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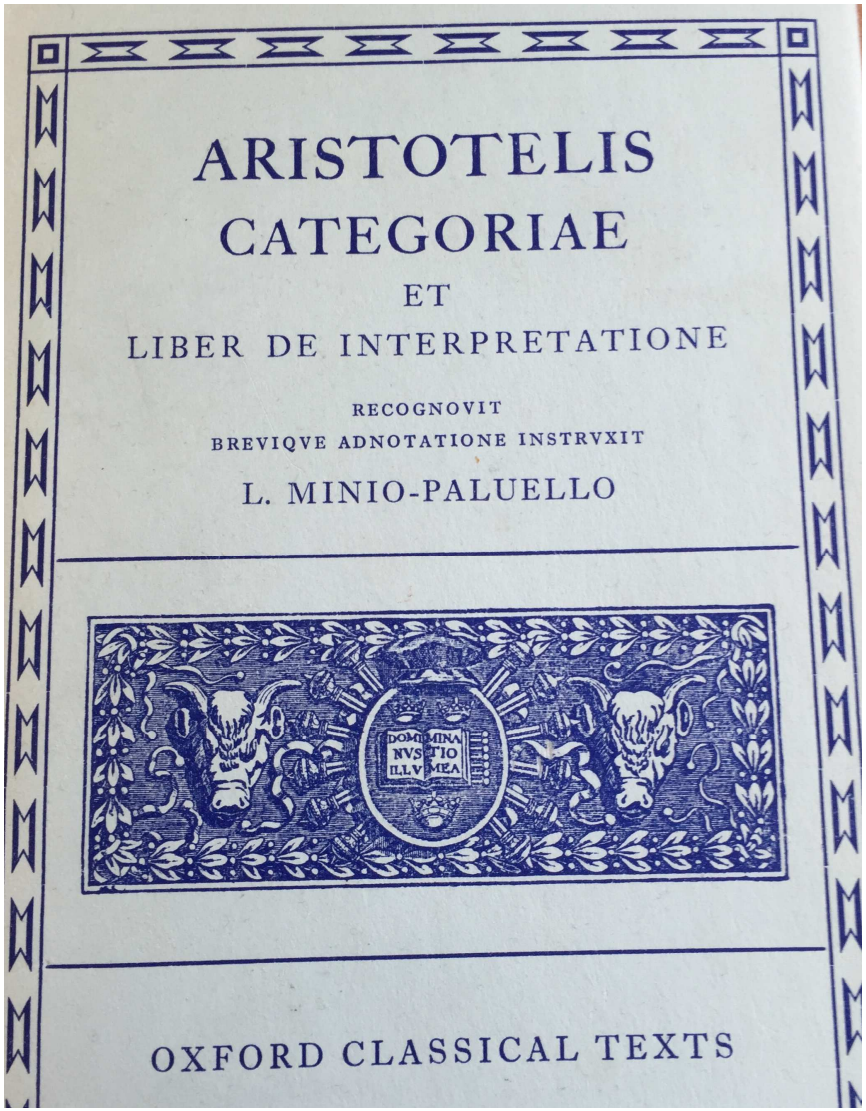
… the first genuine scientist in history ... every scientist is in his debt (Encyclopædia Britannica)



