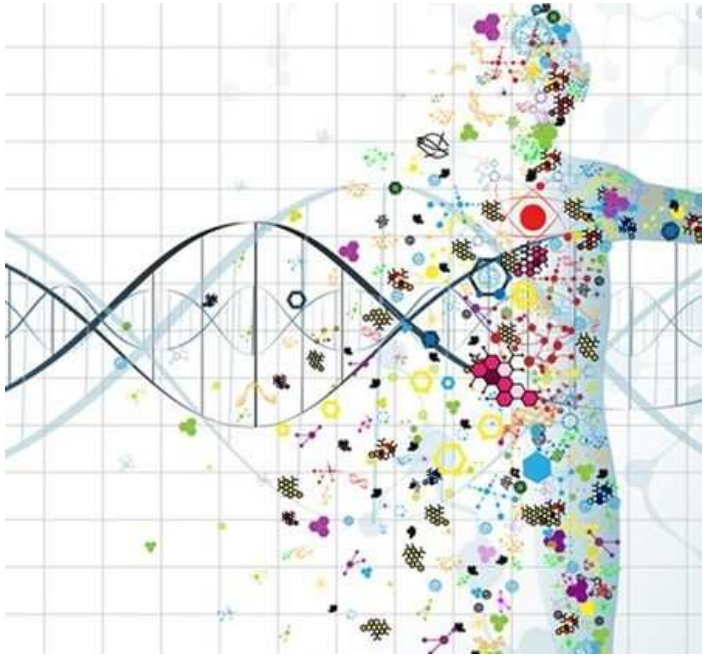
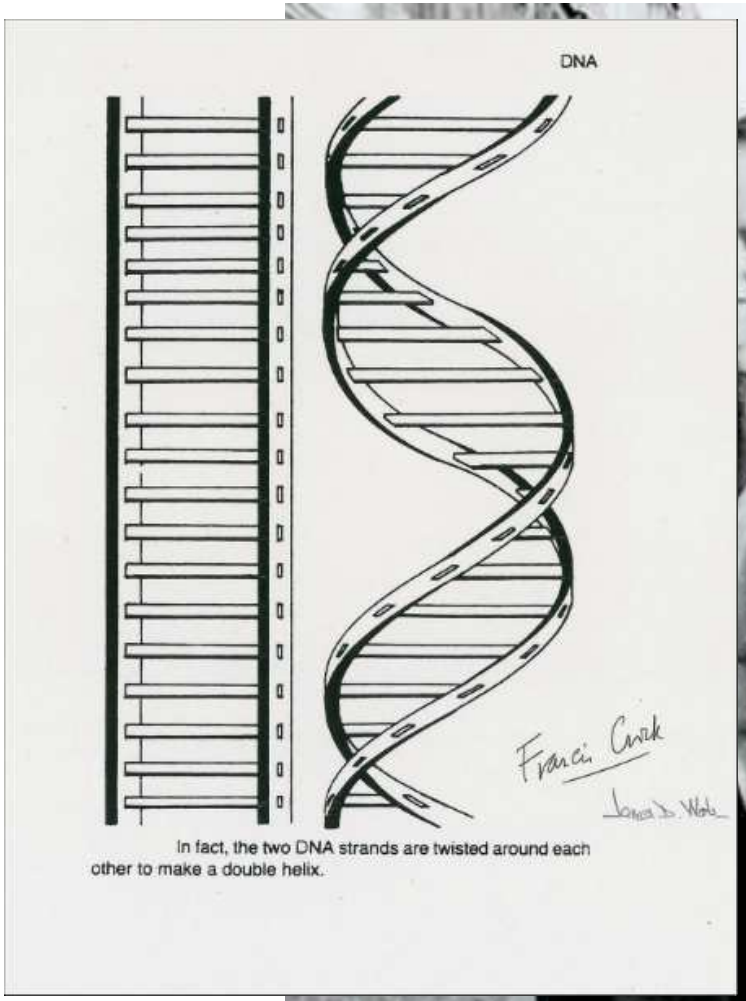
Οι εφαρμογές της Γενετικής στη σύγχρονη Παιδιατρική



Ελευθερία Παπαδοπούλου
Παιδίατρος-Γενετίστρια
Διευθύντρια Ε.Σ.Υ
Παιδιατρική Κλινική ΠαΓΝΗ

Η Ιστορία της Γενετικής



In fact, the two DNA strands are twisted around each other to make a double helix.

No. 4356 April 25, 1953 NATURE

equipment, and to Dr. G. E. R. Deacon and the captain and officers of R.R.S. *Discovery II* for their part in making the observations.

[1] Young, F. B., Gerrard, H., and Jevons, W., *Phil. Mag.*, **40**, 149 (1920).

[2] Longuet-Higgins, M. S., *Mon. Not. Roy. Astro. Soc., Geophys. Supp.*, **5**, 285 (1949).

[3] Von Arx, W. S., Woods Hole Papers in Phys. Oceanog. Meteor., **11** (3) (1950).

[4] Ekman, V. W., *Arkiv. Mat. Astron. Fysik.* (Stockholm), **2** (11) (1905).

MOLECULAR STRUCTURE OF NUCLEIC ACIDS

A Structure for Deoxyribose Nucleic Acid

WE wish to suggest a structure for the salt of deoxyribose nucleic acid (D.N.A.). This structure has novel features which are of considerable biological interest.

A structure for nucleic acid has already been

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tion. We have as
adjacent residues i
structure repeats aft
is, after 34 A. Th
from the fibre axis i
the outside, cations
The structure is a
is rather high. At
expect the bases t
become more comp
The novel feature
in which the two
purine and pyrimidi
are perpendicular t
together in pairs, a
hydrogen-bonded t
chain, so that the t
z-co-ordinates. One
the other a pyrimi
hydrogen bonds are
1 to pyrimidine p

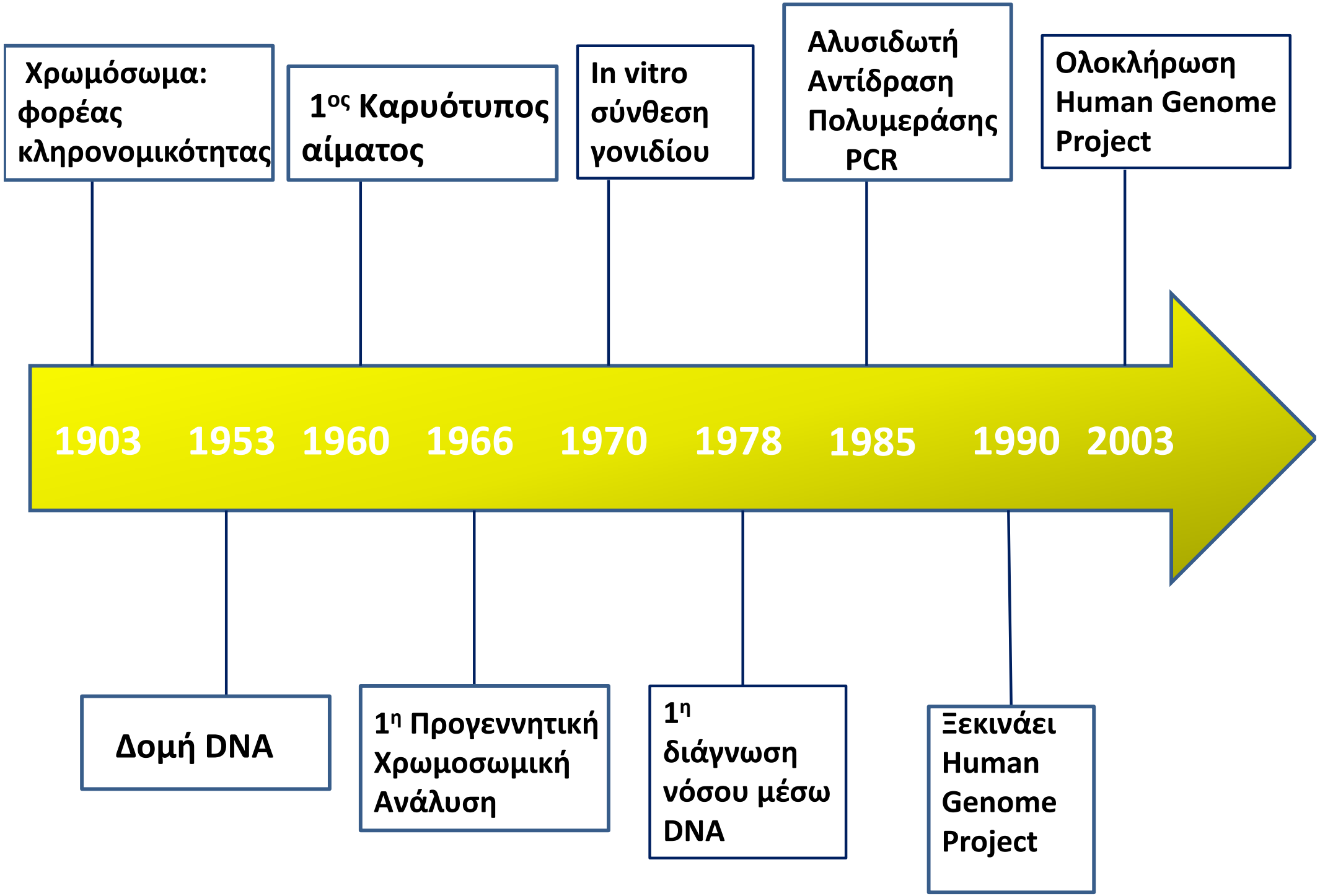
Dawn of gene-editing medicine?

6 November 2015

By James Gallagher

Health editor, BBC News website

Does the smiling face of Layla Richards mark a new era in genetic medicine that could change all our lives?

