# 「萬用模版」
# 英文論文寫作法

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# Why am I qualified?

- 179 papers (1989-2019)
- 4 review articles
- 3 book chapters
- 1 patent
- Review 300+/year
- Proposals/Manuscripts
- Technical writing/Experimental design courses

**Google Play Books**
https://play.google.com/store/books/details?id=bgihDwAAQBAJ
**Readmoo**
https://readmoo.com/book/2101112570001

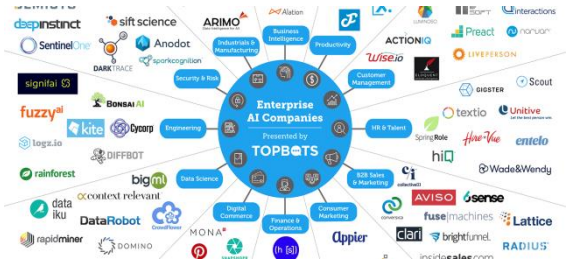
# 「萬用模版」與人工智慧(AI)結合

①事實問題
②研究回顧
③研究縫隙
④研究策略

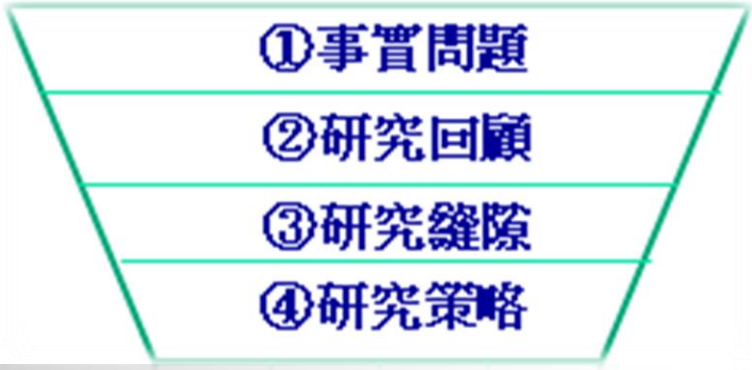
| ④統計方法 | ③量測觀察 | ②研究介入 | ①研究設計 |
|---|---|---|---|
| ④質疑結果 | ③關鍵結果 | ②圖表描述 | ①圖形表格 |

①結果摘述
②圖解文獻
③成就貢獻
④限制改進

| | | | |
|---|---|---|---|
| ①簡介 | ①WHY | ①Question | |
| ②方法 | ②HOW | ②Answer | |
| ③結果 | ③WHAT | ③Findings | |
| ④討論 | ④THEN | ④Meaning | |



# Introduction




- Facts and Problems
- Previous and Current Research
- Gap (Motivation)
- The Present work

# Statins and risk of incident diabetes: a collaborative meta-analysis of randomised statin trials

Lancet 2010; 375:735-42

*Naveed Sattar, David Preiss, Heather M Murray, Paul Welsh, Brendan M Buckley, Anton J M de Craen, Sreenivasa Rao Kondapally Seshasai, John J McMurray, Dilys J Freeman, J Wouter Jukema, Peter W Macfarlane, Chris J Packard, David J Stott, Rudi G Westendorp, James Shepherd, Barry R Davis, Sara L Pressel, Roberto Marchioli, Rosa Maria Marfisi, Aldo P Maggioni, Luigi Tavazzi, Gianni Tognoni, John Kjekshus, Terje R Pedersen, Thomas J Cook, Antonio M Gotto, Michael B Clearfield, John R Downs, Haruo Nakamura, Yasuo Ohashi, Kyoichi Mizuno, Kausik K Ray, Ian Ford*

## Summary

**Background** Trials of statin therapy have had conflicting findings on the risk of development of diabetes mellitus in patients given statins. We aimed to establish by a meta-analysis of published and unpublished data whether any relation exists between statin use and development of diabetes.

**Methods** We searched Medline, Embase, and the Cochrane Central Register of Controlled Trials from 1994 to 2009, for randomised controlled endpoint trials of statins. We included only trials with more than 1000 patients, with identical follow-up in both groups and duration of more than 1 year. We excluded trials of patients with organ transplants or who needed haemodialysis. We used the $I^2$ statistic to measure heterogeneity between trials and calculated risk estimates for incident diabetes with random-effect meta-analysis.

**Findings** We identified 13 statin trials with 91 140 participants, of whom 4278 (2226 assigned statins and 2052 assigned control treatment) developed diabetes during a mean of 4 years. Statin therapy was associated with a 9% increased risk for incident diabetes (odds ratio [OR] 1·09; 95% CI 1·02–1·17), with little heterogeneity ($I^2$=11%) between trials. Meta-regression showed that risk of development of diabetes with statins was highest in trials with older participants, but neither baseline body-mass index nor change in LDL-cholesterol concentrations accounted for residual variation in risk. Treatment of 255 (95% CI 150–852) patients with statins for 4 years resulted in one extra case of diabetes.

**Interpretation** Statin therapy is associated with a slightly increased risk of development of diabetes, but the risk is low both in absolute terms and when compared with the reduction in coronary events. Clinical practice in patients with moderate or high cardiovascular risk or existing cardiovascular disease should not change.

