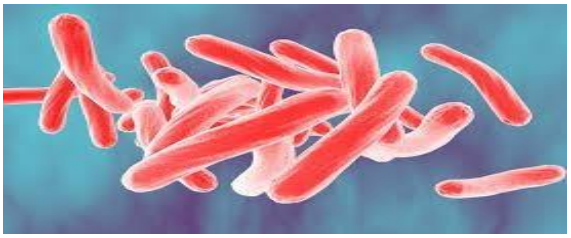
3- Gastrointestinal (GI) infection is most often caused by consuming anthrax-infected meat and is characterized by diarrhea, potentially with blood, abdominal pains, acute

inflammation of the intestinal tract, and loss of appetite. Occasional vomiting of blood can occur. Lesions have been found in the intestines and in the mouth and throat. After the bacterium invades the gastrointestinal system, it spreads to the bloodstream and throughout the body, while continuing to make toxins.

Mycobacterium tuberculosis

General characters: *M. tuberculosis* is an aerobic, Gram negative, non-spore forming, non-motile bacillus with a high cell wall content of high molecular weight lipids, which comprise approximately 60% of the cell wall structure.

According to cell wall composition, mycobacteria stain with acid-fast (Ziehl–Neelsen stain). The high lipid concentration in the cell wall accounts for its resistance to antimicrobial agents, and resistance to killing by acidic and alkaline compounds in both the intra and extracellular environment.



Pathogenesis: Early steps of infection: *M. tuberculosis* is a highly successful bacterial pathogen that mainly targets host macrophages, (key mediators of both innate and adaptive immune response). In lung infections, *M. tuberculosis* is typically inhaled into the body, passes through the airways and reaches the alveolar space. Here, it interacts with dendritic cells, alveolar macrophages and pulmonary epithelial cells, but its optimal hosts are alveolar macrophages and other mononuclear phagocytes.

Toxins: *Mycobacterium tuberculosis* (Mtb) induces necrosis of infected cells to evade immune responses. kill human macrophages by secreting tuberculosis necrotizing toxin (TNT) that induces necrosis.

Disease: TB is caused by *M. tuberculosis* and is one of the most intensively studied human diseases. It can target practically any organ of the body and clinical microbiological studies have been performed for decades. Humans are the only reservoir for the *M. tuberculosis* species, although many animals are also susceptible to infection.

Symptoms: Sweating General signs and symptoms include fever, chills, night sweats, loss of appetite, weight loss, and fatigue. Significant nail clubbing may also occur. If a tuberculosis infection does become active, it most commonly involves the lungs (in about 90% of cases). Symptoms may include chest pain and a prolonged cough producing bloody sputum.

Escherichia coli (E. coli)

General characters: *Escherichia coli* also known as *E coli* are Gram-negative bacilli. They are facultative anaerobes, rod-shaped and non-spore forming. It is commonly found in the gut

of humans and warm-blooded animals. Most strains of *E. coli* are harmless, which are part of the normal flora of the gut, and can benefit their hosts by producing vitamin K_2 and preventing colonization of the intestine with pathogenic bacteria, having a symbiotic relationship.

Virulence factors and toxins: related to bacteria colonization and virulence: including adhesions, toxins, iron acquisition factors, lipopolysaccharides and polysaccharide capsules, which are usually encoded on plasmids. Some strains such as Shiga toxin-producing *E. coli* (STEC), can cause severe foodborne disease.

Disease: Virulent strains can cause gastroenteritis, urinary tract infections and neonatal meningitis. Common signs and symptoms include severe abdominal cramps, diarrhea, vomiting and sometimes fever.

- Enterotoxigenic *E. coli* (ETEC)
- Enteropathogenic *E. coli* (EPEC)
- Enteroinvasive *E. coli* (EIEC)
- Enterohemorrhagic *E. coli* (EHEC)
- Enteroaggregative *E. coli* (EAEC)
- Uropathogenic *E. coli* (UPEC)
- Verotoxin-producing *E. coli*
- *E. coli* O157:H7 is an enterohemorrhagic strain

It is transmitted to humans primarily through consumption of contaminated foods, such as raw or undercooked ground meat products, raw milk, and contaminated raw vegetables and sprouts.

Salmonella spp.

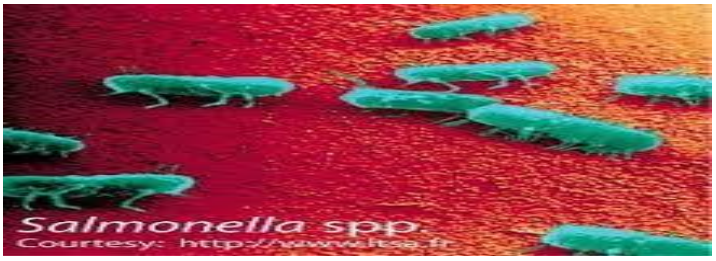
General characters: *Salmonella spp*. are a group of bacteria, which reside in the intestinal tract of human beings and warm blood animals and are capable of causing disease. They are facultative anaerobic, motile, Gram negative rod-shaped bacteria.

Salmonella spp. are members of the *Enterobacteriaceae* group.

The genus *Salmonella* contains 2 species:

- *Salmonella typhi (Typhoid fever)*

- *Salmonella paratyphi*
- Salmonella typhimurium



Virulence factors: Classic virulence factors include virulence-plasmids, toxins, fimbriae and flagella. However, effector proteins involved in survival and have been characterized recently.

Pahogenesis: *Salmonella* can invade different cell types, including epithelial cells, and macrophages, Most infections are due to ingestion of food contaminated by animal feces, or by human feces. cause gastrointestinal disease. They can infect a range of animals, and are zoonotic, meaning they can be transferred between humans and other animals. Typhoidal serotypes include *Salmonella* Typhi and *Salmonella* Paratyphi A, which are adapted to humans and do not occur in other animals.

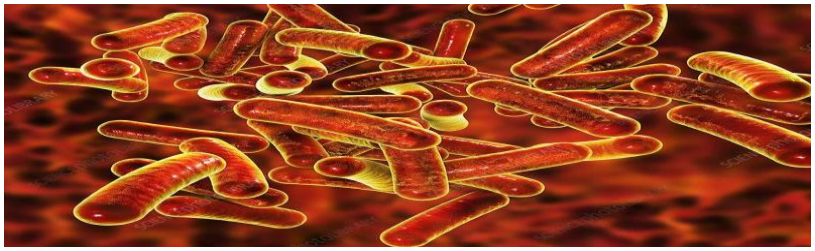
Disease: Salmonellosis in humans is typically transmitted via the alimentary route. Another possible route of transmission is contact with infected animals, such as pets, that are often kept in direct contact with humans, may also constitute a risk group.

The Symptoms of Salmonella

- Diarrhea, fever, and stomach cramps that develop 12 to 72 hours after infection
- Headache
- Nausea, vomiting, loss of appetite

Shigella spp.

Shigella spp. are Gram-negative, non-spore forming rod-shaped bacteria and are members of the family Enterobacteriaceae. The genus *Shigella* is divided into four species based on their O antigen type and biochemical characteristics, i.e.,
S. dysenteriae,
S. flexneri,
S. boydii
S. sonnei.
Shigella spp. are bacteria that cause shigellosis, also known as bacillary dysentery. They are highly infectious organisms.



Virulence factors

Shigella spp. has a virulence plasmid that encodes genes involved in the invasion process and intra cellular spread. Other genes involved in the invasion process are located on the chromosome.

Toxins

Shiga toxins are cytotoxins that because severe gastrointestinal disease caused by *Shigella dysenteriae* serotype 1. (*S. dysenteriae* 1) produces the prototype Shiga toxin. Vascular damage caused by Shiga toxins in the colon, kidneys, and central nervous system may result in hemorrhagic colitis, or more severe conditions such as hemolytic uremic syndrome.

Pathogenesis

Shigella spp. are transmitted by consumption of contaminated food or contaminated water, which is used for drinking and food preparation or fecal contamination of water.
Once ingested, *Shigella spp*. use the acidic environment of the stomach and invade the epithelial cells of the colon to enable infection. *Shigella spp*. multiplies inside the colonic epithelial cells and spread to adjacent cells, leading to the death of the infected cells. The colon becomes inflamed and ulcerated resulting in the bloody mucoid diarrhea.

Disease

The clinical symptoms of shigellosis range from mild diarrhea to severe dysentery, depending on the *Shigella* serotype causing infection, dose and the immunity and age of the host. The incubation period is 1–7 days (usually 3 days) and symptoms typically last for 1–2 weeks.

Initial symptoms include watery diarrhea, fever and fatigue. In more severe cases, as the case for *S. dysenteriae* serotype 1 infection, symptoms include abdominal cramps, nausea and vomiting. All *Shigella spp*. can cause acute bloody diarrhea.

Vibrio cholerae

A Gram-negative, facultative anaerobic, comma-shaped bacterium. The bacterium's natural habitat is saltwater and has a flagellum at one cell pole as well as pili.

