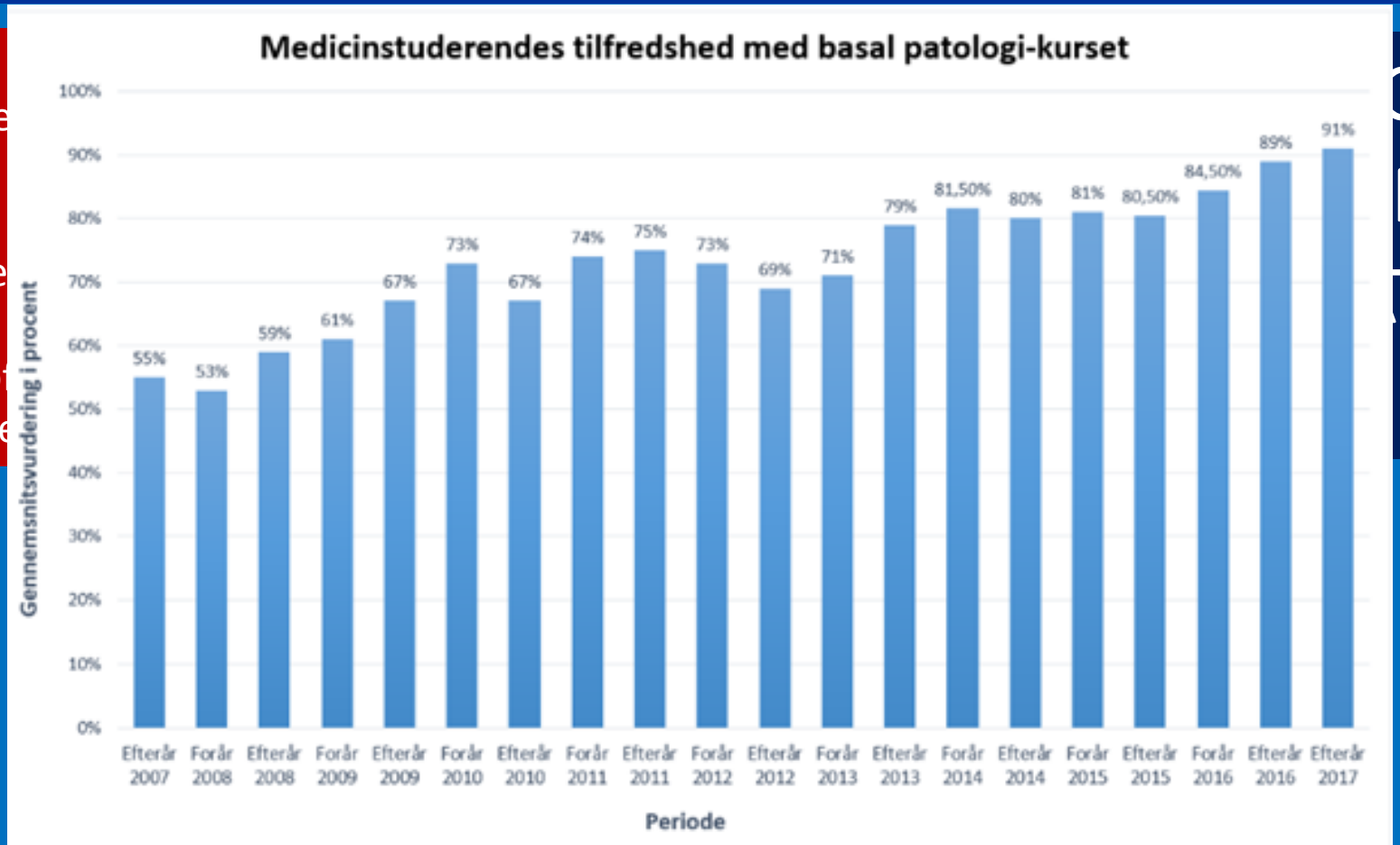
DIGITAL LÆRING

Kursus i almen patologi for stud.med. 5.sem. KU

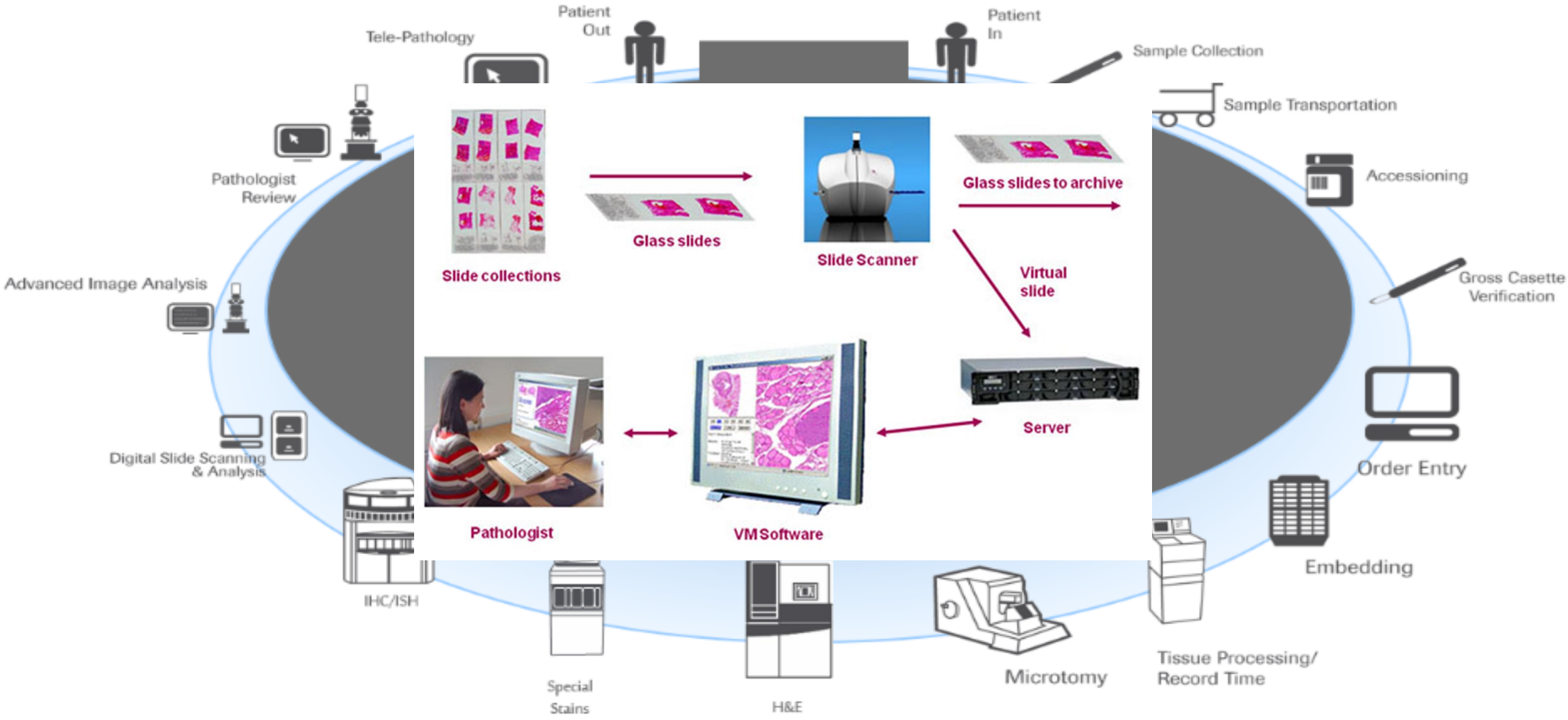
18 FORELÆSNINGER + 14 MIKROSKOPI-TIMER + 12 CASE-TIMER + 8 CAFÉ-TIMER

