

Fine-Grained Information Access and MT for Patents

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Plan of the Talk

- Backgrounds
 - Shifts in Society
 - Semantics-based information access to text and MT
- NLP and Semantics-based information access
 - Deep parsing and entity/event-based access
 - Efficient deep parsers
- MT with Deep Parsing
 - Structural Differences: Predicate-Argument Structures
 - Lexical Differences: Terms and Ontologies
- Concluding remarks and future work

Plan of the Talk

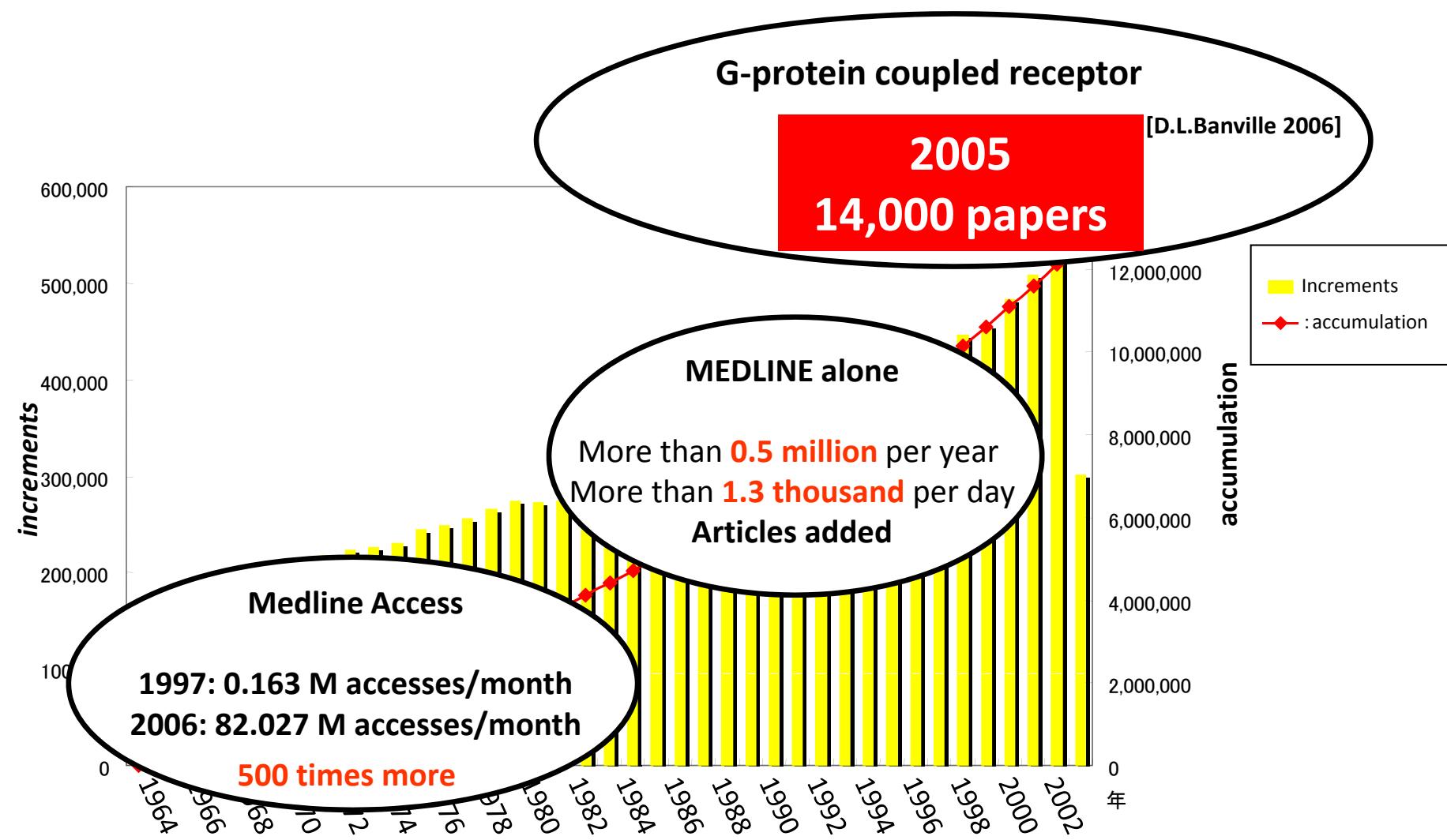
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MT for Patents among Asian Languages

- Shift in society
 - From Labor-based Industrial Society to Knowledge-based Industrial Society
- Shift in the world
 - Globalization
 - From the Economies in the Atlantic-rim to the Economies in the Pacific-rim
 - Translation among (Asian Languages + English)
- Difficulties in MT
 - Translation among European languages to Translation among (Asian Languages + English)
 - Structural and Lexical differences
 - Sentence-oriented languages and Context-oriented languages
- **SMT with great enhancement of semantics-based processing**