Virulence factors and toxins

V. cholerae pathogenicity genes code for proteins directly or indirectly involved in the virulence of the bacteria. During infection, *V. cholerae* secretes cholera toxin, a protein that causes profuse, watery diarrhea (known as "rice-water stool"). Colonization of the small intestine also requires the toxin coregulated pilus (TCP), a thin, flexible, filamentous appendage on the surface of bacterial cells.

Pathogenesis

In the intestinal lumen, *V. cholerae* bacterium uses fimbriae (short pilli) to attach to the intestinal mucosa. After that it secretes cholerae toxin that leads to secreting of water into the intestinal lumen, causing watery stool or rice watery stool.

V. cholerae can cause syndromes ranging from asymptomatic to cholera gravis. Symptoms include watery diarrhea (a grey and cloudy liquid), occasional vomiting, and abdominal cramps.

Disease

V. cholerae bacterium causes Cholera, which is a major infectious disease. Infections are particularly common after ingesting contaminated water or food. Cases are occasionally seen in people, who have eaten raw or undercooked shellfish.

Cholera appears suddenly with painless, watery diarrhea, sometimes accompanied by vomiting.

Infections may be subclinical, mild or severe. Severe fluid loss can be seen in more serious cases; thirst, oliguria, severe dehydration, acidosis, muscle cramps and shock may result. Most cases last approximately 2 to 7 days but death may occur within a few hours if the fluid loss is high.

Treponema spp.

Treponemes are anaerobic, helically coiled, corkscrew-shaped cells (spiral shaped bacteria), 6 to 15 μm long and 0.1 to 0.2 μm wide. They have an outer membrane which surrounds the periplasmic flagella.

The genus *Treponema* contains both pathogenic and nonpathogenic species. Human pathogens cause four treponematoses: syphilis (*T pallidum* subsp *pallidum*), yaws (*T pallidum* subsp *pertenue*), endemic syphilis (*T pallidum* subsp *endemicum*), and pinta (*T carateum*).

Nonpathogenic Treponemes may be part of the normal flora of the intestinal tract, the oral cavity, or the genital tract. Some of the oral Treponemes have been associated with gingivitis and periodontal disease.

Virulence factors

The potential virulence factors of this microorganism include adherence factors, motility, evasion mechanisms from host defenses and cytotoxic factors for host tissues.

Pathogenesis

Treponemes are highly invasive pathogens. Evasion of host immune responses appears to be, at least in part, due to the unique structure of the treponemal outer membrane (i.e., it's extremely low content of surface-exposed proteins). Although treponemes lack classical lipopolysaccharide (endotoxin), they possess abundant lipoproteins which induce inflammatory processes.

Disease

Treponemes cause syphilis, Syphilis is a systemic disease caused by the spirochaete, *Treponema pallidum*. The infection can be classified as congenital (transmitted from mother to child in utero) or acquired (through sex or blood transfusion).

الأسئله البعديه :

Q1:Eunumerate the disease caused by *Salmonella spp* and *Shigella spp*.

رقم المحاضرة: السادسة والسابعة والثامنة .	
عنوان المحاضرة:	علم الفيروسات
اســـم المدرس:	أ.م.د. سوزان عادل
الفئة المستهدفة :	طلاب المستوى الثاني
الهدف العام من المحاضرة :	
الأهداف السلوكية او مخرجات التعلم:	1-التعرف على الخصائص العامه للفيروسات 2- التعرف على الاستجابه المناعيه للاصابات الفيروسيه 3- التعرف على تضاعف الفيروسات
استراتيجيات التيسير المستخدمة	عرض تقديمي،مجسمات ،شرح، صبوره
المهارات المكتسبة	مهارات تعلم على تضاعف الفيروسات وانتشارها ، التوعيه للحد من انتشار الأمراض، مهارات العرض والتقديم.
طرق القياس المعتمدة	الأختبارات التجريبيه، الاسئله و المناقشة، تحليل الردود.

الأسئلة القبليه:

Q1: What's the difference between viruses and bacteria.

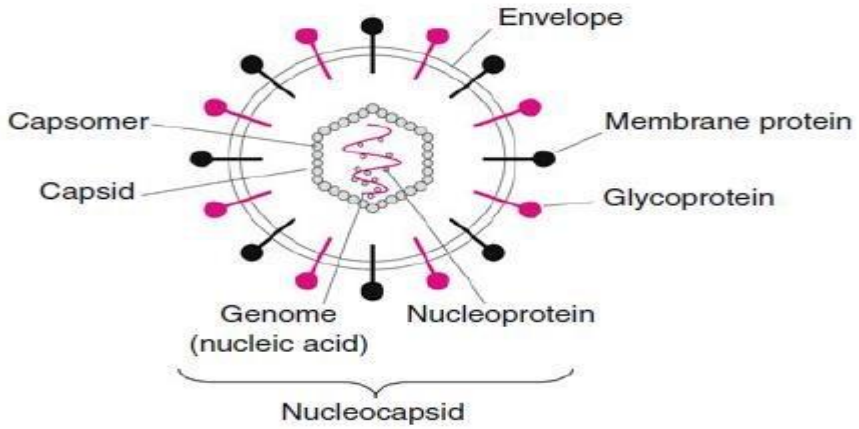
Virology

General structure of virus

Infectious virus particles – also referred to as virions – are constituted of various basic elements.

1- they contain single-stranded or double-stranded, linear, circular or segmented an RNA genome or a DNA genome.

2- The genome forms a nucleo-capsid complex
3- This nucleic acid-protein complex can surround by particular protein structures, which are the capsids (Capsids are rod-shaped or cubic-spherical protein structures).
4- In some cases (such as picornaviruses), the nucleic acid interacts directly with the capsids.
5- In viruses containing an envelope, the capsid layer can be absent (as in corona viruses).
6- The size of the virion (the complete, infective form of a virus outside a host cell, with a core of RNA and a capsid).
7- The site of viral replication within the cell (cytoplasm or nucleus).



The structure of the virus

Differences between virus and bacteria

	Bacteria	Virus
1	Unicellular	Do not have cells
2	Considered as a living organism	Not considered

3	**Larger and visible under light microscope**	**Smaller and visible under electron microscope**
4	**Contain a cell wall with peptidoglycan**	**Contain protein coat rather than a cell wall**
5	**Have, circular Chromosome** DNA	**Has DNA/RNA strand**
6	**Do not need a host organism for reproduction**	**Replicates only inside the host**
7	**Caused localized infections**	**Cause systemic infections**
8	**Can be either beneficial or harmful**	**Usually harmful**
9	**Infections can be prevented by Antibiotics.**	**Spread of viruses can be prevented by vaccines**

Hepatitis virus types

Viral hepatitis caused by infection with one of the five hepatitis viruses, which use the liver as their primary site of replication. Each of these, known as hepatitis A through E viruses (HAV to HEV), belong to different virus families, have unique morphology, genomic organization and replication strategy. These viruses cause similar clinical manifestations during the acute phase of infection but vary in their ability to cause chronic infection.

Hepatitis A virus (HAV)

It is a non-enveloped virus, made up of a capsid of three or four proteins and a single stranded, positive-sense polyadenylated RNA genome

Pathogenesis

Humans are the only host, and hence the only source, of HAV. The virus excreted in large amounts in faces of infected people. The most important mode of transmission is close contact with an infected person, usually in a household or a school. Contaminated water and foods such as seafood, farm products, milk, hamburgers and salads are important modes of transmission. Although blood or blood products can also transmit HAV, they are uncommon. Sexual transmission of HAV had been also reported.

Disease

Hepatitis is a liver disease caused by HAV. Infection with HAV may be asymptomatic or may result in acute hepatitis of variable severity, including fulminant hepatitis. The incubation period is 2–6 weeks. The illness usually begins with a prodromal phase of 1–7 days characterized by non-specific, systemic symptoms, such as fatigue, malaise, low-grade fever, headache, myalgia, arthralgia, loss of appetite, nausea and vomiting, altered taste-sensation and aversion to fatty foods and smoking.

Replication

1- After infection, HAV replicates in the small intestine, from where it reaches the liver through portal circulation.
2- The major site of HAV replication is the hepatocytes.
3- After entry, the genomic RNA is translated into a polyprotein, which is subsequently processed into 11 different proteins.
3- Proteins replicate the genomic RNA.
4- Late in the replication cycle, the capsid proteins package the genomic RNA.
5- The newly formed virions are secreted across the surface of hepatocytes into liver sinusoids and bile canaliculi.
6- Finally, they enter the small intestine and are excreted in faeces.

Vaccine

Two different vaccines are used against hepatitis A, HavrixTM (Glaxo SmithKline) and VAQTATM (Merck). Both contain formalin-inactivated attenuated strains of HAV, are highly immunogenic and safe. For each vaccine, two doses separated by at least 4 weeks are recommended.

Hepatitis B virus (HBV)

Hepatitis B virus (HBV) is an enveloped virus with nucleocapsid that contains double stranded circular DNA genome. The envelope comprises a small amount of lipid and three hepatitis b surface proteins large, medium and small.

