

Language and Information

Jyunichi Tsujii

- School of Science Information Science and Technology
- Graduate School of Interdisciplinary Information Studies

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- **Background**

Japanese
Chinese
Korean
Thai
Malay
Hindi

English
German
French
Spanish
Portuguese
Russian
Greek

Arabic

Language

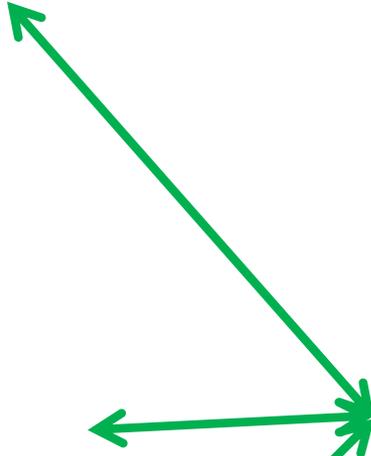
Japanese
Chinese
Korean
Thai
Malay
Hindi

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German
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Russian
Greek

Arabic

Language

meaning
Information
Knowledge



Japanese
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Hindi

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Portuguese
Russian
Greek

Arabic

Language

mean
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Inform
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Knowl
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Physics
Biology
Chemistry

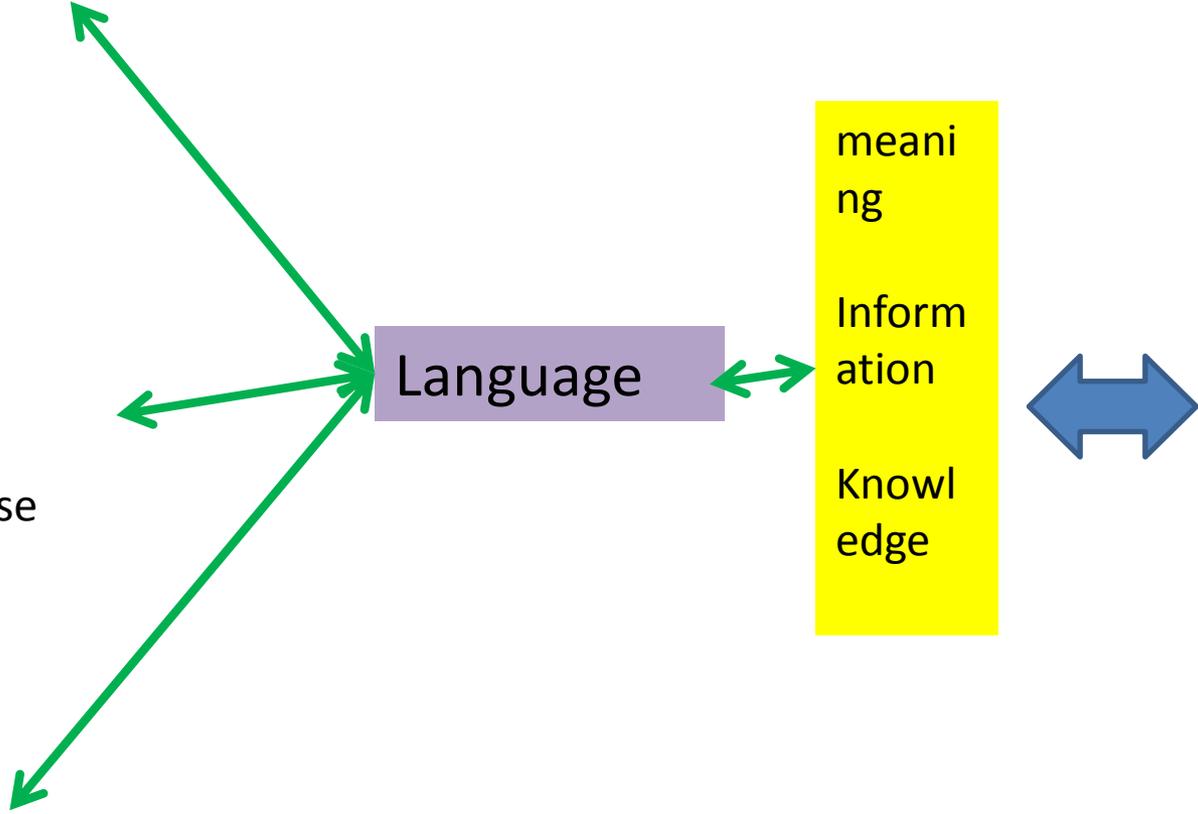
Mathematics

Economics
Politics

Sports
Movie
Game

Daily Life
Cooking
Music

The Whole of Creation



Japanese
Chinese
Korean
Thai
Malay
Hindi

English
German
French
Spanish
Portuguese
Russian
Greek

Arabic

Language



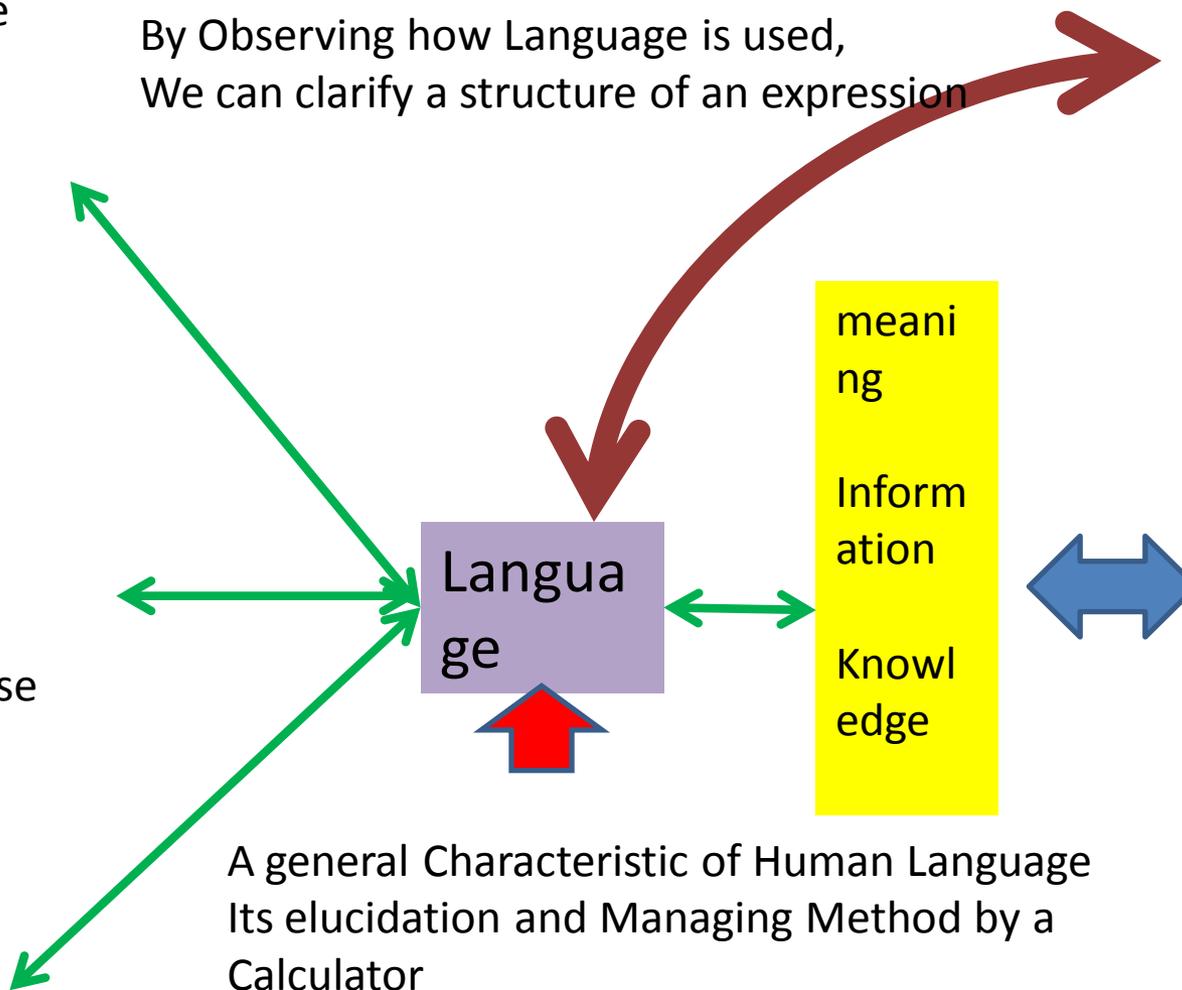
A general Characteristic of Human Language
Its elucidation and Managing Method by a
Calculator

Japanese
Chinese
Korean
Thai
Malay
Hindi

English
German
French
Spanish
Portuguese
Russian
Greek

Arabic

By Observing how Language is used,
We can clarify a structure of an expression



Physics
Biology
Chemistry

Mathematics

Economics
Politics

Sports
Movie
Game

Daily Life
Cooking
Music

The Whole of Creation

Language and (Meaning ▪ Context ▪ Memory ▪ Structure ▪ Interpretation)