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Increase in Medline

NaCTeM

www.nactem.ac.uk

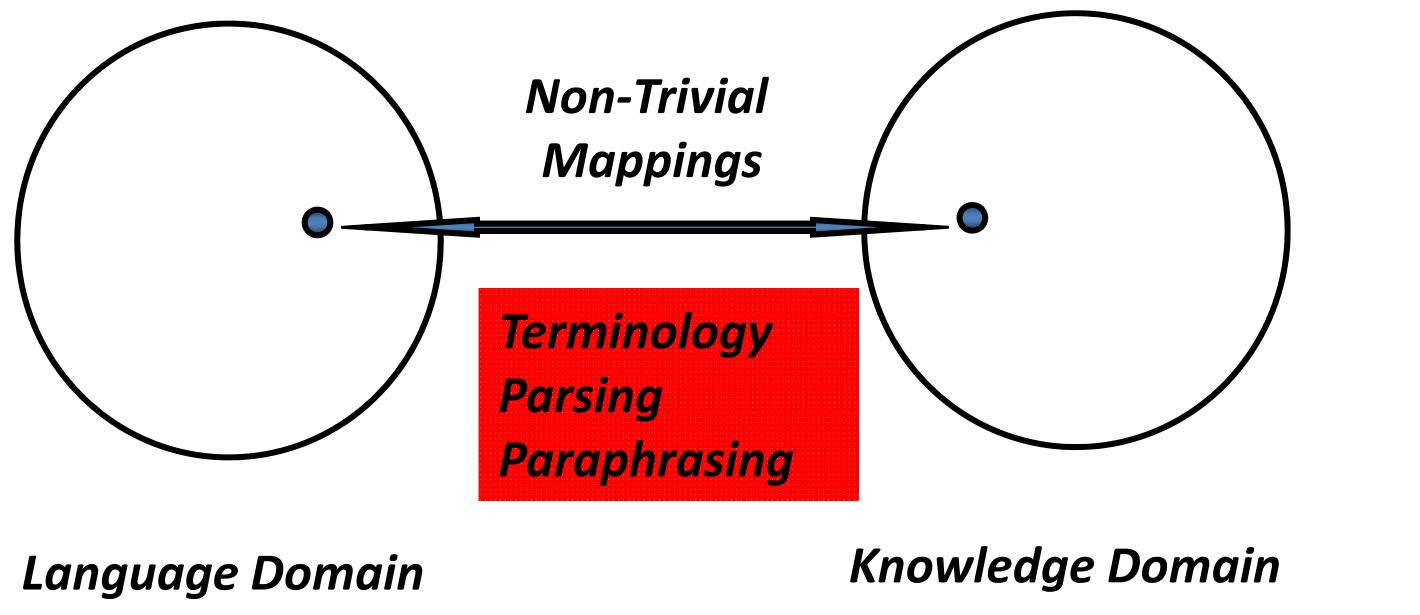
- First such centre in the world
- Funding: JISC, BBSRC, EPSRC
- Consortium investment
 - Chair in TM (Prof. J. Tsujii, Univ. Tokyo)
- Location: Manchester Interdisciplinary Biocentre (MIB) www.mib.ac.uk funded by the Wellcome Trust
- Initial focus: biomedical academic community
- Extend services to industry
- Extend focus to other domains (social sciences)

Consortium

- Universities of Manchester, Liverpool
- Service activity run by MIMAS (National Centre for Dataset Services), within MC (Manchester Computing)
- Self-funded partners
 - San Diego Supercomputing Center
 - University of California, Berkeley
 - University of Geneva
 - University of Tokyo
- Strong industrial & academic support
 - IBM, AZ, EBI, Wellcome Trust, Sanger Institute, Unilever, NowGEN, MerseyBio, ...

Semantics-based fine-grained information access

*From surface diversities and ambiguities
to
conceptual invariants*



*Concepts and Relationships
among Them*

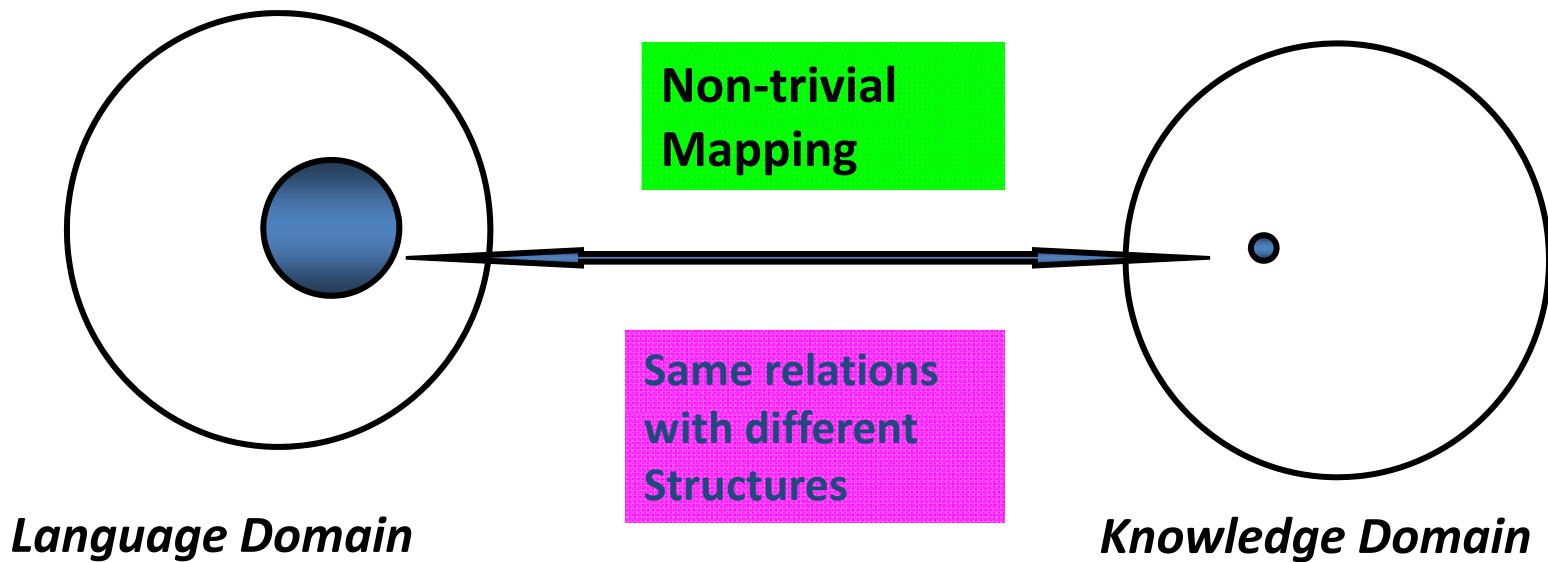
*Motivated
Independently of language*

[A] protein activates [B] (Pathway extraction)

Transcription initiation by the sigma(54)-RNA polymerase holoenzyme requires a full-strength ^σ70 promoter lacking an inherent σ site to associate with oskar PHO2 promoter to activate transcription, and transcription of PHO5 gene.

