شَرَفُ الْعِلْمِ وَ شَرَفُ الْمَكَانِ

د / فيصل بن أحمد العلاف
أستاذ علم الوراثة والطب الجزيئي المشارك
كلية الطب – جامعة أم القرى

الملتقى الأول للبحث العلمي – 20 -21 ربيع الأول 1438هـ

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المرجعية العلمية
الأثر على المجتمع والإقتصاد
تطوير المهارات
تهيئة البيئة البحثية
تطوير الطرائق
توطين التقنية
نقل المعرفة
المشاركة في المؤتمرات العلمية
عدد وجودة النشر
الإكتشاف ات العلمية

الْبَرْنَامَجُ الْوَطَنِيُّ لِلتَّعَرُّفِ عَلَى الْاعْتِلَالَاتِ الْوِرَاثِيَّةِ الْمُسَبِّبَةِ لِمَرَضِ ارْتِفَاعِ كُولِيسْتُرُولِ الدَّمِ الْعَائِلِيِّ فِي الْمُجْتَمَعِ السُّعُودِيِّ وَتَطْوِيرُ اخْتِبَارٍ لِلتَّشْخِيصِ الْجُزَيْئِيِّ

(08-BIO34-10)

ارْتِفَاعِ كُولُسْتُرُولِ الدَّمِ الْوِرَاثِيِّ الْعَائِلِيِّ

أَثْبَتَتْ الدِّرَاسَاتُ الْمُتَزَايِدَةُ الَّتِي أُجْرِيَتْ عَلَى بَعْضِ الْمُجْتَمَعَاتِ أَنَّ مَرَضَ ارْتِفَاعِ كُولُسْتُرُولِ الدَّمِ الْوِرَاثِيِّ الْعَائِلِيِّ لاَ يُشَخَّصُ بِدِقَّةٍ تَعْكِسُ الْعَدَدَ الْفِعْلِيَّ لِلْمُصَابِينَ فِي الْمُجْتَمَعِ ، كَمَا أَنَّهُ لاَ يُعَالِجُ بِصُورَةٍ كَافِيَةٍ بَعْدَ التَّشْخِيصِ. وَيَرْتَبِطُ الْمَرَضُ بِشَكْلٍ مُبَاشِرٍ بِارْتِفَاعِ نِسْبَةِ مُعَدَّلاَتِ أَمْرَاضِ الْقَلْبِ، وَالشَّرَايِينِ، وَالْوَفَاةِ الْمُفَاجِئَةِ، وَبِالتَّالِي؛ فَإِنَّ تَأَخُّرَ التَّشْخِيصِ، وَالْعِلاَجِ لَهُ عَوَاقِبُ اجْتِمَاعِيَّةٌ، وَاقْتِصَادِيَّةٌ سَلْبِيَّةٌ لاَ يُمْكِنُ تَجَاهُلُهَا.

وَمِنْ الْمُمْكِنِ الْوِقَايَةُ مِنْ الْمَرَضِ، وَعِلاَجُهُ إِذَا تَمَّ تَشْخِيصُه وَاكْتِشَافُه فِي سِنٍّ مُبَكِّرَةٍ، وَالطَّرِيقَةُ الْمُثْلَى وَالأَكْثَرُ دِقَّةً لاِكْتِشَافِ هَذَا الْمَرَضِ هِيَ: بِالْفَحْصِ الْجُزِيئِيِّ الْوِرَاثِيِّ للحمض النووي المعروف باسم ال دي إن ايه.

فِي السَّنَوَاتِ الْماضِيَةِ لَمْ يَكُنْ هَذَا النَّوْعُ مِنْ الاِخْتِبَارَاتِ مُتَوَفِّرًا فِي الْمَمْلَكَةِ الْعَرَبِيَّةِ السُّعُودِيَّةِ، وَتَكْلُفَةِ الْمَسْحِ الْجِينِيِّ لِلشَّخْصِ الْوَاحِدِ تَزِيدُ عَلَى عَشْرَةِ آلاَفِ رِيَالٍ، وَتَنْخَفِضُ التَّكْلُفَةُ فِي حَالِ تَوَفُّرِ مَعْلُومَاتٍ، وَقَوَاعِدِ بَيَانَاتِ عَنْ عَدَدِ وَنَوْعِ الاِعْتِلاَلاَتِ الْوِرَاثِيَّةِ الْمُنْتَشِرَةِ فِي الْمُجْتَمَعِ.

والجدير بالذكر أنه لا تُوجَدُ إِحْصَاءَاتٌ دَقِيقَةٌ عَنْ مُعَدَّلِ الإِصَابَةِ بِالْمَرَضِ فِي الْمُجْتَمَعِ السُّعُودِيِّ، لَكِنَّ نَمَطَ الْحَيَاةِ، وَالْعَادَاتِ وَالتَّقَالِيدَ الاِجْتِمَاعِيَّة، وَالنِّسْبَةَ العَالِيَة لِلتَّزَاوُجِ بَيْنَ الأَقَارِبِ الَّتِي تَزِيدُ عَلَى 50% مَعَ ارْتِفَاعِ مُعَدَّلِ الإِصَابَةِ بِأَمْرَاضِ الْقَلْبِ، وَالشَّرَايِينِ تُوحِي بِأَنَّ عَدَدَ الْمُصَابِينِ يَزِيدُ عَلَى خَمْسِينَ مُصَابٍ فِي فِئَةِ الأَطْفَالِ، وَيَتَرَاوَحُ مَا بَيْنَ 46 - 230 أَلْفِ مُصَابٍ مِنْ فِئَةِ الْكِبَارِ مُعْظَمُهُمْ غَيْرُ مُدْرِكِينَ لإِصَابَتِهِمْ.

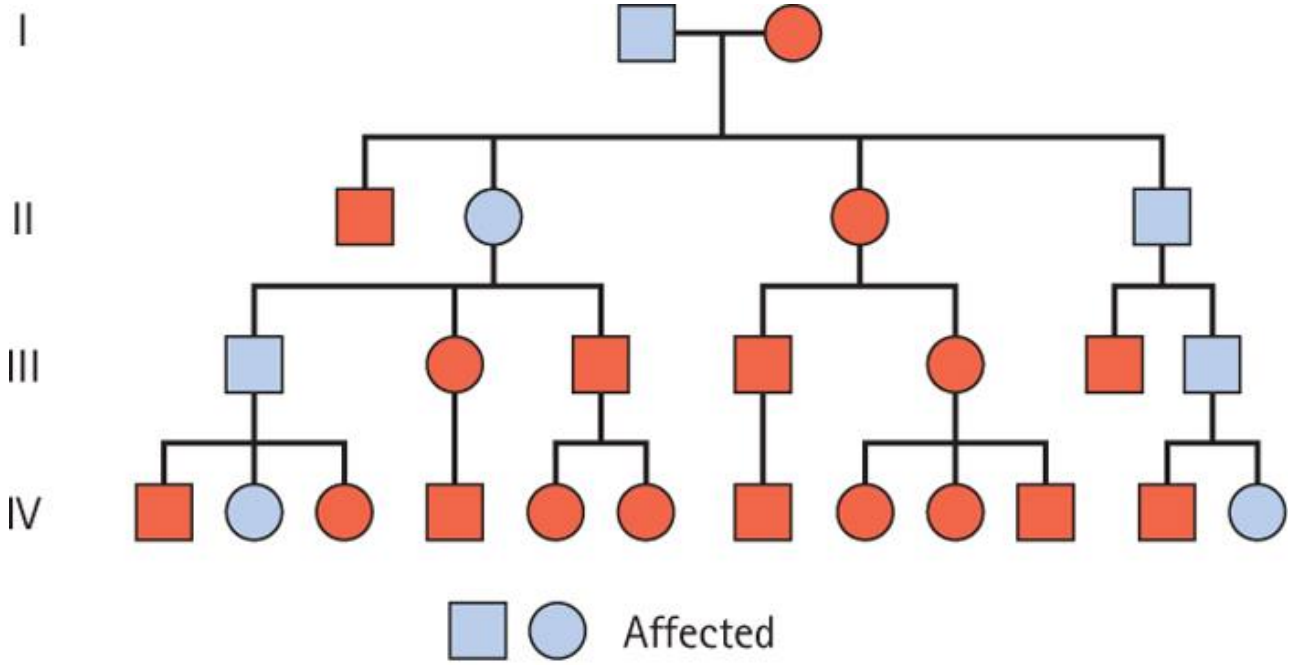
فِي السَّنَوَاتِ الْخَمْسِ الْمَاضِيَةِ قُمْنَا بِدِرَاسَةٍ وَطَنِيَّةٍ؛ لِلتَّعَرُّفِ عَلَى الأَعْتِلاَلاَتِ الْوِرَاثِيَّةِ الْمُسَبِّبَةِ لِمَرَضِ اِرْتِفَاعِ كُولِيسْتُرُولِ الدَّمِ الْعَائِلِيِّ فِي الْمُجْتَمَعِ السُّعُودِيِّ، وَتَطْوِيرِ اخْتِبَارَاتِ التَّشْخِيصِ الْجُزِيئِيِّ بِمَا يُقَلِّلُ التَّكْلُفَة، وَيَمْنَحُ الْفُرْصَة لِلْعَائِلَةِ الشَّائِعِ فِيهَا الْمَرَضُ أَنْ يُكْرِمَهَا اللهُ بِطِفْلٍ سَلِيمٍ.

