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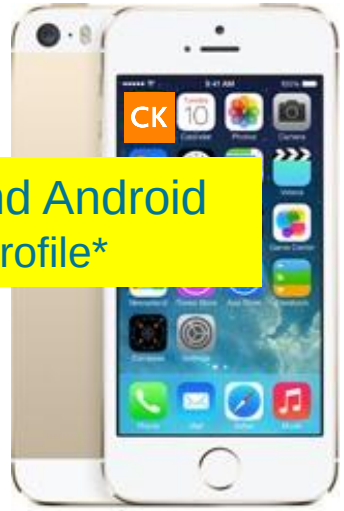

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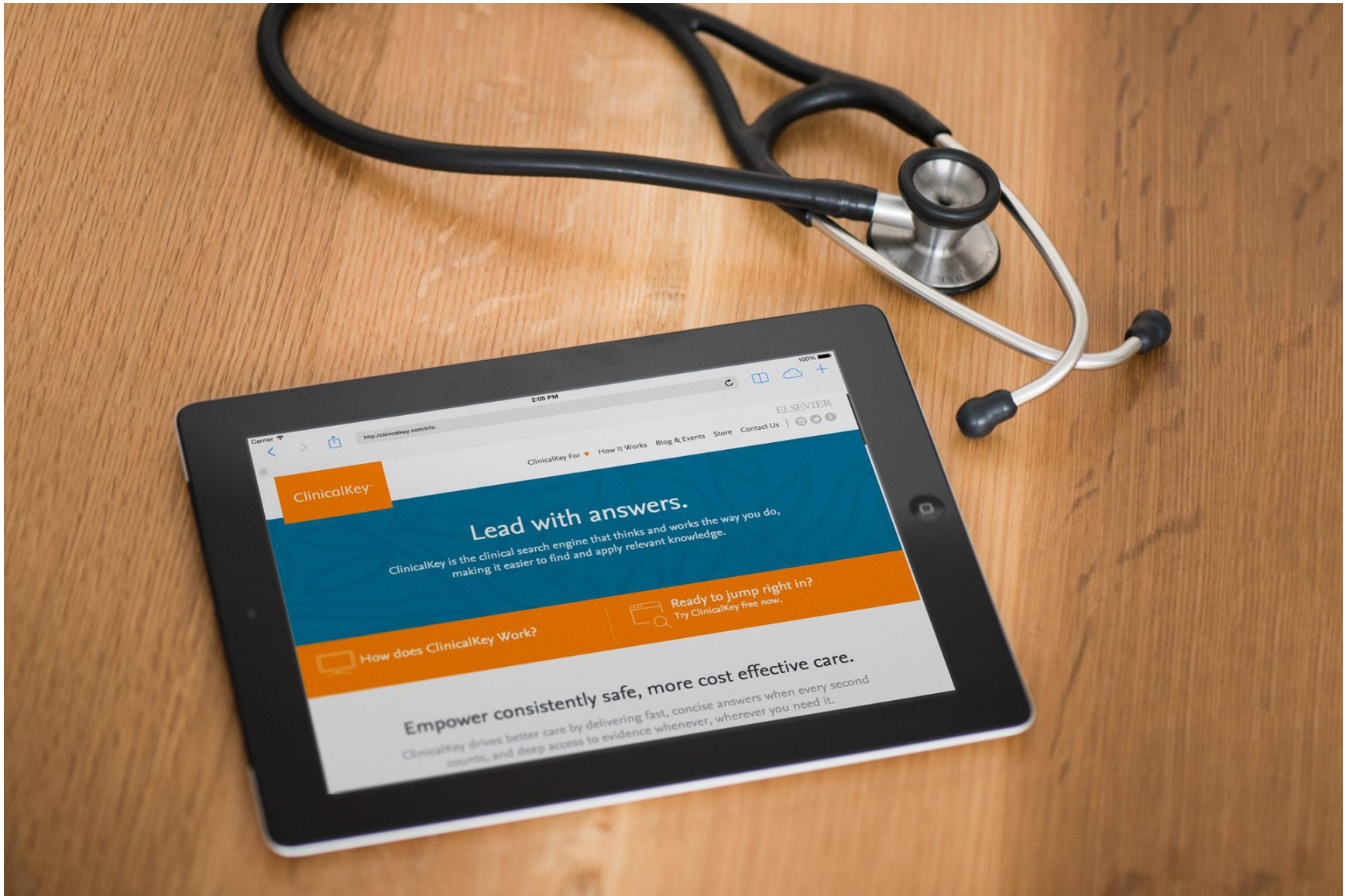