- An Infant, A picture book
- Bilingual
- Justice = to be fair
- Freedom Fighters , Terrorists
- Playing, A Structure of *Amae*

Natural Language as Media

- Awareness, Interpretation, An Expression way of Knowledge
 - Picture : An Instance at a point of Spatiotemporal
 - Symbolic Picture : monotonous, a simplex message
 - Natural Language :
 - Not an Instance, But a generalized recognition、 Interpretation
 - A Structural Combination of Information, Logical Development
- A Means of Communication
 - Command Language : Each system has its own characteristics, Optional Design
 - Natural Language : Generality, Universal Use (Impediment Removal)

The Role of Natural Language

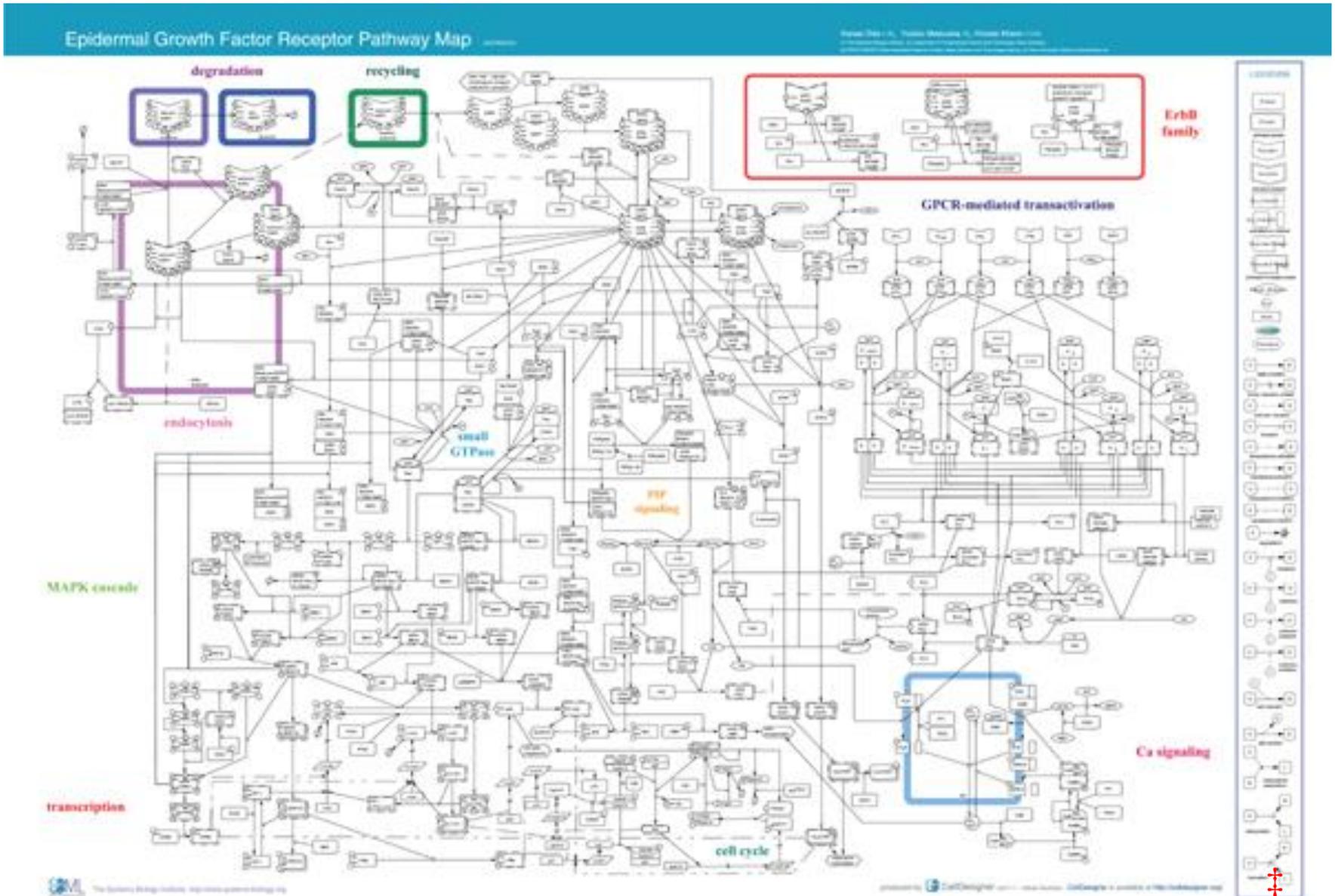
- Integration of Information, Global Understanding by **Structured**
- Communication of an Intellectual agent who has a different awareness
- An Universal will indication way for Various IT apparatus
- Knowledge Sharing, An Integration of a data base
- One of Multimedia, An Integration of multimedia

Ex: An Integration of Information

An Integration of Information in Life science

- DNA and Protein, A basis of Biological Phenomena
- A Structure and a Function of Protein
- Understanding of Biological Phenomena :
Reciprocal Action between Protein,
Understanding of Networks

Toll-Like Receptor (TLR) pathway



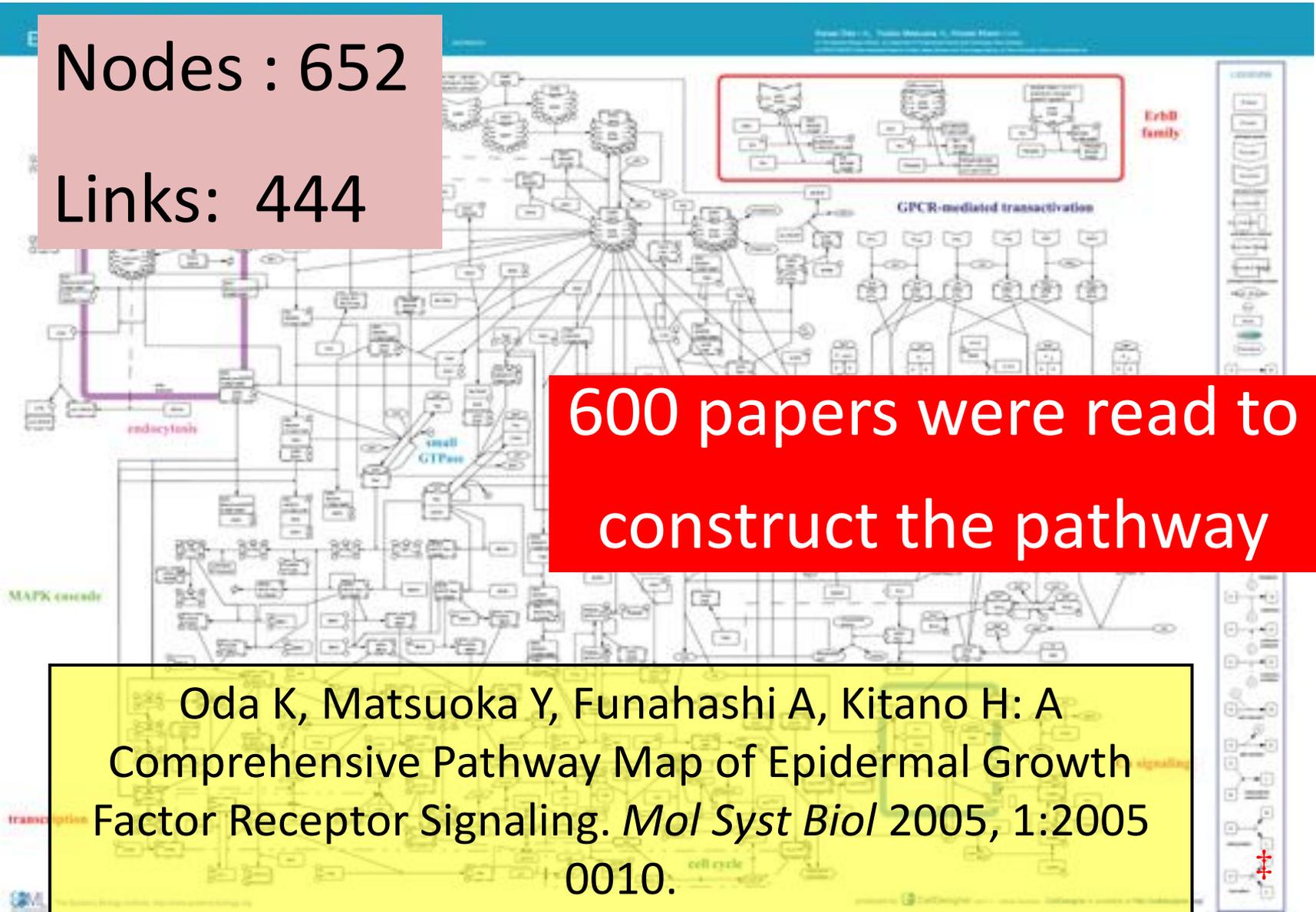
Toll-Like Receptor (TLR) pathway

Nodes : 652

Links: 444

600 papers were read to
construct the pathway

Oda K, Matsuoka Y, Funahashi A, Kitano H: A Comprehensive Pathway Map of Epidermal Growth Factor Receptor Signaling. *Mol Syst Biol* 2005, 1:20050010.