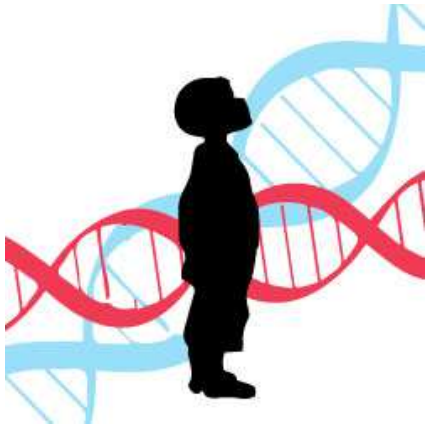


Σταθμοί Εξέλιξης της Γενετικής

Η Επίπτωση των Γενετικών Νοσημάτων

Γενετική νόσος: 53 / 1.000 άτομα *μέχρι τα 25 έτη ζωής*

- ✓ **Μονογονιδιακά (3,6/53)**
- ✓ **Χρωμοσωματικά (2/53)**
- ✓ **Πολυπαραγοντικά (47/53)**



+ Συγγενείς ανωμαλίες

Γενετική νόσος: 79 / 1.000 άτομα *μέχρι τα 25 έτη ζωής*

Baird et al, Health Surveillance Registry, 1998

Γενικό Παιδιατρικό Νοσοκομείο ΗΠΑ
71% εισαγωγών έχουν γενετική βάση
96% χρόνιων νοσημάτων: γενετικά ή με γενετική προδιάθεση

Am. J. Hum. Genet. 74:121–127, 2004

The Burden of Genetic Disease on Inpatient Care in a Children's Hospital

Shawn E. McCandless,[1,2] Jeanne W. Brunger,[3] and Suzanne B. Cassidy[4]

[1]Department of Genetics, Case Western Reserve University, Cleveland; [2]Center for Human Genetics and Department of Pediatrics, University Hospitals of Cleveland, Cleveland; [3]Clinical Genetics Service, Department of Cancer Genetics, Roswell Park Cancer Institute, Buffalo, NY; and [4]Department of Pediatrics, Division of Human Genetics, University of California, Irvine, School of Medicine, Irvine, CA

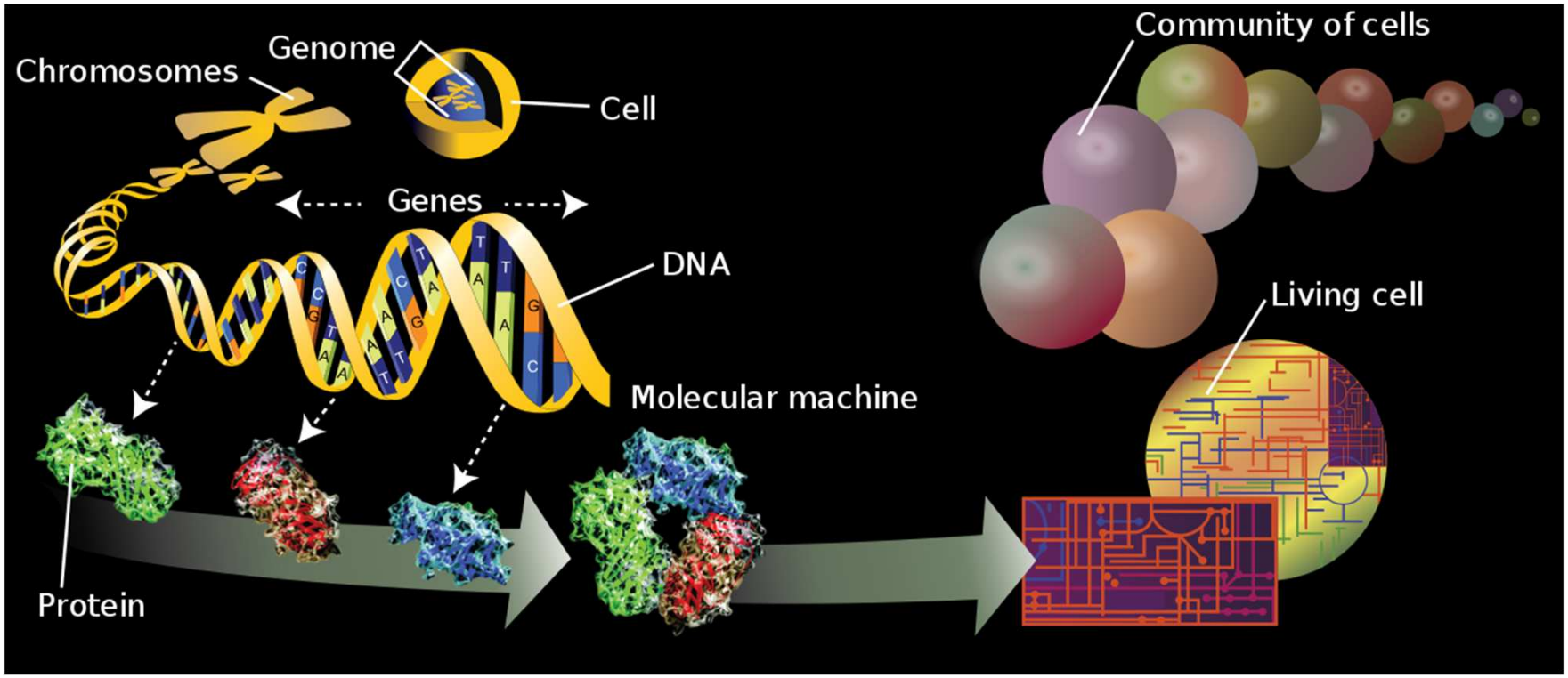
The important role of genetics in pediatric illness has been increasingly recognized, but the true impact has not been well delineated. An important study of pediatric inpatient admissions to a children's hospital in 1978 found a genetic basis for disease in just less than half of admitted patients. We sought to update this study in light of current hospitalization practices and new knowledge about genetics. We systematically reviewed the records of 5,747 consecutive admissions (4,224 individuals), representing 98% of patients admitted in 1996 to Rainbow Babies and Children's Hospital (Cleveland, OH). Each patient was assigned to one of five groups on the basis of the presence or absence of an underlying chronic medical condition and whether that condition had a genetic basis or susceptibility. An underlying disorder with a significant genetic component was found in 71% of admitted children. The vast majority (96%) of underlying chronic disorders in children in this study were either clearly genetic or had a genetic susceptibility. Total charges for 1996 were >$62 million, of which $50 million (81%) was accounted for by disorders with a genetic determinant. The 34% of admissions with clearly genetic underlying disorders accounted for 50% (>$31 million) of the total hospital charges. The mean length of stay was 40% longer for individuals with an underlying disease with a genetic basis than for those with no underlying disease. Charges and length of stay were similar for children with underlying chronic disorders, regardless of the cause. This study begins to quantify the enormous impact of genetic disease on inpatient pediatrics and the health care system. Additional study and frank public discourse are needed to understand the implications on the future health care workforce and on the utilization of health care resources.

Cc

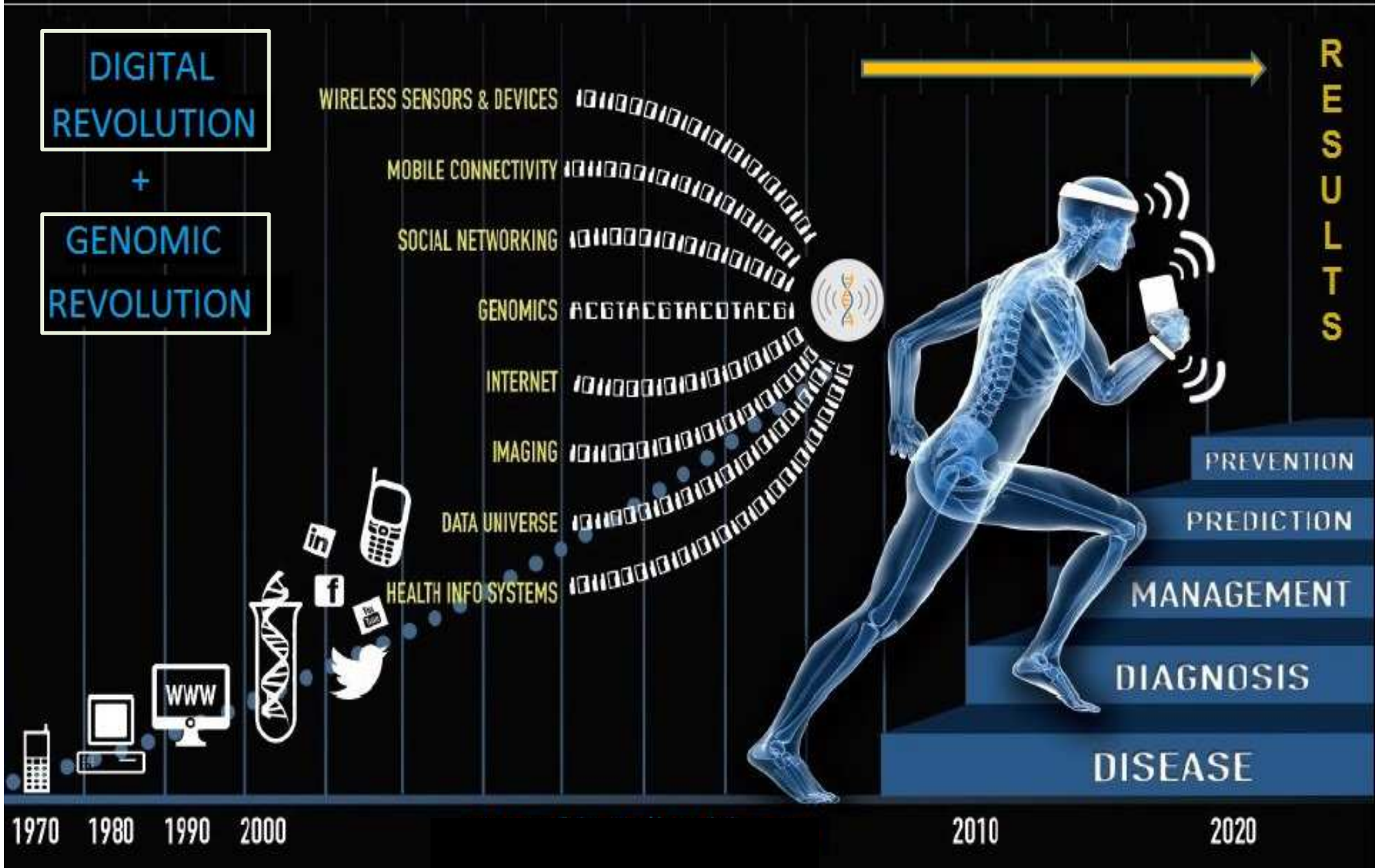
21ος αιώνας: Η εποχή της Γενωμικής Επανάστασης