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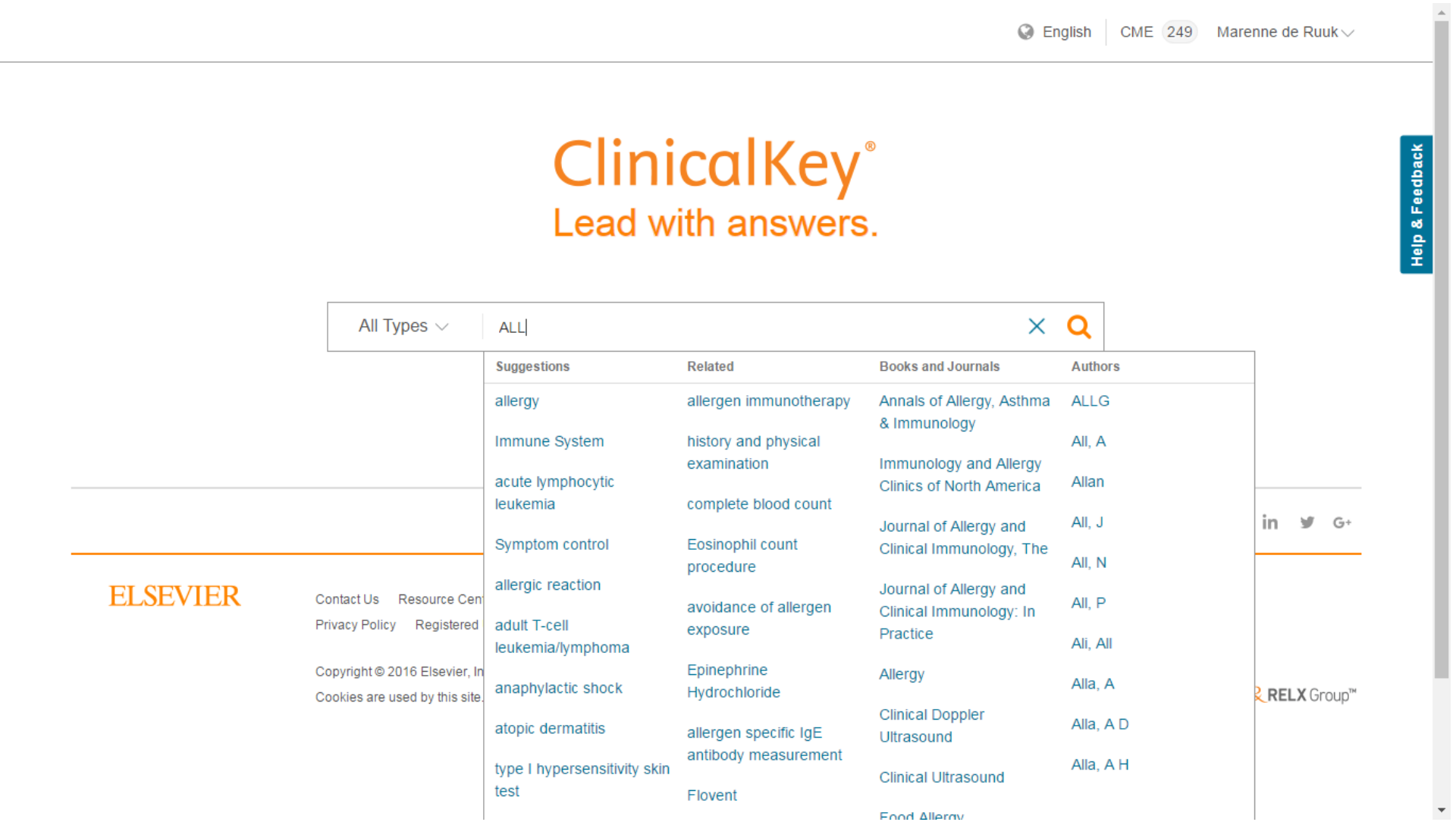
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Ferri, Fred F., M.D., F.A.C.P.. Published January 2, 2016. Pages 61-63.e1. © 2016.

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Definition

Acute lymphoblastic leukemia (ALL) is a malignancy of B or T lymphoblasts characterized by uncontrolled proliferation of abnormal, immature lymphocytes and their progenitors, ultimately replacing normal bone marrow elements.