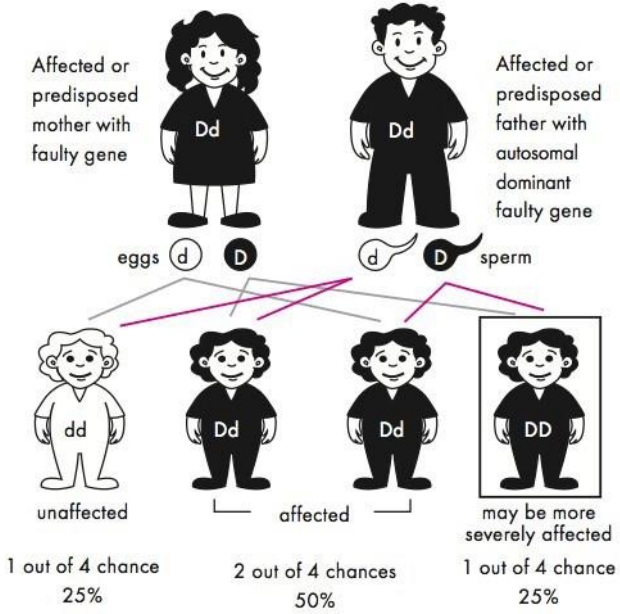
نمط التوريث للصفات السائدة

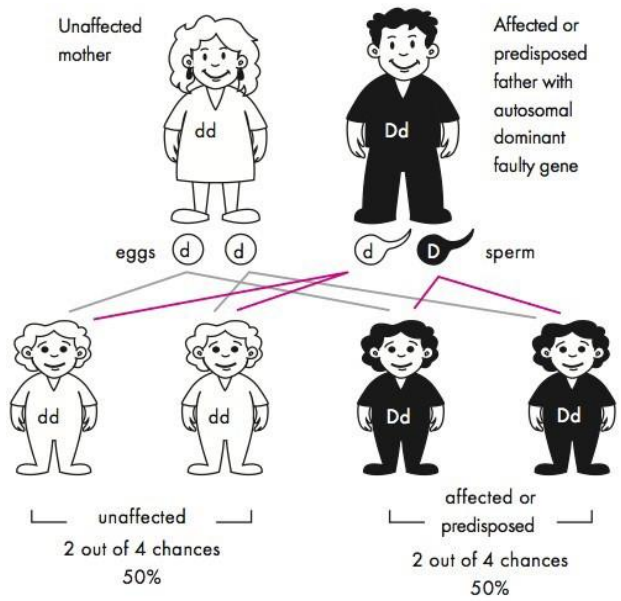
LDLR (the plasma membrane Low Density Lipoprotein Receptor)

ApoB (Apolipoprotein B 100)

PCSK9 (the neural apoptosis regulated convertase 1)

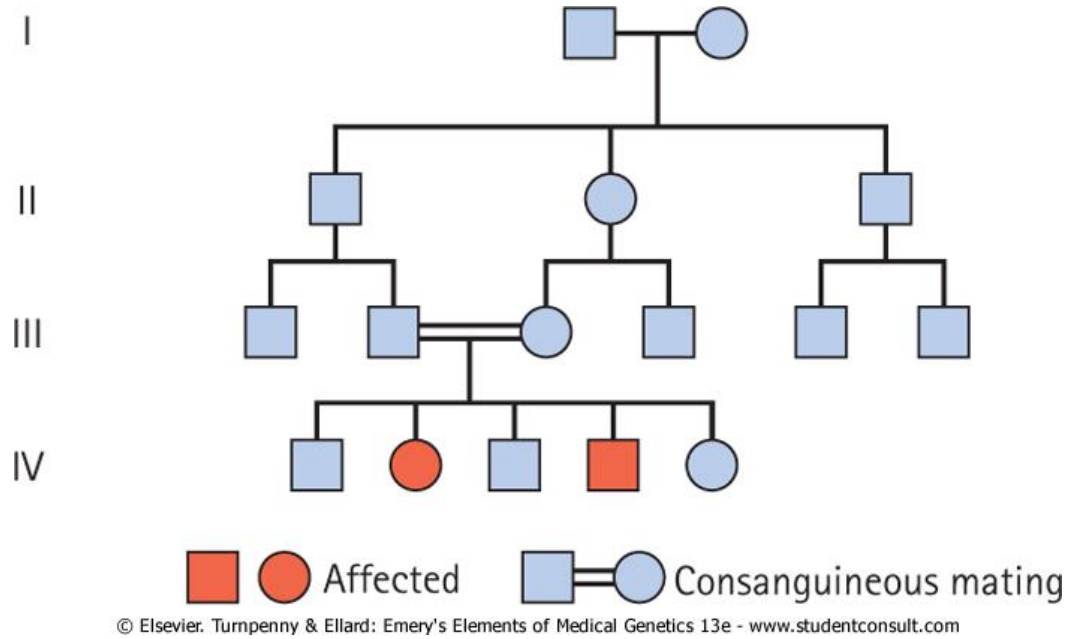


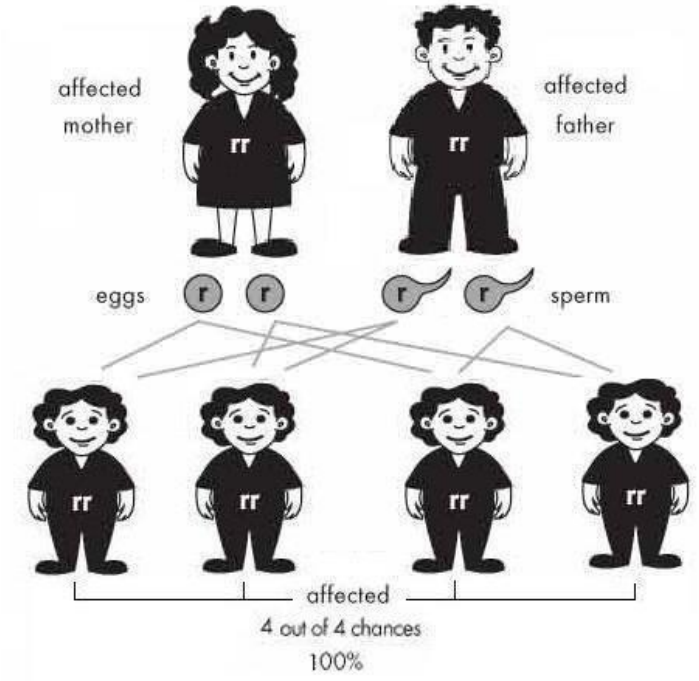


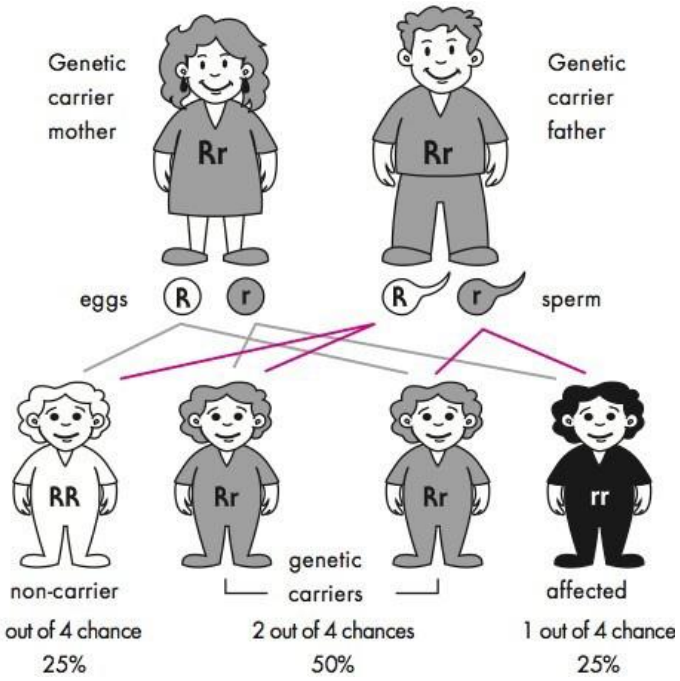


نمط التوريث للصفات المتنحية

LDLRAP1 (the plasma membrane Low Density Lipoprotein Receptor Adaptor Protein 1)







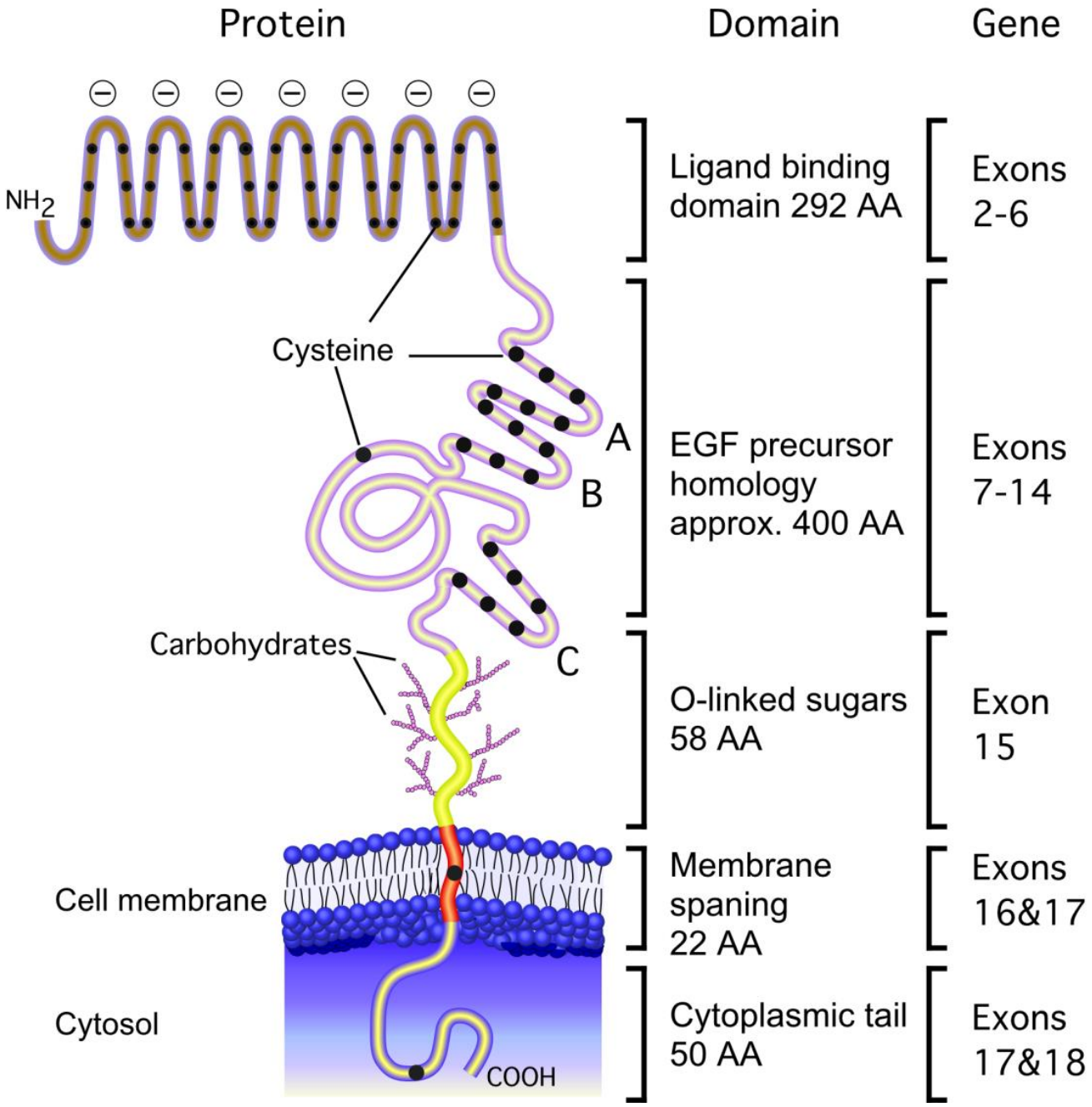
Classes of LDLR mutations and genotype/phenotype correlation