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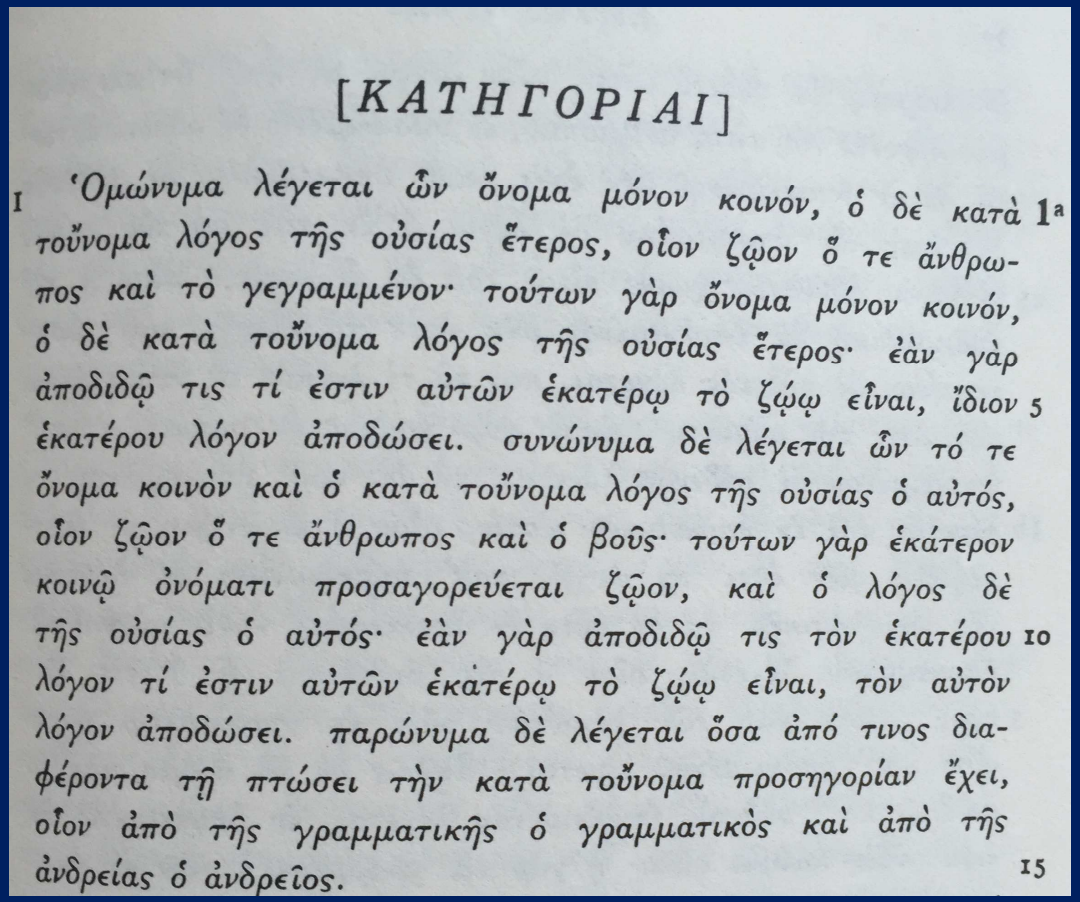


[ΚΑΤΗΓΟΡΙΑΙ]

1 Ὁμώνυμα λέγεται ὧν ὄνομα μόνον κοινόν, ὁ δὲ κατὰ 1ª
τοὔνομα λόγος τῆς οὐσίας ἕτερος, οἷον ζῷον ὅ τε ἄνθρω-
πος καὶ τὸ γεγραμμένον· τούτων γὰρ ὄνομα μόνον κοινόν,
ὁ δὲ κατὰ τοὔνομα λόγος τῆς οὐσίας ἕτερος· ἐὰν γὰρ
ἀποδιδῷ τις τί ἐστιν αὐτῶν ἑκατέρῳ τὸ ζῴῳ εἶναι, ἴδιον 5
ἑκατέρου λόγον ἀποδώσει. συνώνυμα δὲ λέγεται ὧν τό τε
ὄνομα κοινὸν καὶ ὁ κατὰ τοὔνομα λόγος τῆς οὐσίας ὁ αὐτός,
οἷον ζῷον ὅ τε ἄνθρωπος καὶ ὁ βοῦς· τούτων γὰρ ἑκάτερον
κοινῷ ὀνόματι προσαγορεύεται ζῷον, καὶ ὁ λόγος δὲ
τῆς οὐσίας ὁ αὐτός· ἐὰν γὰρ ἀποδιδῷ τις τὸν ἑκατέρου 10
λόγον τί ἐστιν αὐτῶν ἑκατέρῳ τὸ ζῴῳ εἶναι, τὸν αὐτὸν
λόγον ἀποδώσει. παρώνυμα δὲ λέγεται ὅσα ἀπό τινος δια-
φέροντα τῇ πτώσει τὴν κατὰ τοὔνομα προσηγορίαν ἔχει,
οἷον ἀπὸ τῆς γραμματικῆς ὁ γραμματικὸς καὶ ἀπὸ τῆς
ἀνδρείας ὁ ἀνδρεῖος. 15

ΑΓΕΩΜΕΤΡΗΤΟΣ ΜΗΔΕΙΣ ΕΙΣΙΤΩ

ο άρρωστος
… διδάσκει τον γιατρό ;