Curriculum Vitae
Flemming Fryd Johansen, profe

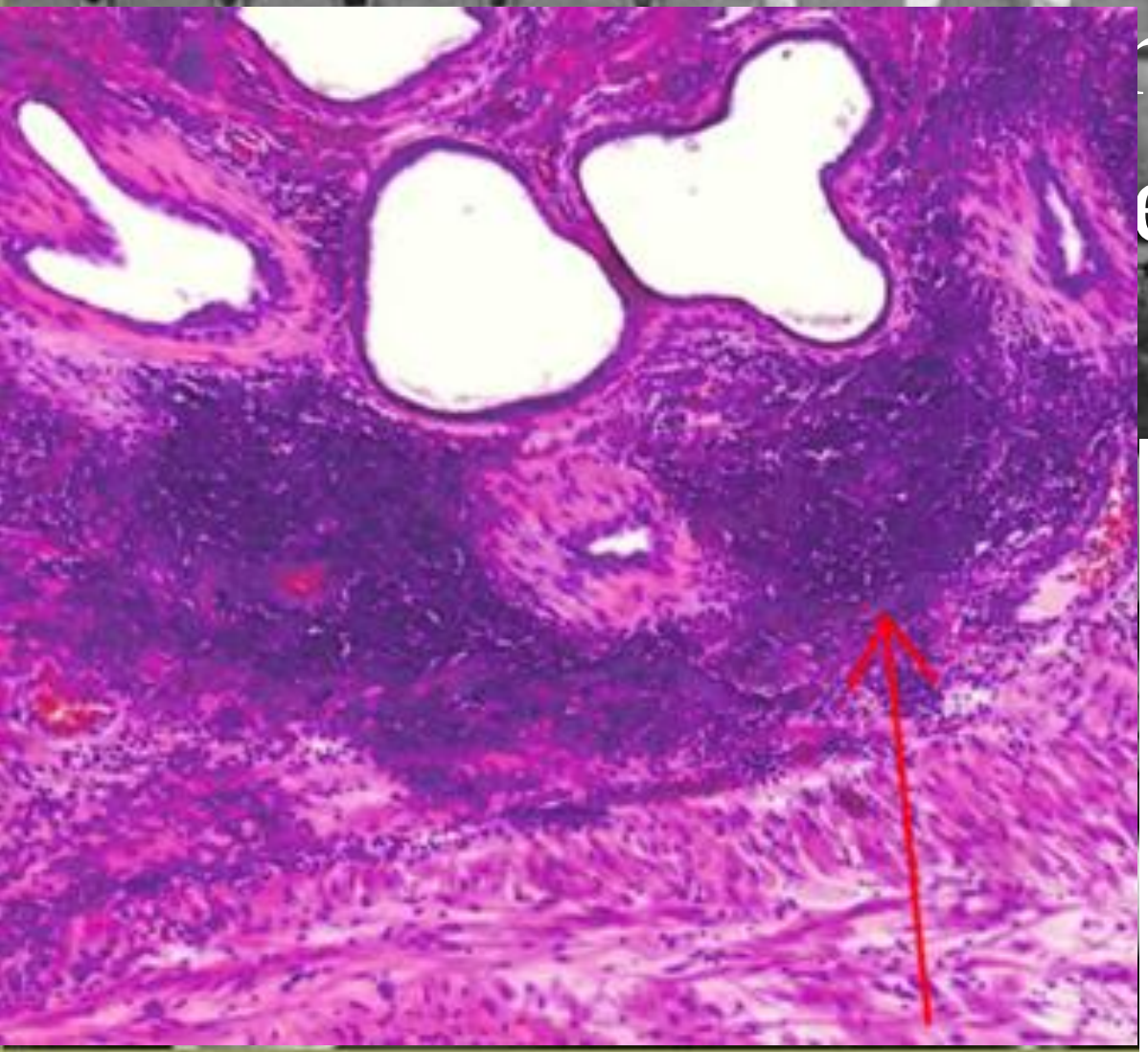
1983	MD (cand.med) KU
1984 –	Assistant & Associate
1993	DMSc (dr.med) KU
2001 –	Leader & organizer of
2010	Professor in experime



Work-flow i moderne digitaliseret patologi-laboratorium

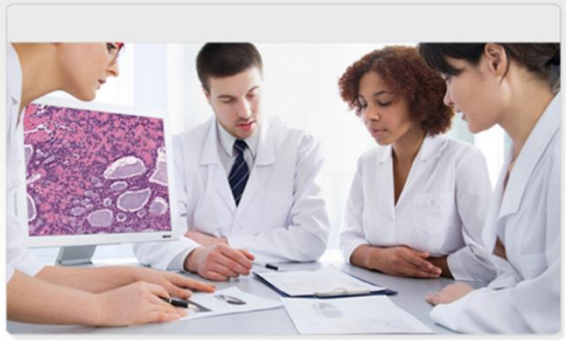


Virtuel Mikroskopi



VIRMIK

UNIVERSITY OF COPENHAGEN



Customer Login

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Password

Login

036 Steatose

Fedtlever, steatosis hepatis. Fedtdegeneration betegner ophobningen af fedt i parenkymceller og kan ses i en lang række celler, dog oftest i leveren. Tilstanden skyldes påvirkning af cellens lipidstofskifte, så der ophobes triglycerid intracellulært. Denne påvirkning kan skyldes en lang række årsager, de hyppigste

Open Case

037a Amyloidose i leveren

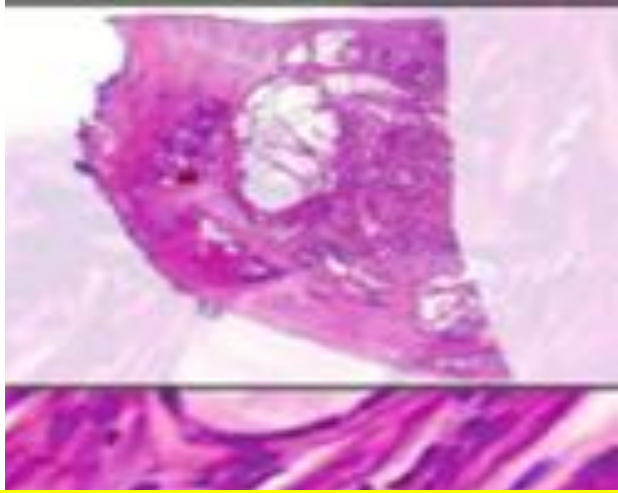
Amyloidosis hepatis. Amyloid er en ekstracellulær aflejring af fibre bestående af abnormt foldet protein. Amyloidose er en tilstand, hvor sådanne aflejringer forårsager inflammation, vævsskade og påvirkning af organfunktionen. Amyloid er i mikroskopet en homogen masse, som i HE-farvningen er mørkrød/lilla. Farves der

Open Case

EKSAMEN i basal patologi, 5. semester bachelor, medicinstudiet, sommer 2015

Eksamenssættet indeholder 1 hovedspørgsmål og 10 multiple choice (MC)-spørgsmål. Hovedspørgsmålet vægter 50%, mens hver af de 10 MC-spørgsmål vægter 5% af den samlede besvarelse. Til hovedspørgsmålet hører to digitale mikroskopi-præparater. Mikroskopi-præparaterne åbnes med VIRMIK, som tilgås via internettet. Systemet fungerer nøjagtigt som det, der er brugt i undervisningen … …

DET ER IKKE TILLADT AT ANVENDE ANDRE BRUGERNAVNE FOR AT LOGGE PÅ VIRMIK USERNAMES – dette



Sammenhæng mellem hvad der er pensum; hvad der undervises; og hvad der eksamineres

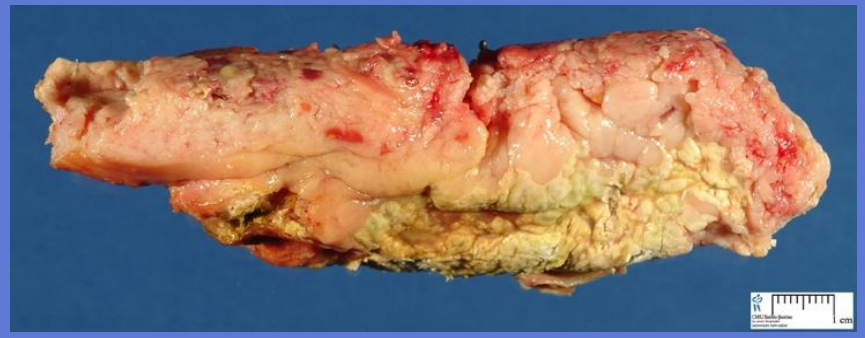
pathologyCLUB 1-4

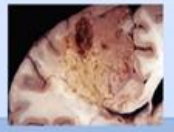
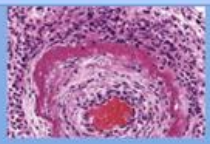
CELLE-PATOLOGI
INFLAMMATION
KREDSLØBS-PATOLOGI
NEOPLASI