[sentence] > ([arg1_activate] > [protein])

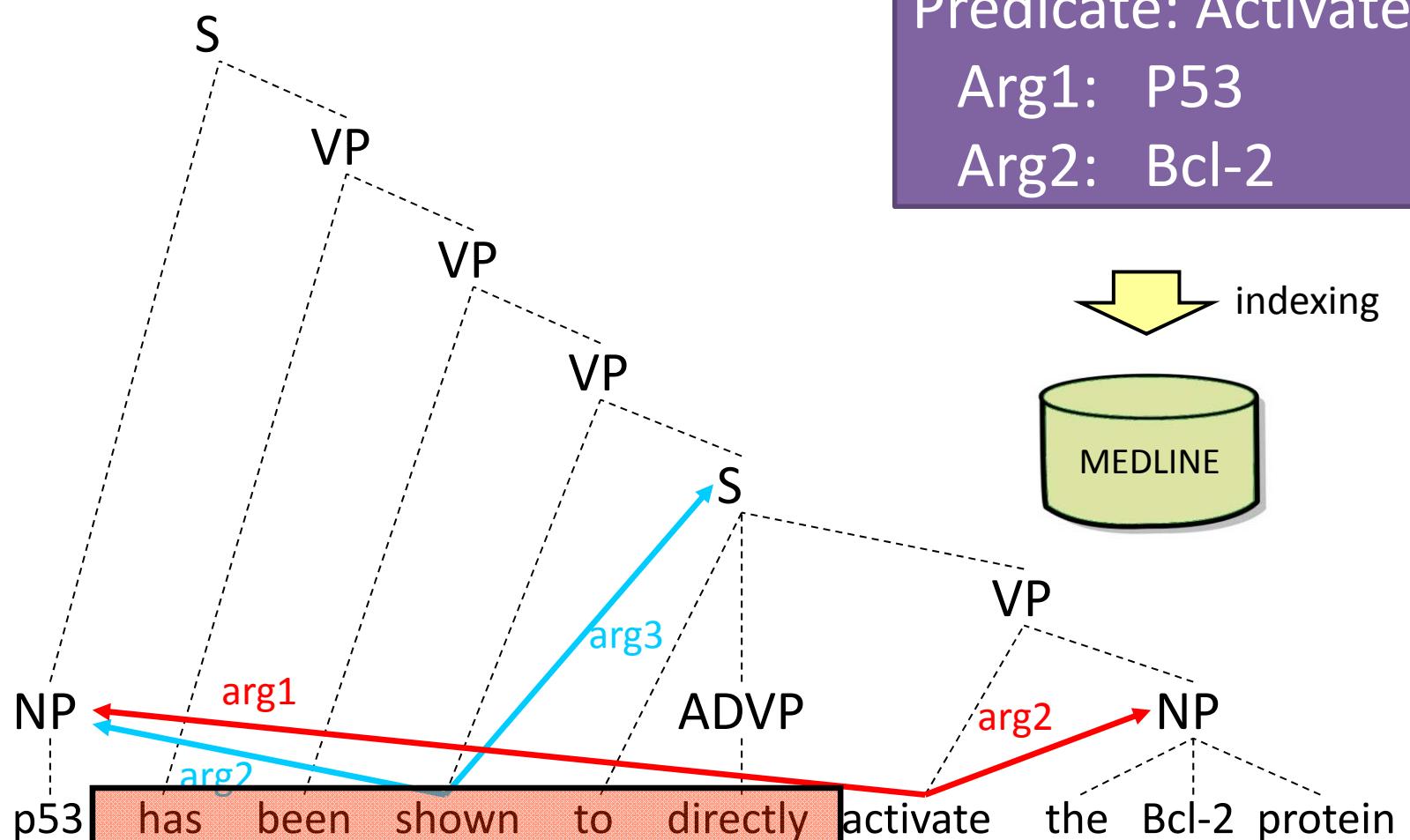
Retrieval using
Regional Algebra



Independently motivated of
Language

Deep Parsing

Parser based on Probabilistic HPSG (Enju)



Semantic Search Keyword Search GCL Search

subject

verb

object

p53

activate

Search!

Clear

Help

[Advanced search](#)Results 1-50 for p53 activate [»Show next](#) [»Show query](#)

1. [PMID: 15446548](#) [XML](#)

The molecules activated by p53 induce apoptosis, cell cycle arrest, and DNA repair to cons

2. [PMID: 15273740](#) [XML](#)

In this report, we demonstrated that human AMID gene promoter was activated by p53 in reporter gene assay.

Passive

3. [PMID: 15020844](#) [XML](#)

Recently, p53 has been shown to directly activate the pro-apoptotic Bcl-2 protein and apoptosis.

Passive and Infinitival Clause

4. [PMID: 15105421](#) [XML](#)

Electrophoretic mobility shift assays reveal that both transcription factors are capable of binding to putative consensus sites, and luciferase reporter assays reveal that E2F1 and p53 can activate transcription from the SIVA promoter.



5. [PMID: 15247038](#) [XML](#)

Although the role of the nuclear factor-kappa B (NF-kappa B) signaling cascade is crucial in ICAM-1 activation, we have shown that p53 directly activates the expression of ICAM-1 in an NF-kappa B-independent manner.

6. [PMID: 15021899](#) [XML](#)

Because the MDM2 gene is transcriptionally activated by p53, it forms part of an autoregulatory feedback loop that directly links the transcriptional activity of p53 with its degradation.

7. [PMID: 15064739](#) [XML](#)

Semantic Retrieval System

Using Deep Syntax

MEDIE

Passive

Passive and Infinitival Clause

MARIE — See what causes cancer?