The Integration of Information in Life Sciences

- DNA and Protein, A basis of Biological Phenomena
- Structure and a Function of Protein
- Understanding of Biological Phenomena : Reciprocal Action between Protein, Understanding of Networks
- Hundreds of thousands of types of Protein
- A Process of Life Evolution, Sharing Protein between Biological Species
- Name of Protein: Freedom Fighters and Terrorists
- A Great Volume of Article Information: MEDLINE, 18 million , Half a million a year, 1300 a day

Demo



Model: NFKappaB_for_PathTextR2
Node: NF-kappaB IkappaB alpha|bind|NFKappaB (re2)

- Manual Annotations (70)**
- Medie Search Results (64)
- Kleio Search Results (200)
- About PathText

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[Ubiquitin-dependent degradation of IkappaBalpha is mediated by a ubiquitin ligase Skp1/Cul 1/F-box protein FWD1.](#)
Journal: **Proceedings of the National Academy of Sciences of the United States of America** 1999 Mar 30;96(12):7385-90. 1 Annotation(s)

[An N-terminal nuclear export signal is required for the nucleocytoplasmic shuttling of IkappaBalpha.](#)
Journal: **The EMBO journal** 1999 Dec 15;18(25):2366-73. 1 Annotation(s)

[Cotranslational dimerization of the Rel homology domain of NF-kappaB1 generates p50-p105 heterodimers and is required for effective p50 production.](#)
Journal: **The EMBO journal** 2000 Sep 13;19(18):4712-22. 1 Annotation(s)

[NF-kappaB-inducing kinase regulates the processing of NF-kappaB2 p100.](#)
Journal: **Molecular cell** 2001 Feb 7;7(2):240-9. 1 Annotation(s)

[Missing pieces in the NF-kappaB puzzle.](#)
Journal: **Cell** 2002 Apr 10;109(Suppl):S81-96. 2 Annotation(s)

[The precursor of NF-kappa B p50 has I kappa B-like functions.](#)
Journal: **Cell** 1992 Oct 16;71(2):224-33. 2 Annotation(s)

[I kappa B/MAD-3 masks the nuclear localization signal of NF-kappa B p65 and requires the transactivation domain to inhibit NF-kappa B p65 DNA binding.](#)
Journal: **Molecular biology of the cell** 1992 Dec 16;3(12):1213-9. 2 Annotation(s)

[Click here for details](#)

Manual Annotations generated using the PathText Manual Annotation Tool. See pathtext.org for details.



Model: NFKappaB_for_PathTextR2
Node: NF-kappaB I kappaB alpha|bind|NFKappaB (re2)

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Journal: **Proceedings of the National Academy of Sciences of the United States of America** 1999 Mar 30;96(12):7385-90. 1 Annotation(s)

[An N-terminal nuclear export signal is required for the nucleocytoplasmic shuttling of I kappaBalpha.](#)
Journal: **The EMBO journal** 1999 Dec 11;18(23):682-93. 1 Annotation(s)

[Cotranslational dimerization of the Rel homology domain of NF-kappaB1 generates p50-p105 heterodimers and is required for effective p50 production.](#)
Journal: **The EMBO journal** 2000 Sep 11;19(18):5412-22. 1 Annotation(s)

[NF-kappaB-inducing kinase regulates the processing of NF-kappaB2 p100.](#)
Journal: **Molecular cell** 2001 Feb 7;7(2):241-9. 1 Annotation(s)

[Missing pieces in the NF-kappaB puzzle.](#)
Journal: **Cell** 2002 Apr 10;109(Suppl):S81-96. 2 Annotation(s)

[The precursor of NF-kappa B p50 has I kappa B-like functions.](#)
Journal: **Cell** 1992 Oct 16;71(2):223-53. 2 Annotation(s)

[I kappa B/MAD-3 masks the nuclear localization signal of NF-kappa B p65 and requires the transactivation domain to inhibit NF-kappa B p65 DNA binding.](#)
Journal: **Molecular biology of the cell** 1992 Dec 16;3(12):1339-52. 2 Annotation(s)

[Click here for details](#)

Manual Annotations generated using the PathText Manual Annotation Tool. See pathtext.org for details.



Model: NFKappaB_for_PathTextR2
Node: NF-kappaB I kappaB alpha|bind|NFKappaB (re2)

- Manual Annotations (70)
- Medie Search Results (64)
- Kleio Search Results (200)
- About PathText

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Ubiquitin-dependent degradation of I kappaB alpha is mediated by a ubiquitin ligase Skp1/Cul 1/F-box protein FWD1.

Journal: **Proceedings of the National Academy of Sciences of the United States of America** 1999 Mar 30;96(7):7385-90. 1 Annotation(s)

Authors: Hatakeyama S, Kitagawa M, Nakayama K, Shirane M, Matsumoto M, Hattori K, Higashi H, Nakano H, Okumura K, Onoé K, Good RA, Nakayama K.

PubMed: 10097128 [View PubMed Page](#)

Publication Abstract

Activation of the transcription factor nuclear factor kappa B (NF-kappaB) is controlled by proteolysis of its inhibitory subunit (I kappaB) via the ubiquitin-proteasome pathway. Signal-induced phosphorylation of I kappaB alpha by a large multisubunit complex containing I kappaB kinases is a prerequisite for ubiquitination. Here, we show that FWD1 (a mouse homologue of Slimb/betaTrCP), a member of the F-box/WD40-repeat proteins, is associated specifically with I kappaB alpha only when I kappaB alpha is phosphorylated. The introduction of FWD1 into cells significantly promotes ubiquitination and degradation of I kappaB alpha in concert with I kappaB kinases, resulting in nuclear translocation of NF-kappaB. In addition, FWD1 strikingly evoked the ubiquitination of I kappaB alpha in the in vitro system. In contrast, a dominant-negative form of FWD1 inhibits the ubiquitination, leading to stabilization of I kappaB alpha. These results suggest that the substrate-specific degradation of I kappaB alpha is mediated by a Skp1/Cull 1/F-box protein (SCF) FWD1 ubiquitin-ligase complex and that FWD1 serves as an intracellular receptor for phosphorylated I kappaB alpha. Skp1/Cullin/F-box protein FWD1 might play a critical role in transcriptional regulation of NF-kappaB through control of I kappaB protein stability.

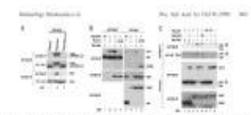


Fig. 1. Ubiquitination of I kappaB alpha is mediated by FWD1. (A) Western blot analysis of I kappaB alpha in cells treated with TNF-alpha. (B) Ubiquitination of I kappaB alpha in cells expressing FWD1. (C) Ubiquitination of I kappaB alpha in cells expressing a dominant-negative FWD1. (D) Ubiquitination of I kappaB alpha in cells expressing FWD1 and a dominant-negative FWD1. (E) Ubiquitination of I kappaB alpha in cells expressing FWD1 and a dominant-negative FWD1. (F) Ubiquitination of I kappaB alpha in cells expressing FWD1 and a dominant-negative FWD1. (G) Ubiquitination of I kappaB alpha in cells expressing FWD1 and a dominant-negative FWD1. (H) Ubiquitination of I kappaB alpha in cells expressing FWD1 and a dominant-negative FWD1. (I) Ubiquitination of I kappaB alpha in cells expressing FWD1 and a dominant-negative FWD1. (J) Ubiquitination of I kappaB alpha in cells expressing FWD1 and a dominant-negative FWD1. (K) Ubiquitination of I kappaB alpha in cells expressing FWD1 and a dominant-negative FWD1. (L) Ubiquitination of I kappaB alpha in cells expressing FWD1 and a dominant-negative FWD1. (M) Ubiquitination of I kappaB alpha in cells expressing FWD1 and a dominant-negative FWD1. (N) Ubiquitination of I kappaB alpha in cells expressing FWD1 and a dominant-negative FWD1. (O) Ubiquitination of I kappaB alpha in cells expressing FWD1 and a dominant-negative FWD1. (P) Ubiquitination of I kappaB alpha in cells expressing FWD1 and a dominant-negative FWD1. (Q) Ubiquitination of I kappaB alpha in cells expressing FWD1 and a dominant-negative FWD1. (R) Ubiquitination of I kappaB alpha in cells expressing FWD1 and a dominant-negative FWD1. (S) Ubiquitination of I kappaB alpha in cells expressing FWD1 and a dominant-negative FWD1. (T) Ubiquitination of I kappaB alpha in cells expressing FWD1 and a dominant-negative FWD1. (U) Ubiquitination of I kappaB alpha in cells expressing FWD1 and a dominant-negative FWD1. (V) Ubiquitination of I kappaB alpha in cells expressing FWD1 and a dominant-negative FWD1. (W) Ubiquitination of I kappaB alpha in cells expressing FWD1 and a dominant-negative FWD1. (X) Ubiquitination of I kappaB alpha in cells expressing FWD1 and a dominant-negative FWD1. (Y) Ubiquitination of I kappaB alpha in cells expressing FWD1 and a dominant-negative FWD1. (Z) Ubiquitination of I kappaB alpha in cells expressing FWD1 and a dominant-negative FWD1.