CAFÉ 

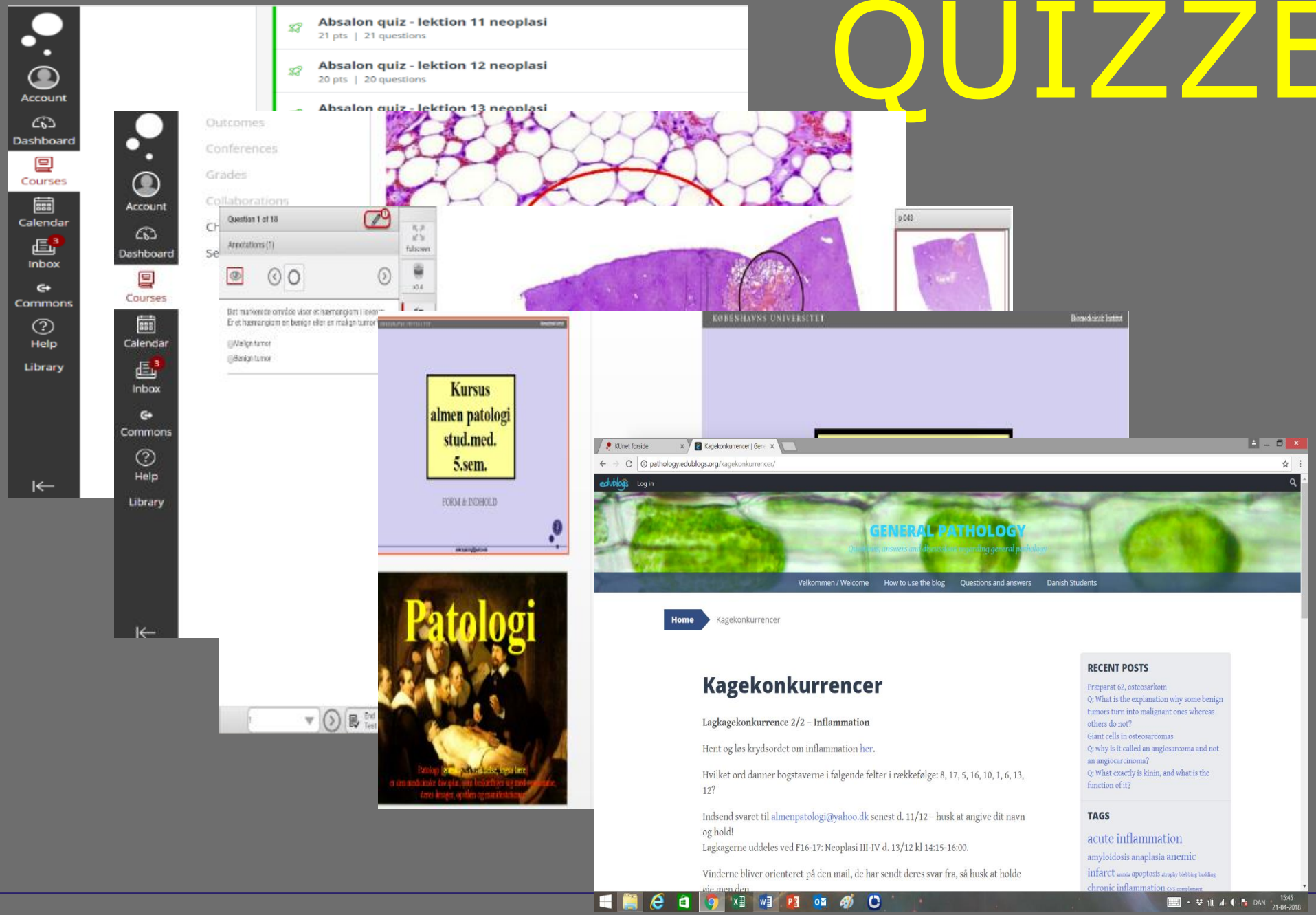
1. I vævet ses en særlig form for nekrose. Beskriv denne og angiv dens betegnelse.

Forslag til svar:
Fedtnekrose. Fremstår som konturer af fedtceller med lillafarvet (basofilt), homogent omdannet cytoplasma og med accentureret cellemembran. Der kan ses egentlig forkalkning (mørkere lilla farve, evt. sort).



Nekroseform	Beskrivelse	Lokalisering	Udseende
Koagulationsnekrose	Cellerne er døde, men vævets arkitektur er bevaret.	Infarkter i solide organer (ikke hjernen)	
Kollikvationsnekrose = Liquefactive	Inflammatorisk nedbrydning til flydende viskøs masse.	Infarkt: i CNS Infektion: fokal	
Kaseøs nekrose	Gulligt-hvidligt udseende ansamling bestående af fragmenterede/lyserede celler; omgivende inflammation = granulom. Udvisket vævsarkitektur.	Tuberkulose foci (oftest lungerne, men ses i hele kroppen).	
Fedtnekrose	Fokale områder med saponificering. Mikroskopisk ses nekrotiske fedtceller omgivet af inflammation.	Pancreas og peritoneum (udløses af pancreas lipase v. akut pancreatit).	
Fibrinoid nekrose	Udfældning af antistoffer og antigener samt fibrinlækage fra karvæggen.	Arterier. Ses bl.a. v. polyarteritis nodosa.	