The null receptor phenotype results from non-sense mutation(s) or deletion(s) in the promoter region within the LDLR gene

The transport deficient receptors are synthesised normally in the endoplasmic reticulum but fail to be transported to the Golgi apparatus for further processing

Binding deficient LDLR is transported normally to the cell membrane but binds LDL only partially

The internalisation defective receptors reach the cell surface and bind LDL, but fail to cluster in clathrin coated pits



Causative Mutations

- **Loss of Function Mutations**

LDLR

Accounts for 85-90%.
Over 1000 mutations reported
No hotspots in some population

ApoB

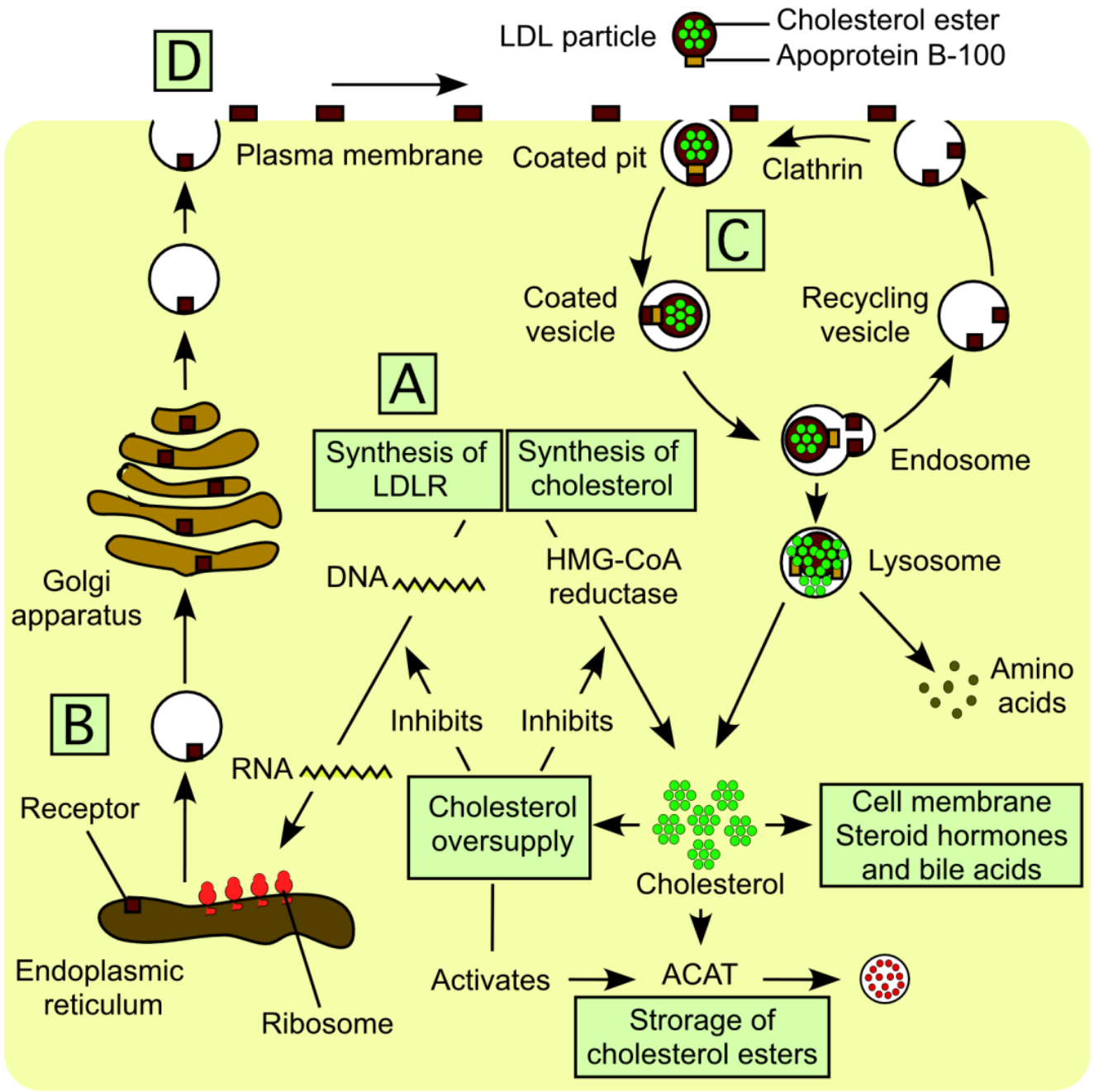
Accounts for 1-5%
Only two mutations is reported

- **Gain of Function Mutations**

PCSK9

Account for 5-10%
Nine mutations are reported

Other candidate Genes: ABCA1, APOA2, APOC3, PON2, ARH, LDLRAP1, APOC2, APOE, and LPL



Selection criteria: Simon Broome Criteria

'Definite' FH – A+B must be present

'Possible' FH – A+C or A+D

A. Total and LDL-Cholesterol

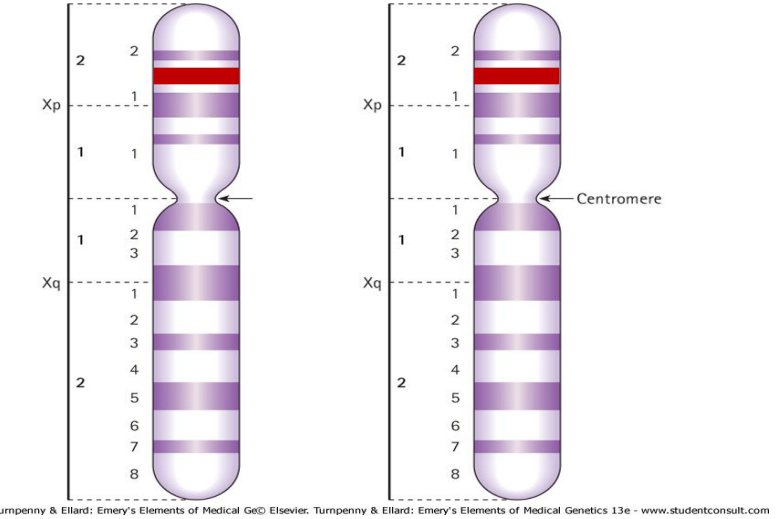
16 years+ : Total cholesterol >7.5mmol/l (>290mg/dl) or LDL-C >4.9mmol/l (>190mg/dl)

Under 16 years: Total cholesterol >6.7mmol/l (>260mg/dl) or LDL-C >4.0mmol/l (>155mg/dl)

B. Tendon xanthomas in patient or 1st (parents, sibling, children) or 2nd (grandparents, uncle, aunt) degree relative

C. FH of myocardial infarction (MI) <60 yrs in 1st degree relative or FH of MI <50 yrs in 2nd degree relative

D. FH of total cholesterol >7.5mmol/l (>290mg/dl) in 1st or 2nd degree relative



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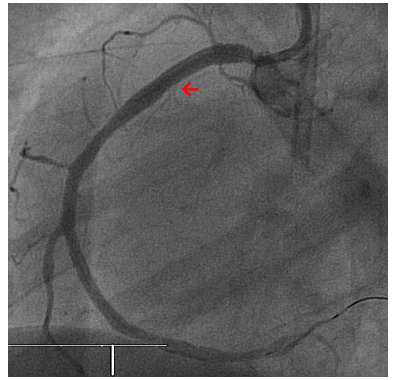