MARIE is a demo system presented by [Tsujii Laboratory](#)

Semantic Search **Keyword Search** GCL Search

subject	verb	object
p53	activate	

[Search!](#) [Clear](#) [Help](#)

[Advanced search](#)

Show	50 results	Help
Output format	<input checked="" type="radio"/> sentence <input type="radio"/> article <input type="radio"/> table	Help
Keywords	<input type="text"/>	Help
Modifiers	<input type="text"/> not	Help
Base form	<input type="checkbox"/> subject <input checked="" type="checkbox"/> verb <input type="checkbox"/> object <input type="checkbox"/> keyword	Help
Ontology	<input type="checkbox"/> subject <input type="checkbox"/> verb <input type="checkbox"/> object	Help
Category	subject any object any	Help

Results 1-50 for p53 activate [»Show next](#) [»Show query](#)

35.82 seconds (5.37% finished)

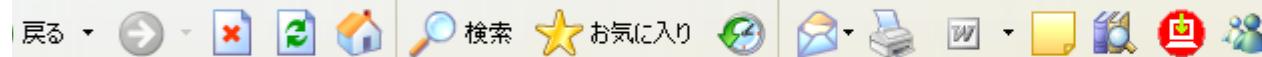
1. [PMID: 11212267](#) [»XML](#)

KAI1/CD82 has been shown to be a metastasis suppressor for several human cancers , and a recent study revealed that wild-type tumor suppressor p53 can directly activate KAI1/CD82 gene expression .

2. [PMID: 11162500](#) [»XML](#)

However , in an in vitro transcription assay with partially purified basal transcription factors , p53 only partially activated transcription from the same binding site and required PAb421 for full activation .

3. [PMID: 10521394](#) [»XML](#)



ス(D) http://nactem2.mc.man.ac.uk/mdecie/search.cgi?search_type=semantic_search&subject=p53&verb=activate&base_form=verb&ontology=

移動 リン

Category subject any object any

help

sults 1-11 for p53 activate [Show query](#)

100.67 seconds (100.00% finished)

1. [PMID: 11483599](#) [XML](#)

We demonstrated that mutant p53 did not activate either the MRP1 promoter or the endogenous gene .

2. [PMID: 12019172](#) [XML](#)

However, luciferase constructs driven by the HDAC5 promoter containing three to six potential binding sites were not activated by p53 , nor was the expression of HDAC5 mRNA induced by p53-activating agents .

3. [PMID: 12048243](#) [XML](#)

This activation occurred by a phosphorylation-independent mechanism involving direct binding of GSK3beta to p53 , which was confined to the nucleus where p53 is localized , and mutated p53 (R175H) bound but did not activate GSK3beta .

4. [PMID: 14557665](#) [XML](#)

Thus , it is likely that the E1B 55-kDa protein sequesters Daxx and p53 in specific nuclear locations , where p53 can not activate transcription .

5. [PMID: 14517211](#) [XML](#)

The DDATHF-stabilized p53 bound to the p21 promoter in vitro and in vivo but did not activate histone acetylation over the p53 binding sites in the p21 promoter that is an integral part of the transcriptional response mediated by the DNA damage pathway .

6. [PMID: 8632013](#) [XML](#)

Two monoclonal antibodies to the N terminus of p53 , PAb1801 and DO-1 , do not activate the latent DNA binding function of p53 but can protect the p53 wild-type conformation at 37 degrees C .

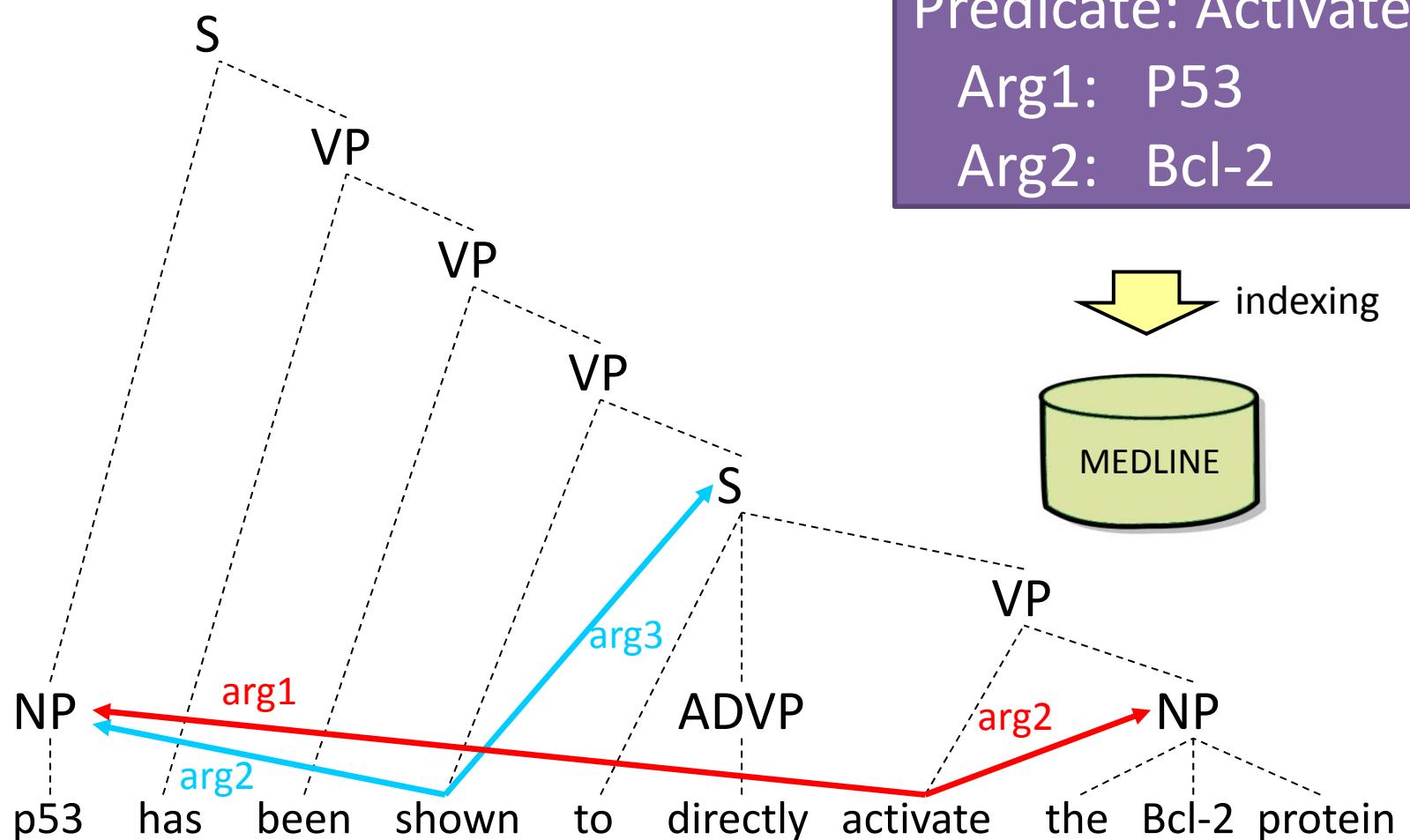
7. [PMID: 9159467](#) [XML](#)