Etiology

• Unknown; increased risk in patients with a previous use of antineoplastic agents (e.g., chemotherapy of non-Hodgkin's lymphoma, Hodgkin's disease, ovarian cancer, myeloma).

• Environmental factors (e.g., ionizing radiation), toxins (e.g., benzene).

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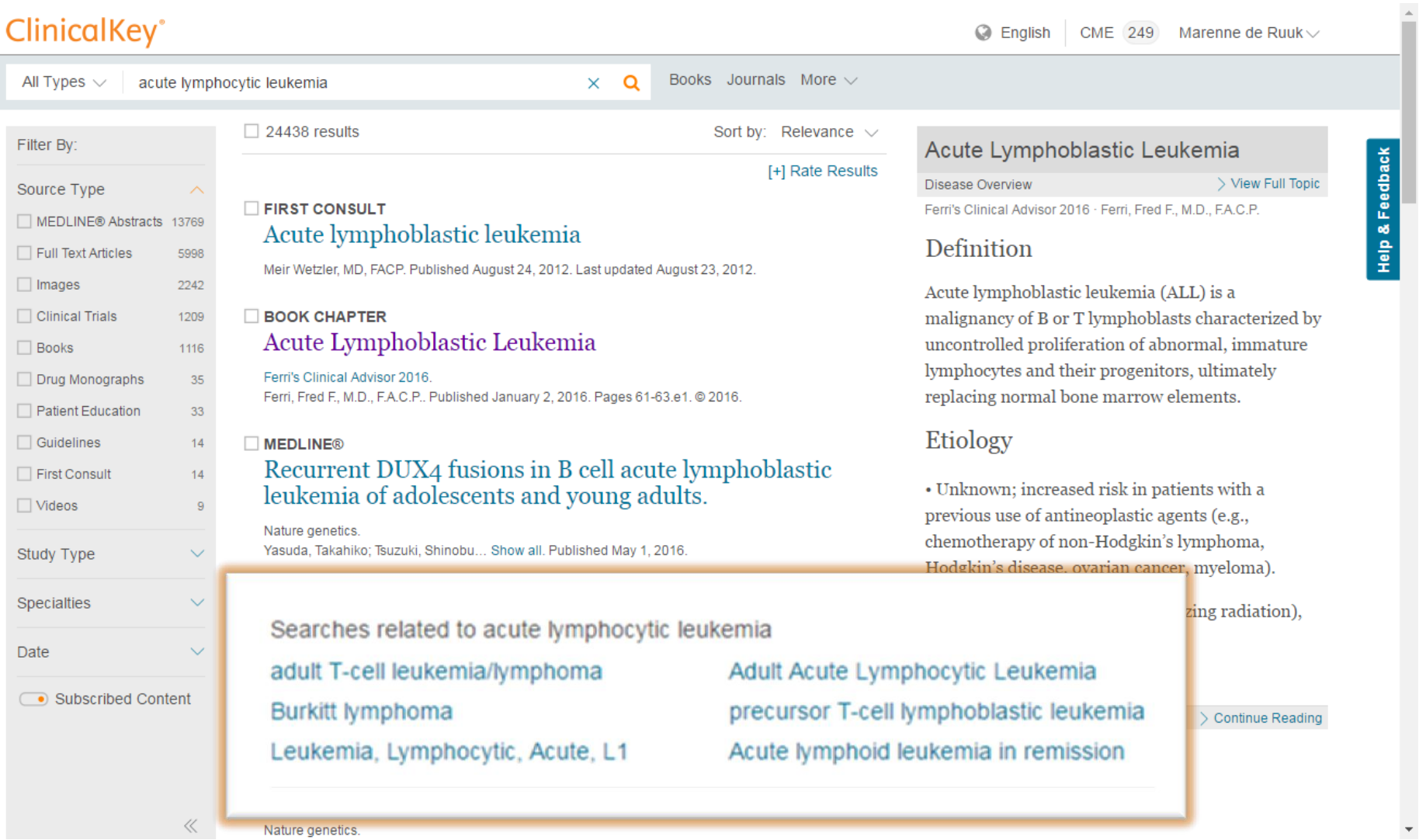
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Fred F. Ferri M.D., F.A.C.P.