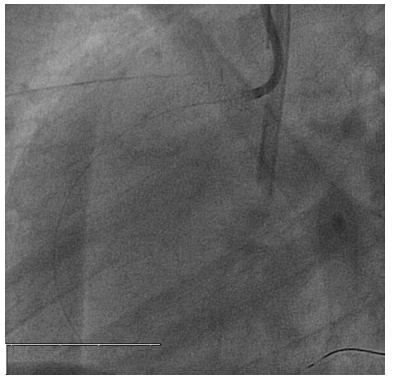

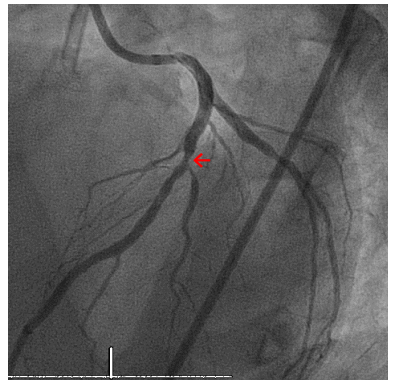

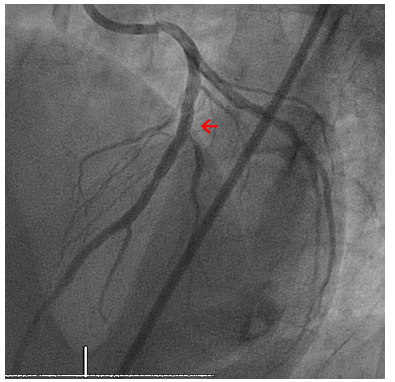

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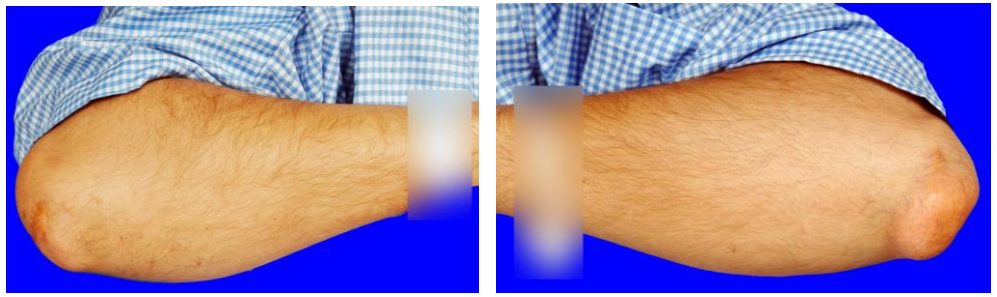
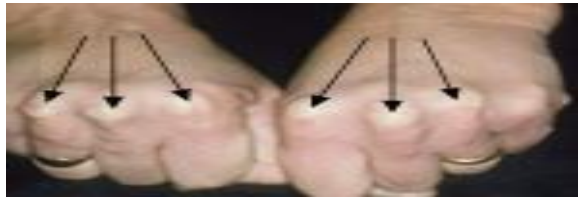
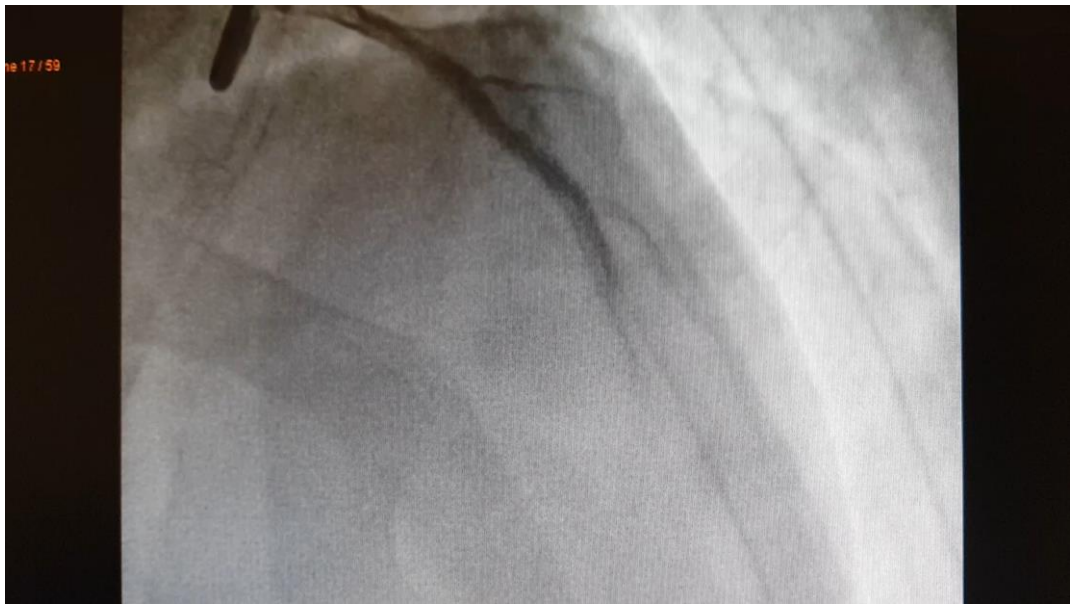
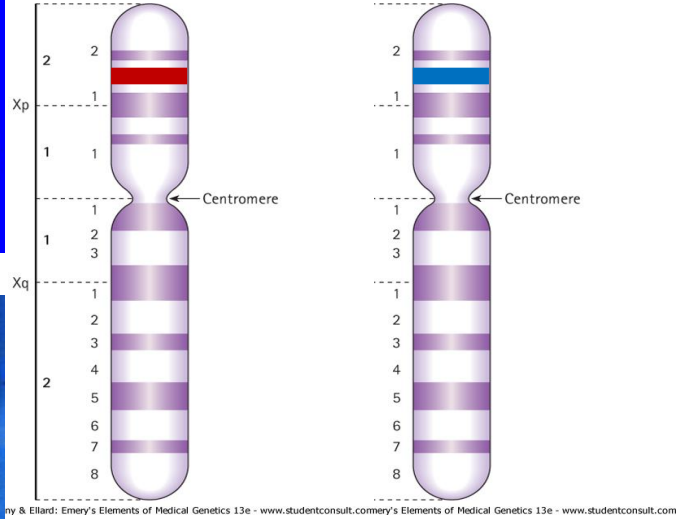


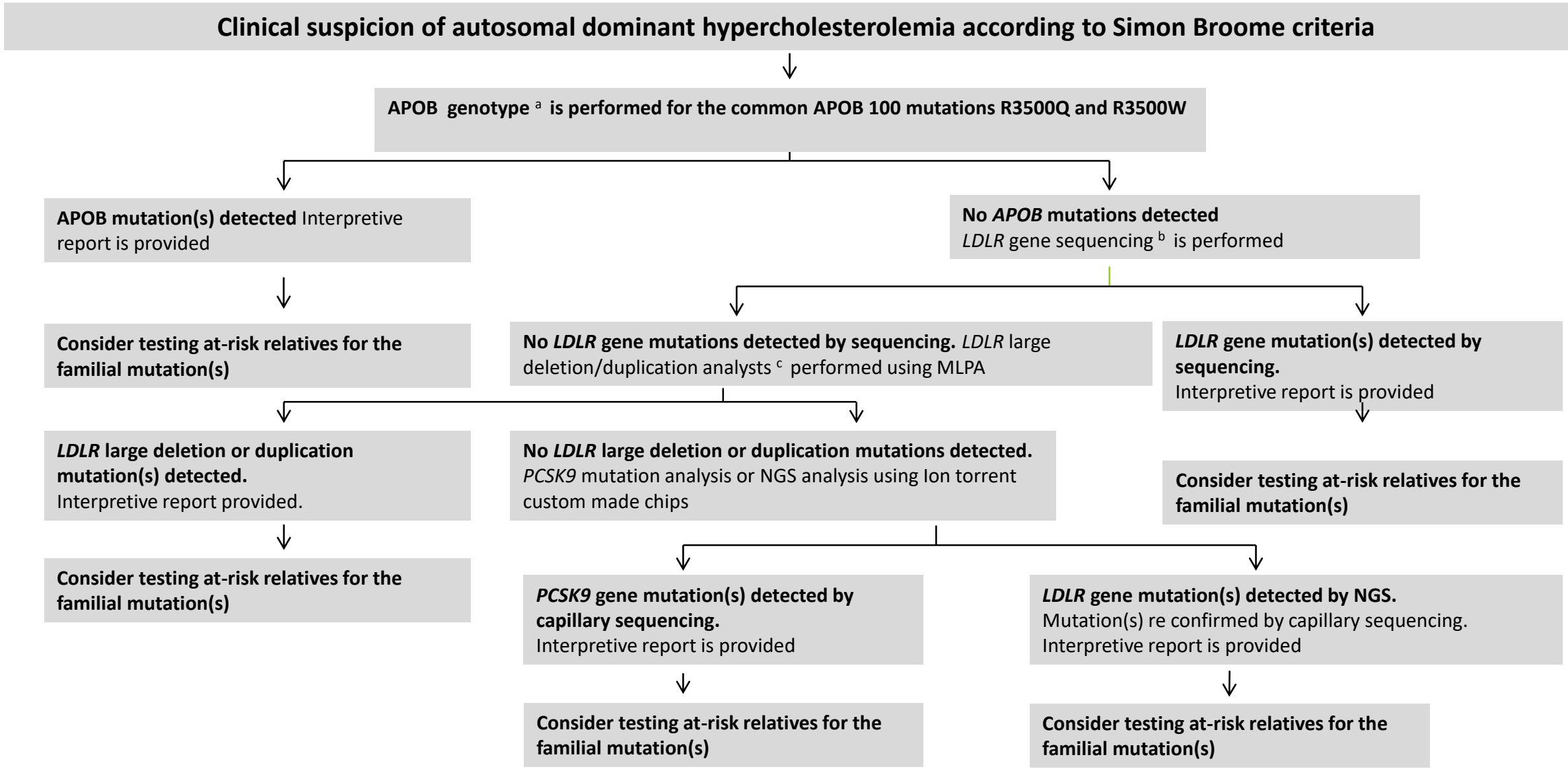






H) Left Anterior Descending (LAD): mid segment significant lesion (red arrow). I) Left Anterior Descending (LAD): post PCI and stent (red arrow). J) Right Coronary Artery totally occluded proximally. K) Right Coronary Artery (RCA): post PCI and stent (red arrow).

Familial Hypercholesterolemia mutation detection Algorithm



Identification of Novel nonsense mutation p.D445X in LDLR gene causes familial hypercholesterolemia

We have identified a novel insertion mutation (c.1332_1333insT) at exon 9 of the LDLR gene (alr family)

This insertion mutation results in a premature stop codon at position 445 in exon 9 of the LDLR gene, which results in truncation of the protein.