από μελέτη μεμονωμένων γονιδίων (Γενετική)

μελέτη όλου του γονιδιώματος (Γενωμική)

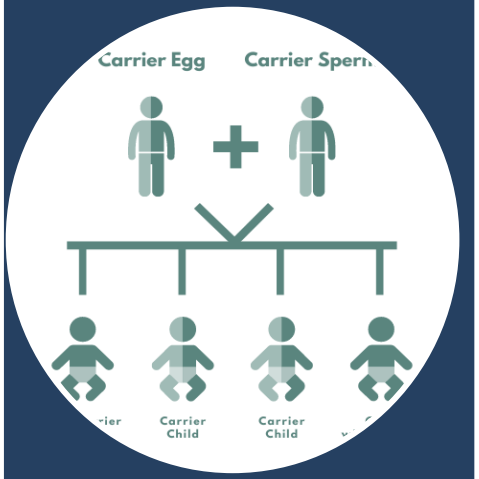


Η Ψηφιακή Επανάσταση στην Υγεία

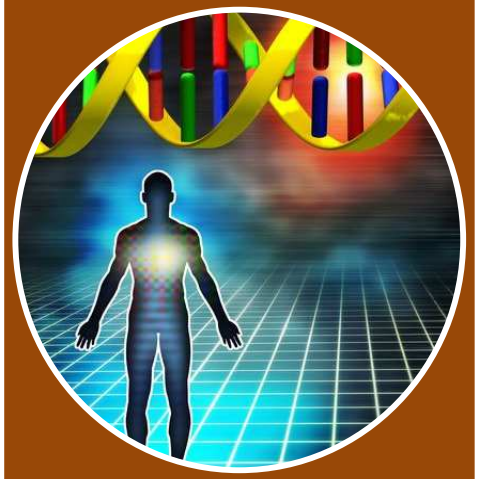


ΔΙΑΓΝΩΣΗ

ΘΕΡΑΠΕΙΑ



SCREENING



ΠΡΟΛΗΨΗ

ΕΦΑΡΜΟΓΕΣ ΤΗΣ ΓΕΝΕΤΙΚΗΣ

ΔΙΑΓΝΩΣΗ ΓΕΝΕΤΙΚΩΝ ΝΟΣΩΝ

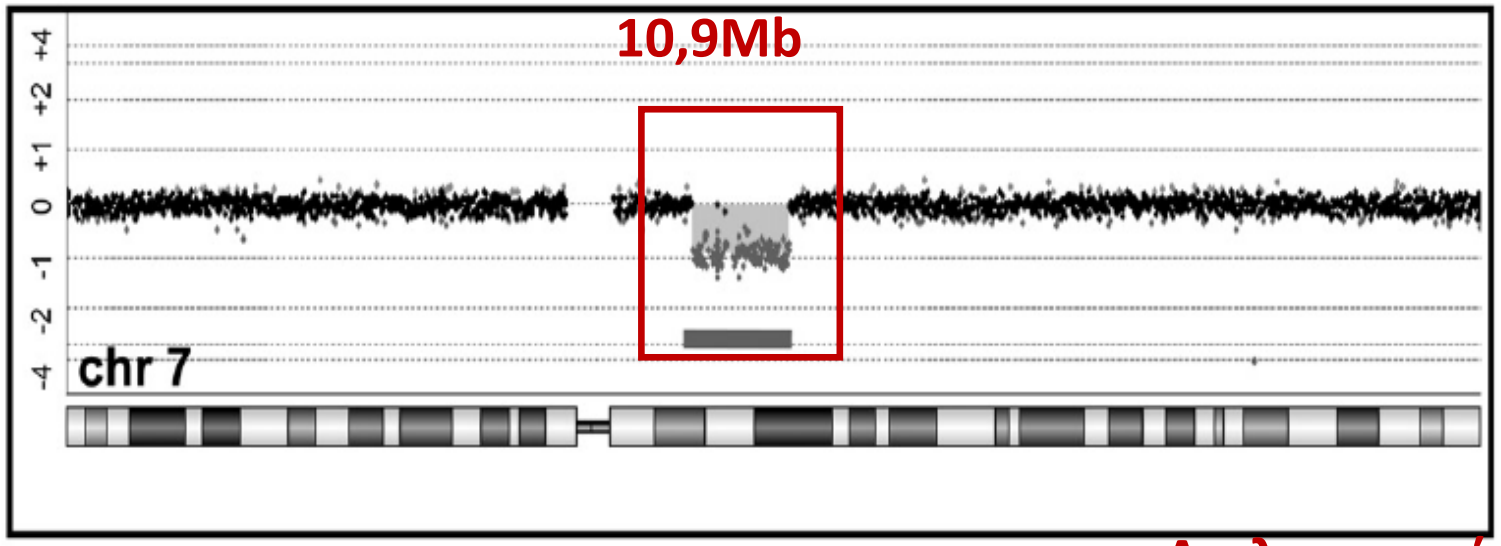
1. Σε νεογνά, βρέφη και παιδιά
2. Προγεννητικά, σε κύτταρα λάχνης ή αμνιακά

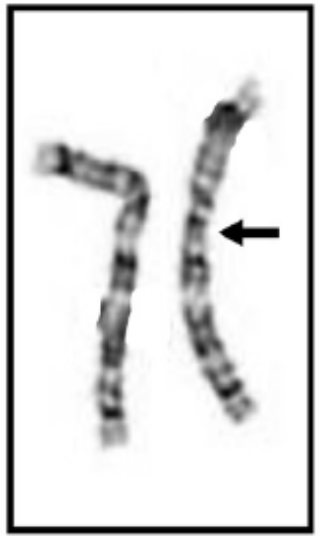
Μοριακός Καρυότυπος
ή Μικροσυστοιχίες Συγκριτικού Γενομικού Υβριδισμού (array CGH)

Συμβατικός Καρυότυπος

A

Έλλειμμα 10,9Mb

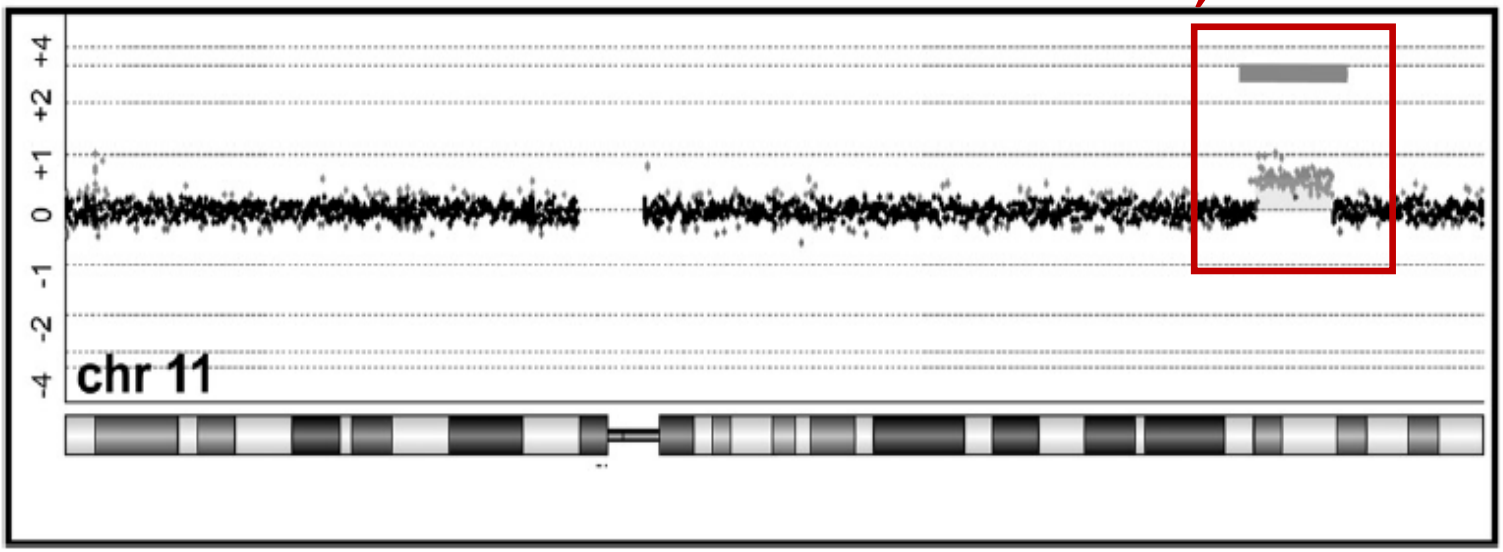




Chrom 7

B

Διπλασιασμός 7,2Mb



Chrom 11

Consensus Statement: Chromosomal Microarray Is a First-Tier Clinical Diagnostic Test for Individuals with Developmental Disabilities or Congenital Anomalies

David T. Miller,[1,*] Margaret P. Adam,[2,3] Swaroop Aradhya,[4] Leslie G. Biesecker,[5] Arthur R. Brothman,[6] Nigel P. Carter,[7] Deanna M. Church,[8] John A. Crolla,[9] Evan E. Eichler,[10] Charles J. Epstein,[11] W. Andrew Faucett,[2] Lars Feuk,[12] Jan M. Friedman,[13] Ada Hamosh,[14] Laird Jackson,[15] Erin B. Kaminsky,[2] Klaas Kok,[16] Ian D. Krantz,[17] Robert M. Kuhn,[18] Charles Lee,[19] James M. Ostell,[8] Carla Rosenberg,[20] Stephen W. Scherer,[21] Nancy B. Spinner,[17] Dimitri J. Stavropoulos,[22] James H. Tepperberg,[23] Erik C. Thorland,[24] Joris R. Vermeesch,[25] Darrel J. Waggoner,[26] Michael S. Watson,[27] Christa Lese Martin,[2] and David H. Ledbetter[2,*]

Chromosomal microarray (CMA) is increasingly utilized for genetic testing of individuals with unexplained developmental delay/intellectual disability (DD/ID), autism spectrum disorders (ASD), or multiple congenital anomalies (MCA). Performing CMA and G-banded karyotyping on every patient substantially increases the total cost of genetic testing. The International Standard Cytogenomic Array (ISCA) Consortium held two international workshops and conducted a literature review of 33 studies, including 21,698 patients tested by CMA. We provide an evidence-based summary of clinical cytogenetic testing comparing CMA to G-banded karyotyping with respect to technical advantages and limitations, diagnostic yield for various types of chromosomal aberrations, and issues that affect test interpretation. CMA offers a much higher diagnostic yield (15%–20%) for genetic testing of individuals with unexplained DD/ID, ASD, or MCA than a G-banded karyotype (~3%, excluding Down syndrome and other recognizable chromosomal syndromes), primarily because of its higher sensitivity for submicroscopic deletions and duplications. Truly balanced rearrangements and low-level mosaicism are generally not detectable by arrays, but these are relatively infrequent causes of abnormal phenotypes in this population (<1%). Available evidence strongly supports the use of CMA in place of G-banded karyotyping as the first-tier cytogenetic diagnostic test for patients with DD/ID, ASD, or MCA. G-banded karyotype analysis should be reserved for patients with obvious chromosomal syndromes (e.g., Down syndrome), a family history of chromosomal rearrangement, or a history of multiple miscarriages.