RNA polymerase II transcriptional activators , like GAL4 , VP16 or p53 , fused to GAL4 DNA-binding domain , did not activate the UAS (G) SNR6 gene .

8. [PMID: 9360984](#) [XML](#)

Deep Parsing

Parser based on Probabilistic HPSG (Enju)



Results on PTB-WSJ

Parser	grammar	Accuracy	Speed
MST parser	dependency	90.02% (LAS)	4.5 snt/sec
Sagae's parser	dependency	89.01% (LAS)	21.6 snt/sec
Berkeley parser	CFG	89.27% (LF1)	4.7 snt/sec
Charniak's parser	CFG	89.55% (LF1)	2.2 snt/sec
Charniak's parser reranker	CFG	91.40 % (LF1)	1.9 snt/sec
Enju parser	HPSG	88.87% (PAS-LF1)	2.7 snt/sec
Mogura parser	HPSG	88.07% (PAS-LF1)	22.8 snt/sec

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Fine-grained tree-to-string translation rule extraction

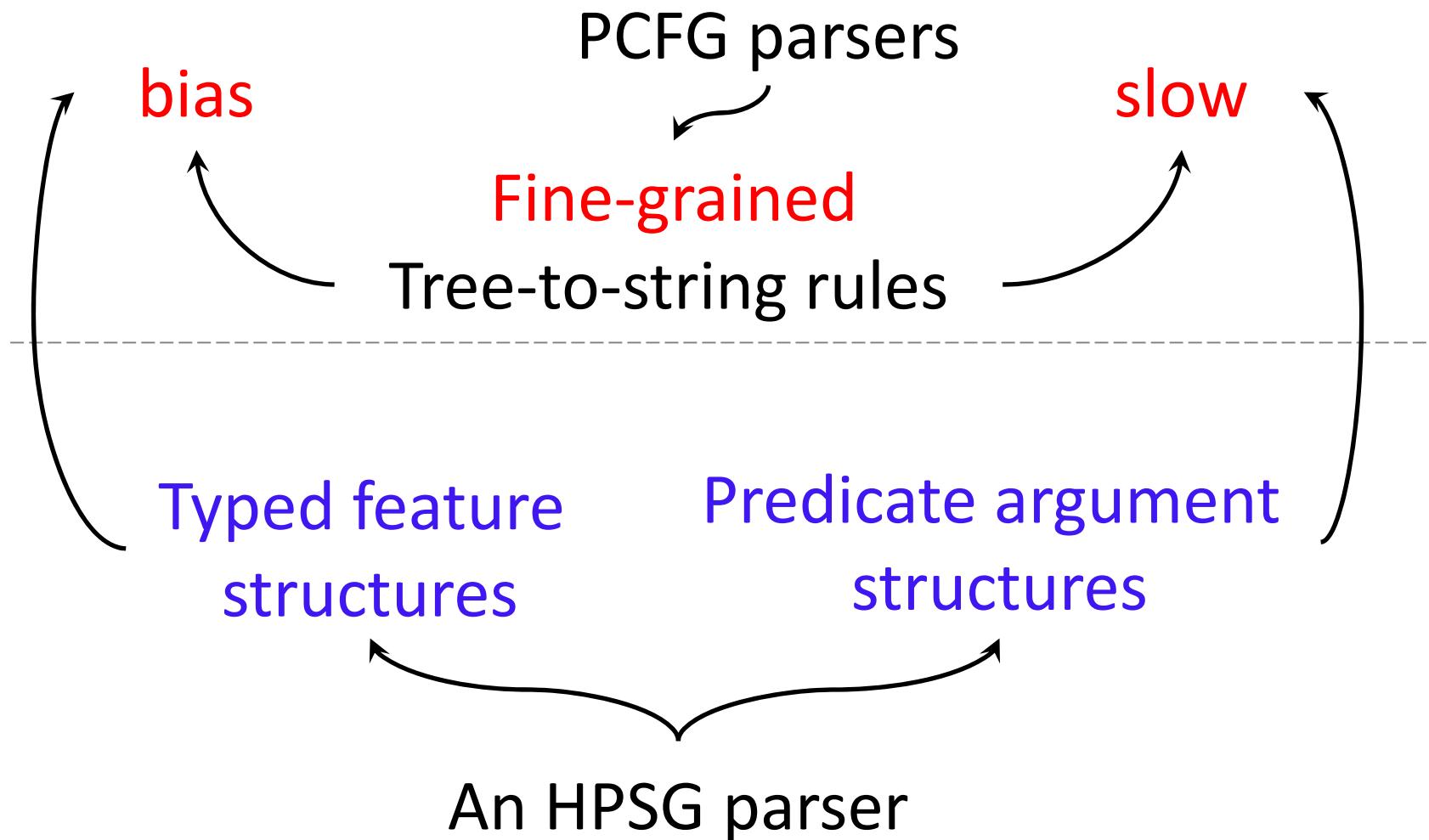
(presented in ACL 2010)