Pathogenesis

Hepatitis B virus is transmitted by cutaneous and mucosal exposure to infected blood and other body fluids (semen and vaginal fluid). The highest concentrations of virus occur in blood and wound secretions. Moderate concentrations of HBV are found in semen and vaginal fluid. Lower concentrations occur in saliva. HBV is not spread by air, food or water. Common modes of transmission include mother to infant, child to child, unsafe injection practices and blood transfusions as well as sexual contact.

Disease

HBV can be a symptomatic disease (acute hepatitis B) or an asymptomatic infection with no sign or symptoms of disease. The hepatitis B virus (HBV) infects 350 million people worldwide, causing maladies ranging from acute hepatitis to chronic hepatitis, cirrhosis and hepato-cellular carcinoma (HCC). Chronic HBV infection with cirrhotic liver is associated with the development of HCC, which are one of the most malignant cancers.

Replication

1- All three envelope (or surface) proteins are encoded, i.e., the large envelope protein, the middle and small (S) envelope proteins.

2- All transcriptional regulatory elements including the promoters, enhancers, and the polyadenylation signal overlap with the protein-coding sequences.

Vaccine

The vaccine had included the development of an effective recombinant vaccine composed of purified HBsAg as well as Ig containing high-titer anti-HBsAg.

Hepatitis C virus (HCV)

General features

Hepatitis C virus is a small, enveloped virus. The genome consists of a single stranded RNA molecule.

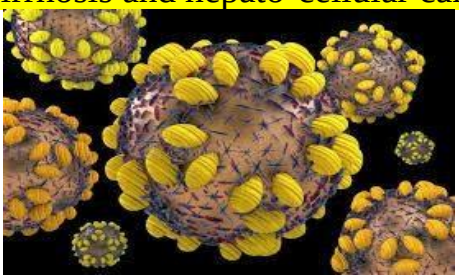
Pathogenesis

HCV is transmitted through large or repeated percutaneous exposures to blood from transfusion and transplantation of infected organs.

Disease

Hepatitis C virus (HCV) is estimated to have infected almost 200 million people, representing almost 3% of world population. In 20–30% of patients HCV causes acute infection, but in the majority of patients, it causes a long-term chronic infection.

Persistent infection with HCV is associated with the development of chronic hepatitis, hepatic steatosis, cirrhosis and hepato-cellular carcinoma (HCC).



Replication

1- The core protein interacts with HCV RNA and is largely involved in nucleocapsid assembly, oligomerization of the capsid protein and encapsidation of the viral genomic RNA.

2- Once the nucleocapsid is formed in the cytoplasm, it acquires the envelope proteins.

3- HCV particles are released.

Vaccine

A combination therapy of interferon with antiviral Ribavirin is usually given to patients. However, its efficiency varies with the HCV genotype and the viral loads at the start of therapy.

Herpes virus

General features

The structure of herpes viruses consists of a relatively large, double-stranded, linear DNA genome encased within a protein called the capsid, which is wrapped in a lipid bilayer called the envelope. The envelope is combined to the capsid by a membrane. This complete particle is known as the virion.

Pathogenesis

1- Transmission of HSV infections occurs through close contact with mucosal fluids, or in genital or oral secretions.

2- Infection occurs by inoculation of virus onto susceptible mucosal surfaces (e.g. Oropharynx, cervix, conjunctivae) or through small cracks in the skin.

3- During the primary infection:

* Virus infects the host at muco-cutaneous surfaces including the cornea, mouth, genital tract and skin.
* Invades the local sensory nerves by propagating via neurons.
* Establishes lifelong latency in the neuron bodies of sensory ganglia.

4- Following a primary infection, the virus enters at the site of primary infection, migrates to cell body of the neuron, and becomes latent in the ganglion.

5- As a result of primary infection; the body produces antibodies to particular type of HSV involved, preventing a subsequent infection of that type at a different site.

Disease

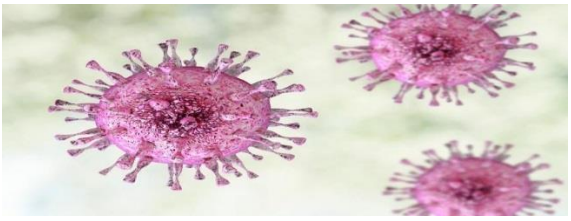
Herpes simplex virus (HSV) types 1 and 2 (HSV-1 and HSV-2) are two pathogenic agents that cause lifelong recurrent immune-pathologic diseases in man, ranging from fatal disseminated disease in newborns, to skin lesion (cold sores), genital ulcerations, **blinding** eye lesions and fatal encephalitis in adults.

1- Exposure to HSV at mucosal surfaces or abraded skin sites permits entry of the **virus** and initiation of its replication in cells of the epidermis and dermis.

2- Initial HSV infection is often subclinical, without apparent lesions.

3- Common infection of the skin or mucosa may affect the face and mouth (orofacial herpes), genitalia (genital herpes), or hands (herpetic whitlow).

4- More serious disorders occur when the virus infects and damage the eyes (herpes keratitis), or invades the central nervous system, damaging the brain (herpes encephalitis).



Replication

Viral replication occurs in ganglia and contagious neural tissue during primary infection only. After initial inoculation of the neural ganglion, virus spreads to other mucosal skin surfaces by centrifugal migration of infectious virions through peripheral sensory nerves.

Vaccine

Several antiviral drugs are effective for treating herpes, including acyclovir, valaciclovir (valacyclovir), famiciclovir and penciclovir. Acyclovir was the first discovered and is now available in generic. Lipopeptide vaccines (i.e., topical ocular and intravaginal) provide a novel strategy that might target ocular and genital herpes and possibly provide protection from this virus.

Varicella-zoster virus

Varicella zoster virus (VZV), also known as human herpesvirus is a herpesvirus with a double-stranded DNA genome. VZV only infects humans, with no animal reservoir. Its main targets are T lymphocytes, epithelial cells and ganglia.

Pathogenesis

1- VZV is highly communicable and spreads by the airborne route, with high transmission rate in temperate countries.

2- The virus spread to others from the respiratory tract.

3- Most viruses come from skin where it is highly concentrated in vesicles and skin cells.

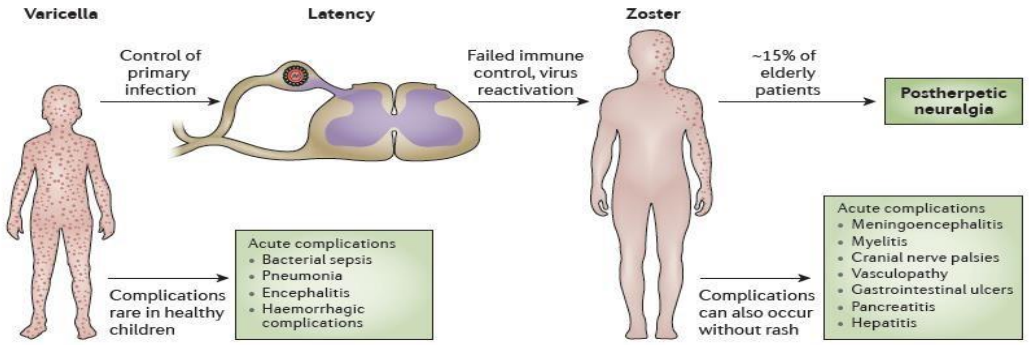
4- VZV particles enter cells by fusion of the virion envelope with the plasma membrane or by endocytosis followed by the transport of capsids and associated virion membrane proteins to the cell nucleus.

Disease

1- Primary infection causes varicella (chickenpox), as VZV becomes latent in ganglionic neurons.

2- As cellular immunity to VZV decreases with advancing age or in immune-compromised individuals, VZV reactivates to cause zoster (shingles).

3- Zoster can be complicated by chronic pain (postherpetic neuralgia (PHN)) and other serious neurological and ocular disorders (for example, meningoencephalitis, myelitis), as well as multiple visceral and gastrointestinal disorders, including ulcers, hepatitis and pancreatitis.



Different phases of varicella zoster virus infection

Replication

varicella zoster virus (VZV) gene transcription is occur in a cascade that leads to the synthesis of viral proteins that are classified as immediate-early, early, and late, based on the time course of their expression after virus entry.

Vaccine

Live attenuated vOka consists of a mixture of distinct VZV genotypes, with 42 single nucleotide polymorphisms.

Cytomegalovirus (CMV)

Cytomegalovirus (CMV) is a member of the human herpesvirus family. It has large, linear, double-stranded DNA. The genome divided into a unique long (U_L) region and a unique short region. The U_L region contains two genes whose protein products are important in antiviral therapies.

Pathogenesis

Cytomegalovirus (CMV) predominantly transmitted via:

- Breastfeeding
- Fomite spread
- Contact with other children
- The cervix during parturition

Infection with CMV can also occur via:

- Inhalation
- Sexual contact
- Blood transfusions
- Transfer with transplanted organs
- Transmission from mother to unborn child

Disease

1- Human cytomegalovirus (HCMV) causes severe illness and death in people, whose immune systems are weak, including organ and bone marrow transplant recipients, HIV infected people, those on immunosuppressive drugs and newborns infected during pregnancy, with recognized syndromes of fever, hepatitis, pneumonitis, encephalitis and retinitis.