Introduction

Scope and Purpose

Clinical genetic testing, including chromosome analysis, is a standard practice for patients with diagnoses including unexplained developmental delay/intellectual disability (DD/ID), autism spectrum disorders (ASD), and multiple congenital anomalies (MCA). These categories of disorders account for the largest proportion of cytogenetic testing because of their high prevalence in the population. The incidence of DD/ID in the general population approaches 3%,[1] and ASD affects ~1:150 individuals.[2,3] Most patients lack sufficient specific history or features from physical examination to suggest a specific genetic (or non-genetic) cause. Published guidelines for testing such patients have emphasized (1) testing for chromosomal abnormalities by G-banded karyotyping and (2) testing for common single-gene disorders, such as fragile X syndrome.[4]

Microarray-based genomic copy-number analysis is now a commonly ordered clinical genetic test for this patient population and is offered under various names, such as "chromosomal microarray" (CMA) and "molecular

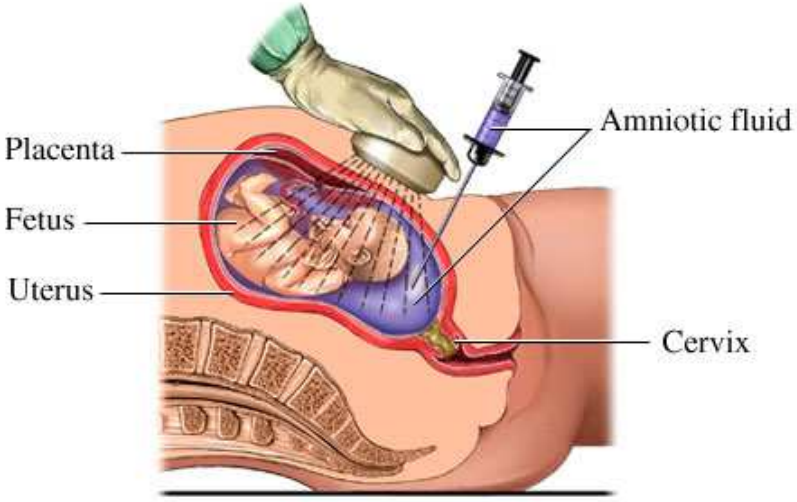
[1]Division of Genetics and Department of Laboratory Medicine, Children's Hospital Boston and Harvard Medical School, Boston, MA, USA; [2]Department of Human Genetics, Emory University School of Medicine, Atlanta, GA, USA; [3]Department of Pediatrics, University of Washington School of Medicine, Seattle, WA, USA; [4]GeneDx, Gaithersburg, MD, USA; [5]National Human Genome Research Institute, National Institutes of Health, Bethesda, MD, USA; [6]Department of Pediatrics, Human Genetics, Pathology and ARUP Laboratories, University of Utah School of Medicine, Salt Lake City, UT, USA; [7]Wellcome Trust Sanger Institute, Cambridge, UK, USA; [8]National Center for Biotechnology Information, Bethesda, MD, USA; [9]National Genetics Reference Laboratory (Wessex), Salisbury UK; [10]Department of Genome Sciences and Howard Hughes Medical Institute, University of Washington School of Medicine, Seattle, WA, USA; [11]Institute for Human Genetics and Department of Pediatrics, University of California, San Francisco, San Francisco, CA, USA; [12]Rudbeck Laboratory, Uppsala University, Uppsala, Sweden; [13]Department of Medical Genetics, University of British Columbia, and Child & Family Research Institute, Vancouver, British Columbia, Canada; [14]Department of Pediatrics and McKusick-Nathans Institute of Genetic Medicine, Johns Hopkins University School of Medicine, Baltimore, MD, USA; [15]Department of Obstetrics and Gynecology, Drexel University College of Medicine and Children's Hospital of Philadelphia, Philadelphia, PA, USA; [16]Department of Genetics, University Medical Centre Groningen, University of Groningen, The Netherlands; [17]Department of Pediatrics/Human Genetics, The Children's Hospital of Philadelphia, University of Pennsylvania School of Medicine, Philadelphia, PA, USA; [18]Center for Biomolecular Science and Engineering, University of California, Santa Cruz, Santa Cruz, CA, USA; [19]Department of Pathology, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, USA; [20]Department of Genetics and Evolutionary Biology, University Sao Paulo, Brazil; [21]The Centre for Applied Genomics and Program in Genetics and Genetic Biology, The Hospital for Sick Children and Department of Molecular Genetics, University of Toronto, Ontario, Canada; [22]Department of Pediatric Laboratory Medicine, The Hospital for Sick Children, Toronto, Ontario, Canada; [23]Laboratory Corporation of America, Research Triangle Park, NC, USA; [24]Laboratory Medicine and Pathology, Mayo Clinic, Rochester, MN, USA; [25]Center for Human Genetics, Universiteit Leuven, Leuven, Belgium; [26]Department of Human Genetics and Pediatrics, University of Chicago, Chicago, IL, USA; [27]American College of Medical Genetics, Bethesda, MD, USA
*Correspondence: david.miller2@childrens.harvard.edu (D.T.M.), david.ledbetter@emory.edu (D.H.L.)
DOI 10.1016/j.ajhg.2010.04.006.

ο Μοριακός Καρυότυπος μετά τη γέννηση αποτελεί εξέταση επιλογής σε άτομα με:

- **ανεξήγητη νοητική υστέρηση**
- **αναπτυξιακή διαταραχή**
- **αυτισμό**
- **πολλαπλές συγγενείς ανωμαλίες**

ο Μοριακός Καρυότυπος παρέχει 15-20% υψηλότερη διαγνωστική κάλυψη σε σχέση με συμβατικό καρυότυπο

Προγεννητικός Μοριακός Καρυότυπος



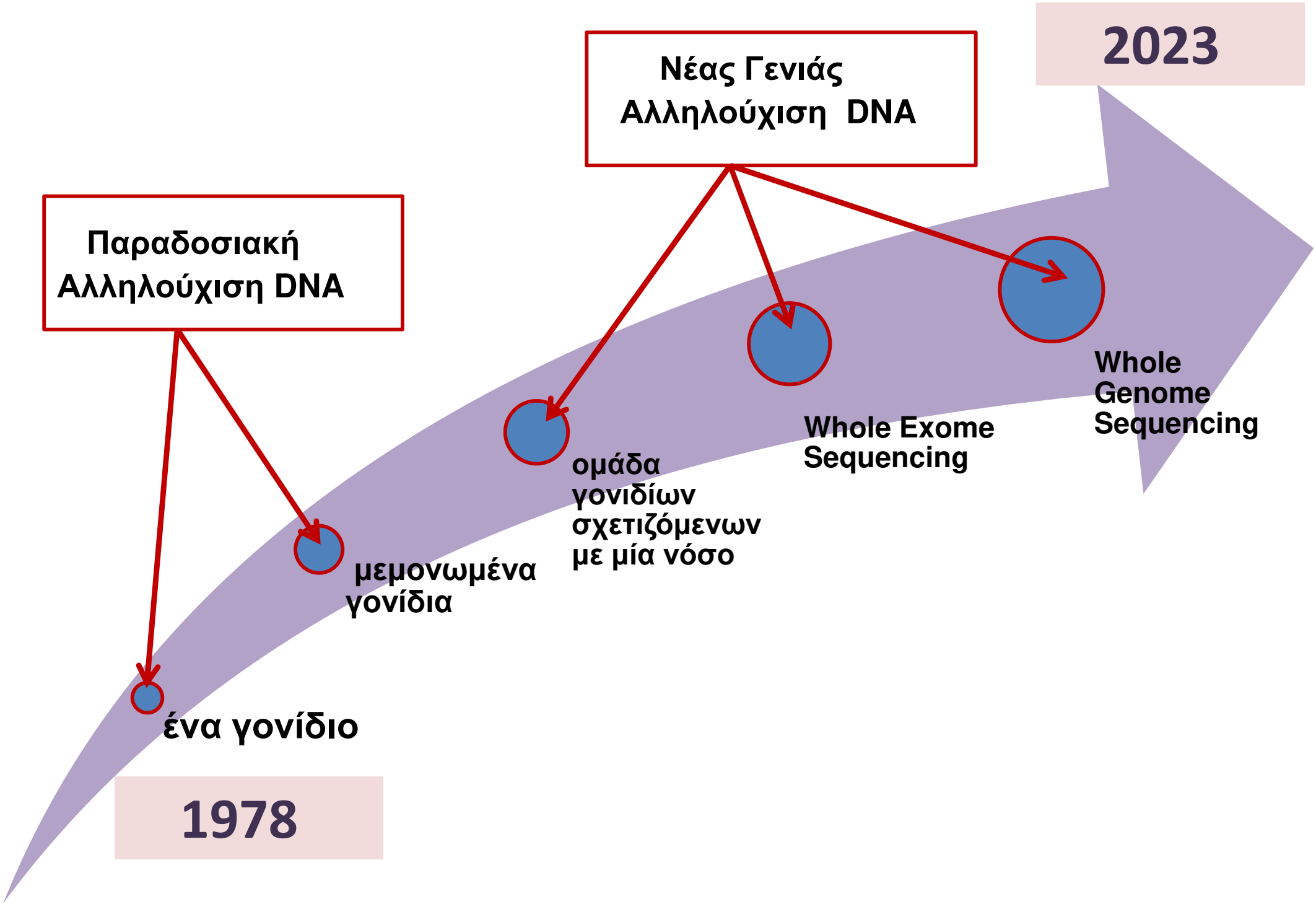
The American College of Obstetricians and Gynecologists
WOMEN'S HEALTH CARE PHYSICIANS