Xianchao Wu (吳 先超)

Takuya Matsuzaki, Jun'ichi Tsujii

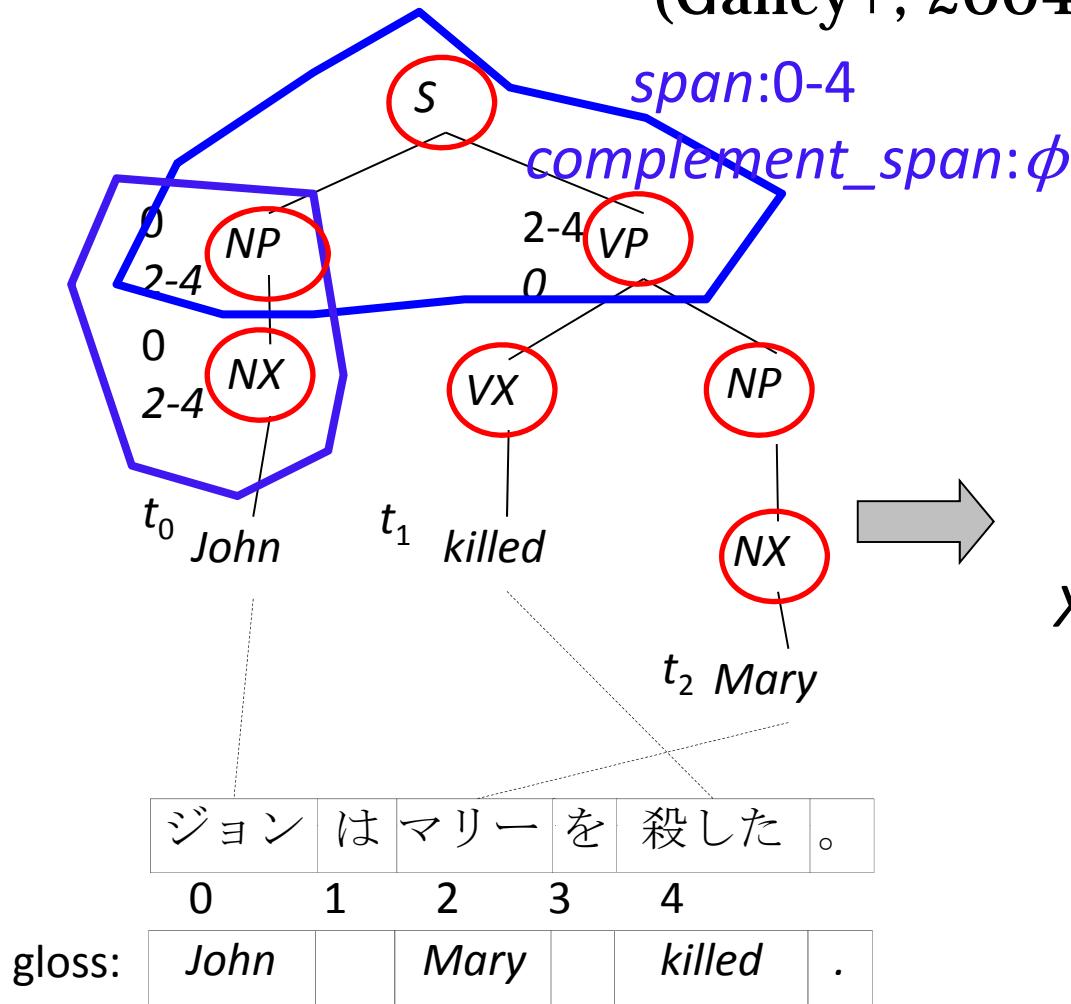
The University of Tokyo
2010.07.23

Outline



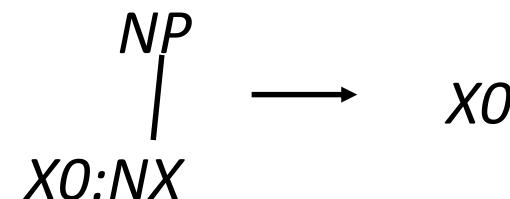
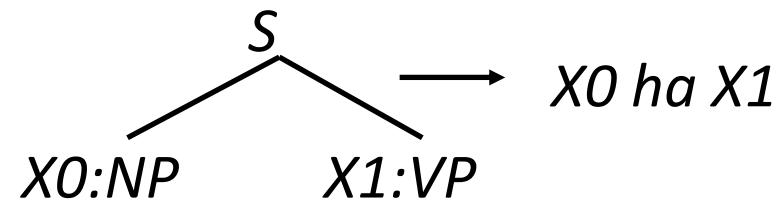
Minimal rule extraction : GHKM

(Galley+, 2004)