Annotation by Kanae Oda
Hence, FWD1 binds to I kappaB alpha when it is complexed with NF-kB.

Page: 3



Model: NFKappaB_for_PathTextR2
Node: NF-kappaB I kappa B alpha|bind|NFKappaB (re2)

- Manual Annotations (70)**
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[An N-terminal nuclear export signal is required for the nucleocytoplasmic shuttling of I kappa Balpha.](#)
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[Cotranslational dimerization of the Rel homology domain of NF-kappaB1 generates p50-p105 heterodimers and is required for effective p50 production.](#)
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[NF-kappaB-inducing kinase regulates the processing of NF-kappaB2 p100.](#)
Journal: **Molecular cell** 2001 Feb 7;6(2):240-9. 1 Annotation(s)

[Missing pieces in the NF-kappaB puzzle.](#)
Journal: **Cell** 2002 Apr 10;109(Suppl 1):S81-96. 2 Annotation(s)

[The precursor of NF-kappa B p50 has I kappa B-like functions.](#)
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[I kappa B/MAD-3 masks the nuclear localization signal of NF-kappa B p65 and requires the transactivation domain to inhibit NF-kappa B p65 DNA binding.](#)
Journal: **Molecular biology of the cell** 1992 Dec 16;3(12):1233-42. 2 Annotation(s)

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Manual Annotations generated using the PathText Manual Annotation Tool. See pathtext.org for details.

Authors: Ganchi PA, Sun SC, Greene WC, Ballard DW.

PubMed: 1493333 View PubMed Page

Publication Abstract

The active nuclear form of the NF-kappa B transcription factor complex is composed of two DNA binding subunits, NF-kappa B p65 and NF-kappa B p50, both of which share extensive N-terminal sequence homology with the v-rel oncogene product. The NF-kappa B p65 subunit provides the transactivation activity in this complex and serves as an intracellular receptor for a cytoplasmic inhibitor of NF-kappa B, termed I kappa B. In contrast, NF-kappa B p50 alone fails to stimulate kappa B-directed transcription, and based on prior in vitro studies, is not directly regulated by I kappa B. To investigate the molecular basis for the critical regulatory interaction between NF-kappa B and I kappa B/MAD-3, a series of human NF-kappa B p65 mutants was identified that functionally segregated DNA binding, I kappa B-mediated inhibition, and I kappa B-induced nuclear exclusion of this transcription factor. Results from in vivo expression studies performed with these NF-kappa B p65 mutants revealed the following: 1) I kappa B/MAD-3 completely inhibits NF-kappa B p65-dependent transcriptional activation mediated through the human immunodeficiency virus type 1 kappa B enhancer in human T lymphocytes, 2) the binding of I kappa B/MAD-3 to NF-kappa B p65 is sufficient to retarget NF-kappa B p65 from the nucleus to the cytoplasm, 3) selective deletion of the functional nuclear localization signal present in the Rel homology domain of NF-kappa B p65 disrupts its ability to engage I kappa B/MAD-3, and 4) the unique C-terminus of NF-kappa B p65 attenuates its own nuclear localization and contains sequences that are required for I kappa B-mediated inhibition of NF-kappa B p65 DNA binding activity. Together, these findings suggest that the nuclear localization signal and transactivation domain of NF-kappa B p65 constitute a bipartite system that is critically involved in the inhibitory function of I kappa B/MAD-3. Unexpectedly, our in vivo studies also demonstrate that I kappa B/MAD-3 binds directly to NF-kappa B p50. This interaction is functional as it leads to retargeting of NF-kappa B p50 from the nucleus to the cytoplasm. However, no loss of DNA binding activity is observed, presumably reflecting the unique C-terminal domain that is distinct from that present in NF-kappa B p65.

Annotation by Kanae Oda

The NF-KB p65 subunit provides the transactivation activity in this complex and serves as an intracellular receptor for a cytoplasmic inhibitor of NF-KB, termed IKB.

Page: 1

I κ B/MAD-3 Masks the Nuclear Localization Signal of NF- κ B p65 and Requires the Transactivation Domain to Inhibit NF- κ B p65 DNA Binding

Parham A. Ganchi, Shan-Cong Sun, Warner C. Greene, and David W. Ballard*

Division of Hematology and Oncology, Department of Medicine, San Francisco General Hospital, University of California, San Francisco, California 94143

Received August 19, 1992; accepted October 1, 1992.

The active nuclear form of the NF- κ B transcription factor complex is composed of two DNA binding subunits, NF- κ B p65 and NF- κ B p50, both of which share extensive N-terminal sequence homology with the v-rel oncogene product. The NF- κ B p65 subunit provides the transactivation activity in this complex and serves as an intracellular receptor for a cytoplasmic inhibitor of NF- κ B, termed I κ B. In contrast, NF- κ B p50 alone fails to stimulate kappa B-directed transcription, and based on prior in vitro studies, is not directly regulated by I κ B. To investigate the molecular basis for the critical regulatory interaction between NF- κ B and I κ B/MAD-3, a series of human NF- κ B p65 mutants was identified that functionally segregated DNA binding, I κ B-mediated inhibition, and I κ B-induced nuclear exclusion of this transcription factor. Results from in vivo expression studies performed with these NF- κ B p65 mutants revealed the following: 1) I κ B/MAD-3 completely inhibits NF- κ B p65-dependent transcriptional activation mediated through the human immunodeficiency virus type 1 kappa B enhancer in human T lymphocytes, 2) the binding of I κ B/MAD-3 to NF- κ B p65 is sufficient to retarget NF- κ B p65 from the nucleus to the cytoplasm, 3) selective deletion of the functional nuclear localization signal present in the Rel homology domain of NF- κ B p65 disrupts its ability to engage I κ B/MAD-3, and 4) the unique C-terminus of NF- κ B p65 attenuates its own nuclear localization and contains sequences that are required for I κ B-mediated inhibition of NF- κ B p65 DNA binding activity. Together, these findings suggest that the nuclear localization signal and transactivation domain of NF- κ B p65 constitute a bipartite system that is critically involved in the inhibitory function of I κ B/MAD-3. Unexpectedly, our in vivo studies also demonstrate that I κ B/MAD-3 binds directly to NF- κ B p50. This interaction is functional as it leads to retargeting of NF- κ B p50 from the nucleus to the cytoplasm. However, no loss of DNA binding activity is observed, presumably reflecting the unique C-terminal domain that is distinct from that present in NF- κ B p65.

INTRODUCTION

Members of specific cellular genes, including those in the NF- κ B gene family, are induced in a variety of cellular responses and are essential for the development and function of the immune system. The NF- κ B transcription factor complex is composed of two DNA binding subunits, NF- κ B p65 and NF- κ B p50, both of which share extensive N-terminal sequence homology with the v-rel oncogene product. The NF- κ B p65 subunit provides the transactivation activity in this complex and serves as an intracellular receptor for a cytoplasmic inhibitor of NF- κ B, termed I κ B. In contrast, NF- κ B p50 alone fails to stimulate kappa B-directed transcription, and based on prior in vitro studies, is not directly regulated by I κ B. To investigate the molecular basis for the critical regulatory interaction between NF- κ B and I κ B/MAD-3, a series of human NF- κ B p65 mutants was identified that functionally segregated DNA binding, I κ B-mediated inhibition, and I κ B-induced nuclear exclusion of this transcription factor. Results from in vivo expression studies performed with these NF- κ B p65 mutants revealed the following: 1) I κ B/MAD-3 completely inhibits NF- κ B p65-dependent transcriptional activation mediated through the human immunodeficiency virus type 1 kappa B enhancer in human T lymphocytes, 2) the binding of I κ B/MAD-3 to NF- κ B p65 is sufficient to retarget NF- κ B p65 from the nucleus to the cytoplasm, 3) selective deletion of the functional nuclear localization signal present in the Rel homology domain of NF- κ B p65 disrupts its ability to engage I κ B/MAD-3, and 4) the unique C-terminus of NF- κ B p65 attenuates its own nuclear localization and contains sequences that are required for I κ B-mediated inhibition of NF- κ B p65 DNA binding activity. Together, these findings suggest that the nuclear localization signal and transactivation domain of NF- κ B p65 constitute a bipartite system that is critically involved in the inhibitory function of I κ B/MAD-3. Unexpectedly, our in vivo studies also demonstrate that I κ B/MAD-3 binds directly to NF- κ B p50. This interaction is functional as it leads to retargeting of NF- κ B p50 from the nucleus to the cytoplasm. However, no loss of DNA binding activity is observed, presumably reflecting the unique C-terminal domain that is distinct from that present in NF- κ B p65.