Society for Mamaternal-Fetal Medicine

COMMITTEE OPINION

- **Μοριακός καρυότυπος** συστήνεται **σε κάθε περίπτωση εμβρύου** με **1 ή περισσότερες δομικές ανωμαλίες** διαπιστωμένες με υπέρηχους, που προχωράει σε επεμβατικό προγεννητικό έλεγχο

- Ο **μοριακός καρυότυπος** θεωρείται **1ης γραμμής εξέταση** για έλεγχο **αποβολών και θνησιγενών εμβρύων**

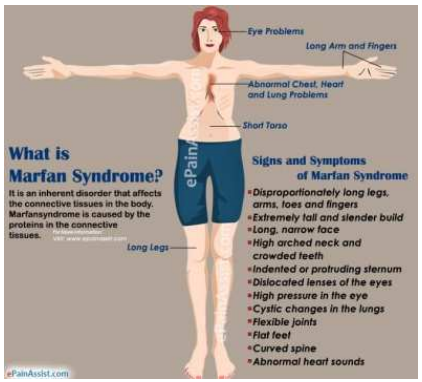


Η εξέλιξη στην διάγνωση των μονογονιδιακών νόσων

Παραδοσιακή Αλληλούχιση DNA

Ένδειξη: Χαρακτηριστικός φαινότυπος , 1 ή λίγα υπεύθυνα γονίδια

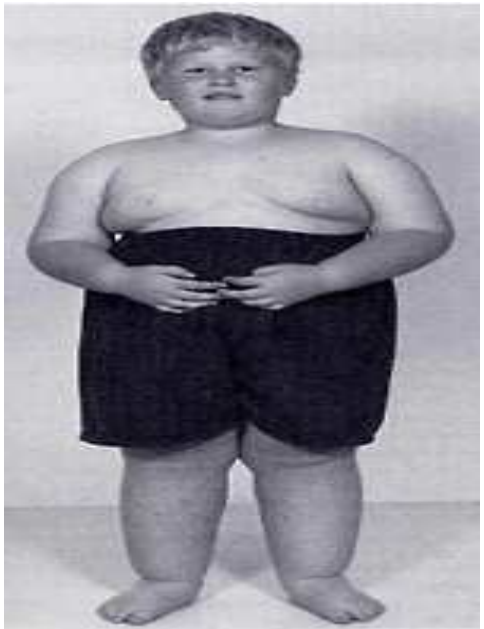
1. **Μενδέλειας κληρονομικότητας (AD, AR, X-linked)**



2. **Επανάληψη τριπλέτας τρινουκλεοτιδίων (Σ. FRAX)**

3. **Αποτυπωμένα γονίδια**

(Σ.Prader Willi & Σ.Angelman)



Νέας Γενιάς Αλληλούχιση DNA

Whole Exome Sequencing (WES)

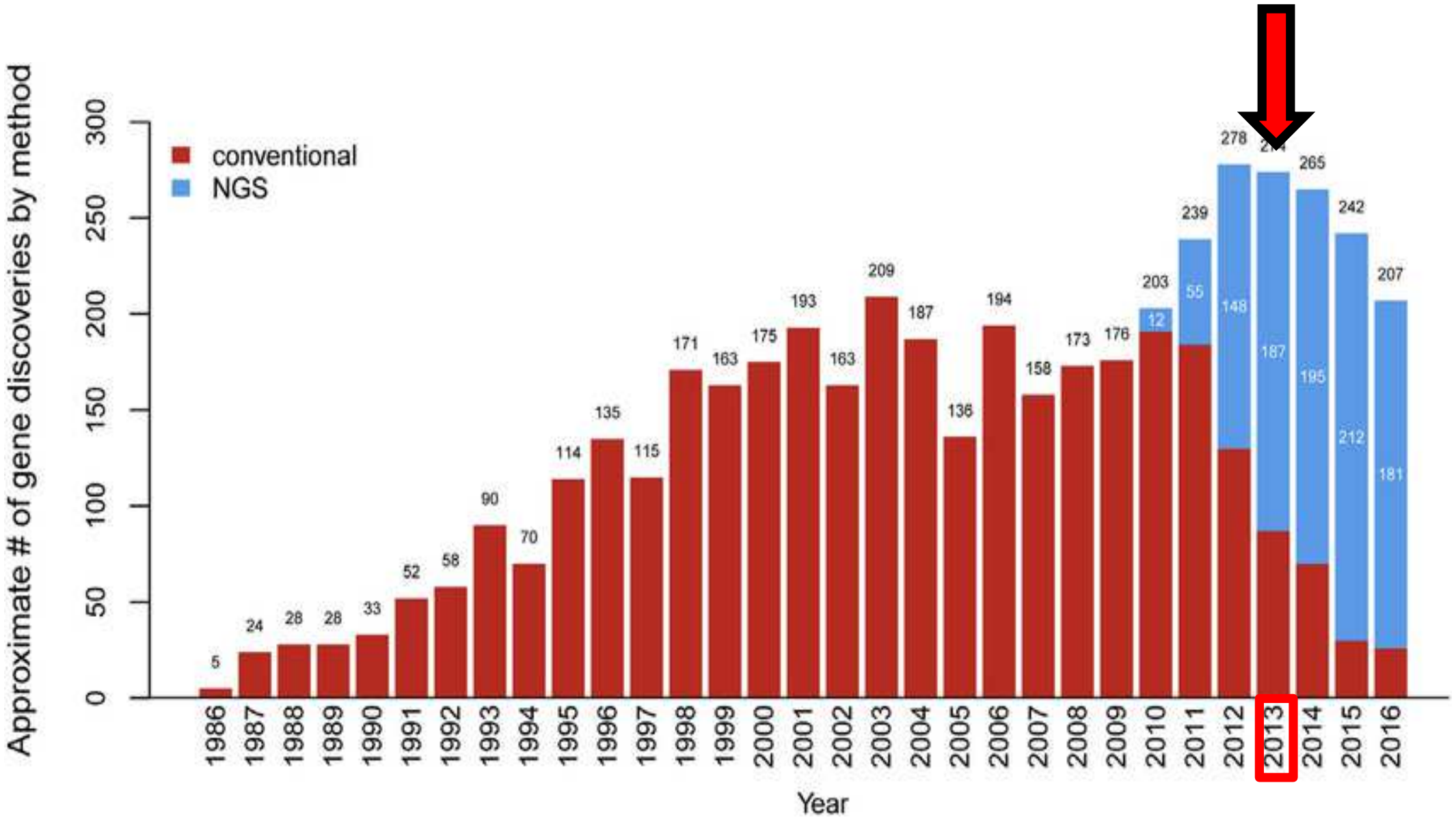
Το Γονιδίωμα που κωδικοποιεί Πρωτεΐνες

Παράλληλη Αλληλούχιση ≈ 20.000 Γονίδια

Προοπτικές

Περιορισμοί

- ✓ Δυσκολία στην ανάγνωση αποτελεσμάτων
- ✓ Ανίχνευση νέων γονιδίων υπεύθυνων για νόσο
- ✓ Παρουσία Πολυμορφισμών Αγνώστου Σημασίας (VUS)
- ✓ Μείωση κόστους αλληλούχισης ανά Mb DNA
- ✓ Τυχαίες Μεταλλάξεις – Ζητήματα Ηθικής


Approximate # of gene discoveries by method
conventional
NGS
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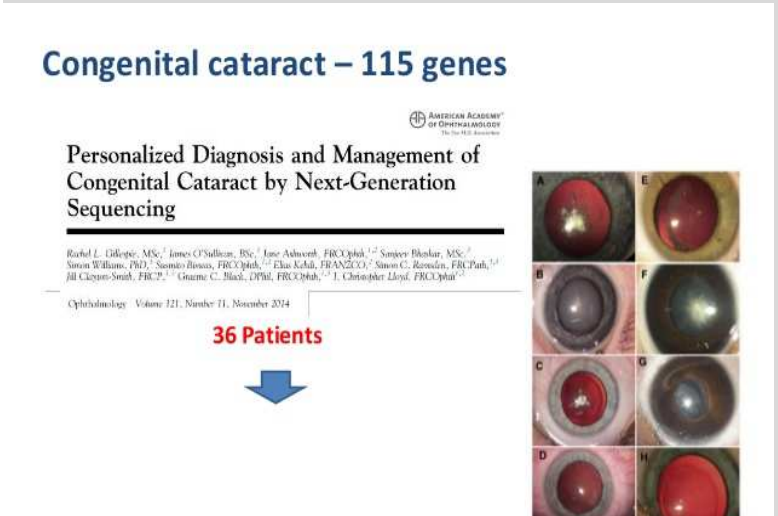
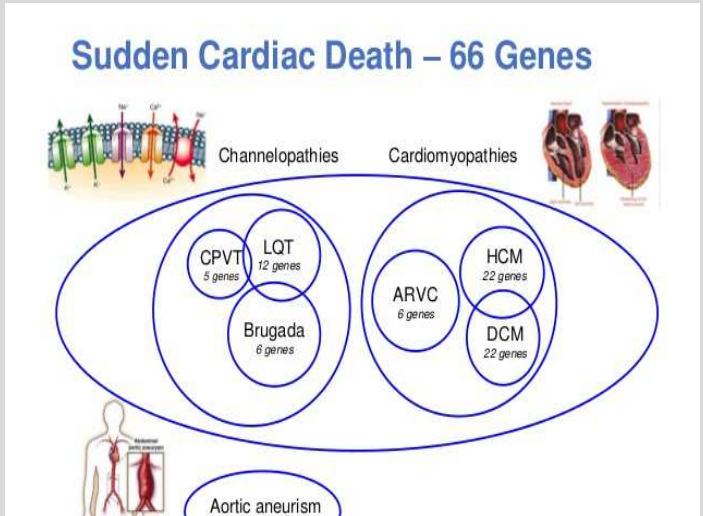
Year