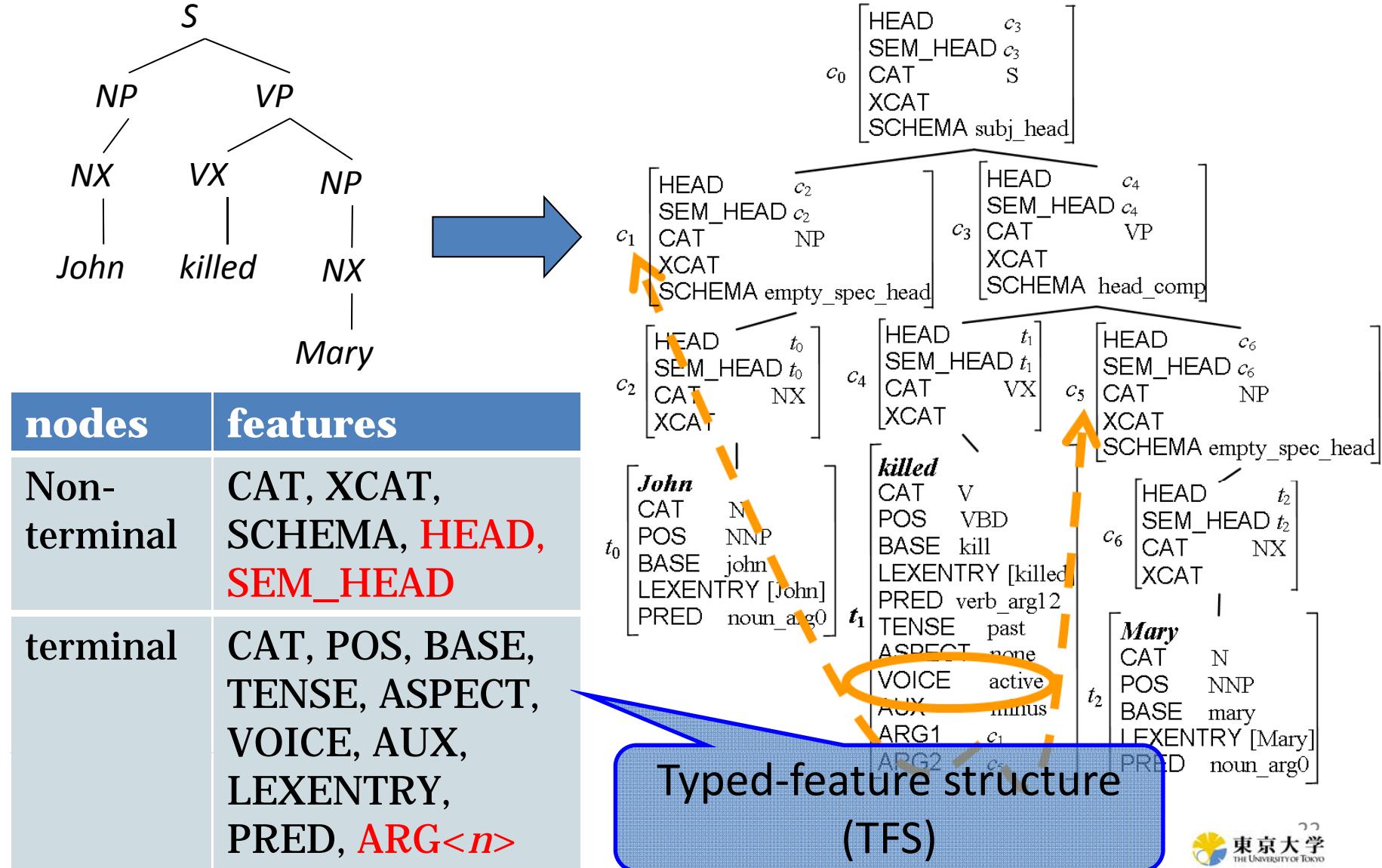


NO overlapping

Admissible nodes



HPSG for syntax-based SMT



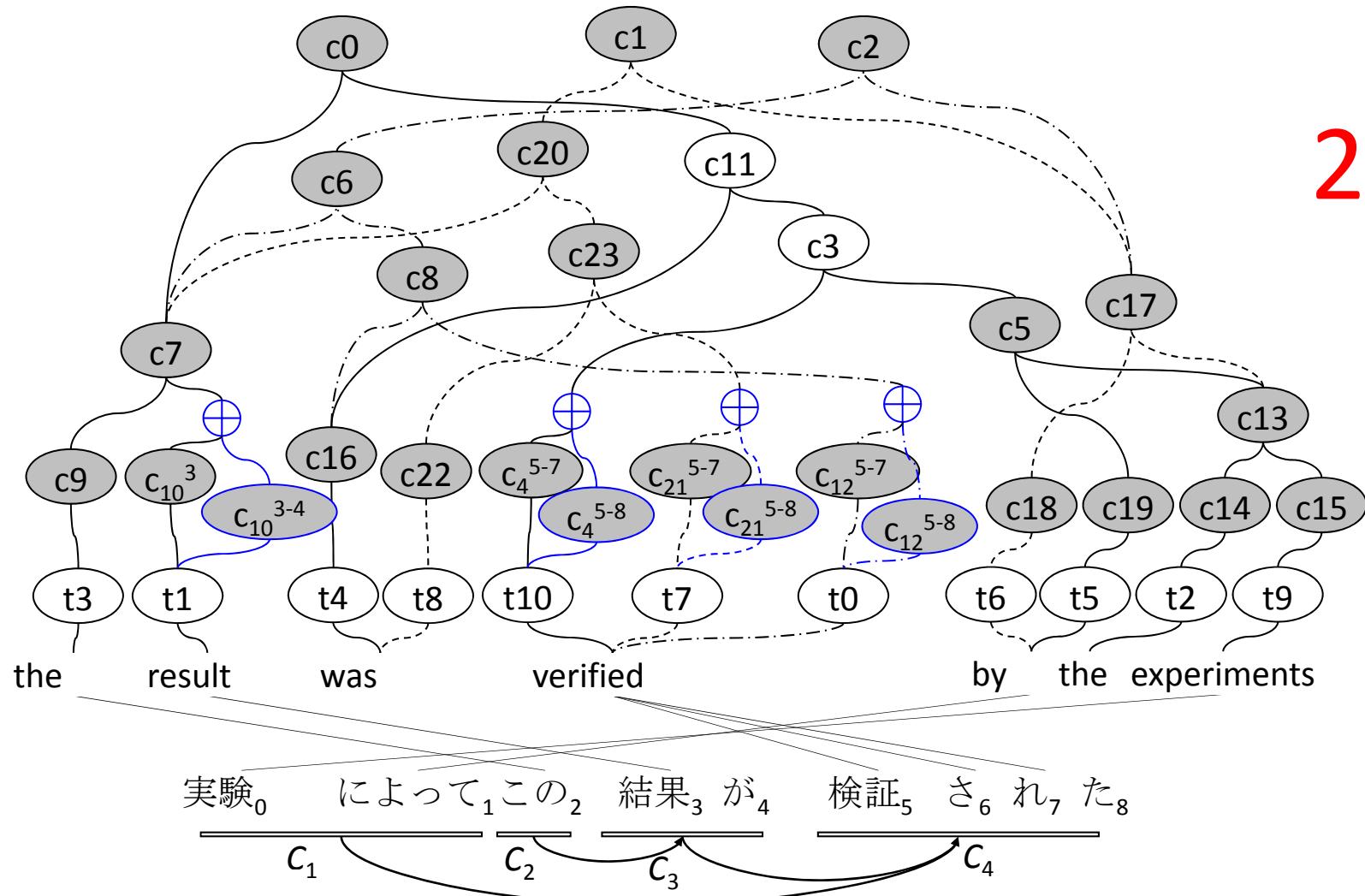
Additional comparison

Systems	En2Jp	Jp2En
Joshua (Li+, 2009)	21.79	19.73
Forest-based	24.75**	22.67**

**= $p<0.01$

Remove the ambiguous alignments

22



Remove the ambiguous alignments

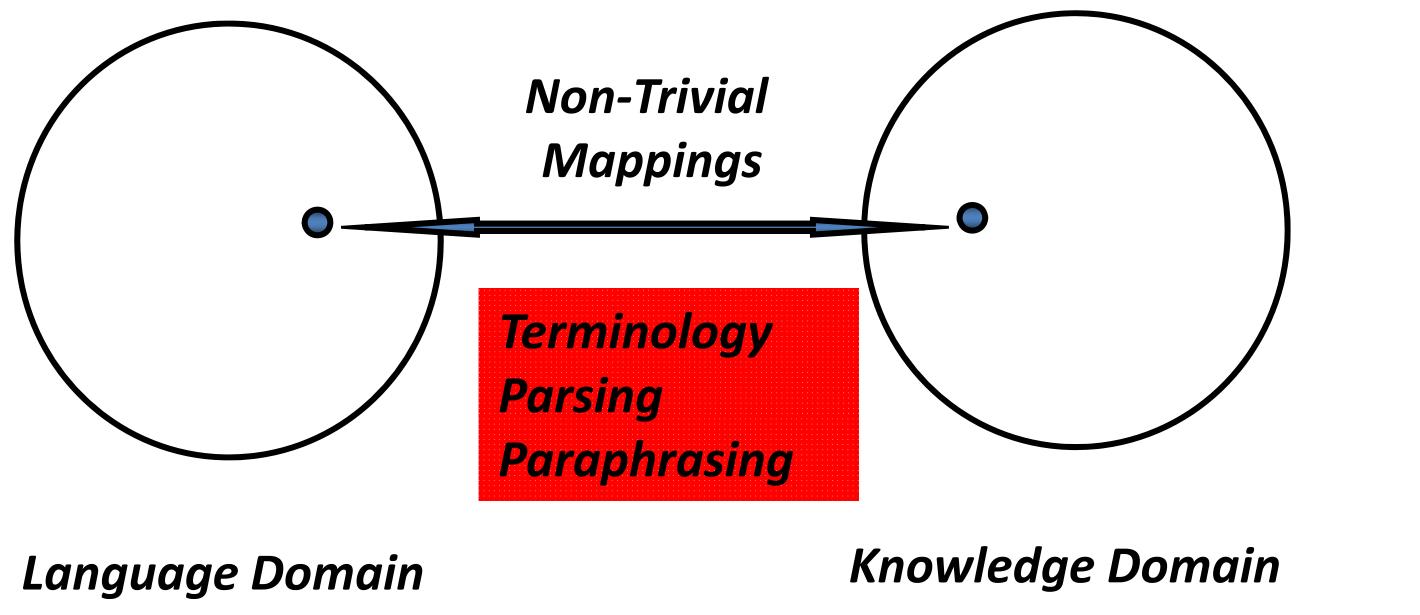
Systems	En2Jp
Forest-based rules (Wu+, ACL 2010)	24.75
+ forest-based decoder	25.83
+ function words	28.44**

**= $p<0.01$

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Annotation of GENIA corpus – Term&POS

PMID:1984449

Induction of NF-KB during monocyte differentiation by HIV type 1 infection.

The production of human immunodeficiency virus type 1 (HIV-1) in macrophage progeny was followed in the U937 promonocytic cell line after stimulation either with retinoic acid or PMA, and purified human monocytes and macrophages. Electrophoretic mobility shift assays and Southwestern blotting experiments were used to detect the binding of cellular transactivation factor NF-KB to the double repeat-KB enhancer sequence located in the long terminal repeat. PMA treatment and not retinoic acid treatment of the U937 cells acts in inducing NF-KB expression in the nucleus. In nuclear extracts from monocytes or macrophages induction of NF-KB occurred only if the cells were previously infected with HIV-1. When U937 cells were infected with HIV-1, no induction of NF-KB was observed unless the cells were previously infected with HIV-1.

Ontology Protégé 3.1.1 (file:D:\cygwin\)

File Project OWL Code Window Tools Help

Individuals Classes Functions Projects

Ter
an
2000-400
abstracts

Event Annotation - Example

2) the binding of >I kappa B/MAD-3< T36 to >NF-kappa B p65< T37 is sufficient to retarget >NF-kappa B p65< T38 from the >nucleus< T39 to the >cytoplasm< T40.

EVENT E5

TYPE : Binding

THEME : T36

THEME : T37

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,

EVENT E6

TYPE : Localization

THEME : T38

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,

EVENT E7

TYPE : Positive regulation

THEME : E6

CAUSE : E5

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,