QUIZZER


Absalon quiz - lektion 11 neoplasi
21 pts | 21 questions
Absalon quiz - lektion 12 neoplasi
20 pts | 20 questions
Account
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Grades
Collaborations
Question 1 of 18
Kursus almen patologi stud.med. 5.sem.
Patologi
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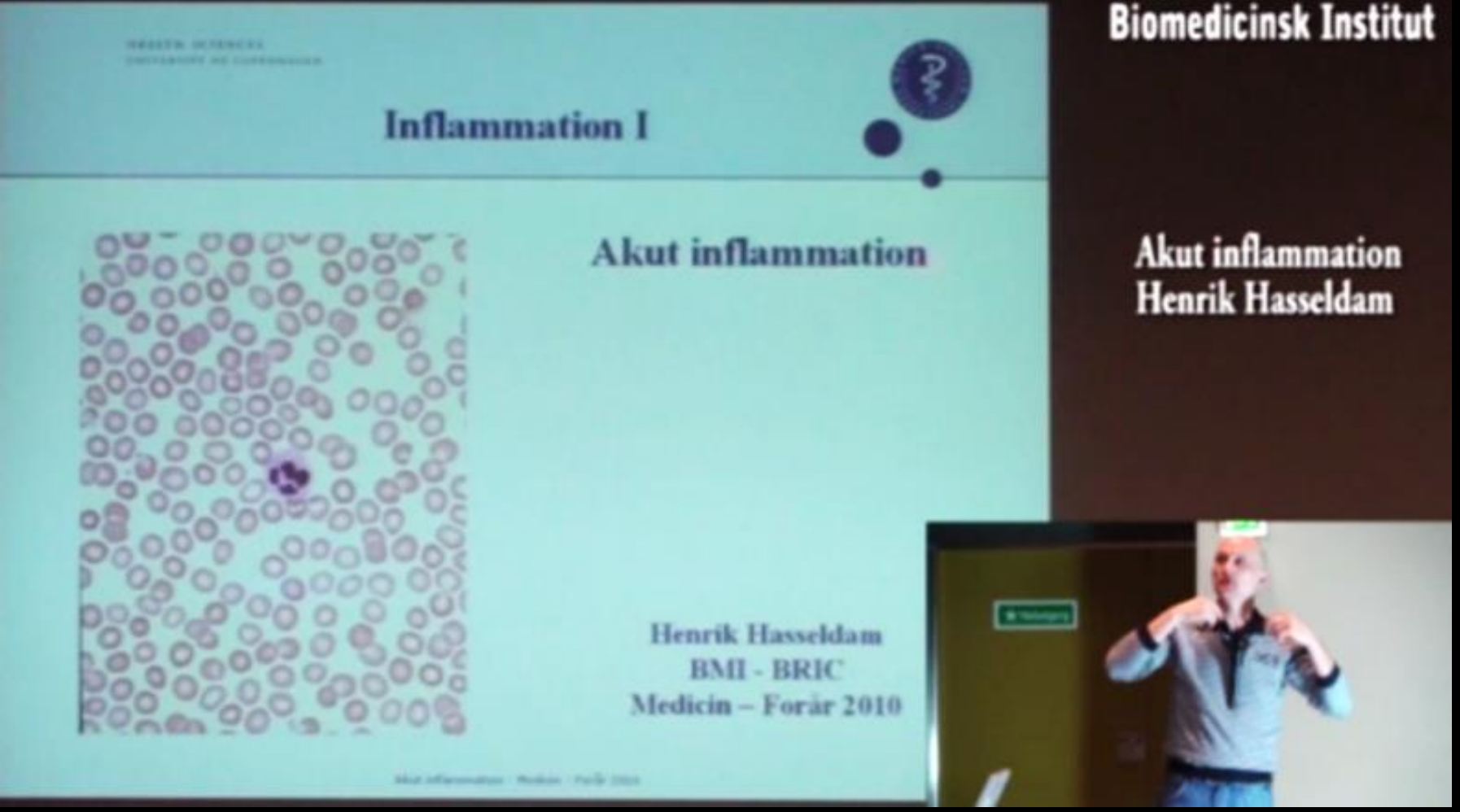
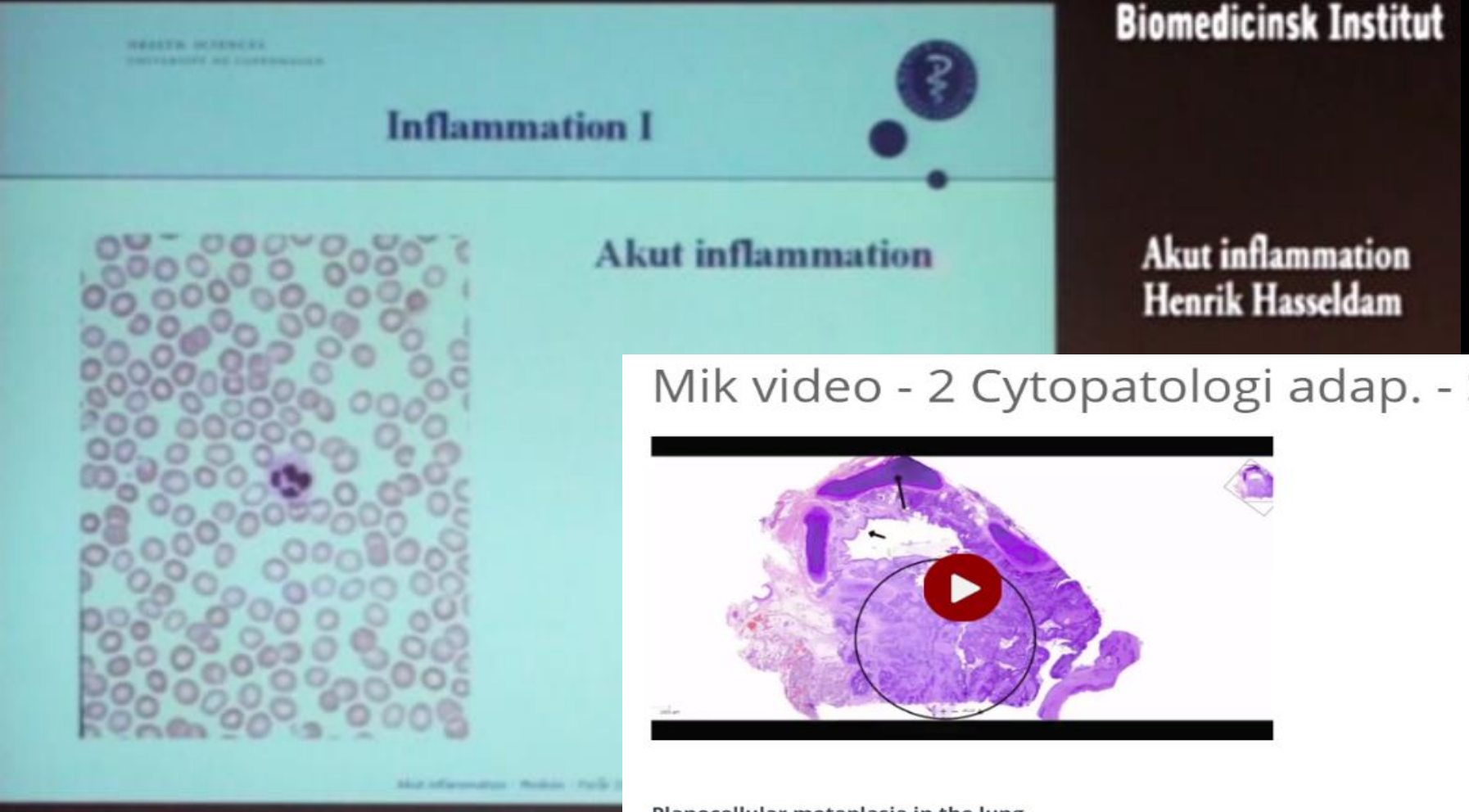
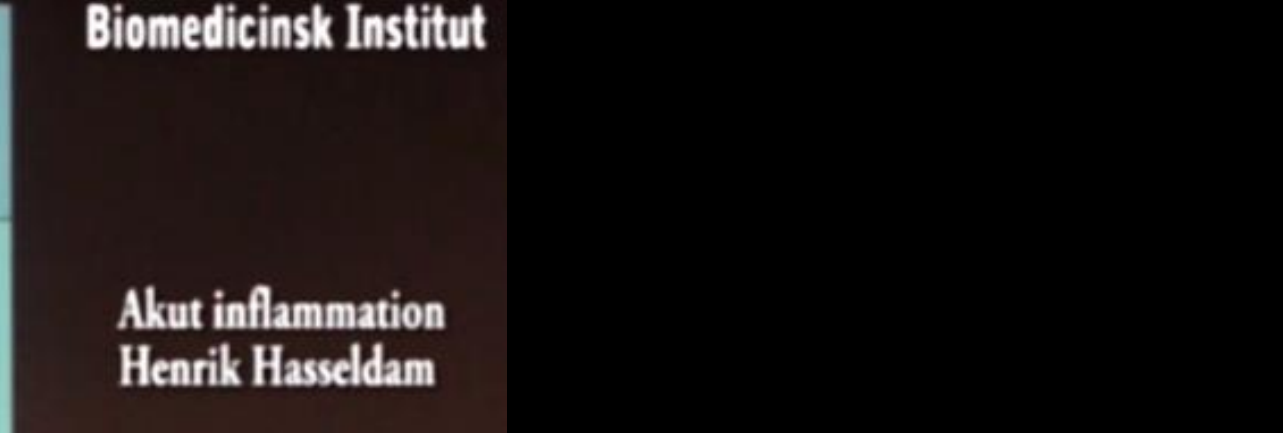
pathology.edublogs.org/kagekonkurrencer/
GENERAL PATHOLOGY
Velkommen / Welcome
How to use the blog
Questions and answers
Danish Students
Home
Kagekonkurrencer
Kagekonkurrencer
Lagkagekonkurrence 2/2 – Inflammation
Hent og løs krydsordet om inflammation her.
Hvilket ord danner bogstaverne i følgende felter i rækkefølge: 8, 17, 5, 16, 10, 1, 6, 13, 12?
Indsend svaret til almenpatologi@yahoo.dk senest d. 11/12 – husk at angive dit navn og hold!
Lagkagerne uddeles ved F16-17: Neoplasi III-IV d. 13/12 kl 14:15-16:00.
Vinderne bliver orienteret på den mail, de har sendt deres svar fra, så husk at holde øje med den.
RECENT POSTS
Præparat 62, osteosarkom
Q: What is the explanation why some benign tumors turn into malignant ones whereas others do not?
Giant cells in osteosarcomas
Q: why is it called an angiosarcoma and not an angiocarcinoma?
Q: What exactly is kinin, and what is the function of it?
TAGS
acute inflammation
amyloidosis anaplasia anemic
infarct anoxia apoptosis atrophy blebbing budding
chronic inflammation



VIDEO & LIVE STREAMING

‘CHAT’