2- After primary infection with CMV, the virus becomes latent and can be reactivated to produce a secondary infection, particularly during episodes of immunosuppression.

3- Cytomegalovirus is secreted in saliva, urine and breast milk.

Cytomegalovirus inside human cell

Replication

Human cytomegalovirus (HCMV) infects and replicates in a wide variety of cells, including epithelial cells of gland and mucosal tissue, smooth muscle cells, fibroblasts, macrophages, dendritic cells, hepatocytes and vascular endothelial cells.

HCMV undergoes latency in myeloid cells of the bone marrow leading to a life-long infection with sporadic reactivation.

Vaccine

A recombinant HCMV glycoprotein B (gB) vaccine has been shown to have some efficacy in prevention of infection in young women and adolescents.

Human Immunodeficiency Virus (HIV virus)

Human Immunodeficiency Virus (HIV) are grouped into two types, HIV-type 1 (HIV-1) and HIV-type 2 (HIV-2). The worldwide main agent of AIDS is HIV-1, while HIV-2 is restricted to some regions of Western and Central Africa.

The retrovirus genome is composed of two identical copies of single-stranded RNA molecules and is characterized by the presence of structural genes.

Pathogenesis

Human Immunodeficiency Virus (HIV) cannot survive outside the bloodstream or lymphatic tissue. Furthermore, virus is easily inactivated by the exposure to common

detergents and disinfectants. Thus, virus transmission requires the directed exposition to infected blood or secretions in the presence of skin damage, i.e. by needles or sharp tools, or abrasions in mucosal tissues within sexual intercourses. Transmission of HIV is highly dependent on the:

1- Biologic properties of the virus
2- Its concentration in the infected body fluid
3- Host susceptibility

HIV is mainly integrated or replicating into the infected cells, which are the main vehicles of virus transmission.

Disease

Both viruses potentially cause AIDS, though disease of the central nervous system may be more frequent in HIV-2 infection. In addition, HIV-2 appears less virulent than HIV-1 and infection course takes longer to progress to AIDS. Ranging from few days to few weeks since exposure to HIV, most of the infected individuals present symptoms, e.g. flu-like illness, fever, maculopapular rash, oral ulcers, lymphadenopathy, pharyngitis, weight loss and myalgia.

It has been reported that individuals who display more severe and durable symptoms in the course of acute infection tend to progress more rapidly to AIDS. The symptomatic phase of acute HIV-1 infection lasts between 7 and 10 days, and rarely longer than 14 days.

Replication

Human Immunodeficiency Virus (HIV) viruses are characterized by other accessory/ regulatory genes that responsible of modulating virus replication. Among these, the tat gene encodes for a protein (Tat) that is expressed after infection and promotes the expression of HIV genes. The Rev protein, coded by the rev gene, ensures the export from nucleus to cytoplasm of the genomic RNA.

The HIV replication cycle can be summarized in six steps;

1) Binding and entry
2) Uncoating
3) Reverse transcription
4) Provirus integration
5) Virus protein synthesis and assembly
6) Budding

Vaccine

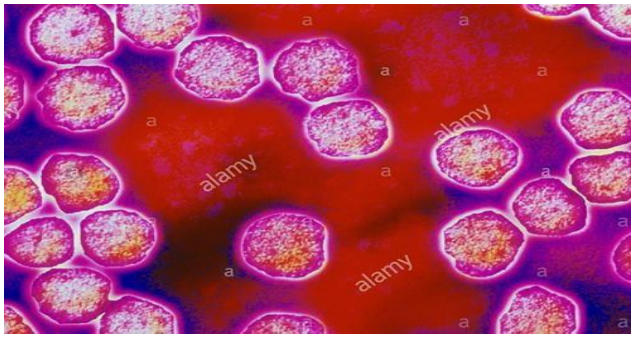
An effective HIV vaccine should induce powerful and durable immunity to prevent infection in healthy individuals and/or reduce viral replication and viral load in infected people, slowing or halting disease transmission and progression. These vaccines include:

1- Live attenuated and inactivated virus vaccines

2- Protein subunit vaccines

Rubella virus

The name rubella is derived from Latin word where rubella stands for “Little Red”. Rubella was initially considered as scarlet fever or measles. The rubella virus is roughly spherical in shape of diameter 40 - 60 nm. The virus carries a positive sense single stranded RNA genome enclosed within lipid capsid. The virus consists of three structural proteins, two envelope proteins glycoproteins E1 and E2 and one core protein C protein surrounding the genome.



Pathogenesis

1- The disease is contagious from 7 days after appearance of rash.
2- Postnatal rubella spreads by airborne respiratory droplets that result from coughing and sneezing, by direct contact with nasopharyngeal fluid of an infected person or from urine of infants with congenital rubella syndrome (CRS).
3- Infected individuals may be contagious as early as a week before the appearance of the rubella rash, and for up to a week after it first appears.
4- Children born with CRS may transmit the virus to others for more than a year.
5- Rubella cases typically peak in late winter or early spring.

Disease

1- The infection starts from initial appearance of rash on face and then gradually spreads down the neck.
2- Infection occurs due to inhalation of aerosols and infects the upper respiratory tract where the virus enters the cell through cell - mediated endocytosis.
3- In case of: children Rash beginning on the face, which spreads to the rest of the body, low fever of less than 38˚C and posterior cervical lymphadenopathy.
4- In older children and adults’ additional symptoms may be present including: swollen glands, Coryza (cold like symptoms) and aching joints (especially in young women).
5- Serious problems can occur including brain infections, bleeding problems, birth defects (Congenital), inflammation of lymph nodes, cataracts, maculopapular rashes, heart defects and hearing loss.

Replication

1- Rubella virus (RV) is characterized by slow replication, which is reflected in the long viral latent period of 8 to 12 h.

2- During viral replication, RV genomic RNA serves as a messenger for the nonstructural proteins and as a template for the synthesis of negative-polarity RNA strand.

3- RNA is packaged with the RV capsid protein to form nucleocapsids.

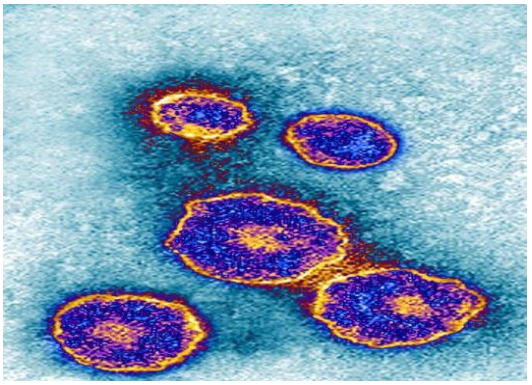
Vaccine

MMR II (Measles, Mumps and Rubella virus vaccine live) is the vaccine which is recommended to boost immune system and prevent serious, life threatening diseases. MMR II consists of live. Attenuate strains Measles, Mumps and Rubella virus.

Mumps virus

General features

The virus is enveloped, has roughly spherical particles and containing a nonsegmented negative strand RNA molecule. Virions are sensitive to treatment with lipid solvents, nonionic detergents, formaldehyde, oxidizing agents, and heat.



Pathogenesis

The virus is transmitted by respiratory droplets or by direct contact with infected respiratory secretions (e.g., kissing or shared utensils) or by contact with items in the environment contaminated with infected secretions.

Disease

1- Mumps or Epidemic parotitis is the common name of mumps disease. Symptoms include parotitis or swelling of sublingual or submandibular salivary glands for 2 or more days. Parotitis accompanied by fever, sore throat, and systemic symptoms of malaise and fever.

2- Less common manifestations, with or without parotitis, include benign orchitis, aseptic meningitis.

Replication

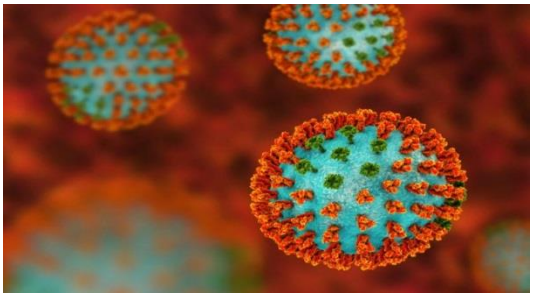
Mumps mostly colonizes and replicates along the upper respiratory tract. After entering the respiratory system, the virus locally replicates. To target tissues of the salivary glands and central nervous system, viremic dissemination occurs.

Vaccine

Live attenuated mumps virus vaccine is incorporated into combined MMR vaccine. For prevention of mumps, 2 doses of MMR vaccine are recommended routinely for children with the first dose at 12–15 months of age and the second dose at 4–6 years of age (school entry).

Orthomexovirus (Influenza virus)

Helical enveloped virus. It has Linear, 8 segmented RNA. These are important human pathogens as they cause both outbreaks and pandemics (infrequently) that kill thousands of people.