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Acute lymphoblastic leukemia (ALL) is a malignancy of B or T lymphoblasts characterized by uncontrolled proliferation of abnormal, immature lymphocytes and their progenitors, ultimately replacing normal bone marrow elements.

Synonyms

Lymphoid leukemia

ALL

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Acute General Rx

Table A1-24 summarizes the preferred approach to the treatment of adult ALL patients. For younger adults, induction therapy usually consists of an anthracycline, vincristine, l -asparaginase, and a corticosteroid.

TABLE A1-24
Preferred Approach to the Treatment of Adult Acute Lymphocytic Leukemia Patients

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AC, Cytosine arabinoside; *ALL,* acute lymphocytic leukemia; *CR,* complete remission; *HDM,* high-dose

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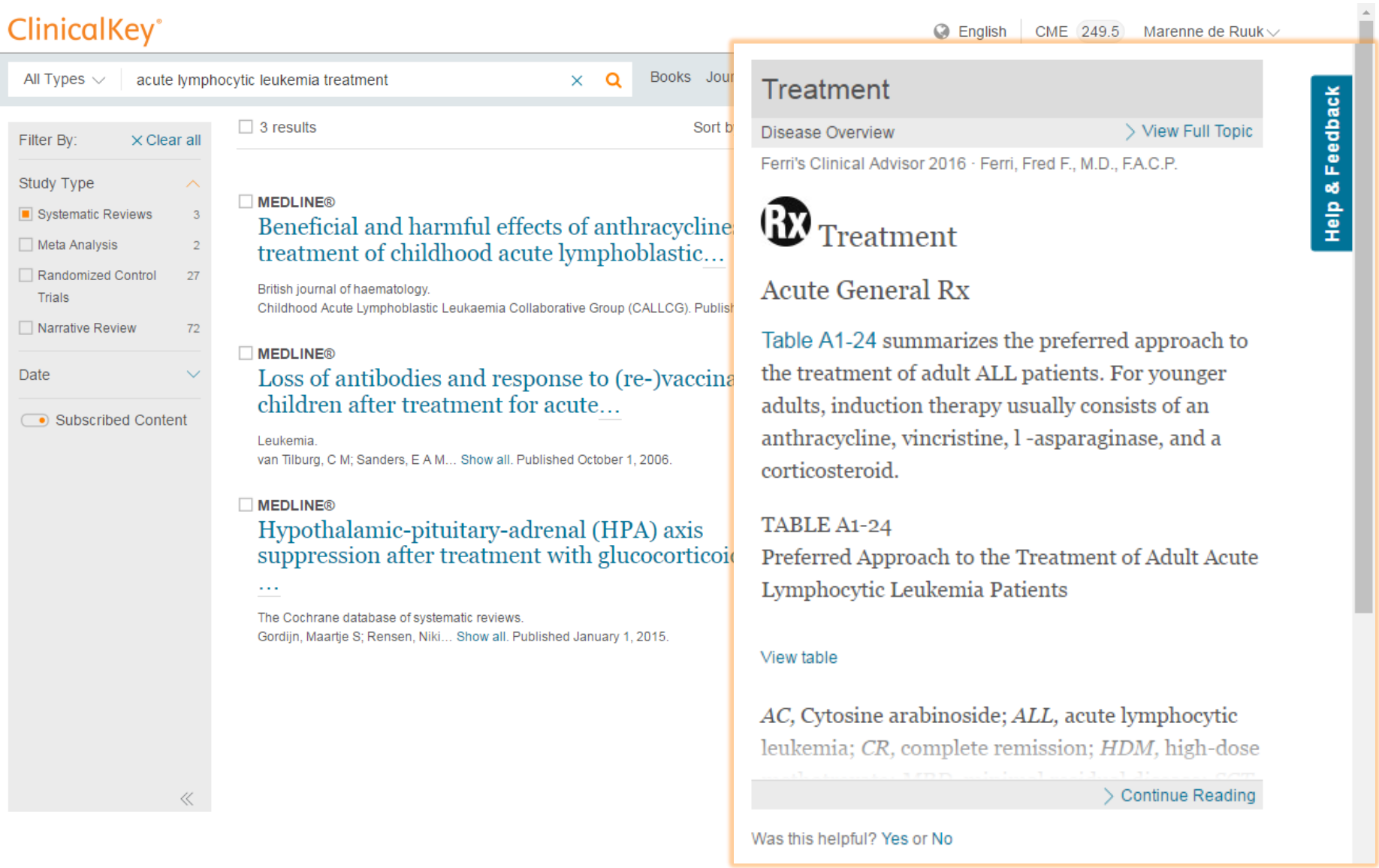
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AC, Cytosine arabinoside; ALL, acute lymphocytic leukemia; CR, complete remission; HDM, high-dose
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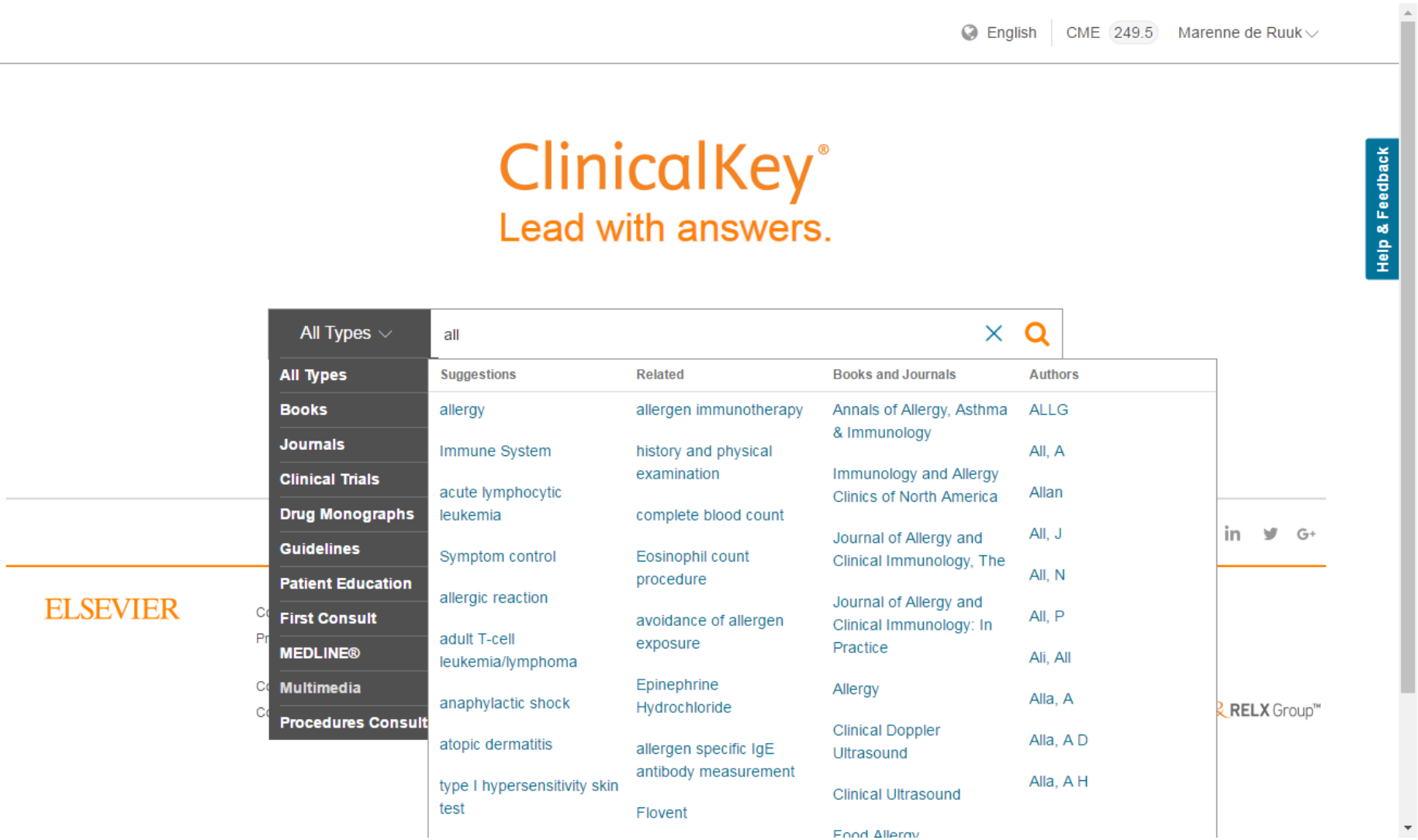
Rx Treatment

Acute General Rx

Table A1-24 summarizes the preferred approach to the treatment of adult ALL patients. For younger adults, induction therapy usually consists of an anthracycline, vincristine, l -asparaginase, and a corticosteroid.