EMNE VIDEO

Mik video - 2 Cytopatologi adap. - Specimen 7

Planocellular metaplasia in the lung

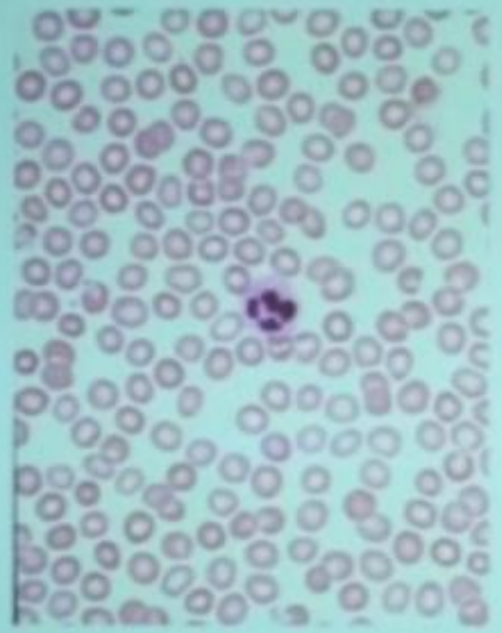
Here we have example of planocellular metaplasia in the lung from a patient with planocellular carcinoma. At low magnification, we see a bronchus with cartilage and epithelia towards the lumen. The major part of this section is occupied by the planocellular carcinoma that has invaded the lung tissue. Note that planocellular carcinoma is always preceded by metaplasia because squamous epithelium is not normally found in a healthy lung.

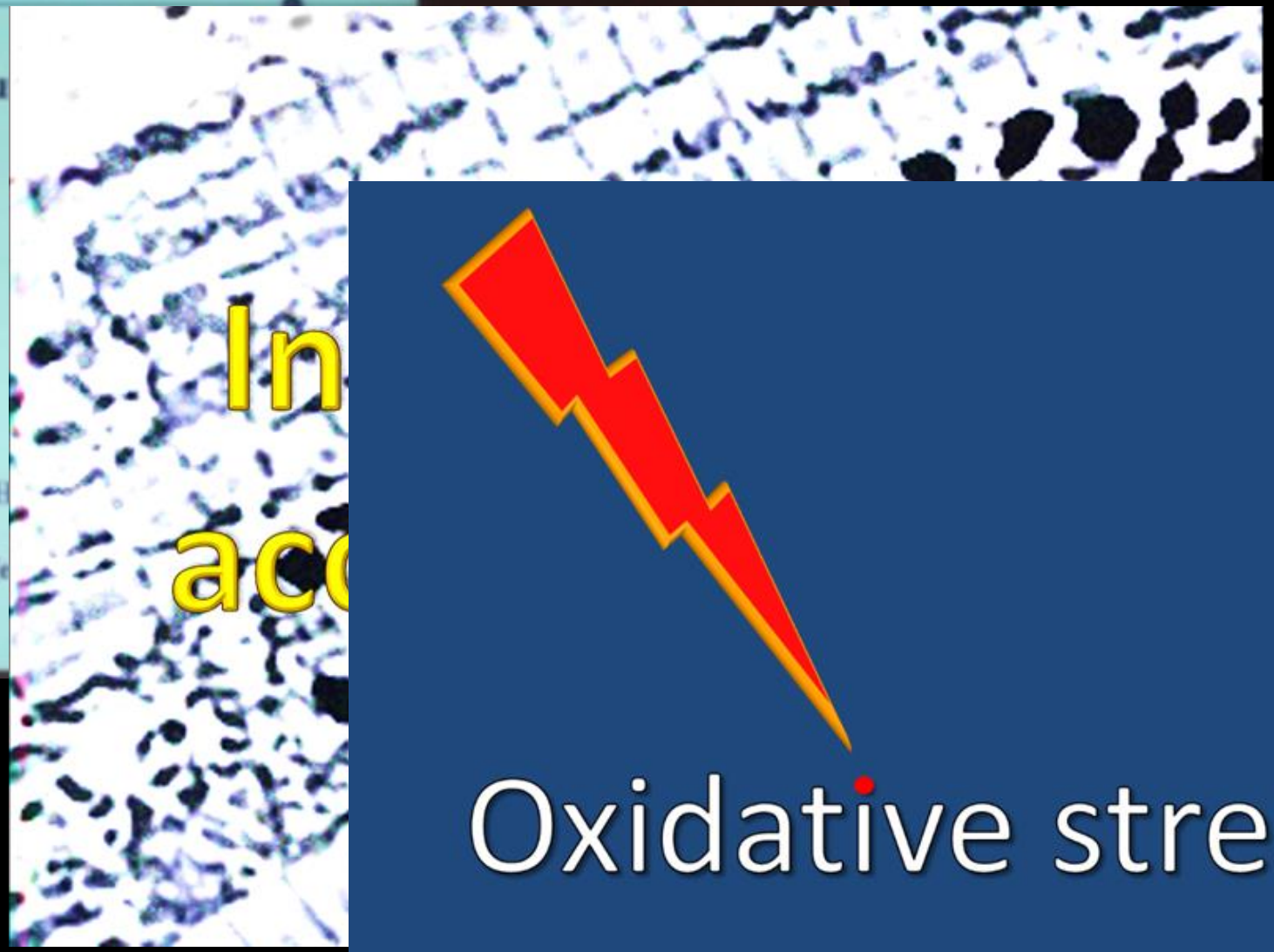
You can also see peribronchial glands here, adipocytes here, and compressed alveoli here.

The normal respiratory epithelium is pseudo-stratified with cilia. However due to metaplasia the pseudo-stratified epithelium has transformed into squamous epithelium. The transformation is not complete because cilia are still found. It can be a little difficult to identify that this is squamas epithelium but there are some degree of flattening towards the lumen. The metaplasia depends on changes in protein expression, without gene mutation, and thus metaplasia is reversible.

Biomedicinsk Institut

Inflammation I





Oxidative stress

EMNE VIDEO

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Welcome | How to use the blog | Questions and answers | Danish Students

- Cytopathology
- Inflammation
- Circulatory disturbances
- Neoplasia

Welcome

Neoplasi, neoplasi, neoplasi.

Har du fuldstændig styr på neoplasierne, eller mangler du lidt opsummering? Så kan du finde svar på mange spørgsmål her: http://pathology.edublogs.org/neoplasia/

Det er også muligt at stille nye spørgsmål i boksen nedenfor. De bliver løbende postet og besvaret af kursets undervisere.

RECENT POSTS

Giant cells in osteosarcomas
Q: why is it called an angiosarcoma and not an angiocarcinoma?
Q: What exactly is kinin, and what is the function of it?
Udspecificering af pensum (CNS patologi)
Q: classical and alternatively activated macrophages

TAGS

acute inflammation
amyloidosis anaplasia anemic
infarct apoptosis
chronic inflammation
criteria of malignancy deep venous

Question on the transition between benign and malignant tumors in relation to mutations.

February 26, 2013 | Neoplasia | criteria of malignancy, reversible/irreversible adaptations

We have discussed the transition between benign and malignant tumors in relation to mutations, and further mutations in relation to the various forms of adaptations described in pathology

One teacher explains that all human cancers have 5-10 rate limiting mutations which make them malignant. If the cells have fewer mutations than required to be an invasive cancer, they might be hyperplasic, dysplastic, or form a benign tumor. Each of these stages may become malignant. For myeloma and lipoma transition is rare, for polyps in the colon more frequent and for villous adenoma in the colon even more frequent.

Another teacher explains that metaplasia is an adaptative and reversible process where one "well-differentiated" cell type is replaced by another "well-differentiated" cell type from the same germ layer. By well-differentiated "I mean not permanently changed for example by genetic mutations". Dysplasia occurs after genetic changes including formation and activation of oncogenes or disappearance or inactivation of tumor suppressor genes and is a precursor of malignancy.

between benign and malignant tumors in relation to mutations."

Ben Vainer
March 5, 2013 at 11:49 pm

It is a question on how to define a mutation. A mutation is not a genetic alteration that is discovered and corrected by the cell – a mutation is a permanent alteration in the gene (i.e. not reversible). The adaptive changes, i.e. atrophy, hypertrophy, hyperplasia and metaplasia, are caused by changes in the external environment and does not need genetic changes in the cell type in question to occur.
It is important to stress, that dysplasia is NOT an adaptive change, but occurs only after permanent genetic mutation in the genome. In some books dysplasia is mentioned as an adaptive change, but this is possibly because early dysplasia is "reversible", that is the cells are shed off the surface and removed – the genetic change is still permanent.