Pathogenesis

1- Typically, influenza is transmitted from infected mammals through the air by coughs or sneezes, creating aerosols containing the virus, and from infected birds through their droppings.

2- Influenza can also be transmitted by saliva, nasal secretions, feces and blood.

Infections occur through contact with these bodily fluids or with contaminated surfaces.

3- Out of a host, flu viruses can remain infectious for about one week at human body temperature, over 30 days at 0°C (32 °F), and indefinitely at very low temperatures (such as lakes in northeast Siberia). They can be inactivated easily by disinfectants detergents.

Disease

1- Uncomplicated Influenza: Symptoms include chills, headache, dry cough, muscular aches. These may be induced by influenza A or B. In contrast, influenza C causes a common cold illness, Coryza.

2- Pneumonia: complications occur only in the elderly and debilitated. Influenza infection enhances the susceptibility of patients to bacterial superinfection, due to loss of ciliary clearance, dysfunction of phagocytic cells.

3- Reye's syndrome: an acute encephalopathy of children and adolescents (2-16 yrs).

Types of Influenza virus:

- Influenza A virus causes worldwide epidemics.
- Influenza B virus causes major outbreaks of Influenza.
- Influenza C virus cause mild respiratory tract infections and no outbreaks.

Replication

1- After viral hemagglutinins interact with the surface receptors, the virus enters the cell in vesicles and uncoats mediated by the M2 proteins and is facilitated by the low pH within the endosome/vesicle.

2- The viral nucleocapsid enters the cytoplasm and migrates to the nucleus where the genome RNA (8 segments) gets transcribed into mRNA by the viral RNA polymerase (transcriptase).

3- Most RNA's move to cytoplasm, some remain in the nucleus to serve as a template for the synthesis of negative polarity strand RNA genomes for the progeny, by a different subunit of viral RNA polymerase (replicase).

Vaccine

Vaccines are composed of either inactivated or live attenuated virions of the H1N1 and H3N2 human influenza A viruses, as well as those of influenza B viruses.

Because the antigenicity's of the wild viruses evolve, vaccines are reformulated annually by updating the seed strains.

Names of virus associated with human cancer

1- Human papillomavirus

2- HPV and cervical cancer

3- Epstein-Barr virus (EBV)

4- Hepatitis B virus (HBV) and hepatitis C virus (HCV)

5- Human immunodeficiency virus (HIV)

6- Human herpes virus 8 (HHV-8)

الأسئله البعديه:

Q1: define :

A-Replication

B- Vaccine

Q2: explain the pathogenicity of each type of viruses .

رقم المحاضرة:التاسعه و العاشره والحاديه عشر

.

عنوان المحاضرة:	علم الفطريات
اســـم المدرس:	أ.م.د. سوزان عادل
الفئة المستهدفة :	طلاب المستوى الثاني
الهدف العام من المحاضرة :	
الأهداف السلوكية او مخرجات التعلم:	1-التعرف على الأعفان والخمائر 2-التعرف على أنواع الاصابات الفطريه -3
استراتيجيات التيسير المستخدمة	مجسمات، عرض تقديمي،شرح ، صبوره
المهارات المكتسبة	مهارة التعلم على تشخيص الأنواع الفطريه، مهارة التعلم على تشخيص الاصابات الفطريه، مهارات العرض والتقديم
طرق القياس المعتمدة	الاختبارات التجريبيه، الاسئله والمناقشه، تحليل الردود

الأسئلة القبليه :

Q1: How we can classify fungi?
Q2: What's the importance of fungal cell wall?

Mycology

Mycology : is the branch of biology concerned with the study of fungi, including their **genetic** and biochemical properties, their taxonomy and their use to humans as a sourcefor tinder, medicinals (e.g., **penicillin**), food (e.g., **beer**, **wine**, **cheese**, edible mushrooms) and entheogens, as well as their dangers, such as poisoning or infection.

Fungi : Are plants that has lack chlorophyll and reproduced by spores .

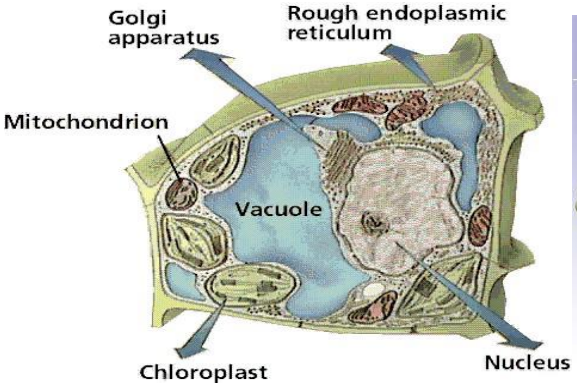
General characteristics :

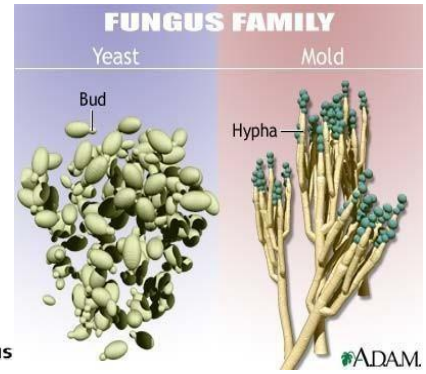
1. All fungi are **eukaryotic** organism .
2. Most Fungi are **obligate** or **facultative** aerobic .
3. Nutritional requirement to growth of fungi is simple, sometimes need enrich media .
4. Each fungal cell has at lest one **nucleus** and **nuclear membrane** ,endoplasmicreticulum , mitochondria and secretory apparatus.
5. Optimum temperature of growth of fungi 28C° .
6. Growth in pH (2-9), it growth well in acidic PH .
7. Fungi may reproduced sexually or asexually
8. Fungi grow in two basic forms as **yeast** and **molds** .

Classification of fungi according to the morphology :

1- **Molds :**- Most fungi consist of microscopic branching filaments, called (**hyphae**). These are normally divided septa in to cells . **e.g :** *Rhizopus*

2- **Yeast :**- When fungi appear **unicellular**, spherical or oral shaped and reproduce by budding are generally called yeast . . **e.g :** *Cryptococcus neoformans*





Fungi Cell **Yeast** **Mold**

Dimorphic fungi : The term dimorphic is used to describe a fungus which occurs in twodifferent forms occording to the environmental culture. Appear filaments at 22C but appear yeast on the culture media at 37C or in the humanbody.

For example some pathogenic fungi are (**Mycelia**) in culture and yeast like in infectedtissues.

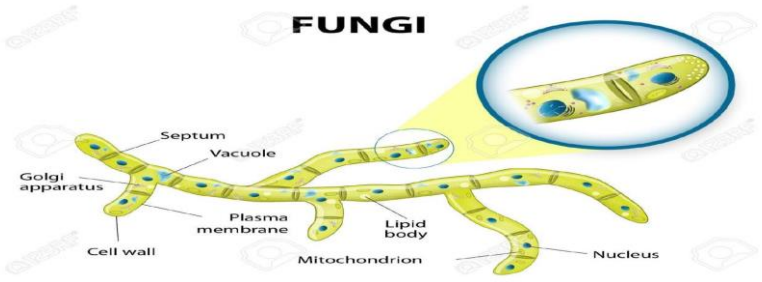
A mycosis:- (plural: *Mycoses*) is a fungal infection of animals, including humans.

There are three types of mycosis :

1. Superficial mycosis: ***Candida albicans***

2. Subcutaneous mycosis: ***Mycetoma***

3. Systemic mycosis : ***Crytococcus***



Typical fungi cell (Fungal hyphae)

Cutaneous fungal infection (Superficial fungal infection)

1- Infections caused by pathogenic fungi and limited to the human hair, nails, epidermis, and mucosa are referred to as superficial fungal infections.

2- They are important because of their worldwide distribution, frequency, person-to person transmission, and morbidity.

3- Severe infections may be the first indication of an underlying immunodeficiency.

4- Dermatophytosis (tinea or ringworm), pityriasis versicolor (formerly tinea versicolor) and candidiasis (moniliasis) are the three most common types of superficial fungal infections.

5- The dermatophytes are a large group that can infect human skin, hair, and nails; they are found in soil (geophilic organisms), on animals (zoophilic) and on humans (anthropophilic).

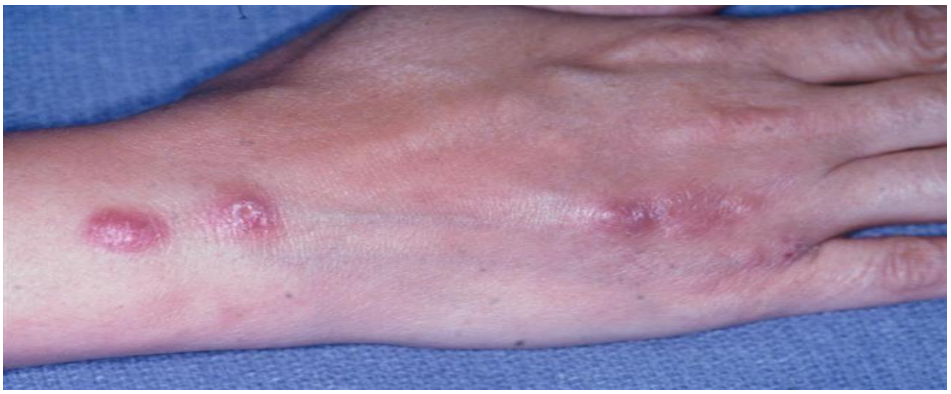
6- These fungi require keratin for growth and, therefore, they are unable to infect mucosal surfaces.