OPEN

Citation: Human Genome Variation (2014) 1, 14021; doi:10.1038/hgv.2014.21
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www.nature.com/hgv

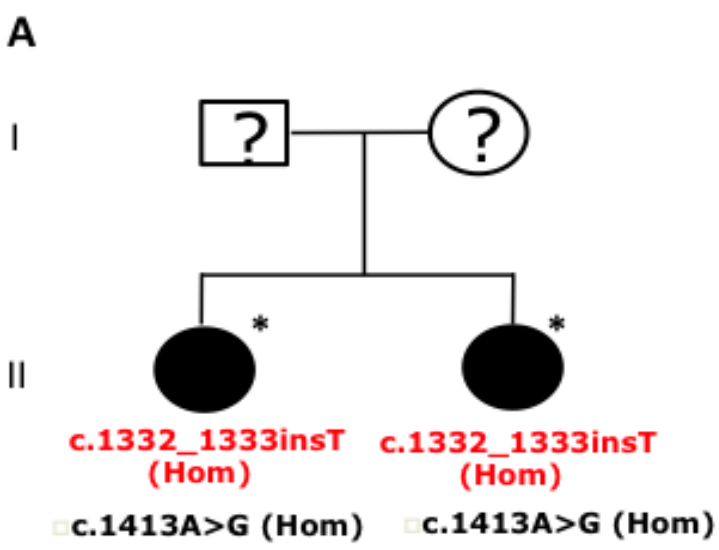
ARTICLE

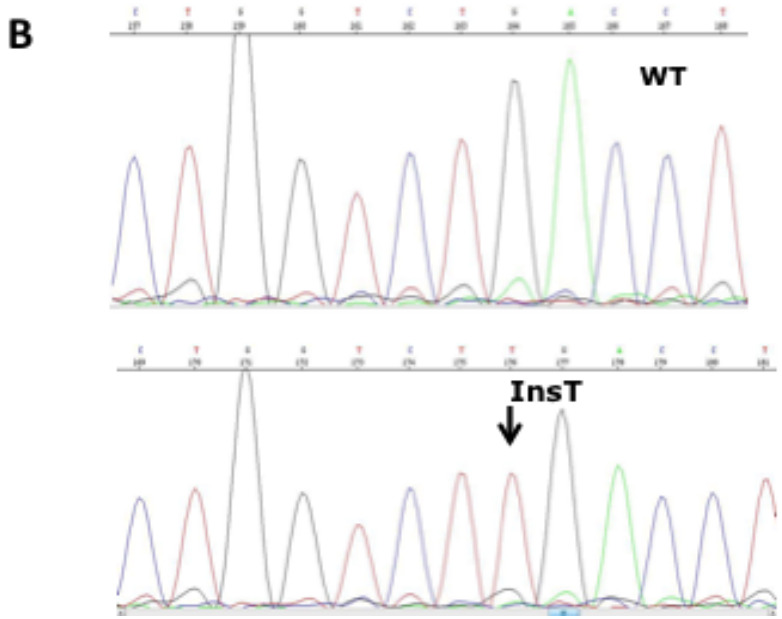
Identification of a novel nonsense variant c.1332dup, p.(D445*) in the *LDLR* gene that causes familial hypercholesterolemia

Faisal A Al-Allaf[1,2,3,6], Mohammad Athar[1,2,6], Zainularifeen Abduljaleel[1,2], Abdellatif Bouazzaoui[1,2], Mohiuddin M Taher[1,2], Rakan Own[1], Ahmad F Al-Allaf[4], Iman AbuMansour[1], Zohor Azhar[1], Faisal A Ba-hammam[1], Hala Abalkhail[5] and Abdullah Alashwal[5]

Familial hypercholesterolemia (FH) is an autosomal dominant disease predominantly caused by a mutation in the low-density lipoprotein receptor (*LDLR*) gene. Here, we describe two severely affected FH patients who were resistant to statin therapy and were managed on an apheresis program. We identified a novel duplication variant c.1332dup, p.(D445*) at exon 9 and a known silent variant c.1413A>G, p.(=), rs5930, NM_001195798.1 at exon 10 of the *LDLR* gene in both patients.

Human Genome Variation (2014) **1**, 14021; doi:10.1038/hgv.2014.21; published online 20 November 2014





Development of Next Generation Sequencing chip to Identify Genetic Variants Causative of FH

Ldlr, ApoB, Pcsk9, Abca1, Apoa2, Apoc3, Apon2, Arh, Ldlrrap1, Apoc2, ApoE, and Lpl

LDLR Exon 14 (c.2026delG, p. Gly676Fs), Novel mutation)
Glycine – Alanine

ARTICLE IN PRESS
GENE-40394; No. of pages: 9; 4C:
Gene xxx (2015) xxx–xxx
Contents lists available at ScienceDirect
Gene
journal homepage: www.elsevier.com/locate/gene

Research paper

Next generation sequencing to identify *novel* genetic variants causative of autosomal dominant familial hypercholesterolemia associated with increased risk of coronary heart disease

Faisal A. Al-Allaf [a,b,c,*,1], Mohammed Athar [a,b,*,1], Zainularifeen Abduljaleel [a,b,*,1], Mohiuddin M. Taher [a,b], Wajahatullah Khan [d], Faisal A. Ba-hammam [a], Hala Abalkhail [e], Abdullah Alashwal [e,1]

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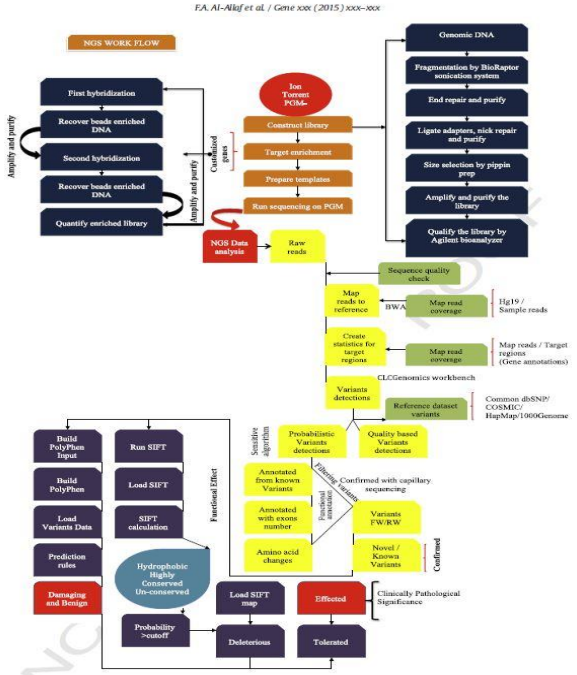
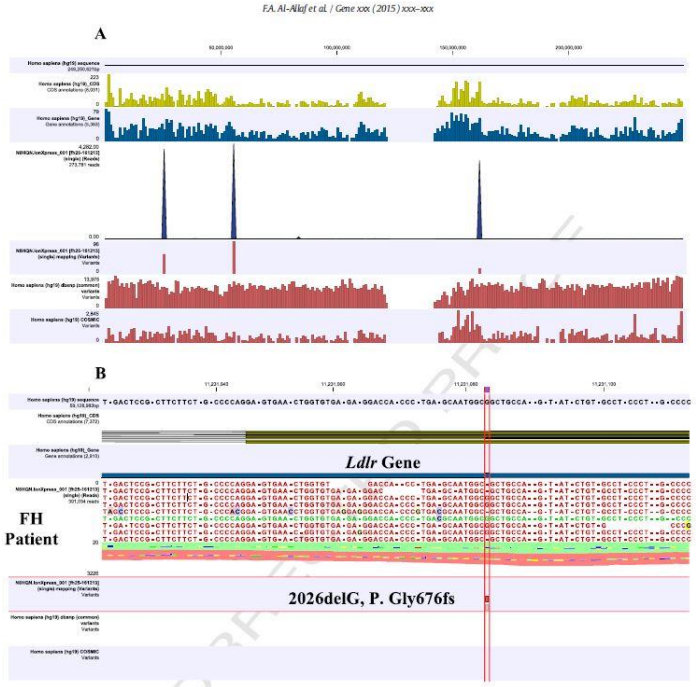
[d] Department of Basic Sciences, College of Science and Health Professions, King Saud Bin Abdulaziz University for Health Sciences, PO Box 3124, Riyadh 11426, Saudi Arabia
[e] Department of Pediatrics, MBC 58, King Faisal Specialist Hospital and Research Centre, P.O. Box 3354, Riyadh 11211, Saudi Arabia





Identification of a novel causative frame shift mutation at the LDLR Exon 14 (c.2027delG, p. Gly676fs)