Is this answer enough? – otherwise, please feel free to write again.

Reply

Marie Kveiborg
March 5, 2013 at 11:53 pm

Additional note to Johannes' Q:

Malignancy is not defined by number of mutations, but is based on the degree and severity to which the histological and cytological criteria of malignancy are observed. Yet, malignancy is thought to arise as a result of accumulating

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GENERAL PATHOLOGY

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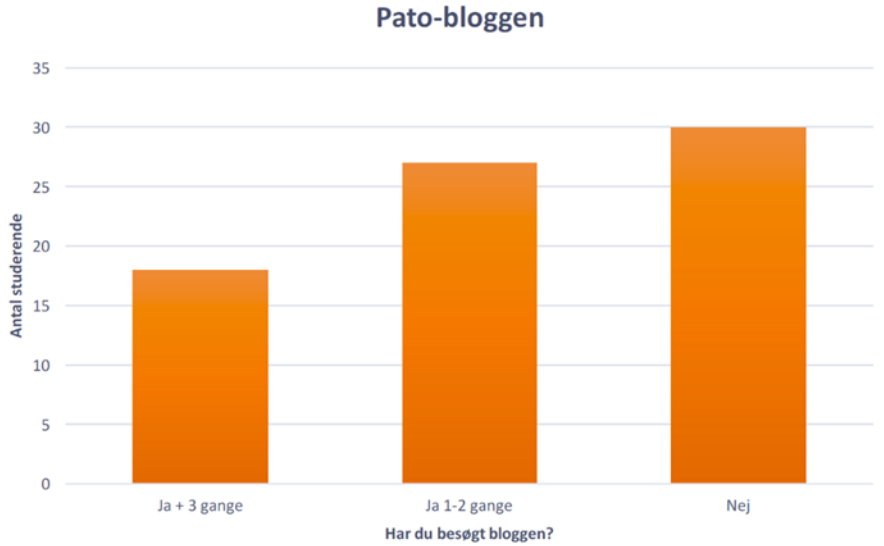

nde postet og

TAGS

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criteria of malignancy deep venous

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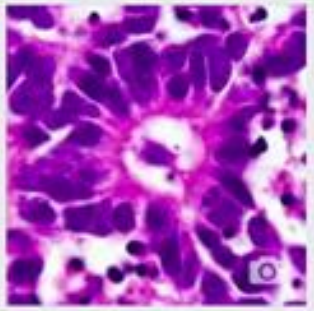
Side | Indbakke 1 | Notifikationer 4 | Indblik | Værktøjer til offentliggørelse | Indstillinger | Hjælp



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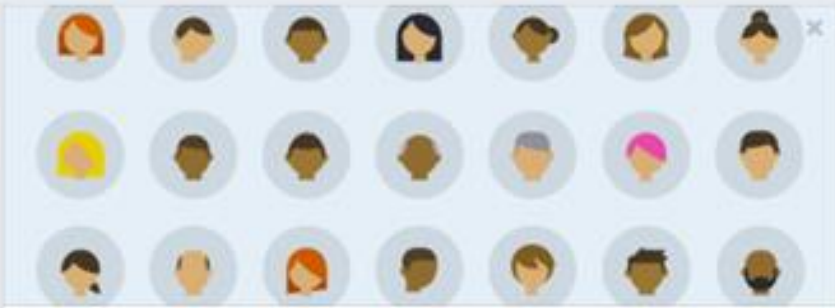
Synes godt om | Følg | Del | ...

Opret et opslag

Skriv noget ...



Du har fået 200 synes godt om for side. Godt gået!

Opret et opslag, hvor du takker folk, som synes godt om din side.

Opret opslag

Denne uge

18 ↑	0	17 ↑
Rækkevidde for opslag	Klik til website	Opslagsinteraktion

+ Tilføj en knap

Jakob Bjerager synes godt om dette.

Udvid din målgruppe på Facebook, og nå ud til flere personer, som er interesseret i din virksomhed.

Inviter venner

Vores historie

Denne side har til formål at oplyse medicinstuderende på femte semester ved KU om pensumrelevant inf...

+ Afslut din historie for at fortælle folk mere om din virksomhed.

Svarrate på 75 %, svartid på 22 timer
Svar hurtigere for at slå badget til

204 Synes godt om + 9 denne uge
Jakob Bjerager

KONTAKT SIDER | SE ALLE

KONTAKTER


Michael Christensen


Niels Thorkild Levinsen


Nickolai Damgaard


Wladek Marchwinski

Ellis Hinz-Berg 14t


Paola Ceoldo 9t


Troels Riis Jørgensen


Judith Frei 2t


Jette Guldfeldt 11t


Marianne Marianne Thyge...

Peter Kjærgaard 2d

Søg

DAN 11:04 22-04-2018

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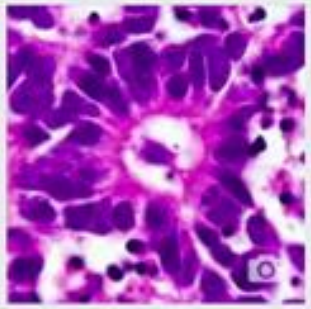

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Fællesskab
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Administrer promoveringer

Synes godt om | Følg | Del | ...