TABLE A1-24
Preferred Approach to the Treatment of Adult Acute Lymphocytic Leukemia Patients

	Low-Risk ALL	High-Risk ALL	Very High-Risk ALL	Mature B-ALL
Definition	*B-lineage* WBC <30,000/mL Time to CR <4 wk No Pro-B/ t(4;11) *T-lineage* Thy ALL Molecular CR	*B-lineage* WBC >30,000/mL Time to CR >4 wk Pro-B/ t(4;11) *T-lineage* Early T, mature T	Ph/BCR-ABL positive	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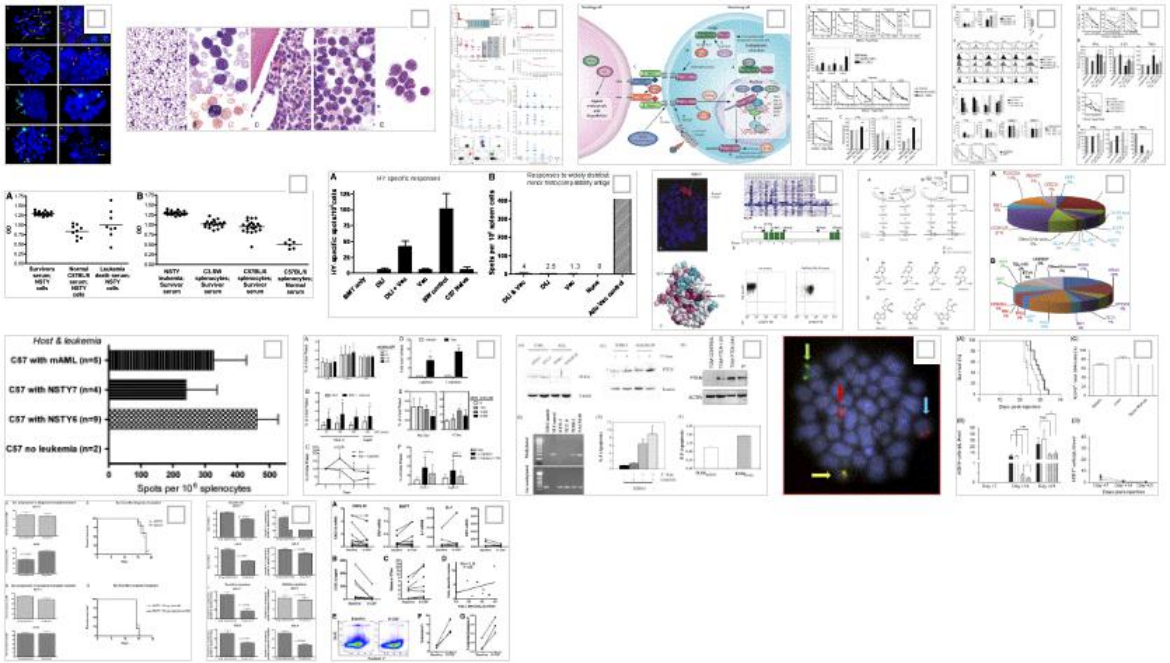

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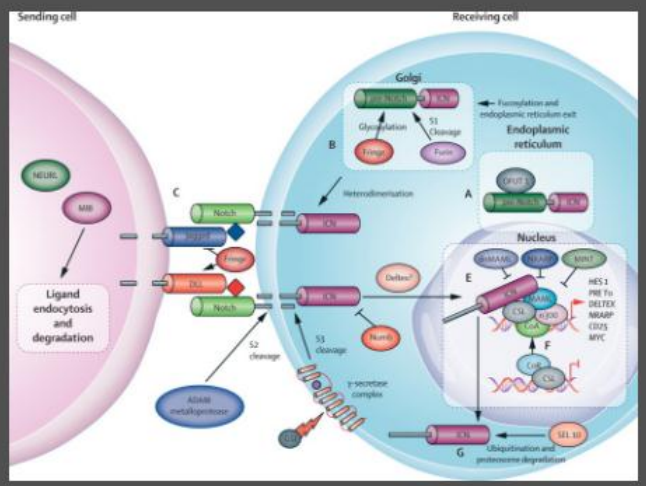

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Acute lymphoblastic leukaemia

Lancet, The.

Pui, Ching-Hon, Prof; Robison, Leslie L, Prof; Look, A Thomas, Prof.. Published March 22, 2008. Volume 371, Issue 9617. Pages 1030-1043. © 2008.

