

Dr.Diala

➤ Sheet 4

Page 2 :

some proteins that are found in the inner membrane--1-emerin #2- laminin binding protein also called laminin B receptor (LBR)

LIP واحذفو

#3- link ---> LINC بالاحرف الكبيرة

Page 3

emery-dreifuss muscular dystrophy غيروها ل "muscular dystrophy" اخر سطر

Page 4 :

heart failure السطر الخامس اخر كلمة

Page 10

Ran-GDP binds the importin to release .. بالمربع اللي تحت مكتوب

مهم جدا ل Ran-GTP ل Ran-GDP غيرو

Page 10

Ran-GTP: binds the exportin to activate the binding of the exportin to the transported protein اخر نقطة مش مكمل .. كملوها

Page 11

بالمربع الموجود بنص الصفحة , رقم 2 مش مكمل .. كملوها

conformational changes --> exposure of NLS or NES

➤ Sheet 22

Page 10,

Line 7,

"For example, if they are involved in movement as in the case of LAMELLIPODIA formation,..."

(Not pseudopodia)

➤ Sheet 23

Page 5

the spectrin composed of two alpha and one beta and alpha domain having two ca binding domaine

➤ Sheet 29

➤ The difference between meiosis in males and females, that meiosis produces 4 sperms in males, but in females it produces only 1 ovum and 3 polar bodies (Inactive-X-chromosome).--

- don't mix them with the "barr body" which is inactivated X chromosome.

4

➤ Sheet 31

Colorblindness is "X-linked RECESSIVE" disease, not dominant.

➤ Sheet 32

page 7

The sheet says that a mosaic karyotype would be 46, XY, +21 [10]
but the book and slides say it's 46, XY[10]

➤ Sheet 33

خطوا محلها galactosidase صفحة ٢؛ النقطة السوداء قبل الأخيرة، بس بدل كلمة - ١
galactokinase

لازم تكون lactase intolerant صفحة ٤؛ داخل المستطيل في الأسفل، النقطة السوداء الأولى، بدل - ٢
lactose intolerant

'cause' لازم يكون في كلمة common صفحة ١٠؛ النقطة السوداء الثانية، بس بعد كلمة - ٣

➤ Sheet 35

Genetics 35 (correction)/page 12/line 12:
A1 instead of A2

➤ Sheet 36



Nisreen Asha

1 min



Clarifications for sheet 36 genetics:

- Page 7, Adoption studies:

Second paragraph, 9th line; it won't be precise to say that it all depends on GENETICS.

- Page 9, last paragraph: (the picture below)

In addition, a small number of FH cases are caused by a ¹⁾ mutations in the gene that encodes PCSK9, an enzyme that plays a key role in degrading LDL receptors. People who have a ²⁾ mutation in PCSK9 actually have a very good lipid profile, very low LDL and very high HDL because the LDL receptors won't be destroyed and would remain on the surface, so now a new drug has been developed that inhibits PCSK9, which can help the general population (those with high cholesterol levels), as it would increase LDL receptors and thus decrease the circulating LDL levels.

1) Gain of function mutation

2) Loss of function mutation