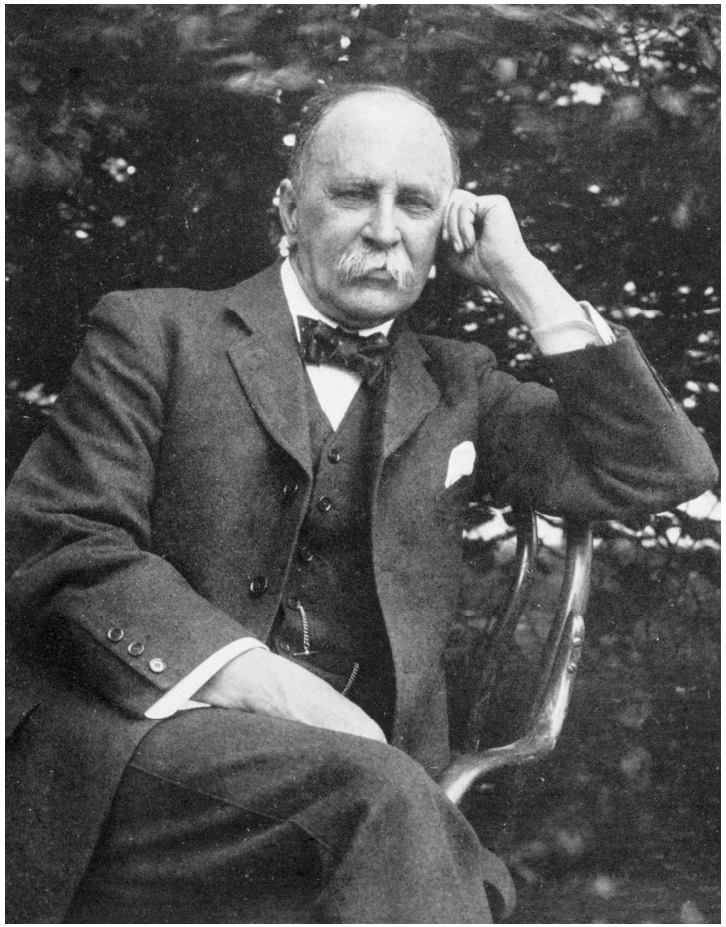
Η ΠΑΡΑΤΗΡΗΣΗ «ΤΟΥ ΕΠΙΣΤΗΜΟΝΑ» ΔΙΔΑΣΚΕΙ

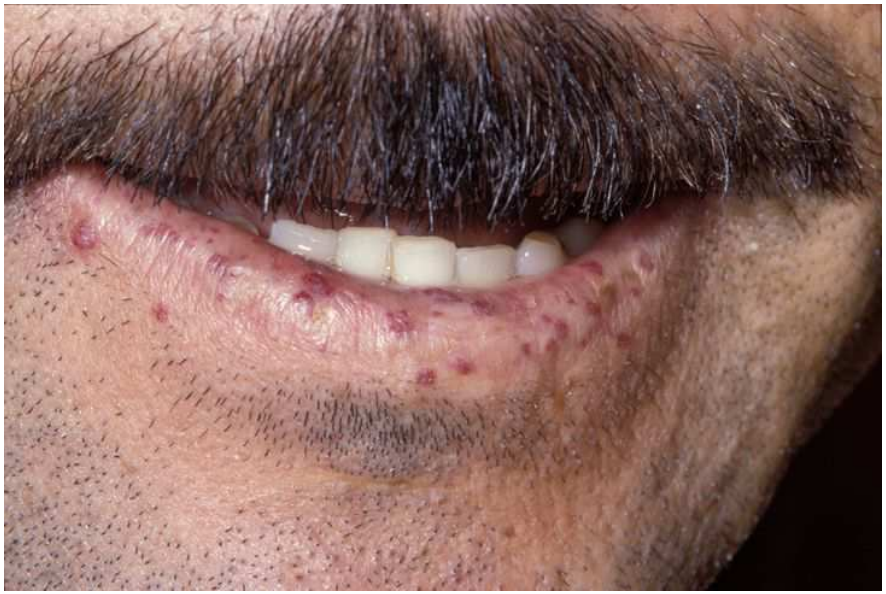
Sir William Osler

1849 - 1919

1849 - 1919

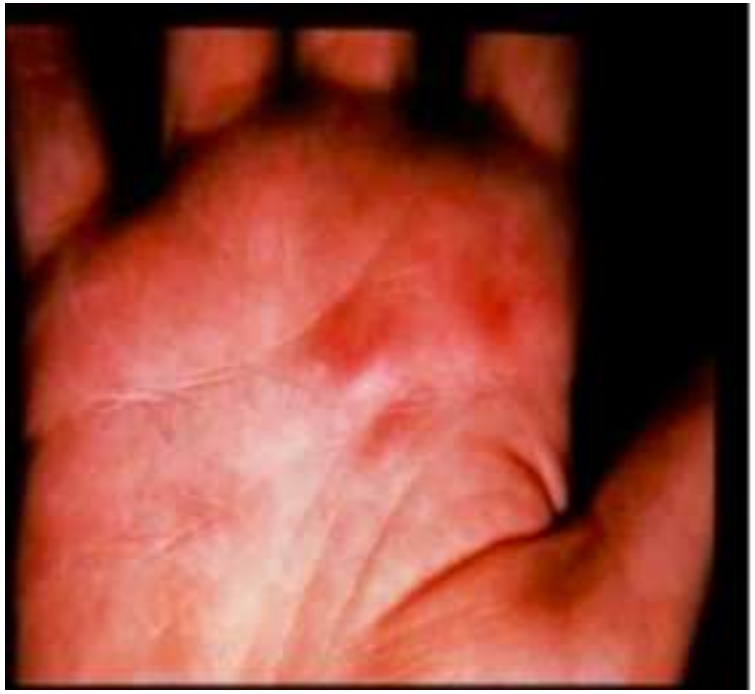
Osler took a patient-centred approach to teaching.
By teaching at the bedside, he was able to demonstrate, watch