Contents lists available at ScienceDirect

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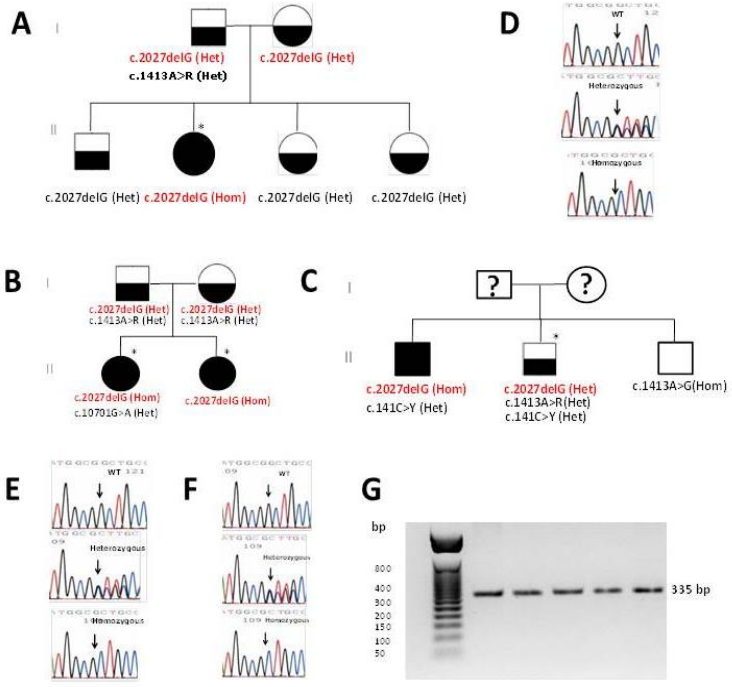
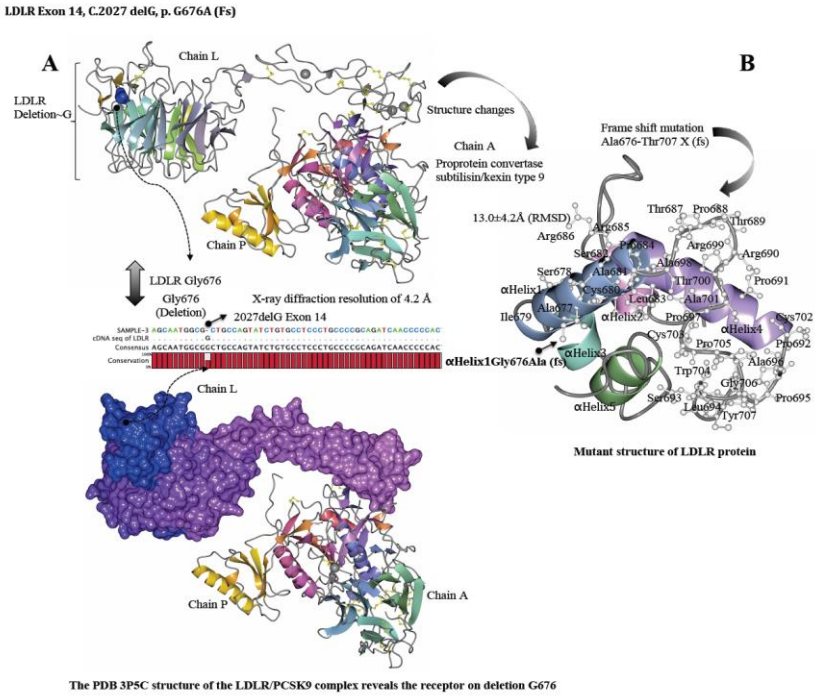
Identification of a recurrent frameshift mutation at the *LDLR* exon 14 (c.2027delG, p.(G676Afs*33)) causing familial hypercholesterolemia in Saudi Arab homozygous children

Faisal A. Al-Allaf [a,b,c,*,1], Abdullah Alashwal [d,1], Zainularifeen Abduljaleel [a,b,1], Mohiuddin M. Taher [a,b], Shahid S. Siddiqui [e], Abdellatif Bouazzaoui [a,b], Hala Abalkhail [d], Rakan Aun [a], Ahmad F. Al-Allaf [f], Iman AbuMansour [a], Zohor Azhar [a], Faisal A. Ba-Hammam [a], Wajahatullah Khan [g], Mohammad Athar [a,b,*,1]



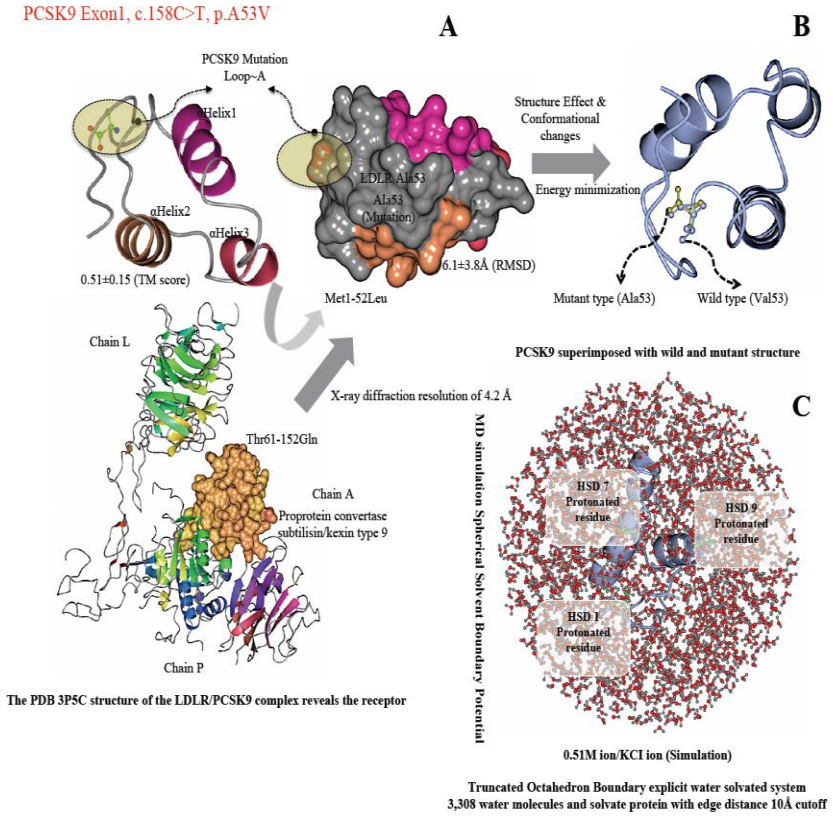


Identification and treatment of patients with homozygous FH

Current Vascular Pharmacology, 2015, *13*, 1-12

Identification and Treatment of Patients with Homozygous Familial Hypercholesterolaemia: Information and Recommendations from a Middle East Advisory Panel

Abdullah Al-Ashwal[1], Fahad Alnouri[2], Hani Sabbour[3], Abdulraof Al-Mahfouz[4], Nasreen Al-Sayed[5], Maryam Razzaghy-Azar[6], Faisal Al-Allaf[7], Khalid Al-Waili[8], Yajnavalka Banerjee[9], Jacques Genest[10], Raul D Santos[11] and Khalid Al-Rasadi[8,*]



Compound heterozygous mutation with novel missense/frameshift DNA sequence variants in the LDLR in Saudi patients suffering sever hypercholesterolaemia

ACTA ABP BIOCHIMICA POLONICA

Epub: No 2016_1283
Vol. 63, 2016
https://doi.org/10.18388/abp.2016_1283

Regular paper

Compound heterozygous LDLR variant in severely affected familial hypercholesterolemia patient

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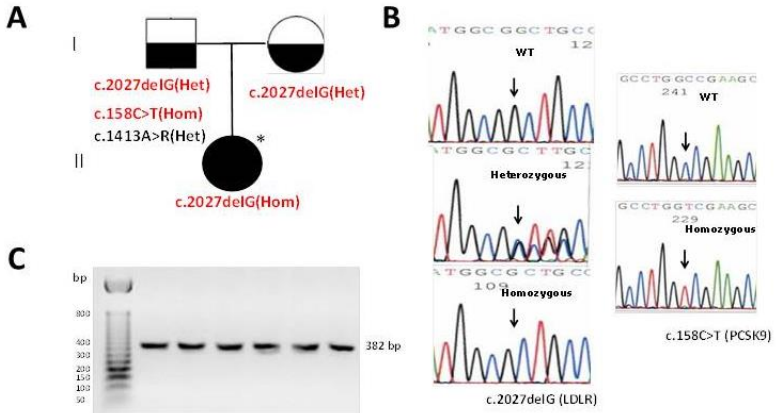
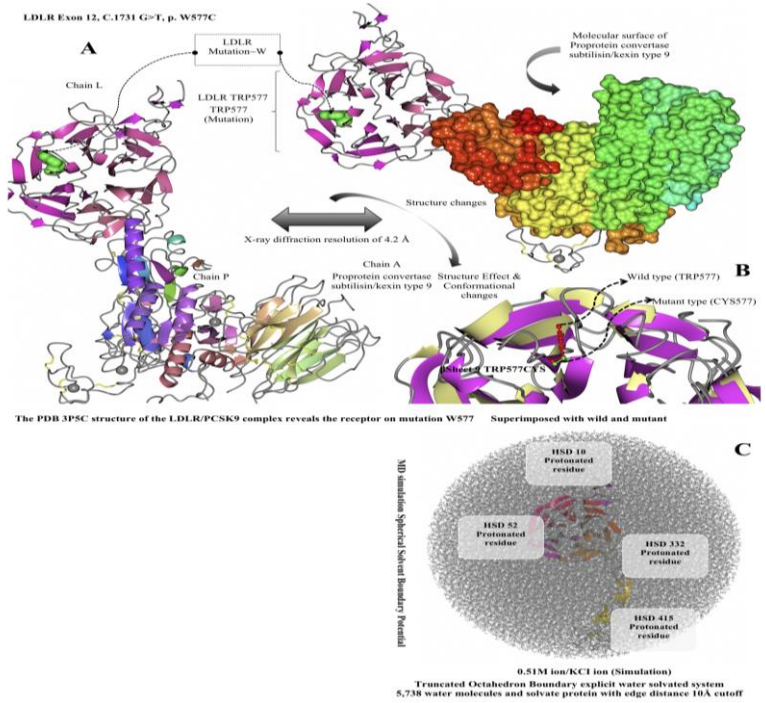




TUNIS

c.1477_1479delTCTinsAGAGACA, p.(S493Rfs*44) (**FH-Souassi**), Slimane et al., 2001
c.1845+1G>A(**FH-Tunis**), Jelassi et al., 2009
c.1186+1G>A, p.(E380_G396), Jelassi et al., 2008
c.267C>G, p.C89W, Jelassi et al., 2009
C.443G>C, p.(C148S), Jelassi et al., 2009
C.796G>A , p.(D266N), Jelassi et al., 2009
C.1027G>T, p.(G343C), Jelassi et al., 2009
c.2446A>T, p.(K816*), Slimani et al., 2009
c.2299delA, p.(M767Cfs*21), Jelassi et al., 2012
12684 bp del (ex2-5), Jelassi et al., 2012
2364 bp del (ex5-6), Jelassi et al., 2012

c.1545T>G, p.(F515L), Jelassi et al., 2011
c.2009A>G, p.(G670E), Jelassi et al., 2011
c.520C > T, p.(P174S), Jelassi et al., 2012