7- These fungi are found all over the world, although the specific species, and subsequent clinical presentation, vary from region to region.

Dermatophytosis is labeled by the involved area of the body (eg, *tinea corporis*, *tinea capitis*¸ *tinea pedis, tinea unguium*). Pityriasis versicolor is caused by the yeast form of a dimorphic fungus that is considered part of the normal human skin flora.

8- Candidiasis is caused by a yeast-like fungus of the genus *Candida* (most commonly *C. albicans*) that is part of the microflora in the human gastrointestinal tract (including the mouth) and the vagina.

9- Symptoms and signs of candidiasis manifest with a change in the normal host immune system.



Subcutaneous fungal infection

1- Subcutaneous fungal infection are a group of infections involving the skin, subcutaneous tissue, fascial planes, bones, or various organs systems.

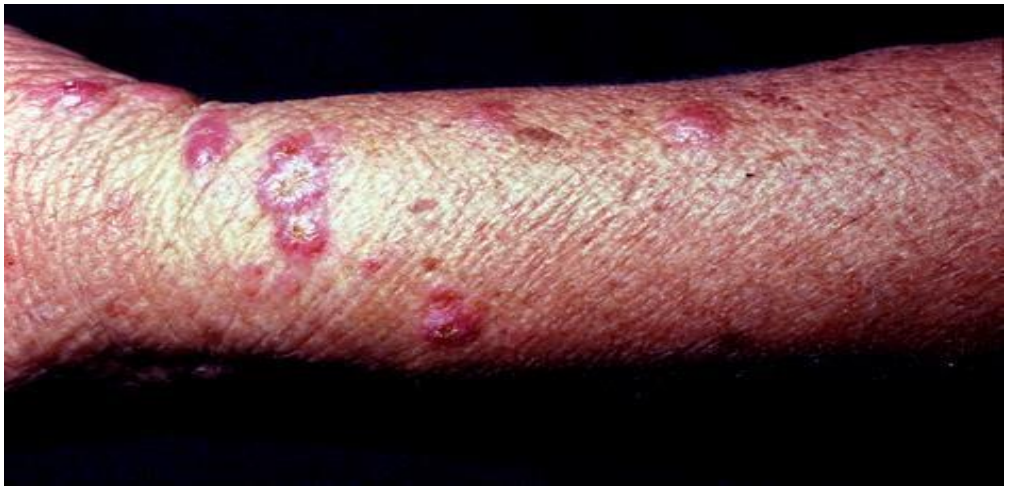
2- These include sporotrichosis, chromoblastomycosis and phaeohyphomycosis, mycetomas, subcutaneous zygomycosis (entomophthoromycosis and mucormycosis), and lobomycosis. Sporotrichosis remains the commonest subcutaneous mycosis

3- These are prevalent in tropics/subtropics due to high temperature and humidity conducive to the growth of fungi.

4- The rural population is particularly at risk of being infected. The majority of patients are between 30 and 50 years of age.

5- The disease may either remain localized or involve adjacent tissues. Wide spread dissemination may occur in immunocompromised host.

6- Treatment varies depending upon the site of infection, severity of the disease, and the pathogen involved.



Systemic fungal infection

1- Systemic fungal infection is an increasing cause of mortality and morbidity in patients with haematological malignancies and other conditions associated with profound immunosuppression.

2- Two groups of patients are at high risk of such infection, these are recipients of allogenic stem cell transplants and patients receiving intensive chemotherapy for de novo and relapsed acute leukaemias.

3- The majority of such infections are caused by *Aspergillus* and *Candida* species.

4- The etiology of systemic fungal infections can be classified into two groups:

endemic mycoses due to true pathogenic fungi and opportunistic fungal infections due to a vast group of saprophytic fungi.

5- Common symptoms of candidemia (*Candida* infection of the bloodstream) include fever and chills that do not improve with antibiotics.

Candidemia can cause shock and therefore may include symptoms such as low blood pressure, fast heart rate, and rapid breathing. Systemic candidiasis may also affect other parts of the body such as the central nervous system (brain and spinal cord), abdomen, heart, kidneys, liver, bones, muscles, joints, spleen, and/or eyes.

Opportunistic fungal infection

1- Invasive fungal infections (IFI) have significantly increased in immunocompromised population.

2- Fungal species are widely distributed in soil, plant debris and other organic substrates.

3- Major risk factors for IFI include neutropenia <500 neutrophils/ml for more than 10 days, haematological malignancies, bone marrow transplantation, prolonged (>4 wk) treatment with corticosteroids; prolonged (>7 days) stays in intensive care, chemotherapy, HIV infection, invasive medical procedures, and the newer immune suppressive agents.

4- Other risk factors are malnutrition, solid organ transplantation, severe burns or prolonged stays in intensive care (>21 days), systemic corticosteroids for >7 days, and major surgery. There are also reports of the presence of infection in immunocompetent patients without signs or symptoms of conditions associated with immunocompromised status.

5- Infection can be transmitted by the inhalation of spores (aspergillosis, cryptococcosis, histoplasmosis), percutaneous inoculation in cutaneous and subcutaneous infections (dermatophytosis, madura foot), penetration into the mucosa by commensal organisms such as *Candida albicans*, and the ingestion of a toxin in contaminated food or drink (gastrointestinal disease).

6- Infections may be mild and only superficial or cutaneous (*e.g*. dermatophytosis and *Tinea versicolor*) or may cause life-threatening, systemic illness (*e.g*. candidiasis, aspergillosis and mucormycosis).

7- The clinical manifestations of the disease are related to host immunity and physiological condition. For example, *Candida* spp. can invade a local site

(mucocutaneous or cutaneous candidiasis, onychomycosis) or cause systemic infections (renal, liver abscess, lung and nervous central system).

8- Allergic symptoms were reported in infections with other fungi such as *Aspergillus* spp. (allergic bronchopulmonary aspergillosis).

9- Treatment requires early diagnosis and is difficult because only a few antifungal agents are available, most usually have side effects, and some organisms have developed resistance.

Candida albicans pathogenesis

The polymorphic fungus *Candida albicans* is a member of the normal human microbiome. In most individuals, *C. albicans* resides as a lifelong, harmless commensal. Under certain circumstances, however, *C. albicans* can cause infections that range from superficial infections of the skin to life-threatening systemic infections.

Candida albicans exists often as a harmless commensal at various mucosal sites. As a pathogen, however, *C. albicans* is responsible for a wide range of infections, both mucosal and systemic, in both immunocompetent and immunocompromised individuals. *C. albicans* is acquired at or shortly after birth, often being transmitted from mother to child, and can remain a commensal or cause neonatal infections.

C. albicans can affect the oropharynx and/or the esophagus of persons with dysfunctions of the adaptive immune system.

Pathogenic factors

Several factors and activities have been identified which contribute to the pathogenic potential of this fungus. Among them are:

1- Molecules which mediate adhesion to and invasion into host cells.

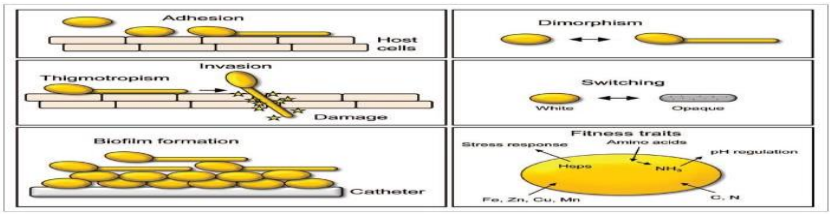
2- The secretion of hydrolases.

3- The yeast-to-hypha transition.

4- Contact sensing and thigmotropism.

5- Biofilm formation.

6- Phenotypic switching and a range of fitness attributes.



Pathogenicity mechanisms of *Candida albicans*

***Cryptococcus neoformans* pathogenesis**

Cryptococcus neoformans (Cn) is a fungal pathogen, commonly found in urban environments that primarily affects immunocompromised individuals through inhalation of spores.

In healthy individuals Cn infection can remain in a latent form for prolonged periods of time. However, in individuals with impaired immune function, the infection may spread to the central nervous system (CNS), causing life-threatening meningitis. Thus, the disease is relatively common in AIDS patients and organ transplant recipients receiving immunosuppressive therapy.

Virulence Factors

1- The capsule of Cn is one of its major virulence factors because it is antiphagocytic required for intracellular replication and the polysaccharide functions as a major modulator of the host immune response

2- Melanin: Melanization is associated with virulence in Cn and other fungi. Melanins are dark pigments exists in cell wall. They absorb light across the UV and visible spectrum, have high physical and chemical strength and can resist degradation, even by strong acids.