and assess students as they examined patients, blood and urine samples using ward micro- scopes, and, following death, he discussed postmortem findings.

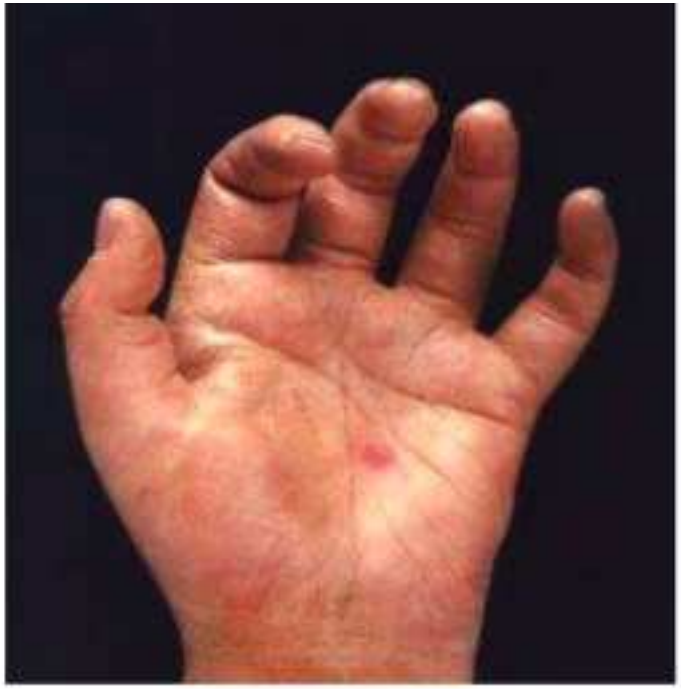




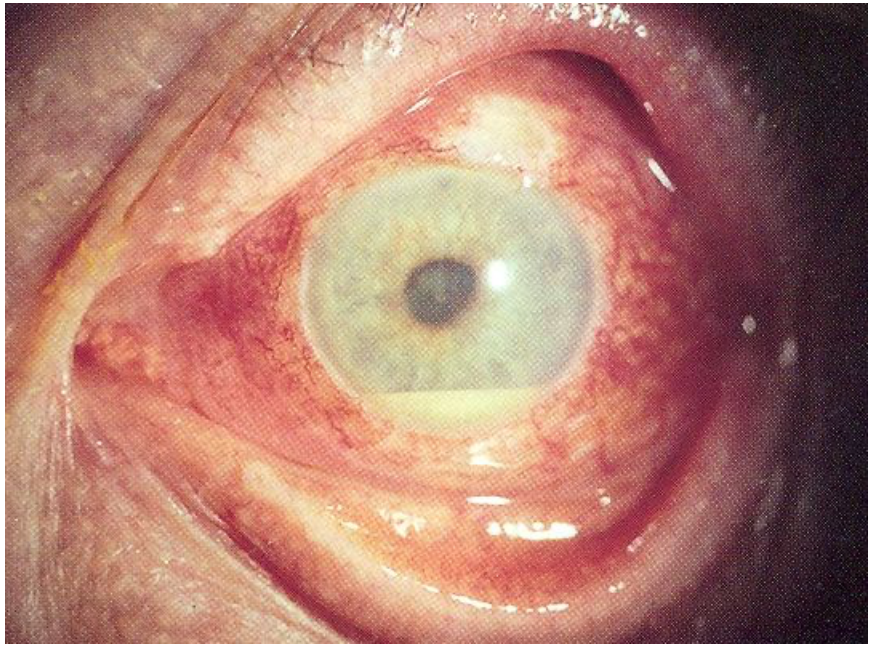
Osler–Weber–Rendu disease
(Hereditary hemorrhagic telangiectasia)