ALGERIA

c.1222G>A, p.(E408K) (**FH Algeria-1**), Hobbs et al. 1992
c.1291G>A, p.(A431T) **(FH Algeria-2),** Hobbs et al. 1990
c.1301C>A, p.(T434K) **(FH Algeria-3),** Hobbs et al. 1992

MOROCO

FH-Moroco-1, El Messal et al., 2003
FH-Moroco-2, El Messal et al., 2003
c.682G>T, p.(E228*), Hobbs et al., 1992
c.400T>C, p.(C134R), El Messal et al., 2003
c.859G>T, p.(G287C), El Messal et al., 2003
c. 1171G>A, p.(A391T), El Messal et al., 2003
c. 2054C>T, p.(P685L), El Messal et al., 2003
c. 2132C>T, p.(C711S), El Messal et al., 2003
c.138C>A, p.(C46*), Chater et al., 2006
c.313+5G>T, Chater et al., 2006
c.1736A>C, p.(D579A), Chater et al., 2006
c.514G>A, p.(D172N), Chater et al., 2006
c.1502C>A, p.(A501E), Chater et al., 2006
c.756_762delCCGGCAG, Chihab et al., 2007

SYRIA

c.550T>C, p.(C184R), Hobbs et al. 1992
c.827G>A, p.(C276Y), Vergopoulos et al. 1998
c.1027G>A, p.(G343S), Vergopoulos et al. 1998
c.1172del1, p.(A370fs), Reshef et al., 1996
c.2043C>A, p.(C681*), Vergopoulos et al. 1998
c.1999T>C, p.(C667R), Lehrman et al. 1987
c.2483A>G, p.(Y828C), Vergopoulos et al 1997
c.89-1G>C, p.(K30T*3), Al-Kateb et al., 2002

OMAN

c.272delG, Al-Hinai et al., 2013
p.(V474I), Al-Waili et al., 2013

BAHRAIN

c.1706-2A>T in SA site of intron 11, Shawar et al., 2012
c.2439G>A, p.(W813*), Lehrman et al., 1985

SAUDI ARABIA

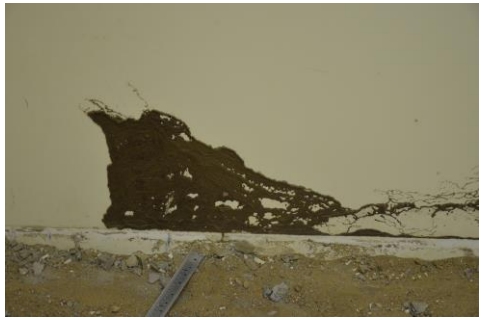
c.2027delG, p.(G676Afs*33), Al-Allaf et al., 2015b
c.2026delG, p.(G676Afs*33), Al-Allaf et al., 2015a
c.1332dup, p.(D445*), Al-Allaf et al., 2014
c.1731G>T, p.(W577C), Al-Allaf et al., 2016 (Unpublished data)
c.2416_2417insG, p.(V806Gfs*11), Al-Allaf et al., 2016 (Unpublished data)
c.622G>A, p.(E208K), Al-Allaf et al., 2016 (Unpublished data)
c.1474G>A, p.(D492N), Al-Allaf et al., 2016 (Unpublished data)
c.1429G>A, p.(D477N), Al-Allaf et al., 2016 (Unpublished data)
c.185C>T, p.(T62M), Al-Allaf et al., 2016 (Unpublished data)
c.1783C>T, p.(R595W), Al-Allaf et al., 2016 (Unpublished data)
c.1706-2A>T in SA site of intron 11, Al-Allaf et al., 2016 (Unpublished data)
c.2439G>A, p.(W813*), Lehrman et al., 1985
c.313C>T, p.(P105S), Alharbi et al., 2015
c.1171G>A, p.(A391T), Alharbi et al., 2015
c.9835A>G, p.(S3279G), Al-Allaf et al., 2016 (Unpublished data)
c.158C>T, p.(A53V), Al-Allaf et al., 2016 (Unpublished data)
c.658-7C>T, Al-Allaf et al., 2016 (Unpublished data)
c.799+3A>G, Al-Allaf et al., 2016 (Unpublished data)

LEBANON

c.2043C>A, p.(C681*), Lehrman et al. 1987
c.761A>C, p.(Q254P), Abifadel et al., 2009
c.1066G>T, p.(D356Y), Abifadel et al., 2009
c.1073G>A, p.(C358Y), Abifadel et al., 2009
c.1329G>A, p.(W443*), Abifadel et al., 2009
c.1352T>G, p.(I451T), Abifadel et al., 2009
c.2476C>T, p.(P826S), Abifadel et al., 2009
c.1171G>A, p.(A391T), Fahed et al., 2011

c.406C>T, p.(Q136*), Garcia et al., 2001
c.[605C>A, p.(P202H), Garcia et al., 2001
748-608G>A, (W249ins62*), Wilund et al 2002
c.89-1G>C, p.(K30T*3), Lind et al., 2004

1432



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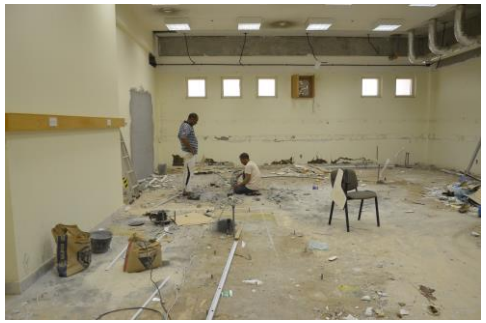
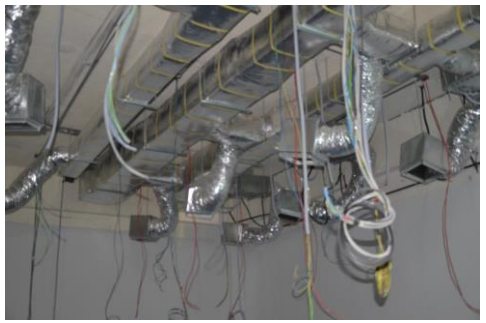
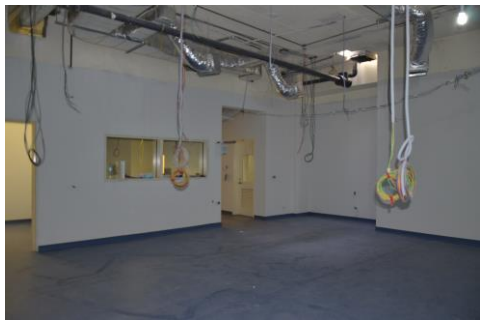
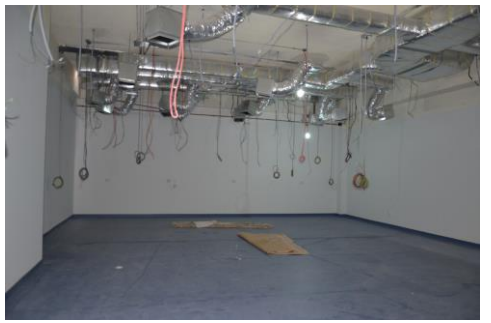
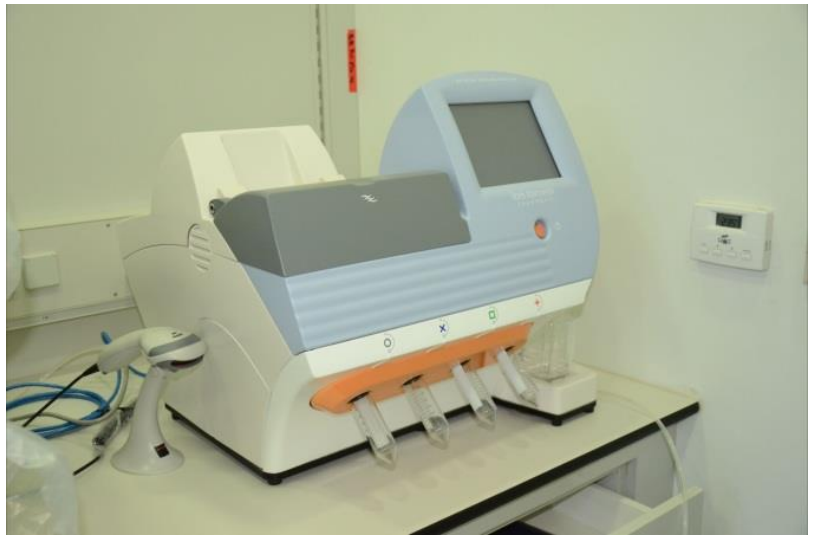
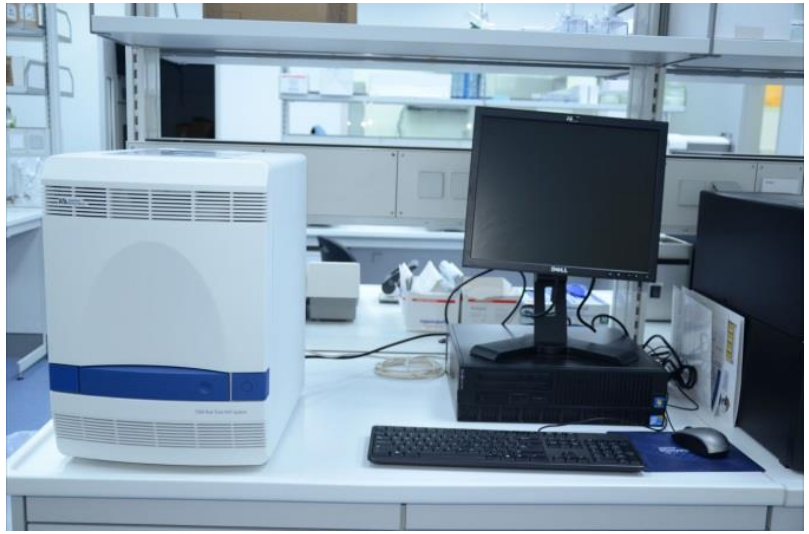