3- Phospholipase B: is a secreted protein that is found in all Cn serotypes. The mechanism of action could involve damage to host tissues, nutrient acquisition, and immune modulation through alteration of lipid molecules.

الأسئلة البعديه :

Q1: Define fungal infection and classify based on the site of infection.

رقم المحاضرة:	
عنوان المحاضرة:	
اســـم المدرس:	
الفئة المستهدفة :	
الهدف العام من المحاضرة :	
الأهداف السلوكية او مخرجات التعلم:	-1 -2 -3
استراتيجيات التيسير المستخدمة	
المهارات المكتسبة	
طرق القياس المعتمدة	

رقم المحاضرة: الثانية عشر، الثالثة عشر، الرابعة عشر، الخامسه عشر.	
عنوان المحاضرة:	علم الطفيليات
اســـم المدرس:	ا.م.د. سوزان عادل
الفئة المستهدفة :	طلاب المستوى الثاني
الهدف العام من المحاضرة :	
الأهداف السلوكية او مخرجات التعلم:	1-التعرف على تركيب خلية الطفيلي ودورها في الامراضيه 2-التعرف دورة حياة الطفيلي 3- التعرف على أنواع المضائف
استراتيجيات التيسير المستخدمة	مجسمات ،شرح ، صبوره ، عرض تقديمي
المهارات المكتسبة	مهارت التعرف على دورة حياة الطفيلي، مهارات تعرف على تشخيص الاصابات الطفيليه
طرق القياس المعتمدة	الاختبارات التحريريه ،تحليل الردود خلال المناقشه

الأسئله القبليه :

Q1:Explain the infect stage of the following :
A-*Giardia lamblia*
B- *Enterobius vermicularis*

Parasitology

Parasitology : Is the science which deal with living organisms which live temporary or permanently on or within other organisms for the purpose of procuring food and shelter.

Medical Parasitology : Is the science which deals with the parasites which cause human infections and the diseases they produce .

Parasites : Organisms that infect other living beings. They live in or on the body of another living beings called **host** and obtain shelter and nourishment from it .

The host : It is the organisms or animale which parasite live on or in it .

Types of hosts :

1. **Definitive (**final**) host :** The host in which the adult stage lives or the sexual mode of reproduction takes place.
2. **Intermedial host :** The host in which the larval stage of the parasite lives or the asexual multiplication takes place .
3. **Reservoir host :** It is an animal that can harbor the parasite and can be potential store of infection for man .
4. **Vector :** It is usually on arthropods (insect) that carries the parasite to it's host.There are **2** types :

A. **Mechincal vector :** Only transport the parasite without any role of life cycle, likefly .
B. **Biological vector :** The parasite undergo development or multiplication in the body of it.

The parasitic disease required the following Factors:

1-Source of infection .

2-Method of transmission .

3-Suitable host

4-Presence of vectors .

Entamoeba histolytic

Multiply:by binary fission .

Disease : Amoebic dysentery , Intestinal amoebiasis , Amoebic hepatitis .

Habitat : large intestine of human .

Geographical distribution : Cosmopolitation

Morphology : two stage, **A.** Cyst→ non motile . **B.** Trophozoite → motile .

Site in host : Lumen and wall of large intestine in human and Monkeys.

Source of infection : Cyst in food and water from feces of human .

Infective stage : Mature cyst with 4 nuclei .

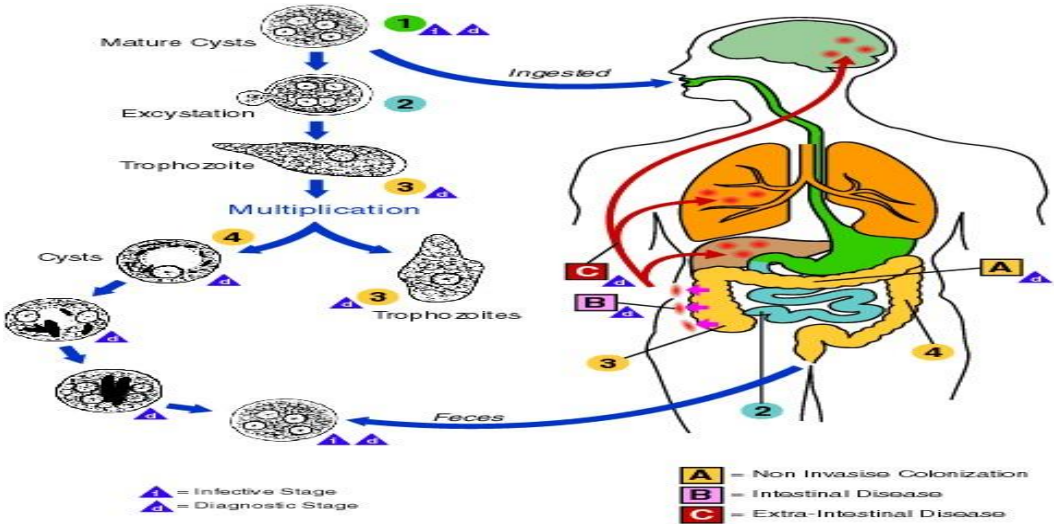
Diagnostic stage : Trophozoite and cyst .

Mode of infection : Oral route by ingestion mature cyst contaminated foods or drinks .

Morphology : Have 2 stages :

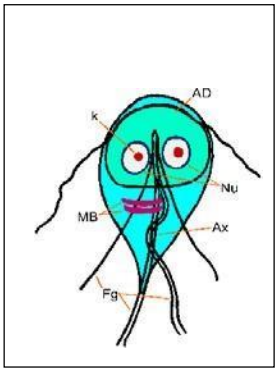
1. **Trophozoite stage :**
2. **Cyst stage:**

Diagnosis sample : Stool examination to identify **trophozoite** or **cyst**



Life cycle of Entamoeba histolytica

Intestinal flagellates



Giardia lamblia

Disease : Giardiasis

Habitate : Upper part of small intestine.

Host : Final host (human) , Intermediat host : No .

Infect stage : (Mature cyst with **4** nucleic)

Diagnostic stage : Trophozoite and Cyst .

Mode of infection : Oral-route . (by ingestion mature Cyst with contaminated food)

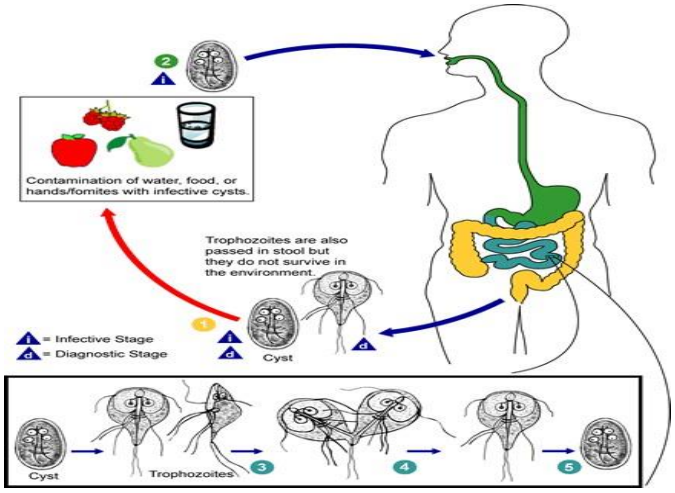
Morphology : Have 2 stages:

1. **Trophozoite stage :**

2. **Cyst stage:**

- Found in diarrheic stool and immature cyst found in normal stool in a large number . **Diagnosis :** Stool____direct smear_____ iodine and Microscopic examination reveal trophpozoite and cyst.

-



Life cycle of Giardia lamblia

Blood and tissue flagellates

(Haemoflagellates **)**

Class : Flagellates

Genus Leishmania

Genus Leishman include 3 speices infected human :

1- ***Leishmania tropica***

2- ***Leishmania donovani***

3- ***Leishmania brazillensis***

Example **:** ***Leishmania tropica***

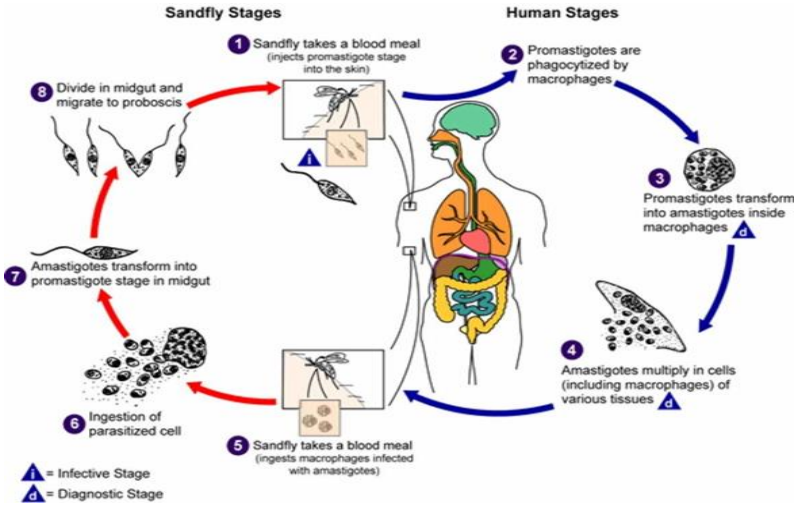
Disease : Cutanous Leishmaniasis, tropica sore, Baghdad boil . It cause 2 types of Lesion

Leishmania tropica major (Wet-type lesion) .