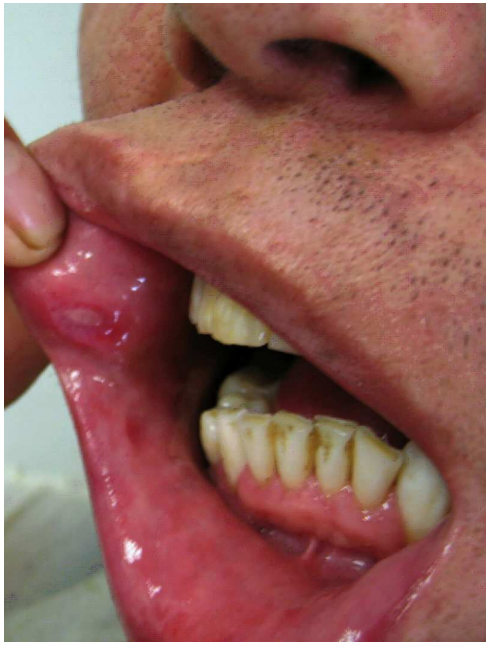
Osler’a nodes





Νόσος Βενέδικτου Αδαμαντιάδη (1930) – Behçet (1937)





1952: Bruton's syndrome or X-linked agammaglobulinemia

PEDIATRICS®

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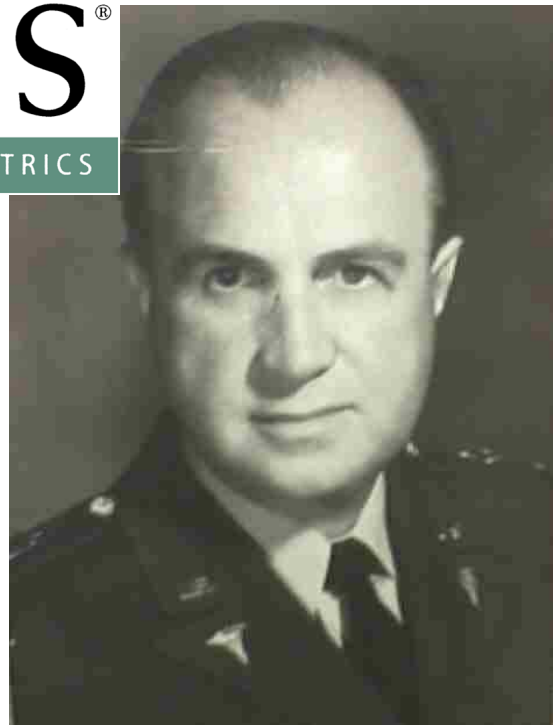
AGAMMAGLOBULINEMIA
OGDEN C. BRUTON
Pediatrics 1952;9;722



NATURE 1993

* Division of Medical and Molecular Genetics, UMDS of Guy's and St Thomas's Hospitals, Guy's Tower, London SE1 9RT, UK
‡ Center for BioTechnology, Karolinska Institute, NOVUM, S-14157 Huddinge, Sweden
§ Unit for Applied Cell and Molecular Biology, Umeå University, S-901 87 Umeå, Sweden
|| Molecular Immunology Unit, Institute of Child Health, 30 Guilford Street, London WC1N 1EH, UK

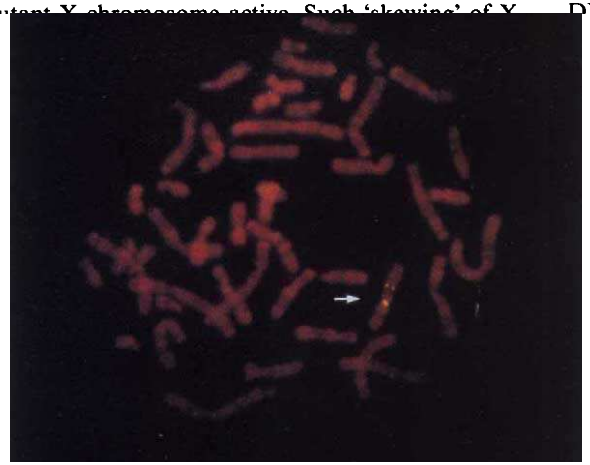
X-linked agammaglobulinaemia (XLA) is a human immunodeficiency caused by failure of pre-B cells in the bone marrow to develop into circulating mature B cells. A novel gene has been isolated which maps to the XLA locus, is expressed in B cells, and shows mutations in families with the disorder. The gene is a member of the *src* family of proto-oncogenes which encode protein-tyrosine kinases. This is, to our knowledge, the first evidence that mutations in a *src*-related gene are involved in human genetic disease.