Annotation by Kanae Oda

These studies clearly demonstrate that NF-KB p65 contains two domains that function in concert to mediate assembly with IKB/MAD-3, resulting in the inhibition of NF-KB p65 DNA binding activity.

Page: 2

INTRODUCTION

Members of specific cellular genes, including those in the NF- κ B gene family, are induced in a variety of cellular responses and are essential for the development and function of the immune system. The NF- κ B transcription factor complex is composed of two DNA binding subunits, NF- κ B p65 and NF- κ B p50, both of which share extensive N-terminal sequence homology with the v-rel oncogene product. The NF- κ B p65 subunit provides the transactivation activity in this complex and serves as an intracellular receptor for a cytoplasmic inhibitor of NF- κ B, termed I κ B. In contrast, NF- κ B p50 alone fails to stimulate kappa B-directed transcription, and based on prior in vitro studies, is not directly regulated by I κ B. To investigate the molecular basis for the critical regulatory interaction between NF- κ B and I κ B/MAD-3, a series of human NF- κ B p65 mutants was identified that functionally segregated DNA binding, I κ B-mediated inhibition, and I κ B-induced nuclear exclusion of this transcription factor. Results from in vivo expression studies performed with these NF- κ B p65 mutants revealed the following: 1) I κ B/MAD-3 completely inhibits NF- κ B p65-dependent transcriptional activation mediated through the human immunodeficiency virus type 1 kappa B enhancer in human T lymphocytes, 2) the binding of I κ B/MAD-3 to NF- κ B p65 is sufficient to retarget NF- κ B p65 from the nucleus to the cytoplasm, 3) selective deletion of the functional nuclear localization signal present in the Rel homology domain of NF- κ B p65 disrupts its ability to engage I κ B/MAD-3, and 4) the unique C-terminus of NF- κ B p65 attenuates its own nuclear localization and contains sequences that are required for I κ B-mediated inhibition of NF- κ B p65 DNA binding activity. Together, these findings suggest that the nuclear localization signal and transactivation domain of NF- κ B p65 constitute a bipartite system that is critically involved in the inhibitory function of I κ B/MAD-3. Unexpectedly, our in vivo studies also demonstrate that I κ B/MAD-3 binds directly to NF- κ B p50. This interaction is functional as it leads to retargeting of NF- κ B p50 from the nucleus to the cytoplasm. However, no loss of DNA binding activity is observed, presumably reflecting the unique C-terminal domain that is distinct from that present in NF- κ B p65.

RESULTS AND DISCUSSION

Members of specific cellular genes, including those in the NF- κ B gene family, are induced in a variety of cellular responses and are essential for the development and function of the immune system. The NF- κ B transcription factor complex is composed of two DNA binding subunits, NF- κ B p65 and NF- κ B p50, both of which share extensive N-terminal sequence homology with the v-rel oncogene product. The NF- κ B p65 subunit provides the transactivation activity in this complex and serves as an intracellular receptor for a cytoplasmic inhibitor of NF- κ B, termed I κ B. In contrast, NF- κ B p50 alone fails to stimulate kappa B-directed transcription, and based on prior in vitro studies, is not directly regulated by I κ B. To investigate the molecular basis for the critical regulatory interaction between NF- κ B and I κ B/MAD-3, a series of human NF- κ B p65 mutants was identified that functionally segregated DNA binding, I κ B-mediated inhibition, and I κ B-induced nuclear exclusion of this transcription factor. Results from in vivo expression studies performed with these NF- κ B p65 mutants revealed the following: 1) I κ B/MAD-3 completely inhibits NF- κ B p65-dependent transcriptional activation mediated through the human immunodeficiency virus type 1 kappa B enhancer in human T lymphocytes, 2) the binding of I κ B/MAD-3 to NF- κ B p65 is sufficient to retarget NF- κ B p65 from the nucleus to the cytoplasm, 3) selective deletion of the functional nuclear localization signal present in the Rel homology domain of NF- κ B p65 disrupts its ability to engage I κ B/MAD-3, and 4) the unique C-terminus of NF- κ B p65 attenuates its own nuclear localization and contains sequences that are required for I κ B-mediated inhibition of NF- κ B p65 DNA binding activity. Together, these findings suggest that the nuclear localization signal and transactivation domain of NF- κ B p65 constitute a bipartite system that is critically involved in the inhibitory function of I κ B/MAD-3. Unexpectedly, our in vivo studies also demonstrate that I κ B/MAD-3 binds directly to NF- κ B p50. This interaction is functional as it leads to retargeting of NF- κ B p50 from the nucleus to the cytoplasm. However, no loss of DNA binding activity is observed, presumably reflecting the unique C-terminal domain that is distinct from that present in NF- κ B p65.



Model: NFKappaB_for_PathTextR2
Node: TAK1|phosphorylate|IKK (re5)

- Manual Annotations (8)
- Medie Search Results (43)**
- Kleio Search Results (475)
- About PathText

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[Inhibition of inhibitor of nuclear factor-kappaB phosphorylation increases the efficacy of paclitaxel in in vitro and in vivo ovarian cancer models.](#)

Journal: **Clinical cancer research : an official journal of the American Association for Cancer Research** 2004 Nov 1510 227645-54 ●●

[Histamine H1 receptor antagonist blocks histamine-induced proinflammatory cytokine production through inhibition of Ca2+-dependent protein kinase C, Raf/MEK/ERK and IKK/I kappa B/NF-kappa B signal cascades.](#)

Journal: **Biochemical pharmacology** 2005 Feb 169 3433-49 ●●

[Inhibition of inhibitor of nuclear factor-kappaB phosphorylation increases the efficacy of paclitaxel in in vitro and in vivo ovarian cancer models.](#)

Journal: **Clinical cancer research : an official journal of the American Association for Cancer Research** 2004 Nov 1510 227645-54 ●●

[Tea polyphenol epigallocatechin 3-gallate impedes the anti-apoptotic effects of low-grade repetitive stress through inhibition of Akt and NFkappaB survival pathways.](#)

Journal: **FEBS letters** 2006 Jan 9580 1278-84 ●●

[Pertussis toxin-sensitive Gi/o proteins are involved in nerve growth factor-induced pro-survival Akt signaling cascade in PC12 cells.](#)

Journal: **Cellular signalling** 2005 Jul 17 7881-90 ●●

[Propofol inhibits lipoteichoic acid-induced iNOS gene expression in macrophages possibly through downregulation of toll-like receptor 2-mediated activation of Raf-MEK1/2-ERK1/2-IKK-NFkappaB.](#)

Journal: **Chemico-biological interactions** 2009 Oct 30181 3430-9 ●●

[Activation of p21-activated kinase 1-nuclear factor kappaB signaling by Kaposi's sarcoma-associated herpes virus G protein-coupled receptor during cellular transformation.](#)

Journal: **Cancer research** 2003 Dec 1563 248837-47 ●●

[Formyl peptide-receptor like-1 requires lipid raft and extracellular signal-regulated protein kinase to activate inhibitor-kappa B kinase in human U87 astrocytoma cells.](#)

Journal: **Journal of neurochemistry** 2007 Nov 103 41553-66 ●●

[Activation of p38MAPK by repetitive low-grade oxidative stress leads to pro-survival effects.](#)

Journal: **Biochimica et biophysica acta** 2007 Mar 1773 3367-74 ●●

[Thalidomide suppresses the interleukin 1beta-induced NFkappaB signaling pathway in colon cancer cells.](#)

Journal: **Annals of the New York Academy of Sciences** 2002 Nov 973 414-8 ●●



Model: NFKappaB_for_PathTextR2
Node: TAK1|phosphorylate|IKK (re5)

Manual Annotations (8) | **Medie Search Results (43)** | Kleio Search Results (475) | About PathText

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[Inhibition of inhibitor of nuclear factor-kappaB phosphorylation increases the efficacy of paclitaxel in in vitro and in vivo ovarian cancer models.](#)

Journal: **Clinical cancer research : an official journal of the American Association for Cancer Research** 2004 Nov 1510 227645-54 ●●



[Histamine H1 receptor antagonist blocks histamine-induced proinflammatory cytokine production through inhibition of Ca2+-dependent protein kinase C, Raf/MEK/ERK and IKK/I kappa B/NF-kappa B signal cascades.](#)

Journal: **Biochemical pharmacology** 2005 Feb 169 3433-49 ●●

[Inhibition of inhibitor of nuclear factor-kappaB phosphorylation increases the efficacy of paclitaxel in in vitro and in vivo ovarian cancer models.](#)

Journal: **Clinical cancer research : an official journal of the American Association for Cancer Research** 2004 Nov 1510 227645-54 ●●