+ Tilføj en knap

Billeder

Hold dig opdateret om sider, du synes godt om som din side. ×

Se alle

Vores historie

204 personer synes godt om denne side, og 219 personer følger den
Jakob Bjerager

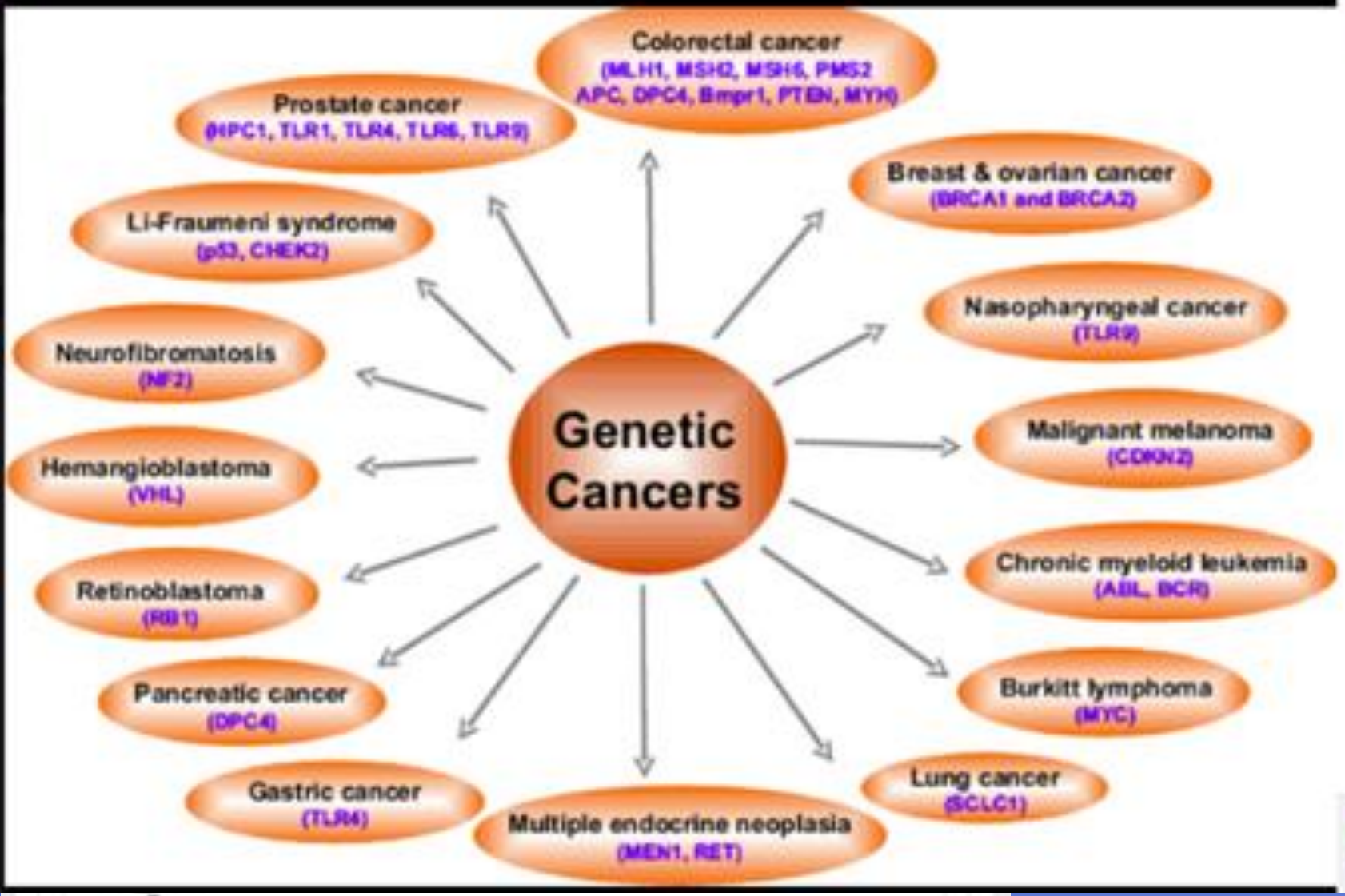
Fællesskab Se alle

KONTAKT SIDER SE ALLE

KONTAKTER
Michael Christensen
Ellis Hinz-Berg 13t
Paola Ceoldo 9t
Judith Frei 3t
Jette Guldfeldt 11t
Marianne Marianne Thyge...
Peter Kjærgaard 2d
Henrik Hasseldam 13t
Mette Gotthardt 23m
Eva Krohn Svendsen 5t
Lone Bartholomæussen

Søg

DAN 11:03 22-04-2018



Basal Patologi KU

Offentliggjort af Adile Orhan [?]

Synes godt om denne side · 10. januar ·

Patients with the Li-Fraumeni syndrome have a 25-fold greater chance of developing a malignant tumour by 50 years of age compared with the general population. The spectrum of tumours that develop in patients with Li-Fraumeni syndrome is much varied. The most common types are sarcomas, breast cancer, leukaemia, brain tumours, and carcinomas of the adrenal cortex.

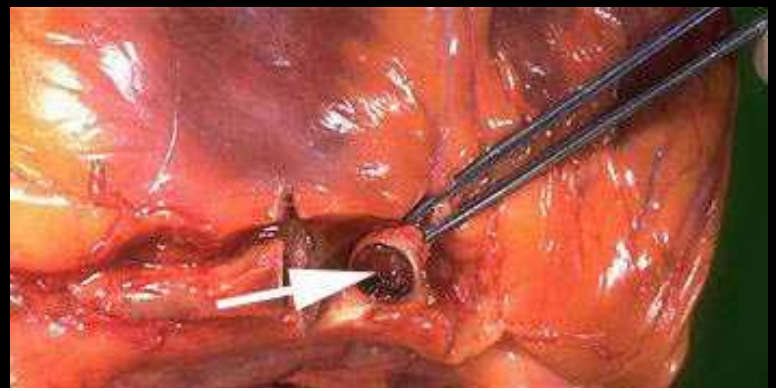
(s. 212, 10th edition of Robbins Basic Pathology)

Tag billede · Tilføj lokation · Rediger

Synes godt om · Kommenter · Del

Din kommentar...

CASE



GRUPPEARBEJDE

Case 1 – Cytopatologi

INFORMATIONSHÆFTE

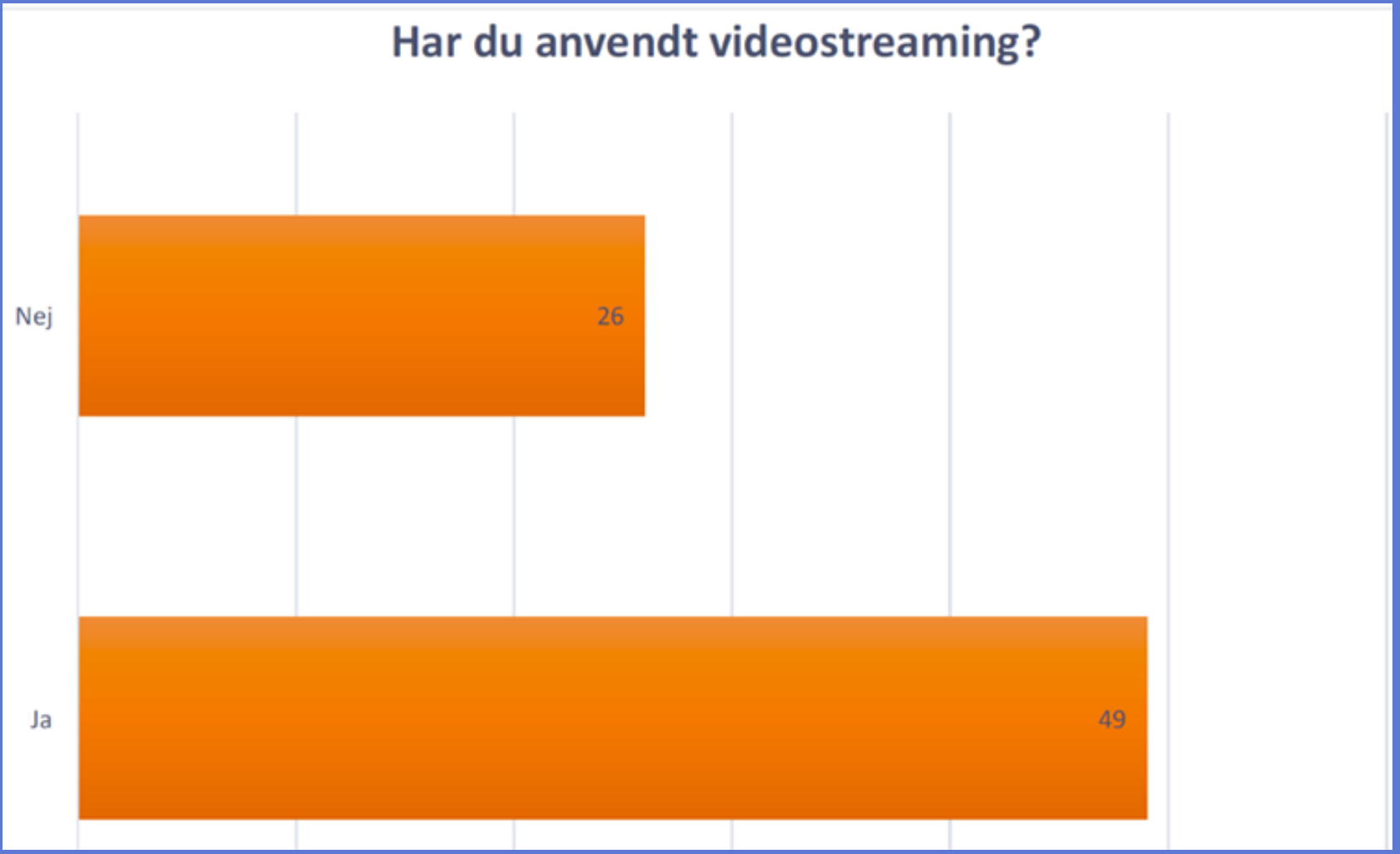
Anbefalet læsning: Kapitel 1

Videoforelæsninger: Cytopatologi I: Cellebeskadigelse, adaptation og nekrose (EFJ) og Cytopatologi II: Celledødsmekanismer (RSR).