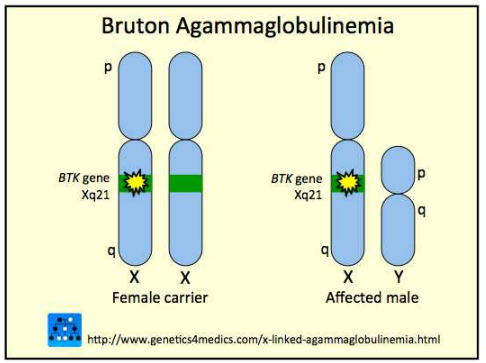
X-LINKED agammaglobulinaemia (XLA; Bruton type; MIM 30030; gene symbol AGMX1) was the first described immunoglobulin deficiency[1]. Affected males lack circulating mature B cells and serum immunoglobulins of all isotypes, and suffer recurrent bacterial infections[2]. The infections, particularly bacterial meningitis and pneumonia, are life-threatening at an early age and require the use of antibiotic and immunoglobulin replacement therapy. Affected males have a normal number of pre-B cells in their bone marrow, suggesting that the XLA defect resides in the pathway of B-cell development[3]. This defect is specific to the B-cell lineage because other lymphocyte populations appear to be normal. Heterozygous females appear immunologically normal, as a result of selection against B cells having the mutant X chromosome active. Such 'skewing' of X

involved in the disease and, therefore, in the process of B-cell development.

Cloning strategy

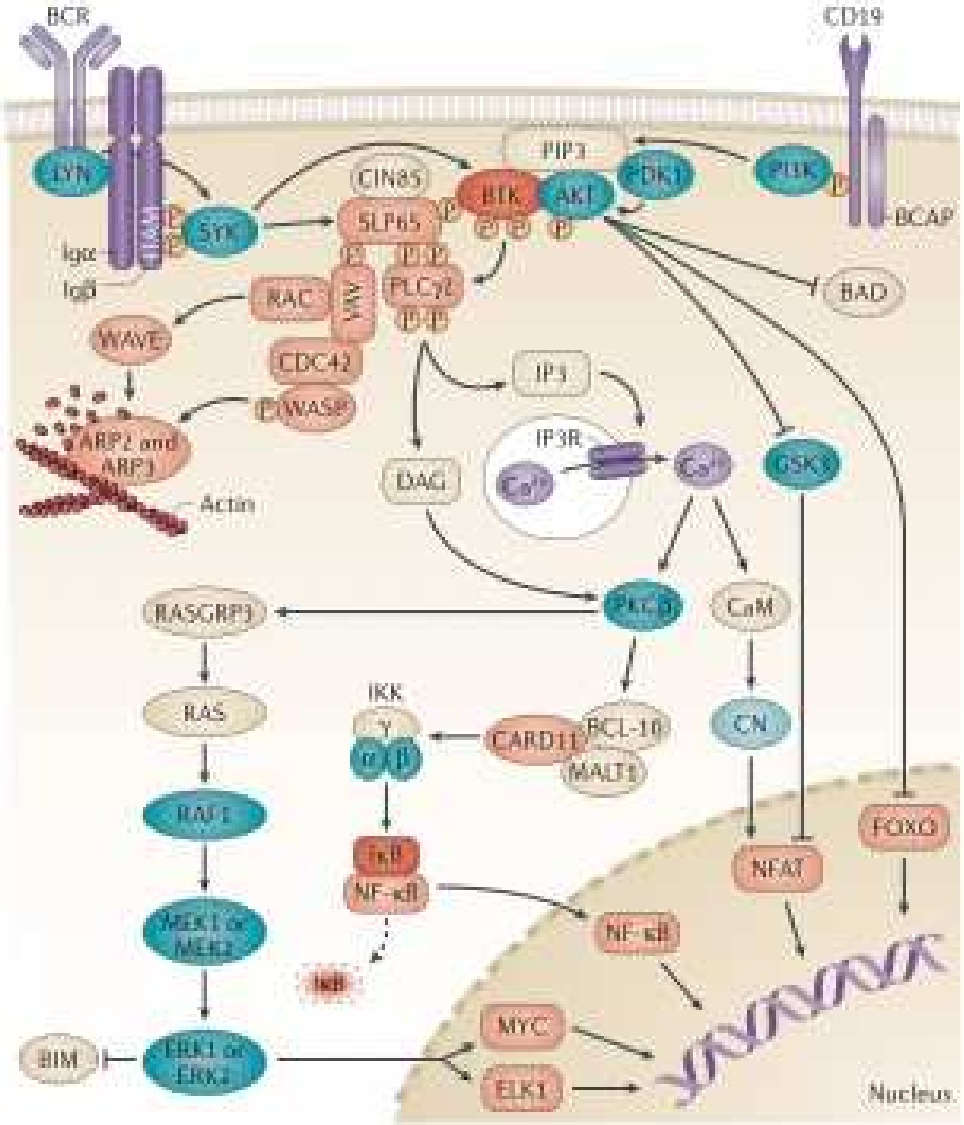
The XLA locus has been mapped to the region Xq21.3–Xq22 (refs 17–21 and R. Lovering *et al.*, manuscript in preparation). The consensus order of loci in the region was established as cen–DXS3–(XLA, DXS178)–DXS94–DXS17–tel, with the polymorphic marker DXS178 showing no recombination with the disease (refs 20, 21 and R. Lovering *et al.*, manuscript in preparation), and DXS3 and DXS94 lying approximately 10 centimorgans (cM) apart (Fig. 1*a*). Recently, the identification of recombinants between XLA and the flanking markers DXS442 and DXS101 resulted in the further localization of XLA to within