[Tea polyphenol epigallocatechin 3-gallate impedes the anti-apoptotic effects of low-grade repetitive stress through inhibition of Akt and NFkappaB survival pathways.](#)

Journal: **FEBS letters** 2006 Jan 9580 1278-84 ●●

[Pertussis toxin-sensitive Gi/o proteins are involved in nerve growth factor-induced pro-survival Akt signaling cascade in PC12 cells.](#)

Journal: **Cellular signalling** 2005 Jul 17 7881-90 ●●

[Propofol inhibits lipoteichoic acid-induced iNOS gene expression in macrophages possibly through downregulation of toll-like receptor 2-mediated activation of Raf-MEK1/2-ERK1/2-IKK-NFkappaB.](#)

Journal: **Chemico-biological interactions** 2009 Oct 30181 3430-9 ●●

[Activation of p21-activated kinase 1-nuclear factor kappaB signaling by Kaposi's sarcoma-associated herpes virus G protein-coupled receptor during cellular transformation.](#)

Journal: **Cancer research** 2003 Dec 1563 248837-47 ●●

[Formyl peptide-receptor like-1 requires lipid raft and extracellular signal-regulated protein kinase to activate inhibitor-kappa B kinase in human U87 astrocytoma cells.](#)

Journal: **Journal of neurochemistry** 2007 Nov 103 41553-66 ●●

[Activation of p38MAPK by repetitive low-grade oxidative stress leads to pro-survival effects.](#)

Journal: **Biochimica et biophysica acta** 2007 Mar 1773 3367-74 ●●

[Thalidomide suppresses the interleukin 1beta-induced NFkappaB signaling pathway in colon cancer cells.](#)

Journal: **Annals of the New York Academy of Sciences** 2002 Nov 973 414-8 ●●



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[Inhibition of inhibitor of nuclear factor-kappaB phosphorylation increases the efficacy of paclitaxel in in vitro and in vivo ovarian cancer models.](#)
Journal: **Clinical cancer research : an official journal of the American Association for Cancer Research** 2004 Nov 15;10(22):2276-84

[Click here for details](#)

Authors: S Mabuchi, M Ohmichi, Y Nishio, T Hayasaka, A Kimura, T Ohta, J Kawagoe, K Takahashi, N Yada-Hashimoto, H Seino-Noda, M Sakata, T Motoyama, H Kurachi, JR Testa, K Tasaka, Y Murata
PubMed: 15569997 [View PubMed Page](#) **Query:** |phosphorylate|IKK

Medie Annotation

Treatment of paclitaxel-sensitive Caov-3 cells with paclitaxel transiently activated the phosphorylation of Akt, the phosphorylation of IkappaB kinase (IKK), and the phosphorylation of inhibitor of NFKappaB (IkappaBalpha).

Subject Verb Object

Publication Abstract

We investigated whether inhibition of nuclear factor-kappaB (NFkappaB) increases the efficacy of paclitaxel in in vitro and in vivo ovarian cancer models. Treatment of paclitaxel-sensitive Caov-3 cells with paclitaxel transiently activated the phosphorylation of Akt, the phosphorylation of IkappaB kinase (IKK), and the phosphorylation of inhibitor of NFkappaB (IkappaBalpha). Paclitaxel also caused a transient increase in NFkappaB activity, followed by a decrease in NFkappaB activity. We show an association between Akt and IKK and show that the phosphorylation of IKK induced by paclitaxel is blocked by treatment with a phosphatidylinositol 3-kinase inhibitor (wortmannin or LY294002). Furthermore, interference of the Akt signaling cascade inhibits the transient induction of IkappaBalpha phosphorylation and NFkappaB activity by paclitaxel. Inhibition of NFkappaB activity by treatment with an IkappaBalpha phosphorylation inhibitor (BAY 11-7085) attenuated both basal and transient induction of IkappaBalpha phosphorylation by paclitaxel. Treatment with BAY 11-7085 also enhanced the inhibition of NFkappaB activity by paclitaxel for up to 24 hours. In addition, treatment with BAY 11-7085 decreased the viability of cells treated with paclitaxel. Moreover, treatment with BAY 11-7085 increased the efficacy of paclitaxel-induced inhibition of intraabdominal dissemination and production of ascites in athymic nude mice inoculated intraperitoneally with Caov-3 cells. These results suggest that paclitaxel transiently induces NFkappaB activity via the phosphatidylinositol 3-kinase/Akt cascade and that combination therapy with paclitaxel and an NFkappaB inhibitor would increase the therapeutic efficacy of paclitaxel.

Rank Abstract: Current Abstract Ranking: Good
Rank Sentence: Current Sentence Ranking: Good

[Histamine H1 receptor antagonist blocks histamine-induced proinflammatory cytokine production through inhibition of Ca2+-dependent protein kinase C, Raf/MEK/ERK and IKK/I kappa B/NF-kappa B signal cascades.](#)
Journal: **Biochemical pharmacology** 2005 Feb 16;76(2):343-51

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Journal: **FEBS letters** 2006 Jan 9;580(1):127-34

[Pertussis toxin-sensitive Gi/o proteins are involved in nerve growth factor-induced pro-survival Akt signaling cascade in PC12 cells.](#)
Journal: **Cellular signalling** 2005 Jul 17;17(7):781-90

[Propofol inhibits lipoteichoic acid-induced iNOS gene expression in macrophages possibly through downregulation of toll-like receptor 2-mediated activation of Raf-MEK1/2-ERK1/2-IKK-NFKappaB.](#)

General : What in IT is changing ?

A Transition of Information Transfer by Language

- Information transfer by manuscript
- Sharing a handwritten text
- Information Sharing among fragmented groups

Transition of Information Transfer by Language

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- Information Sharing among fragmented groups
- Gutenberg's Printing Press
- One Way Information Transfer
- Radio ▪ Television : Mass communication

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- Decentralization of Information Transmission
- An accumulation of Information (archives) ,
Processing, and Indication

Handwritten Text



Printed Text



Text which are electronically **made**,
stored, **processed**, and **circulated**

Handwritten Text



Printed Text



Text which are electronically
**made, stored, processed, and
circulated**

Handwritten Private Text(Diary)



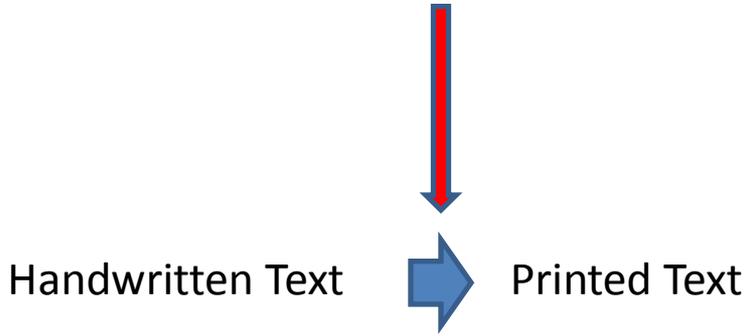
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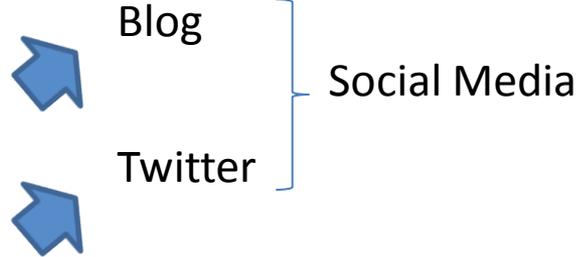


Twitter



Handwritten Private Text(Diary)
mutter, Conversation

Text which are electronically
**made, stored, processed, and
circulated**



ヨハネス・グーテンベルク

出典: フリー百科事典『ウィキペディア (Wikipedia)』

ヨハネス・グーテンベルク(*Johannes Gensfleisch zur Laden zum Gutenberg*、1398年ごろ-1468年2月3日)はドイツ出身の金属加工職人。1445年頃に活版印刷術を発明^{[1][2][3]}。1455年に初めて旧約・新約聖書(ラテン語版)を印刷したことで知られる。これが『グーテンベルク聖書』である。グーテンベルクの開発した印刷システムは急速に普及して、大量の印刷物を生み出し、ルネサンス期における情報伝播の速度を飛躍的に向上させた。

印刷技術は羅針盤、火薬とともに「ルネサンス三大発明」の一つにあげられる。「活版印刷の発明者」としてグーテンベルクは現在でも人気があり、1999年にA&E ネットワークが選定した「紀元1000年代の人」ランキングで一位に選ばれているほどである^[4]。



目次 [非表示]

- 1 生涯
- 2 印刷技術の発明者をめぐって
- 3 グーテンベルクの印刷物
- 4 グーテンベルクの影響
- 5 東アジアの印刷技法との関係
- 6 脚注
- 7 関連項目
- 8 参考書籍

生涯 [編集]