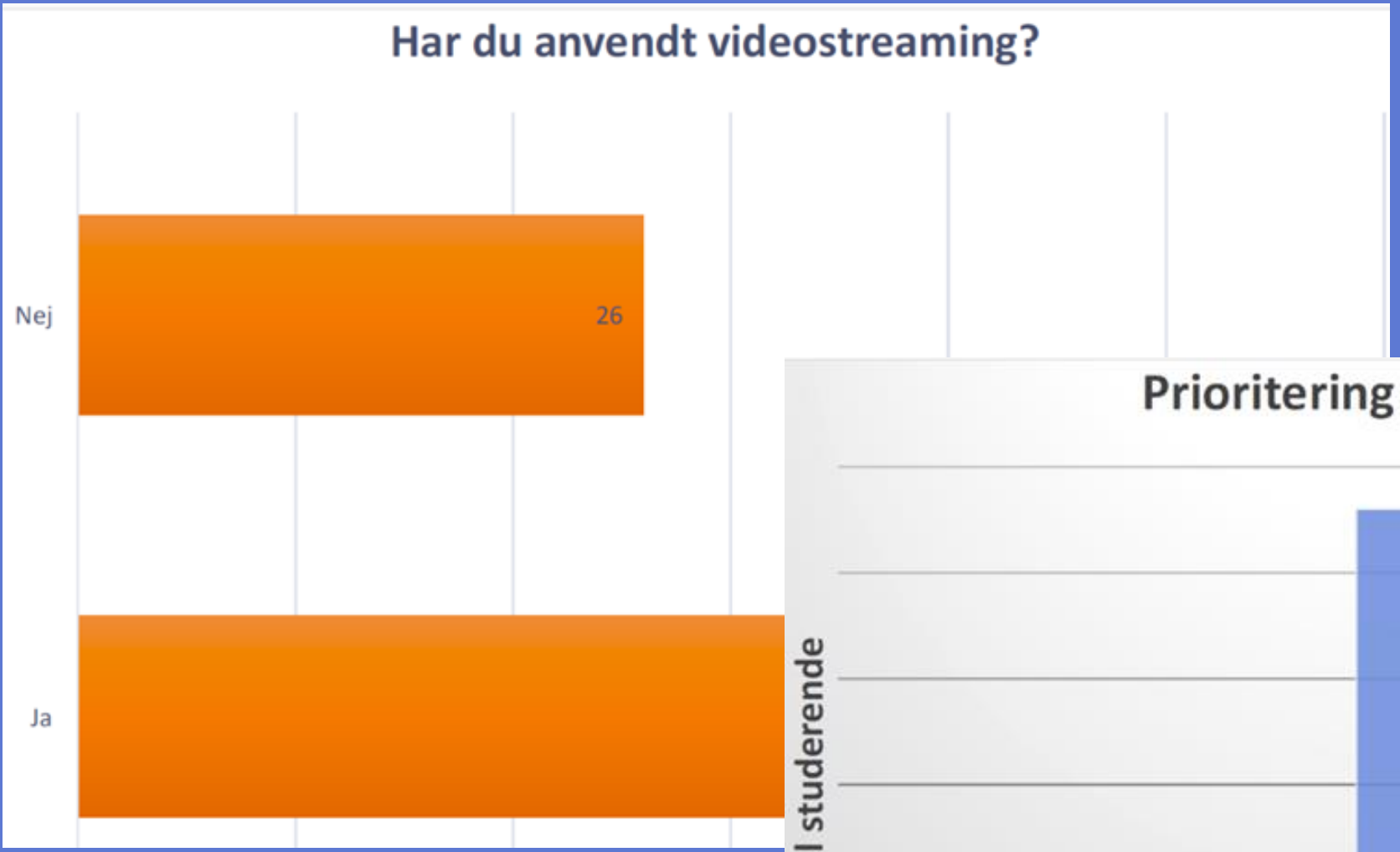
Forberedende spørgsmål:

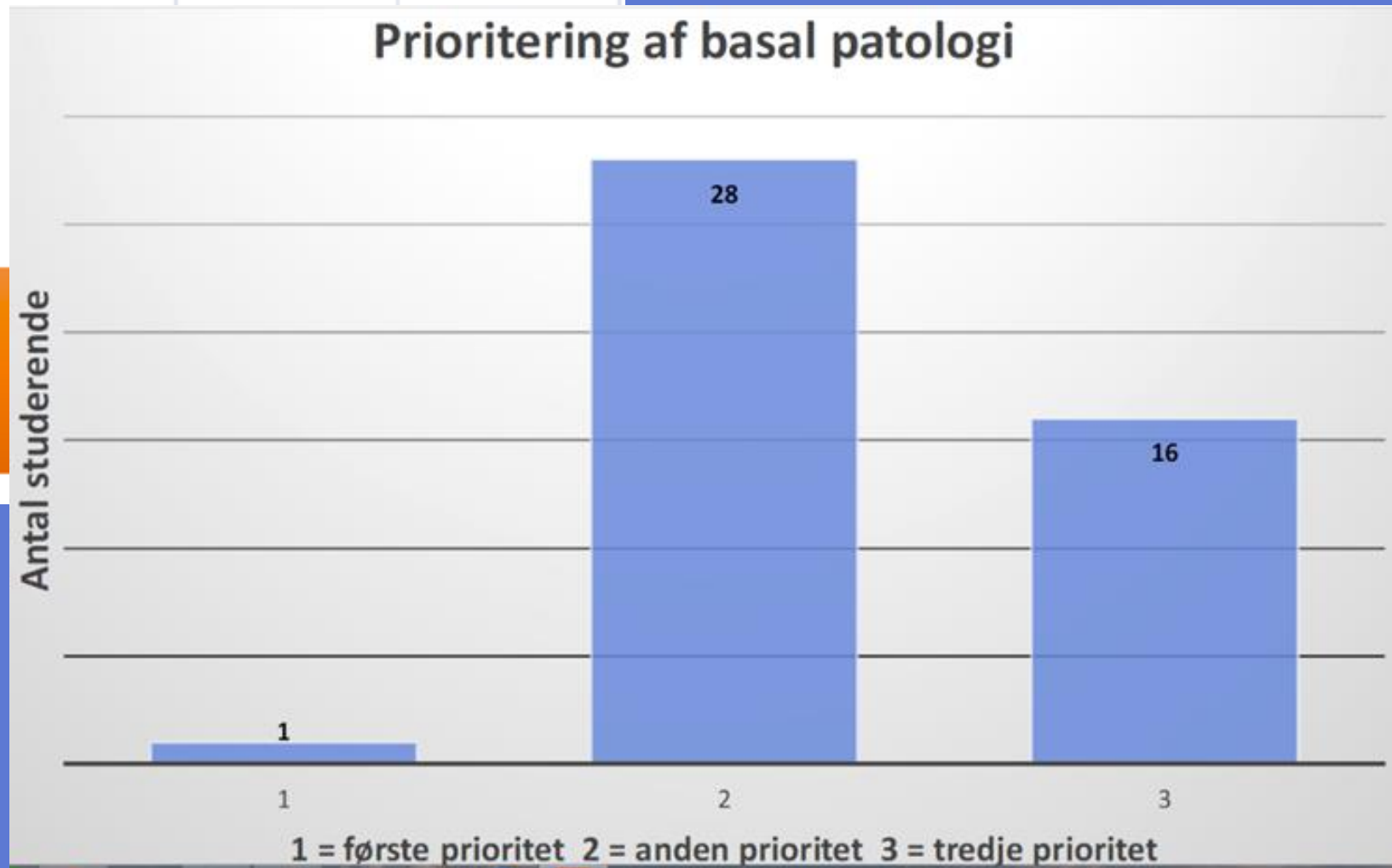
1. Hvad dækker begrebet "cellulær adaptation" over? Find, beskriv og forklar eksempler på hypertrofi, hyperplasi og atrofi.
2. Definér, differentier og forklar ved eksempler metaplasi og dysplasi.
3. Redegør for histopatologiske forandringer ved steatose og hyaline forandringer.
4. Redegør kort for patogenese og find eksempler på typiske organskader ved onkose (=nekrose).
5. Beskriv den indre signalvej ved apoptose.
6. Beskriv den ydre signalvej ved apoptose.
7. Redegør for, og find eksempler på, kollikvationsnekrose og koagulationsnekrose.
8. redegør for, og find eksempler på, kaseøs og hæmoragisk nekrose – samt for våde og tørre gangrene tilstande.

INTERN EVALUERING



INTERN EVALUERING





ERFARINGER MED AT DIGITALISERE UNDERVISNINGEN AF FAGET ALMEN PATOLOGI PÅ MEDICINSTUDIET PÅ KØBENHAVNS UNIVERSITET


Strengths
Hvad er dine 3-4 vigtigste styrker?
Styrker
Strenghts
Svagheder
Weaknesses
Opportunities
Hvad er de vigtigste muligheder eller potentialer i fremtiden for din virksomhed?
Muligheder
Opportunities
Trusler
Threats

That's all for now folks