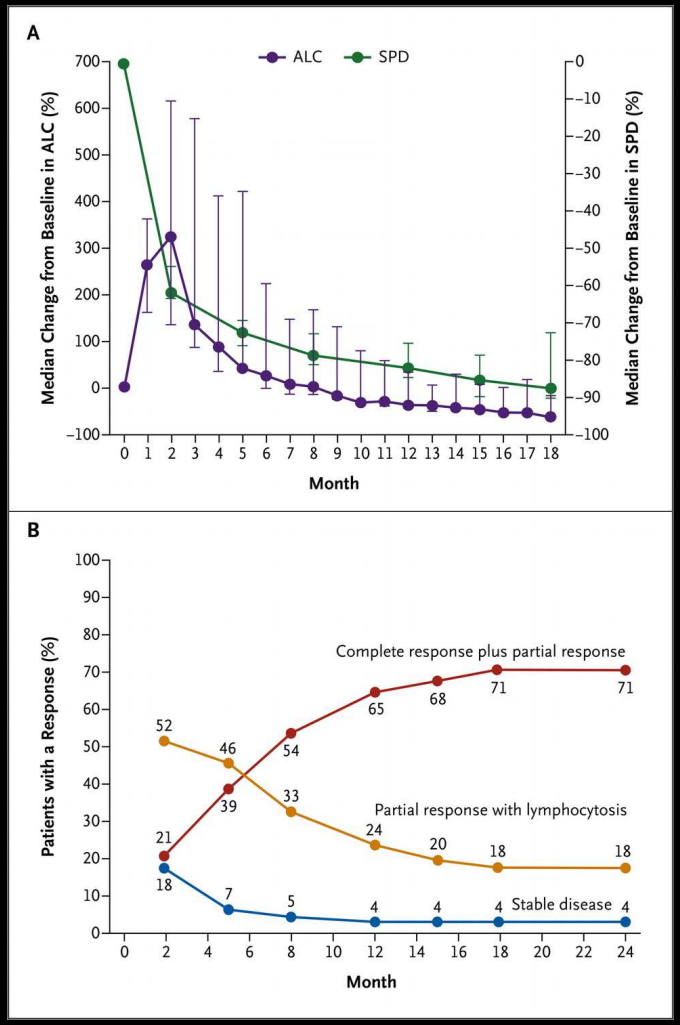


Targeting Bruton's tyrosine kinase in B cell malignancies

Rudi W. Hendriks, Saravanan Yuvaraj and Laurens P. Kil



Response to Ibrutinib over Time.