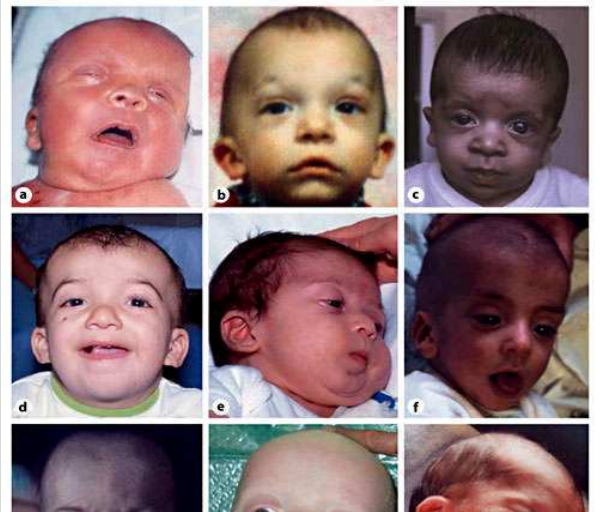
AJHG2017

Ενδείξεις της Νέας Γενιάς Αλληλούχισης

Επιλογή τεστ	Ενδείξεις	Παράδειγμα
Πάνελ Γονιδίων	Σαφώς καθοριζόμενες νόσοι με πολλά υπεύθυνα γονίδια	Συγγενής Καταρράκτης
	Διαταραχές με κοινό σύμπτωμα	Επιληψίες Αιφνίδιος Καρδιακός Θάνατος
	Γονίδια με κοινό μονοπάτι	Σύνδρομα αλληλοεπικαλυπτόμενα με Noonan Syndrome







ΘΕΡΑΠΕΙΑ ΓΕΝΕΤΙΚΩΝ ΝΟΣΩΝ

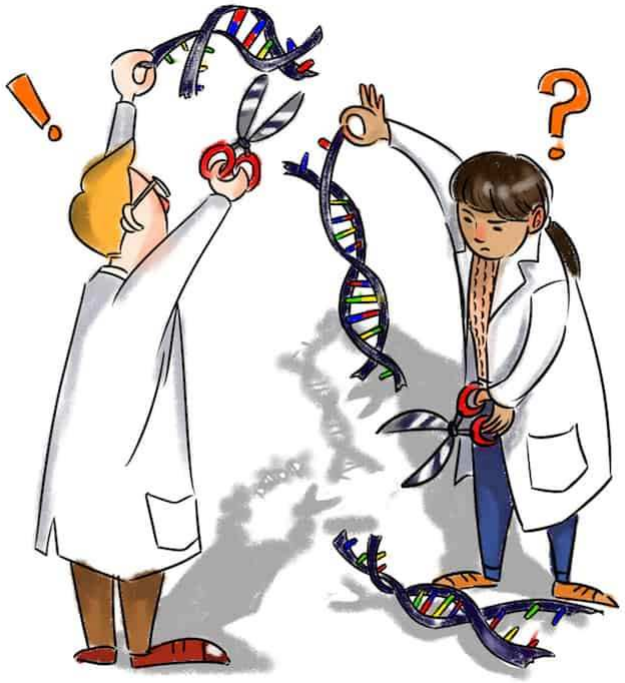
1. Γονιδιακή θεραπεία

2. Γονιδιακή τροποποίηση

3. Γονιδιακή σίγαση

“Πώς η γονιδιακή θεραπεία αναδύεται από τη «σκοτεινή εποχή» της”

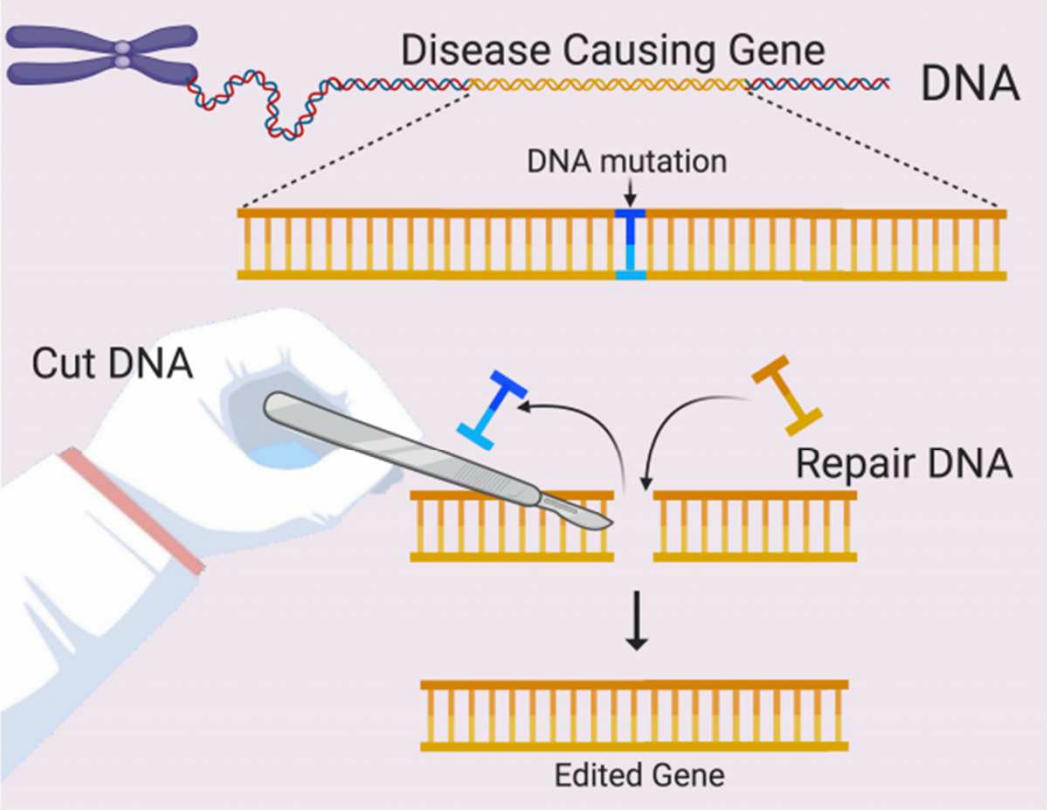
December 2022, Nature



ΓΟΝΙΔΙΑΚΗ ΘΕΡΑΠΕΙΑ: ΚΛΙΝΙΚΕΣ ΕΦΑΡΜΟΓΕΣ

ΘΕΡΑΠΕΙΑ	ΝΟΣΟΣ	ΗΜΕΡΟΜΗΝΙΑ ΕΓΚΡΙΣΗΣ ΑΠΟ FDA
Luxturna	Συγγενής αμαύρωση Leber	*12/2017*
Hemaenix	Αιμορροφιλία Β	*11/2022*
Zynteglo	β-Θαλασσαιμία	*8/2022*
Zolgensma	Νωτιαία μυϊκή ατροφία	*5/2019*
Skysona	Αδρενολευκοδυστροφία	*9/2022*
Elevidys	Μυϊκή δυστροφία Duchenne	*6/2023*

Γονιδιακή Τροποποίηση (Gene Editing)



Κλινικές δοκιμές

- **Θεραπεία καρκίνου**
  με γενετικά τροποποιημένα κύτταρα CAR T
  από το σύστημα CRISPR
  (*από το 2018*)
- **Αιμοποίηση ανθεκτική στον HIV** σε άτομα μολυσμένα

- **Αιμοσφαιρινοπάθειες**
- **Σύνδρομα ανοσοανεπάρκειας**
- **Αιμορροφιλία**
- **Κυστική ίνωση**

SCIENCEINSIDER | ASIA/PACIFIC

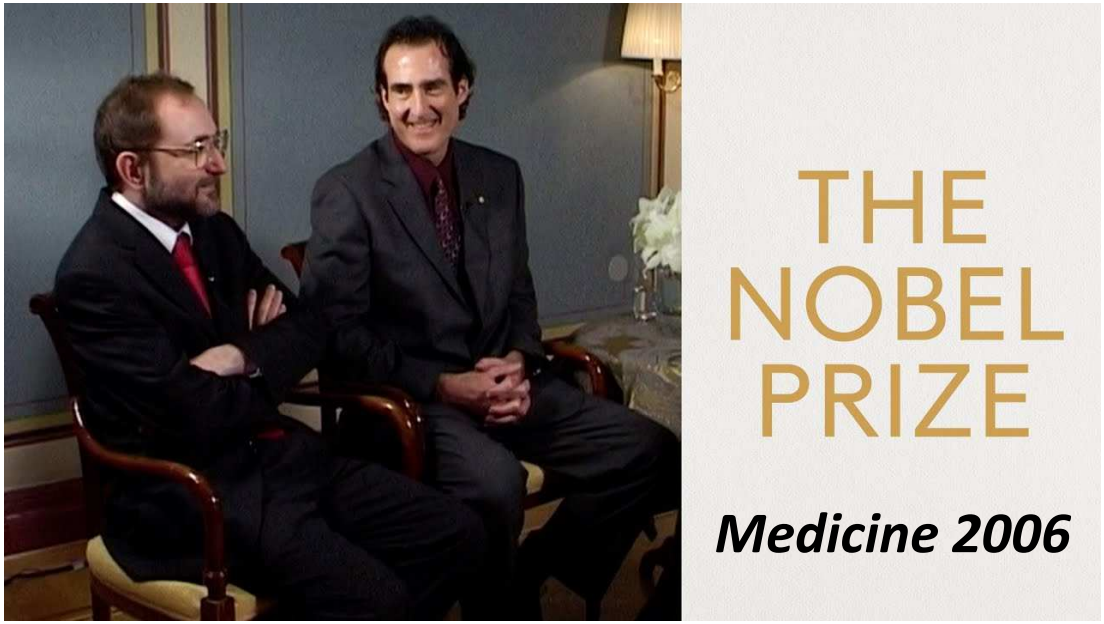
Chinese scientist who produced genetically altered babies sentenced to 3 years in jail

He Jiankui and two collaborators were found guilty of "illegal medical practices"