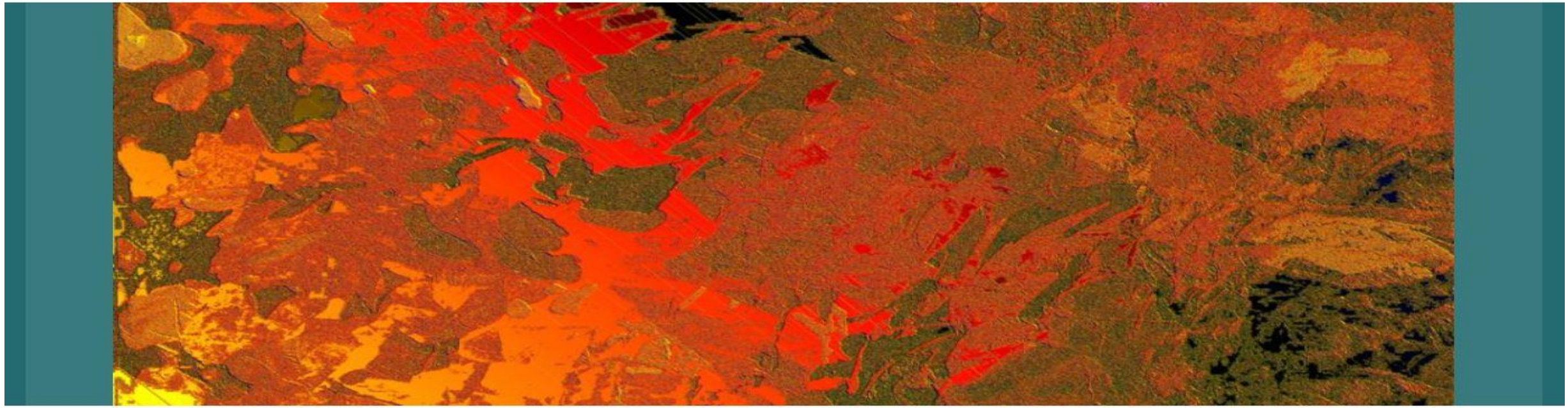


1435

http://www.fh-sa.com

البرنامج الوطني للتعرف على الاعتلالات الوراثية المسببة لمرض ارتفاع كوليسترول الدم العائلي في المجتمع السعودي

Familial hypercholesterolaemia



الرئيسية | الطبيب | المريض | - | - | -

Clinical suspicion of autosomal dominant hypercholesterolemia

LDLR **gene sequencing (Capillary; Sanger) is performed for the common tribes associated mutations panel/hotspot**

No *LDLR* gene mutations detected by sequencing.
NGS (Ion torrent) analysis for customized FH associated genes
(LDLR, APOB, PCSK9 and LDLRAP1)

Gene mutation(s) detected by NGS.
Mutation(s) reconfirmed by capillary sequencing.
Interpretive report is provided

Consider testing at-risk relatives for the familial mutation(s)

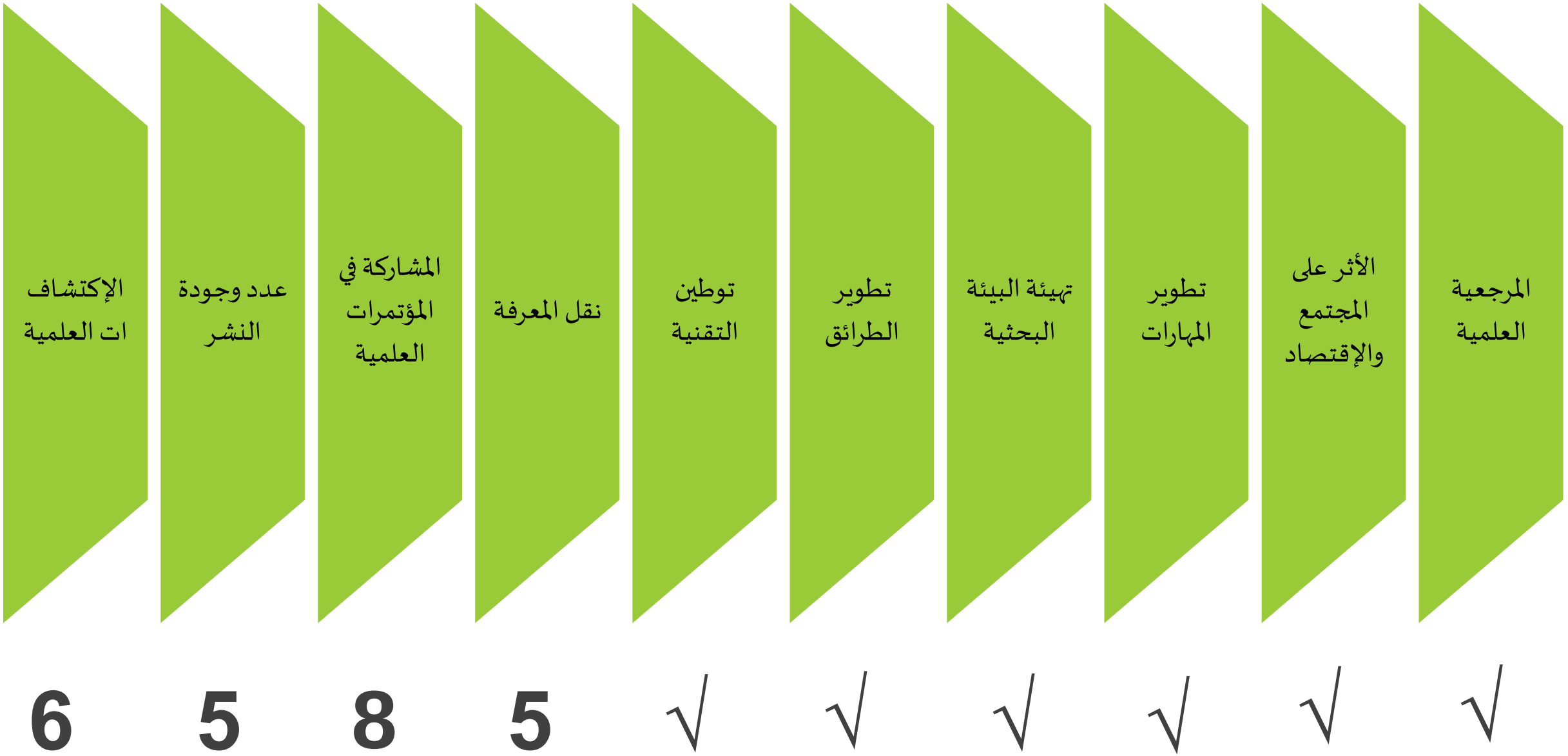
No gene mutation(s) detected in customized genes by NGS.
Whole exome analysis by NGS

Gene mutation(s) detected by NGS.
Mutation(s) reconfirmed by capillary sequencing.
Interpretive report is provided

Consider testing at-risk relatives for the familial mutation(s)

LDLR **gene mutation(s) detected by sequencing.**
Interpretive report is provided

Consider testing at-risk relatives for the familial mutation(s)



المرجعية العلمية	√
الأثر على المجتمع والإقتصاد	√
تطوير المهارات	√
تهيئة البيئة البحثية	√
تطوير الطرائق	√
توطين التقنية	√
نقل المعرفة	5
المشاركة في المؤتمرات العلمية	8
عدد وجودة النشر	5
الإكتشافات العلمية	6

الْخُطَّةُ الْمُسْتَقْبَلِيَّةُ لِلْبَرْنَامَجِ

1. إِيجَادَ مَصَادِرِ تَمْوِيلٍ إِضَافِيَّةٍ لِإِجْرَاءِ فُحُوصَاتٍ لِشَرِيحَةٍ أَكْبَرِ مِنْ الْمَرْضَى.
2. تَطْوِيرَ قَاعِدَةِ الْبَيَانَاتِ الْوَطَنِيَّةِ.
3. نَشْرَ الْوَعْيَ الْمُبَكِّرَ بِالْمَرَضِ، وَأَسْبَابِهِ، وَسُبُلَ تَشْخِيصِهِ، وَعِلَاجِهِ مِنْ أَجْلِ تَقْدِيمِ الْمُسَانَدَةِ، وَالدَّعْمِ لِلْمَرْضَى، وَأُسَرِهِمْ، وَمُجْتَمَعِهِم.
4. نَتَطَلَّعُ إِلَى إِبْرَامِ شَرَاكَاتٍ مَعَ الْمَرَاكِزِ، وَالْمُسْتَشْفَيَاتِ الْعَامَّةِ ،وَالْخَاصَّةِ، وَالشَّرِكَاتِ، وَمُؤَسَّسَاتِ الْمُجْتَمَعِ، وَتَوْسِيعِ دَائِرَةِ التَّعَاوُنِ الْوَطَنِيِّ، والخليجي وَالدَّوْلِيِّ بِمَا يُحَقِّقُ الْأَهْدَافَ والرؤية الْوَطَنِيَّةَ فِي نَقْلِ الْمَعْرِفَةِ، وَتَوْطِينِ التَّقْنِيَةِ، وَاقْتِصَادِيَّاتِ الصِّحَّةِ، وَتَحْسِينِ الْخَدَمَاتِ الْعِلَاجِيَّةِ الَّتِي تَنْعَكِسُ إِيجَابًا عَلَى الْمُجْتَمَعِ.

كثير من التجارب الناجحة تسبقها محاولات فاشلة ...

عَلى قَدرِ أَهلِ العَزمِ تَأتي العَزائِمُ"
" وَتَأتي عَلى قَدرِ الكِرامِ المَكارِمُ

وَتَعظُمُ في عَينِ الصَغيرِ صِغارُها"
" وَتَصغُرُ في عَينِ العَظيمِ العَظائِمُ

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د/ محمد أطهر

د/ زين العارفين عبدالجليل

د/ عبداللطيف بوعزي

أ/ رامي بن سعيد بامرضاح

المعيدين

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الجهة المانحة

برنامج التقنيات الإستراتيجية منحة رقم 08-BIO34-10

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