グーテンベルクはドイツのマイツの貴族の家系に生まれた。父はフリーレ・ゲンスフライシュ・ツァー・ラーデン、母はエルゼ・ヴューリヒであった。父フリーレが1427年ごろにグーテンベルク屋敷と呼ばれた住居を手にいれて、そこで暮らしたことから、一族は以降「ツム・グーテンベルク」という名前も名乗るようになる。13世紀以降、グーテンベルク一族は冶金業と商業に従事していた。父母の間には長男フリーレ(後に市参事会員)、長女エルゼが生まれ、次男として生まれたのがヨハネスであった。(グーテンベルクの人生についてはほとんど知られていなかったが、19世紀にアロイス・キューッペル(Aloys Kuppe)博士が初めて本格的な研究を行い、以降、教会や市の記録をもとにしてグーテンベルク一族の研究が進



ウィキペディア
フリー百科事典

案内

- メインページ
- コミュニティポータル
- 最近の出来事
- 新しいページ
- 最近の更新
- おまかせ表示
- 練習用ページ
- アップロード (ウィキメディア・コモンズ)

ヘルプ

- ヘルプ
- 井戸端
- お知らせ
- バグの報告
- 寄付
- ウィキペディアに関するお問い合わせ

検索

表示 検索

『**ドナトゥス文法書**』(1454年) 中世から近代に至るまでもっともよく用いられたラテン語文法書。4世紀のローマ人で**ヒエロニムス**の師であったアエリウス・ドナトゥス (Aelius Donatus) の著作。

『**贖宥状**』(1454年) 三十行。**ニコラウス5世**がトルコへの戦いに功あるものに示したもの。

『**四十二行聖書**』(1455年) いわゆる『**グーテンベルク聖書**』。180部ほど印刷された。

『**マインツ詩篇**』(1457年) コロフォンにはグーテンベルクの名前はないが、計画の途中までかかっていたと考えられている。

グーテンベルクが自らの工房で印刷していたものとして以下のものがあげられる。

『**贖宥状**』(1454年) 三十一行。ニコラウス5世によるもの。

『**トルコ暦**』(1454年) トルコに対する戦いへの協力をよびかける教皇ニコラウス5世の書簡。

『**トルコ教書**』(1456年) トルコに対する戦いへの協力をよびかける教皇**カリストゥス3世**の書簡。

『**医事暦**』(1456年) 一種のカレンダー。

『**キシアヌス**』(1456年) ドイツにおけるカトリック教会の祝祭日を記した暦。

『**カトリコン**』(1460年) 1286年の**ヨハネス・バルプス**の著作。ラテン語文法書とラテン語辞書を組み合わせたもの。

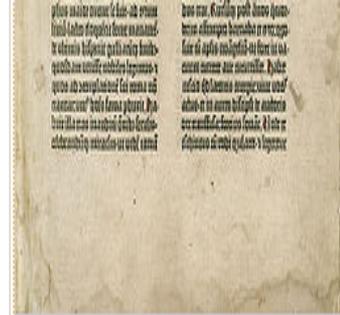
『**信仰大全**』(1460年) **トマス・アクィナス**の著作。

『**贖宥状**』(1461年) **ピウス2世**のもの。十五行および十八行。

『**四十二行聖書**』は**ユネスコ**の推進する歴史的記録遺産のデジタル化計画『**世界の記憶**』プロジェクトに加えられた。

グーテンベルクの影響 [編集]

グーテンベルクが発明した活版印刷技術は急速に普及し、**ニュースや書籍の流通速度を劇的に速めた**。印刷技術はルネサンスの拡大につながり、ひいては**科学革命の土台を作ったとみなされる**。またギリシャやローマの古典書が大量に印刷され出回った。もっとも多かったのはギリシャ語、ラテン語聖書であった。**これらの書物が研究されたことが宗教改革にいたる地下水脈の一つになっていく**。15世紀中に金属活字を用いて印刷された書物は現存数も少ない大変貴重なものとして「**インキュナブラ**」と呼ばれている。



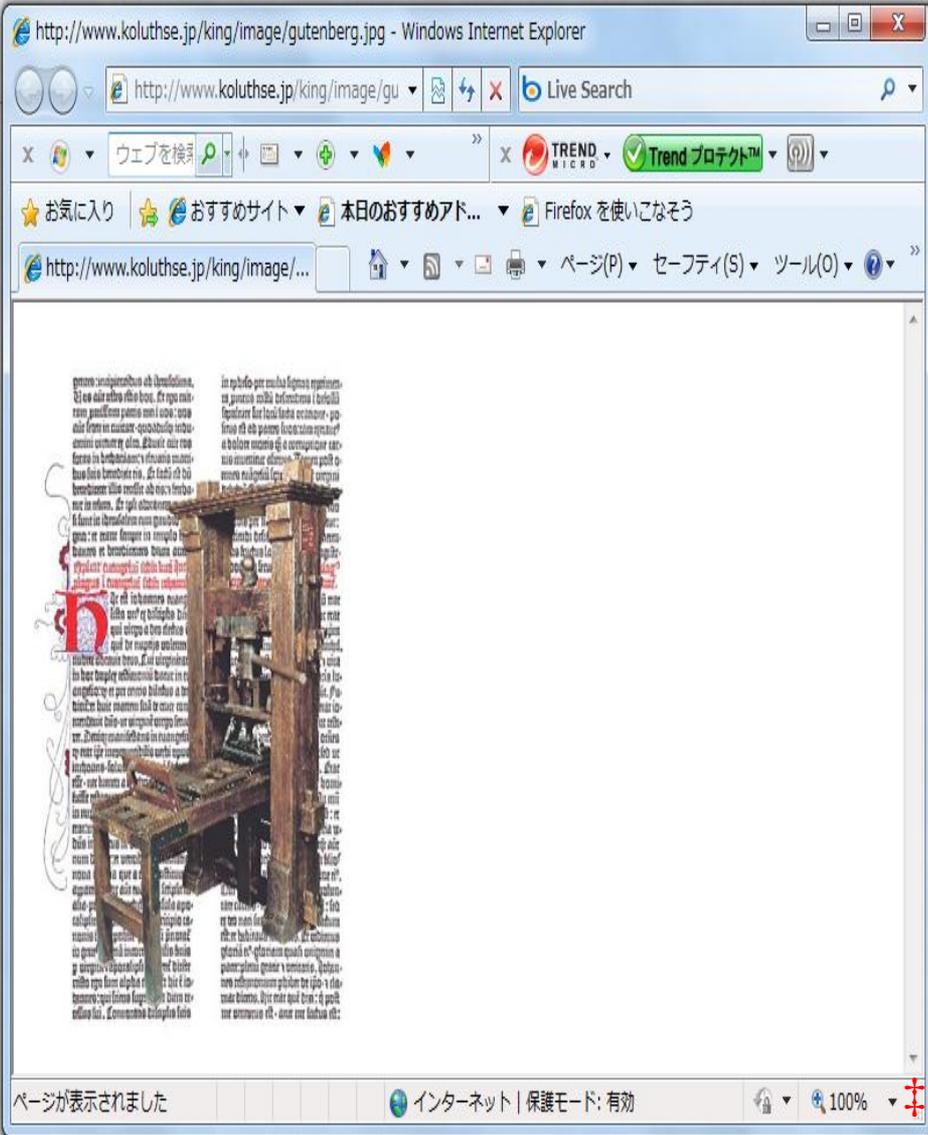
四十二行聖書の冒頭、**ヒエロニムスの書簡**

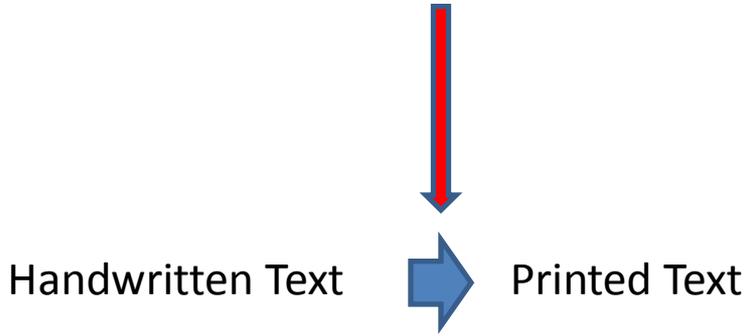
15世紀になると、聖者はグーテンベルグの発明した印刷機によって大量に、しかも正確に印刷され、さらに世界各国の言葉で印刷されるようになりました。グーテンベルグの印刷機で大量に印刷されたドイツ語やフランス語の聖書は、それまでそれぞれの地域でつかわれていた「方言」を標準化し、現代のドイツ語、フランス語の基礎ができました。そしてグーテンベルグの印刷機が、ルネサンスなど文化の基盤を作り、近代への扉を開くことになったのです。



展示品

グーテンベルグ印刷機 復刻機





Handwritten Private Text(Diary)
mutter, Conversation

Text which are electronically
**made, stored, processed, and
circulated**

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WIKIPEDIA
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- article
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Marshall McLuhan

From Wikipedia, the free encyclopedia

"McLuhan" redirects here. For the son of Marshall McLuhan, see Eric McLuhan.