30 DEC 2019 • BY DENNIS NORMILE

He Jiankui, at a Hong Kong meeting in November 2018 where he presented his work, has not been seen in public since then. ANTHONY KWAN/BLOOMBERG/GETTY IMAGES

Γονιδιακή Σίγαση
(RNA interference-Gene Silencing)



Andrew Z. Fire
Craig C. Mello

Κλινικές εφαρμογές

Μυϊκή δυστροφία Duchenne
Eteplirsen (Exondys 51)

Νωτιαία μυϊκή ατροφία
nusinersen (Spinraza)

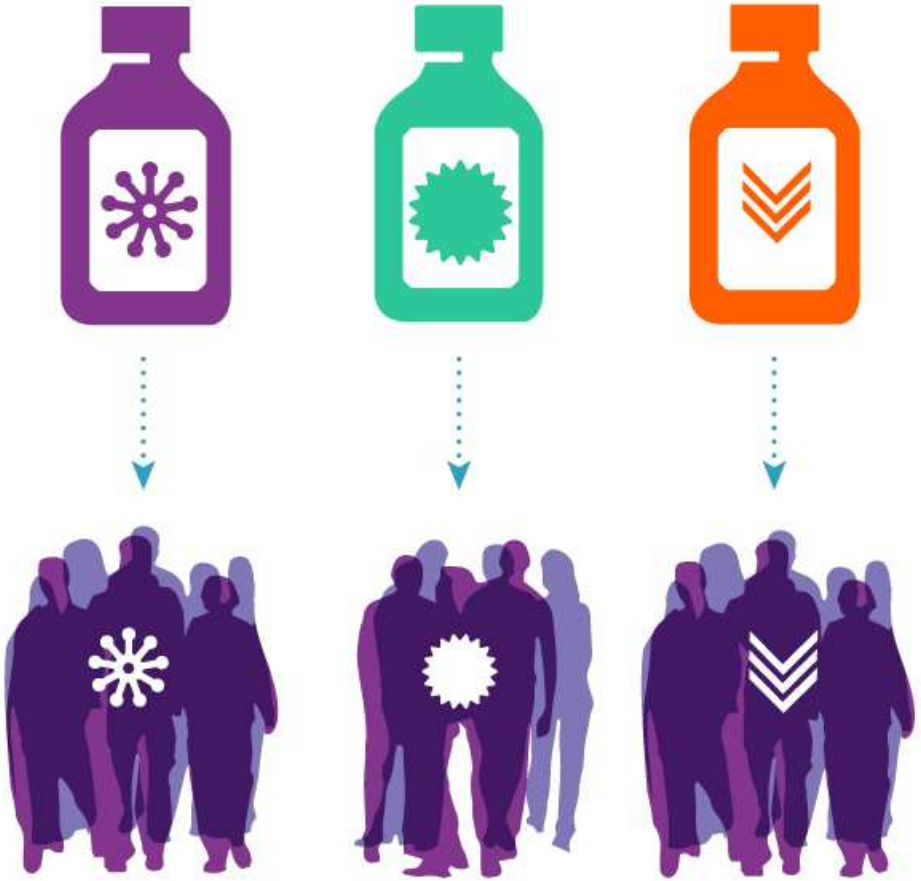
Κλινικές δοκιμές

Αιμορροφιλία
Αιμοσφαιρινοπάθειες
Δυσλιπιδαιμίες
Λοιμώξεις- Ηπατίτιδα Β

Ιατρική Ακριβείας

UNDERSTANDING PRECISION MEDICINE

οι θεραπευτικές αποφάσεις λαμβάνονται βάσει του γενετικού προφίλ του ασθενούς



Η Ιατρική Ακριβείας στον Παιδικό Καρκίνο

PERSPECTIVE article

Front. Oncol.
Sec. Pediatric Oncology
Volume 13 - 2023 | doi: 10.3389/fonc.2023.1279953

Pediatric Precision Oncology: "Better three hours too soon than a minute too late"

Mark Marshall[1*] Jennifer Ivanovich[2] Morgan Schmitt[1] Amy Helvie[3] Lisa Langsford[1] Jennifer Casterline[1] Michael Ferguson[1]

[1] Department of Pediatrics, Indiana University School of Medicine, United States
[2] Department of Medical and Molecular Genetics, Indiana University School of Medicine, United States
[3] The Medical Affairs Company (TMAC), Kennesaw, Georgia, USA, United States

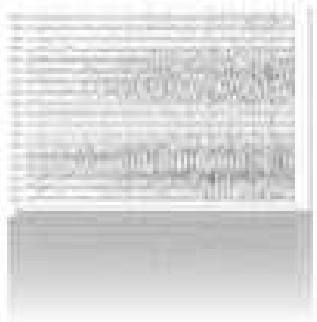
Precision oncology is defined as the selection of an effective treatment for a cancer patient based upon genomic profiling of the patient's tumor to identify targetable alterations. The application of precision oncology towards pediatric cancer patients has moved forward more slowly than with adults but are gaining momentum. Clinical and pharmaceutical advances developed over the past decade for adult cancer indications have begun to move into pediatric oncology, expanding treatment options for young high-risk and refractory patients. As a result, the FDA has approved 23 targeted drugs for pediatric cancer indications, moving targeted drugs into the standard of care. Our precision oncology program is in a medium sized children's hospital, lacking internal sequencing capabilities and bioinformatics. We have developed methods, medical and business partnerships to provide stateof-the-art tumor characterization and targeted treatment options for our patients. We present here a streamlined and practical protocol designed to enable any oncologist to implement precision oncology options for their patients.

Η Ιατρική Ακριβείας στην Παιδική Επιληψία

Καθημερινοί σπασμοί

Ιατρική Ακριβείας

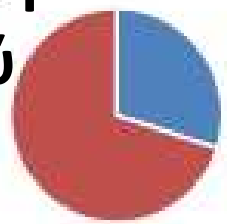
Παιδί με «ανθεκτική» επιληψία



Βελτίωση θεραπείας >20%

Κατανόηση μηχανισμού νόσου

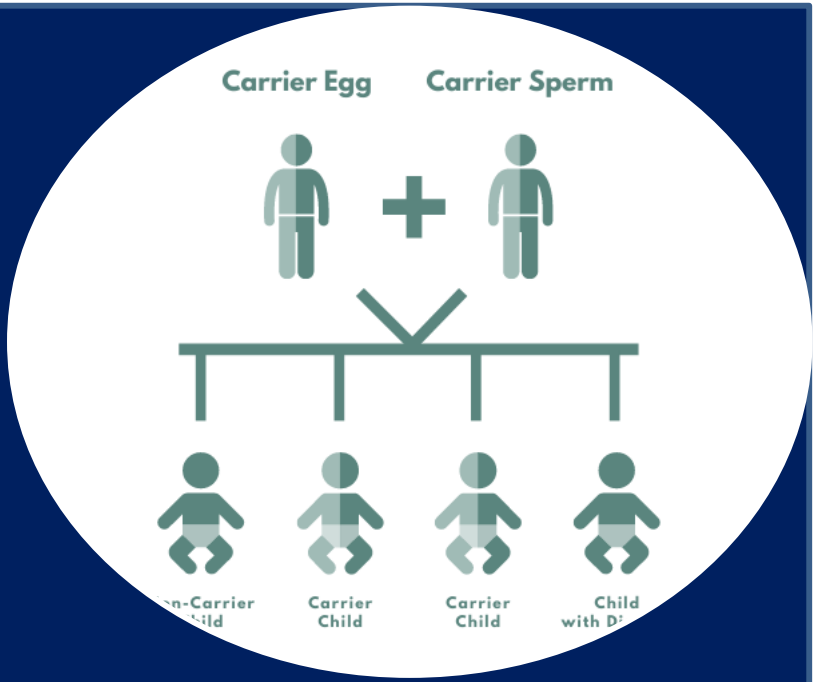
Ανίχνευση γενετικού αιτίου



>30% άγνωστο

Η Ιατρική Ακριβείας στην Παιδονεφρολογία

SCREENING
ΓΕΝΕΤΙΚΩΝ Ν0ΣΩΝ



1. Screening φορέων αναπαραγωγικής ηλικίας

2. Μη επεμβατικός προγεννητικός έλεγχος (NIPT)

3. Νεογνικό screening

Ανίχνευση φορέων αναπαραγωγικής ηλικίας
(Reproductive Carrier Screening)

targeted carrier screening

στοχευμένα σε θετικό οικογενειακό ιστορικό

πχ. SMA

ethnic-based carrier screening

βάσει εθνότητας

πχ. β-θαλασσαιμία σε Ελλάδα και Κύπρο
ή Tay-Sachs σε Ισραήλ

τα συχνότερα υπολειπόμενα γονιδιακά νοσήματα στον ελληνικό πληθυσμό

ΓΟΝΙΔΙΑΚΟ ΝΟΣΗΜΑ	ΣΥΧΝΟΤΗΤΑ ΓΕΝΝΗΣΕΩΝ	ΤΥΠΟΣ ΝΟΣΗΜΑΤΟΣ	ΔΙΑΓΝΩΣΤΙΚΗ ΕΞΕΤΑΣΗ	ΣΥΧΝΟΤΗΤΑ ΦΟΡΕΩΝ
β-Θαλασσαιμία	*	Γονιδιακός υπολειπόμενος	Μοριακός έλεγχος	8-10%
Κυστική ίνωση	1: 2500	Γονιδιακός υπολειπόμενος	Μοριακός έλεγχος	4%
Συγγενής βαρηκοΐα (Cx26)	1:3300	Γονιδιακός υπολειπόμενος	Μοριακός έλεγχος	3,5%
Νωτιαία μυϊκή δυστροφία	1: 10000	Γονιδιακός υπολειπόμενος	Μοριακός έλεγχος	2%