Figure 2 NOTCH signalling in normal thymocytes The NOTCH signalling pathway is complex and involves the coordinated activities of many different molecules. Briefly, NOTCH is synthesised in the endoplasmic reticulum (A) as one protein consisting of an extracellular domain (pre-Notch) and an intracellular domain (ICN), which are transported in tandem to the Golgi (B), where several post-translational modifications take place, including a proteolytic cleavage (S1) that separates the two domains from each other. The resultant heterodimer is then transported to the cell membrane, where NOTCH interacts with ligands (C) and is cleaved twice (D) by the ADAM protease (S2) and a γ-secretase complex (S3), enabling the liberated ICN domain to translocate to the nucleus (E). Nuclear ICN forms a binding/activator complex with a group of cooperating proteins (F), resulting in transcriptional activation of several functionally important genes, including MYC and pre-Tα. Hyperphosphorylation of ICN via interaction of its PEST domain (polypeptide enriched in proline, glutamate, serine, and threonine) with CDK8, MAML, and p300 facilitates ubiquitylation (G) by SEL1 family members, targeting ICN to the proteosome. ADAM=a disintegrin and metalloproteinase domain. DLL=Delta ligand. hes1=hairy/enhancer of split. GSI=γ-secretase inhibition. MAML=mastermind-like proteins. MIB=mindbomb. NEURL=neuralised-like. Nrarp=gene encoding NOTCH-regulated ankyrin-repeat protein. OFUT1=O-fucosyltransferase1. Pre-Tα=pre-TCRα. Deltex=positive regulator of Notch signalling pathway. CS1=DNA binding

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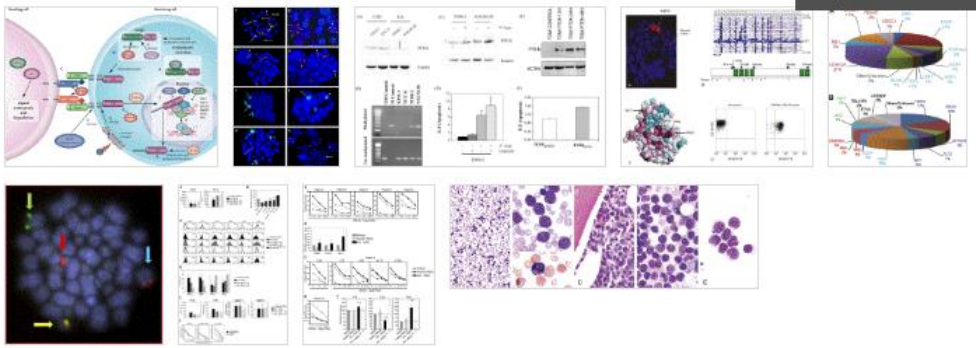