Herbert Marshall McLuhan, CC (July 21, 1911 – December 31, 1980) was a [Canadian educator](#), [philosopher](#), and [scholar](#)—a professor of [English literature](#), a [literary critic](#), a [rhetorician](#), and a [communication theorist](#). McLuhan's work is viewed as one of the cornerstones of the study of [media theory](#).

McLuhan is known for the expressions "[the medium is the message](#)" and "[global village](#)". McLuhan was a fixture in media discourse from the late 1960s to his death and he continues to be an influential and controversial figure. More than ten years after his death he was named the "[patron saint](#)" of [Wired](#) magazine.

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- 1 Life and career
- 2 Major works
 - 2.1 The Mechanical Bride (1951)
 - 2.1.1 Examples of advertisements
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 - 2.2.2 The global village
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 - 2.3.1 "Hot" and "cool" media
 - 2.4 The Medium is the Massage: An Inventory of Effects (1967)
 - 2.5 War and Peace in the Global Village (1968)
 - 2.6 From Cliché to Archetype (1970)
- 3 Key concepts
 - 3.1 Tetrad
 - 3.2 Figure and ground
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- 6 Works cited

Marshall McLuhan



Marshall McLuhan in the early 1970s

Born	Herbert Marshall McLuhan July 21, 1911 Edmonton, Alberta
Died	December 31, 1980 (aged 69) Toronto, Ontario
Main interests	media theory

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 - Community portal
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 - Contact Wikipedia
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- toolbox
- What links here
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 - Special pages
 - Printable version

Though the [World Wide Web](#) was invented thirty years after *The Gutenberg Galaxy* was published, McLuhan may have coined and certainly popularized the usage of the term "[surfing](#)" to refer to rapid, irregular and multidirectional movement through a heterogeneous body of documents or knowledge, e.g., statements like "[Heidegger](#) surf-boards along on the electronic wave as triumphantly as [Descartes](#) rode the mechanical wave." [Paul Levinson](#)'s 1999 book *Digital McLuhan* explores the ways that McLuhan's work can be better understood through the lens of the digital revolution.^[36] Later, [Bill Stewart](#)'s 2007 "Living Internet" website describes how McLuhan's "insights made the concept of a global village, interconnected by an electronic nervous system, part of our popular culture well before it actually happened."^[37]

Cited from Wikipedia : Marshall McLuhan

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- An accumulation of Information (archive) and Processing

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A powerful grammar writing system has been developed. This grammar writing system is called GRADE (GRAMmar DEscriber). GRADE allows a grammar writer to write grammars including analysis, transfer, and generation

GRADE has powerful grammar writing facility. GRADE allows a grammar writer to control the process of ...

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Jun-ichi Tsujii (辻井 潤一)

Publications: [153](#) | Citations: [1335](#) | G-Index: [32](#) | H-Index: [16](#)

Research Interest: [Natural Language & Speech](#), [Bioinformatics and Computational Biology](#), [Artificial Intelligence](#)

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Papers

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Year 2009

Yusuke Miyao, Kenji Sagae, Rune Sætre, Takuya Matsuzaki, **Jun-ichi Tsujii** : [Evaluating contributions of natural language parsers to protein-protein interaction extraction](#), BIOINFORMATICS , 2009 (Citation: 1)

Yoshimasa Tsuruoka, **Jun-ichi Tsujii**, Sophia Ananiadou : [Fast Full Parsing by Linear-Chain Conditional Random Fields](#), EAACL , 2009 (Citations: 1)

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Jun'ichi Tsujii

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Research Interest: [Natural Language & Speech](#), [Bioinformatics and Computational Biology](#), [Machine Learning and Pattern Recognition](#)

University of Manchester, Manchester, United Kingdom



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Akane Yakushiji, Yusuke Miyao, Yuka Tateisi, **Jun'ichi Tsujii** : [Biomedical information ex-traction with predicate-argument structure patterns](#) , 2005 (Citations: 13)

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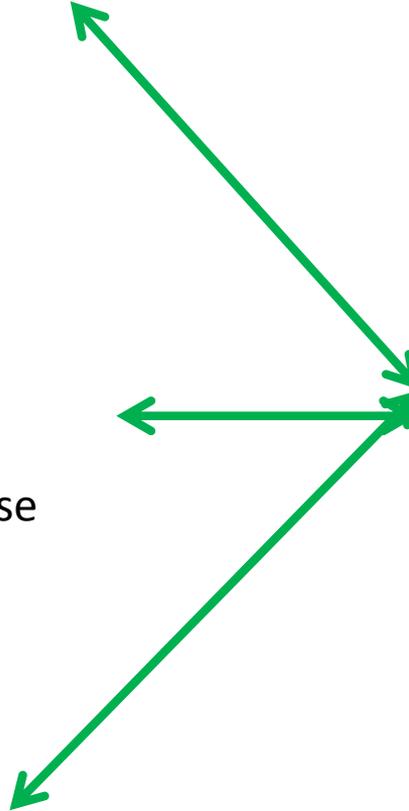
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Music

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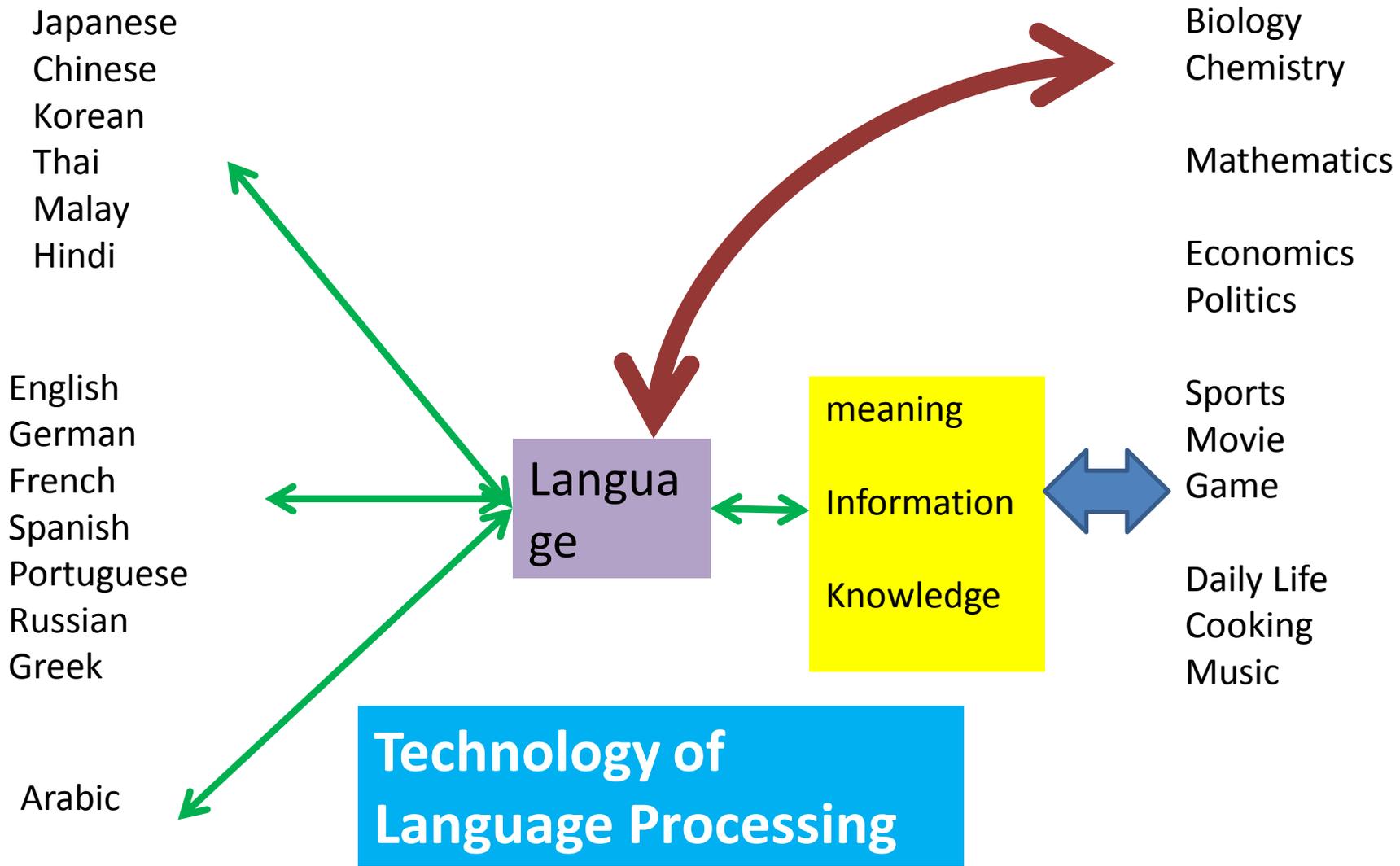
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