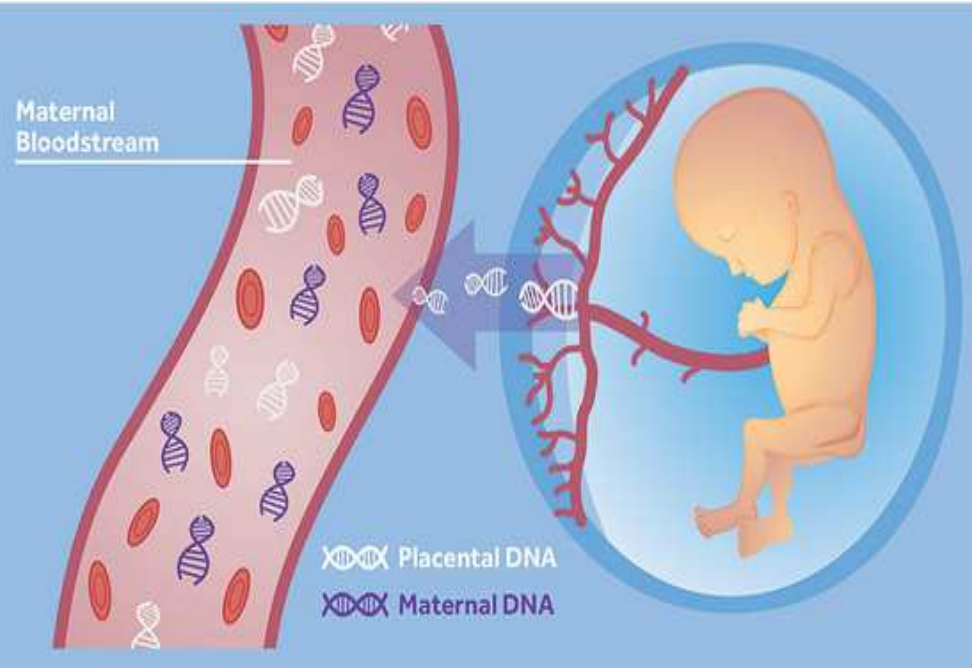
Διευρυμένο screening φορέων γενετικών νόσων

Expanded Carrier Screening

Expanded Carrier Panel	Provider	Number of Diseases/Genes Screened
Counsyl Foresight	Counsyl	176
Invitae Comprehensive Carrier Screen	Invitae	287
Invitae Broad Pan-Ethnic Carrier Screen	Invitae	46
InheriGen	GenPath	180
InherigenTx	GenPath	75
Inheritest	LabCorp	97
Horizon Carrier Screen	Natera	274

ΜΗ ΕΠΕΜΒΑΤΙΚΟΣ ΠΡΟΓΕΝΝΗΤΙΚΟΣ ΕΛΕΓΧΟΣ

Cell Free DNA Test - NIPT



	Ευαισθησία (%)	Ειδικότητα (%)
Trisomy 21	99	100
Trisomy 18	97	100
Trisomy 13	92	100

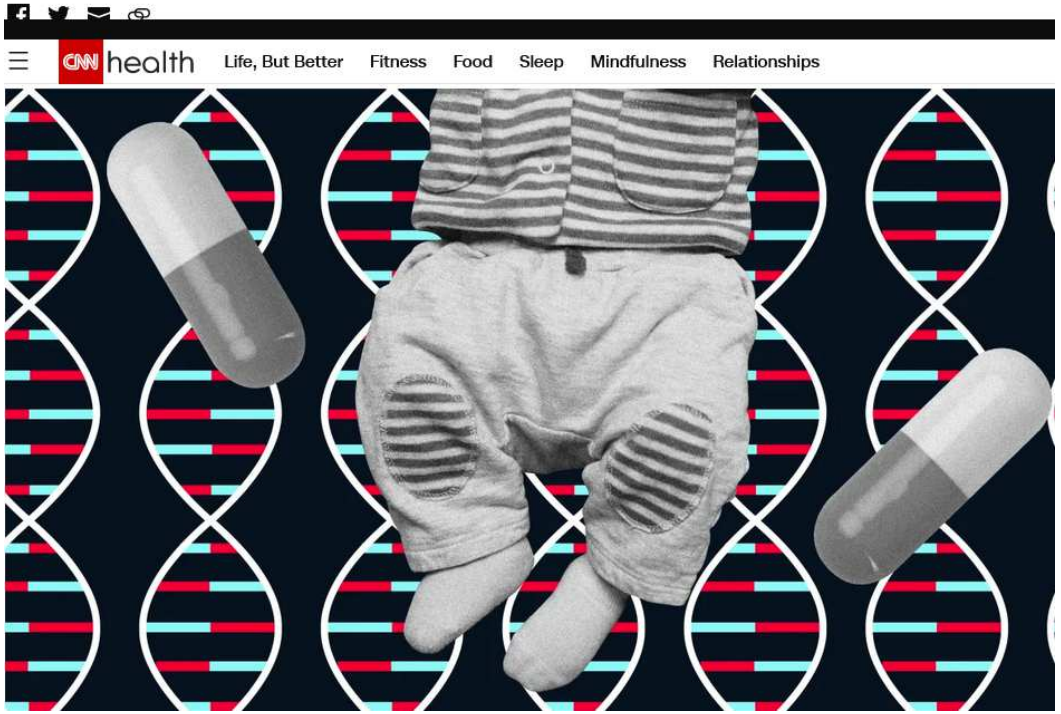
Νεογνικό Screening

Guthrie test

Νεογνικό Γενωμικό Screening

100,000 newborn babies will have their genomes sequenced in the UK. It could have big implications for child medicine

By Thomas Page, CNN
Updated 7:34 AM EDT, Mon March 20, 2023

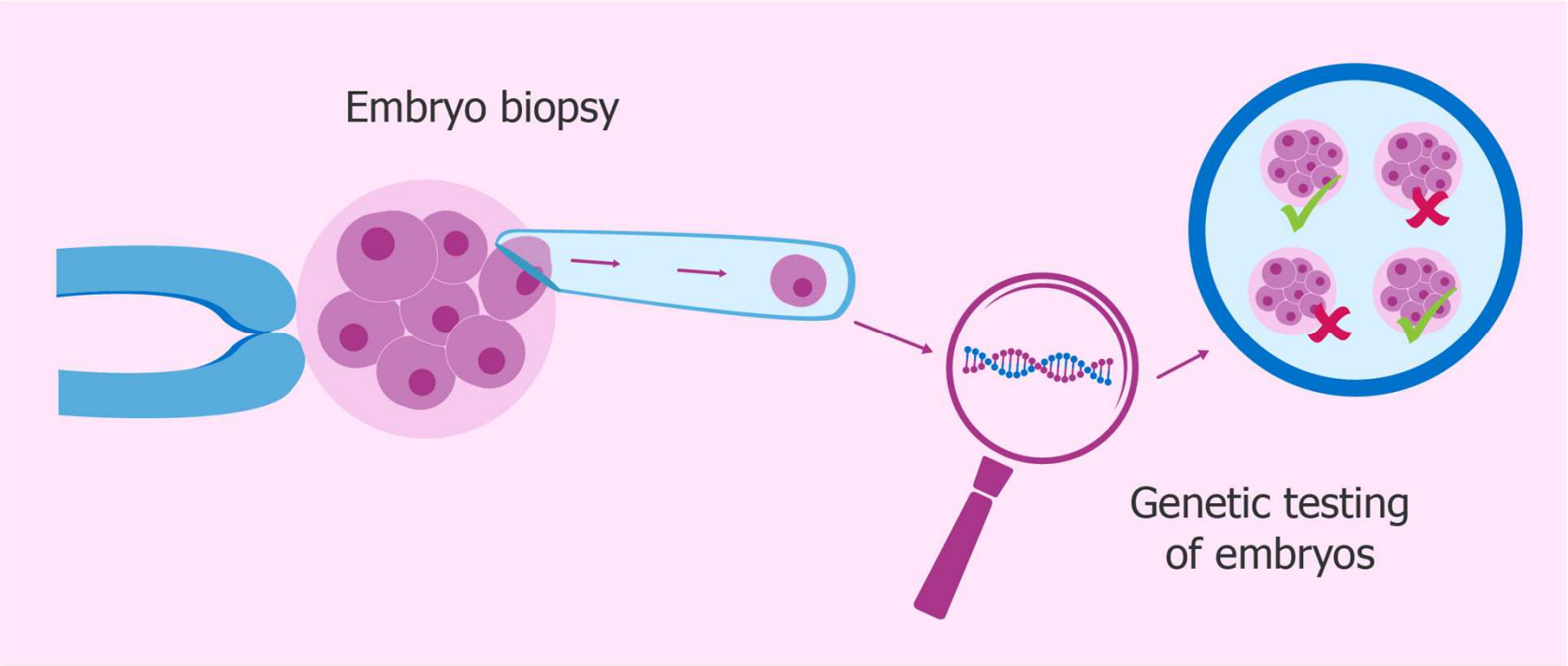


ΠΡΟΛΗΨΗ ΓΕΝΕΤΙΚΩΝ ΝΟΣΩΝ

1. Προεμφυτευτική Γενετική Διάγνωση

2. Προγράμματα πρόληψης νοσημάτων Φθοράς

Προεμφυτευτική Γενετική Διάγνωση (PGD)



Γονιδίωμα

Οικογενειακό ιστορικό

Κληρονομικότητα

Αλλαγές στην αλληλουχία DNA

Περιβάλλον

Ηλικία

Διατροφή

Άσκηση

Ενδομήτριο περιβάλλον

Λοιμώξεις

Επιγενετική

Μεθυλίωση DNA

Τροποποιήσεις ιστόνης

Μικρο-RNAs


JANUARY 10, 2011
Joe Klein: The CIA's Afghan Disaster
Yemen: The New Center Of Terror
Why the Recession Hasn't Been Cool To Teens
TIME
WHY YOUR DNA ISN'T YOUR DESTINY
The new science of epigenetics reveals how the choices you make can change your genes —and those of your kids
BY JOHN CLOUD
www.time.com

Επιγενετική

Πώς η συμπεριφορά μας «αλλάζει» το DNA μας!!

Prenatal	Postnatal

Maternal diet

Undernutrition

Stress, anxiety

Pharmacological treatments

Assisted reproductive technologies

Nutrition

Physical exercise

Chemical compounds

Pharmacological treatments

Unhealthy habits

Η επιγενετική επίδραση της Φυσικής Άσκησης

dementia by up to **30%**

All-cause mortality by **30%**

cardiovascular disease by up to **35%**

hip fractures by up to **68%**

type 2 diabetes by up to **40%**

depression by up to **30%**

colon cancer by **30%**

breast cancer by **20%**

σας ευχαριστώ