Leishmania tropic major (Dry -type lesion) .

Example **:** ***Leishmania donovani***

Disease : Viscular Leishmaniasis or Kala – azar .



Habitat : Tissue of Reticulo – endothelial , system (Liver, spleen, lymphnodes, bonemarrow

Example **:** ***Leishmania brazillensis***

Disease : Muco cutanous Leishmaniasis .

Habitat : Muco cutanous membrane of (nose, Larynx, ear)

Host : Intermediate host **:** Female of **sand fly** .

Final host : Human .

Infective stage : Promastigote stage .

Mode of infection : Through the skin by bitting of infected insect vector (**sand fly**) .

Sample for diagnosis : Blood or tissue (skin according habitate).

Morphology : Leishmania parasite found in 2 forms .

Amastigot stage **:** or (Leishmania form)This stage found in human only .

Promastigote stage :Elongated shape or spindle .Found only in insect.

Sporozoa

Include 4 types

Plasmodium vivax **:** cause Benign tertian malaria
Plasmodium ovale **:** cause tertian malaria
Plasmodium malaria **:** cause quarter malaria
Plasmodium falciparum **:** cause Malignant tertian malaria
Example : ***Plasmodium vivax***

Disease : All species cause malaria .

Host : There are 2 host :

1. Intermediate host vertebrate host **" human"** in blood intra **RBC** cell (**Asexual phase**)
2. .Final host: invertebrate host (insect) called (Female of Anophelas mosquito**)** as avector.**(sexual phase)**.
3. **Vector** : female of Anopheles insect (Mosquito) .
   Infective stage : Sporozoite (salivary gland of Anopheles)

Mode of infection : By biting of Mosquito.
Habitat : In circulatory system of vertebrates .

Clinical aspects : (**fever**, **coldness**, **sweating**) that symptom occur during the

suddenliberation of merozoites into blood stream . and bloody urine

Medical Helminthology

Species :- *Schistosoma*
Schistosoma Haematobium

- *Schistosoma mansoni*

- *Schistosoma Japanicum*

All trematodes pass through a phase of asexual development in the snail host

Disease : Urinary bilharziasis . *S. haematobium*.

Habitat : Depend on the type**.**

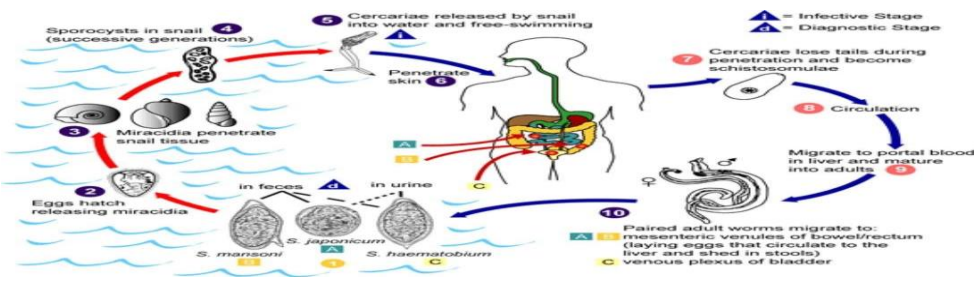
1. **Adults :-** Int the portal vein specially the vesical plexus of man .
2. **Egg :-** passes out in urine and very rarly in faces .
3. **Larval stage :-** in the tissues of snail Bulinus spp.

Morphology : Have two form Male and female .

The larval stage of *Schistosoma SPP*.

1. Miracidium **:** is the Larval stage surrounded by cilia , that infected snail .
2. Sporocyst **:** 2nd Larval stage grow in the snail .
3. Sporocyst **:** Redia , Daughterredia , cercaria.

Final Host: human.
Intermediate host :- ***Snail bulinus truncatus***
Infective stage : Cercaria with forked tail .
Diagnostic stage : Eggs with terminal spine in urine .
Mode of infection : By pentration of cercaria directly through the skin at swimming.



life cycle of schistosoma spp

Enterobius vermicularis

Common name : pin worm
Disease : Enterobiasis or Oxyuriasis or pin worm infection .

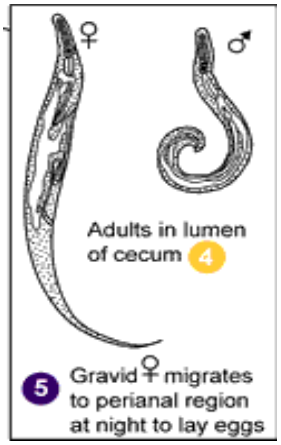
Habitat : Large intestine in man .

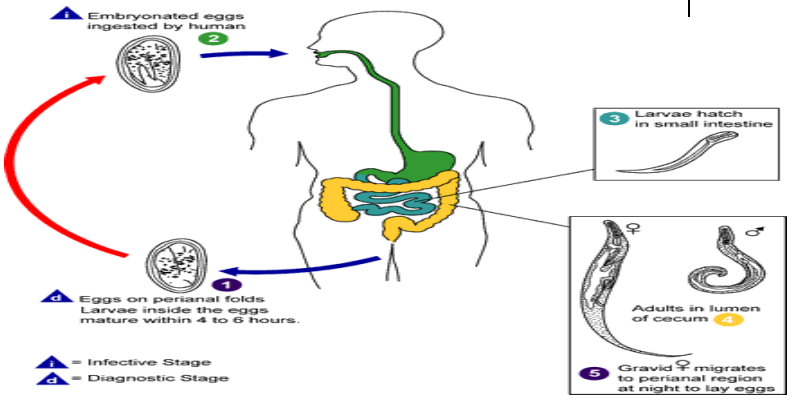
Host : Man
Infective stage : Embryonated egg .

Diagnostic stage : Egg and adult worm .

Mode of infection : Oral – rout by ingestion of eggs .

Diagnosis sample : Stool.





life cycle of Enterobius vermicularis

Ascaris lumbricoides

Common name : roundworm .

Disease : Ascariasis

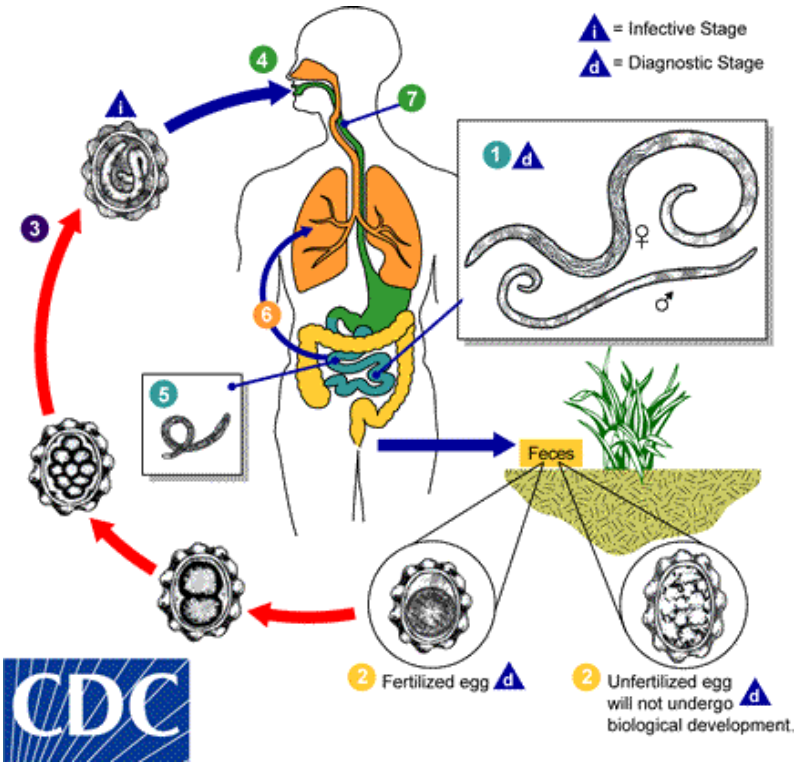
Habitat : Small intestine of man and pigs .

Final host : **Man**

Infective stage : Embryonated egg. (which contain mature **Diagnostic stage :** Unembryonated egg .

Mode of infection : Oral-rout by ingestion of embryonated egg with food or drink .

Diagnosis sample : Stool .



life cycle of Ascaris lumbricoides larvae

الأسئلة البعديه :

Q1: Whats the importance of stool examination of diagnosis parasite.

- **المصادر** الاساسية :
- https://openstax.org/details/books/microbiology/

- **المصادر المقترحة:**
- **https://open.umn.edu/opentextbooks/textbooks/470**
- **روابط مقترحة ذات صلة:**

https://www.amazon.com/Best-Sellers-Microbiology/zgbs/books/689716011