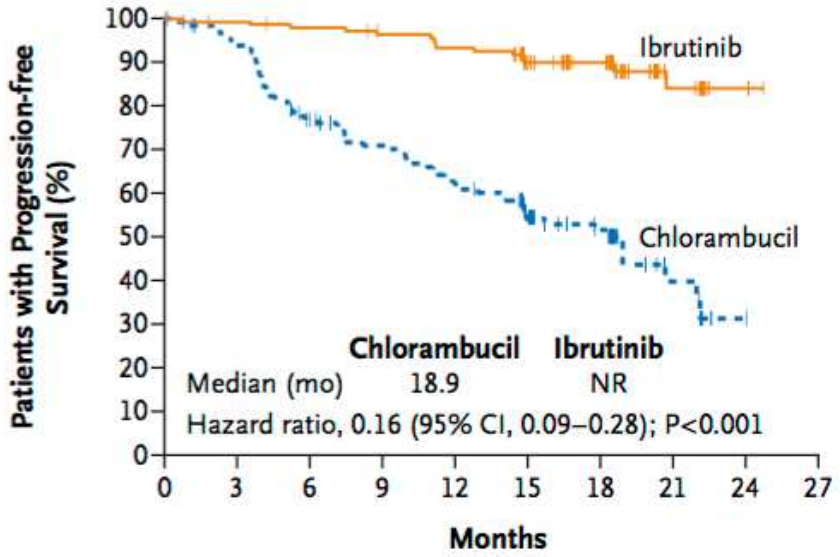


The NEW ENGLAND JOURNAL of MEDICINE

N ENGL J MED 373;25 NEJM.ORG DECEMBER 17, 2015

The New England Journal of Medicine

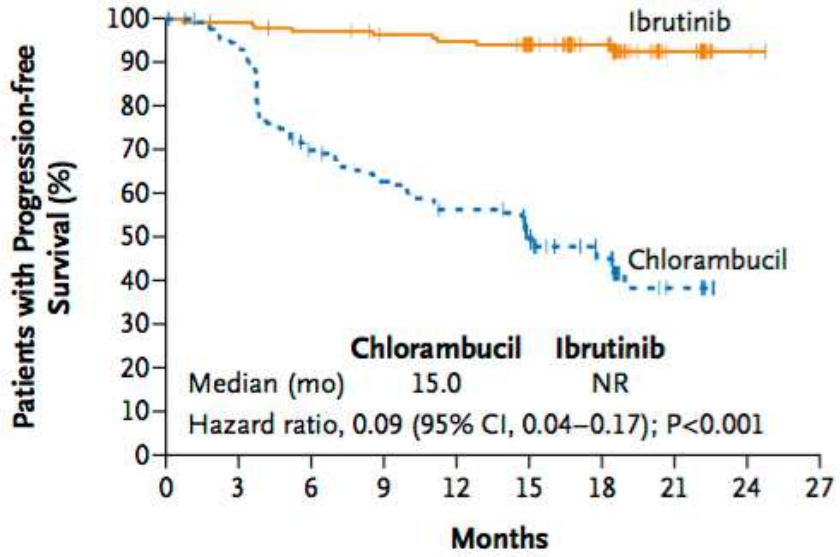
A **Progression-free Survival According to Independent Assessment**



No. at Risk

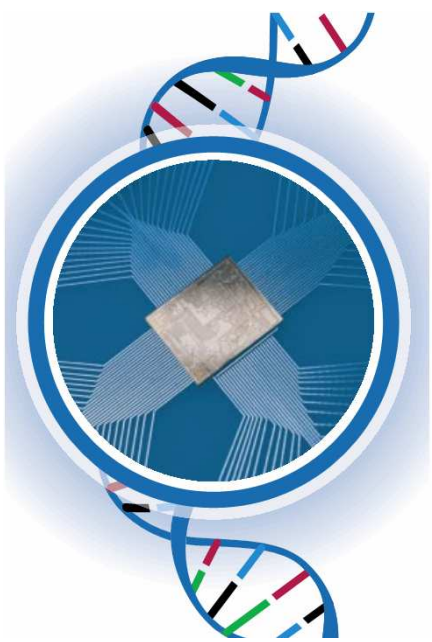
Ibrutinib	136	133	130	126	122	98	66	21	2	0
Chlorambucil	133	121	95	85	74	49	34	10	0	0

B **Progression-free Survival According to Investigator Assessment**



No. at Risk

Ibrutinib	136	133	129	125	123	104	69	22	2	0
Chlorambucil	133	121	88	78	69	46	31	10	0	0



2001 – 2016

Αντικατοπτρισμοί 2016