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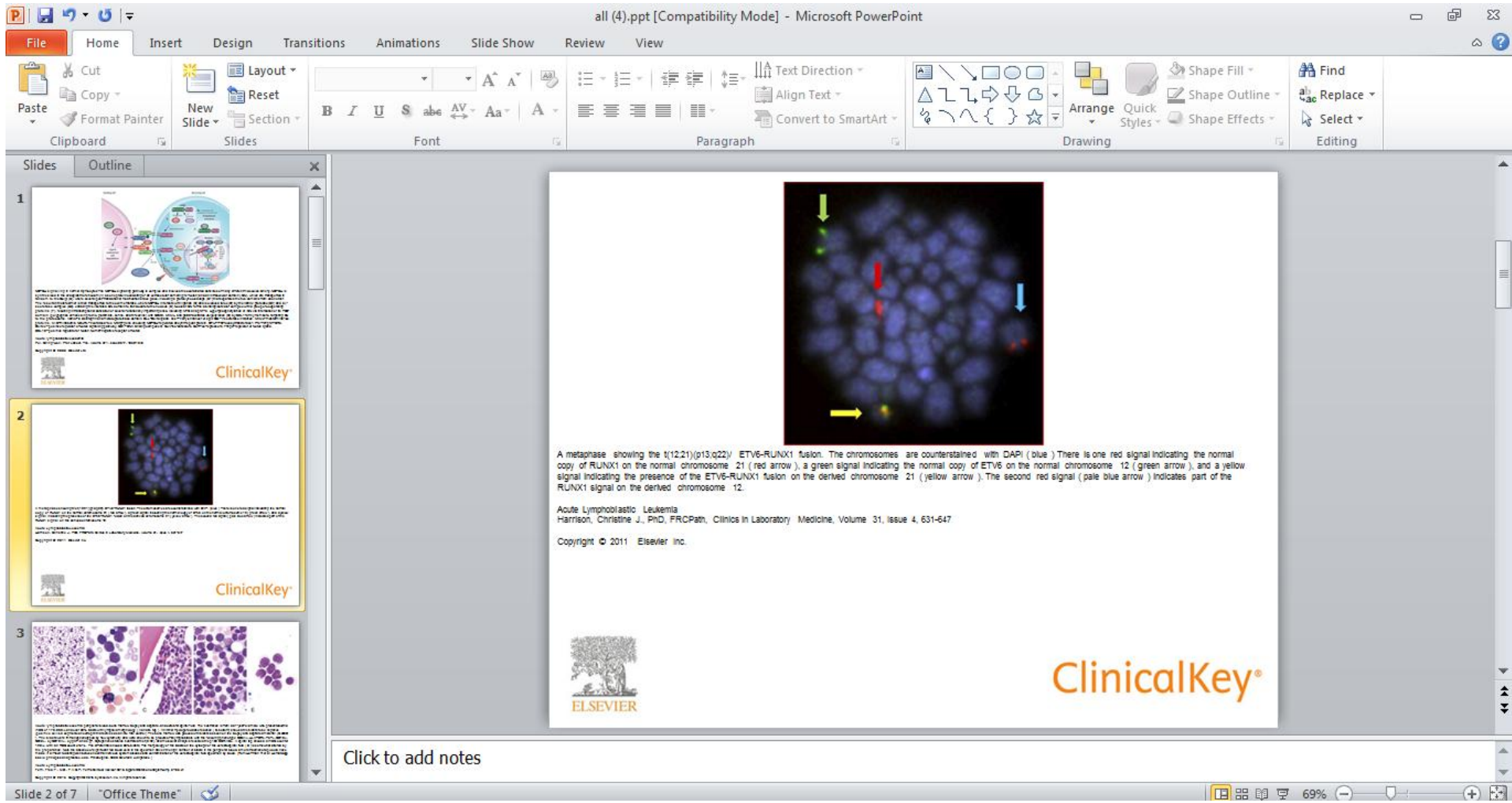
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A metaphase showing the t(12;21)(p13;q22)/ ETV6-RUNX1 fusion. The chromosomes are counterstained with DAPI (blue) There is one red signal indicating the normal copy of RUNX1 on the normal chromosome 21 (red arrow), a green signal indicating the normal copy of ETV6 on the normal chromosome 12 (green arrow), and a yellow signal indicating the presence of the ETV6-RUNX1 fusion on the derived chromosome 21 (yellow arrow). The second red signal (pale blue arrow) indicates part of the RUNX1 signal on the derived chromosome 12.
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Harrison, Christine J., PhD, FRCPath, Clinics in Laboratory Medicine, Volume 31, Issue 4, 631-647
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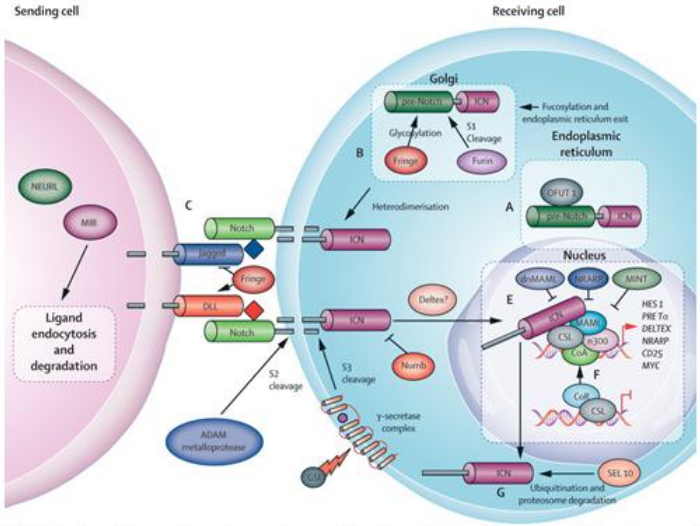

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Pui, Ching-Hon, Prof, Lancet, The, Volume 371, Issue 9617, 1030-1043