**Funding** None.

*Lancet* 2010; 375: 735–42

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See Comment page 700

**British Heart Foundation Glasgow Cardiovascular Research Centre, University of Glasgow, Glasgow, UK** (Prof N Sattar PhD, D Preiss MRCP, P Welsh PhD, Prof J J McMurray MD); **Robertson Centre for Biostatistics, University of Glasgow, Glasgow, UK** (H M Murray MSc, Prof I Ford PhD); **Department of Pharmacology and Therapeutics, Cork University Hospital, Cork, Ireland** (Prof B M Buckley FRCPI); **Department of Gerontology and Geriatrics, Leiden University Medical Centre, Leiden, Netherlands** (A J M de Craen PhD); **Department of Public Health**

# 1-1 Facts and Problems

- Statin therapy is effective for reduction of cardiovascular events[1,2] and is generally recognised as being safe and well tolerated.[3]
- However, researchers of six large randomised placebo-control trials[4–9] have reported conflicting results about the development of diabetes in patients taking such drugs.

# 1-2a. Mini-review

- In the JUPITER[4] trial, 17802 adults with no clinical or biochemical diagnosis of diabetes based on fasting glucose concentrations were assigned rosuvastatin or placebo for a median of 1.9 years.
- Significantly more individuals in the statin group than in the placebo group developed diabetes[10].

# 1-2b. Mini-review

- By contrast, results from the WOSCOP[5] study suggested that pravastatin therapy might reduce the frequency of diabetes.
- These findings have raised questions about the safety of long-term use of statins,[10] and led to calls for a systematic exploration of the possible effect of statin therapy on incident diabetes.[11]

# Literature review

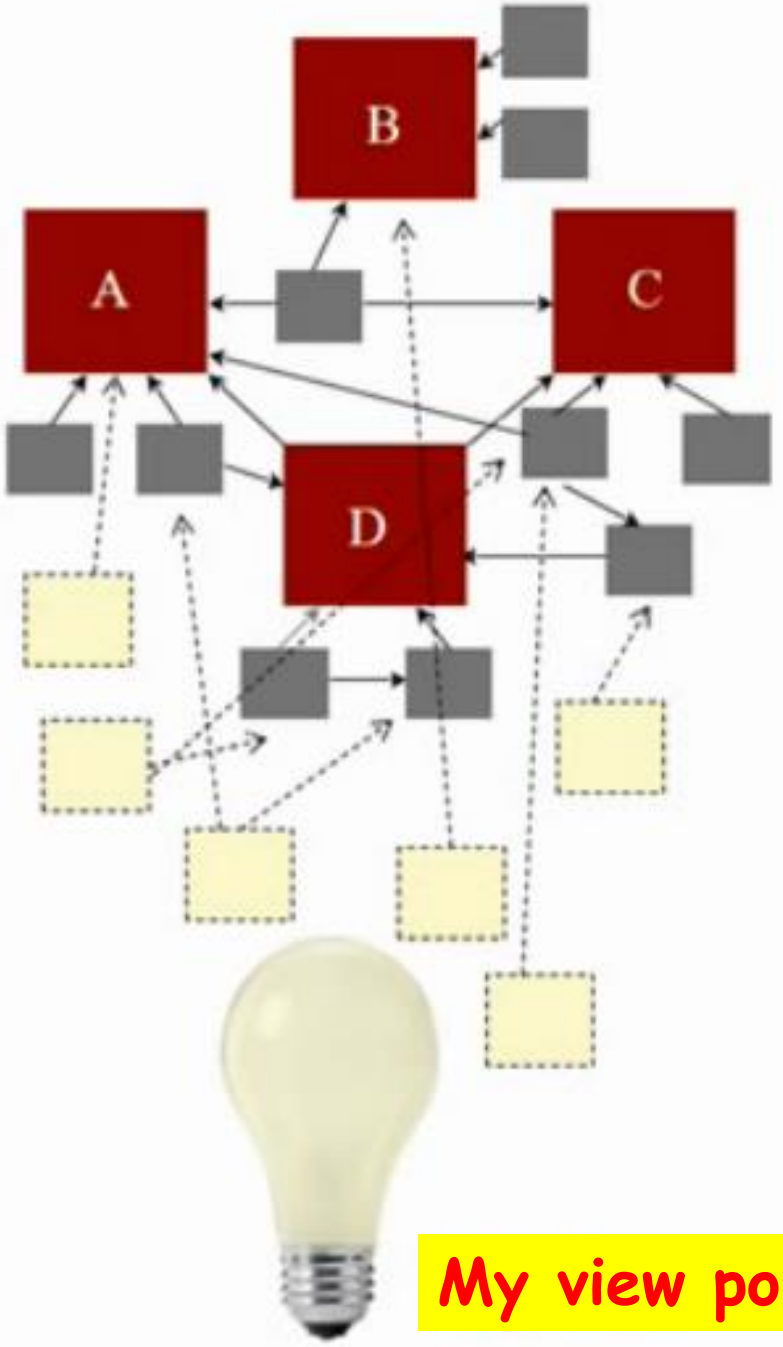
- References to previous & current research
- Providing a transition between previous and current research
- Arranging the order of all references
  - Chronological
  - Different approaches/models
  - General/Specific to your own

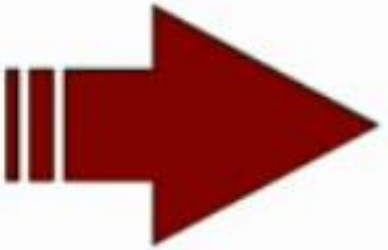
## Purpose of a Literature Review

- Establishes what is already known about a particular topic and what methods have been used in researching the topic
- Prevents you from reproducing what is already known
- Exposes gaps in the literature and helps you position your research

# What should be covered in literature review?

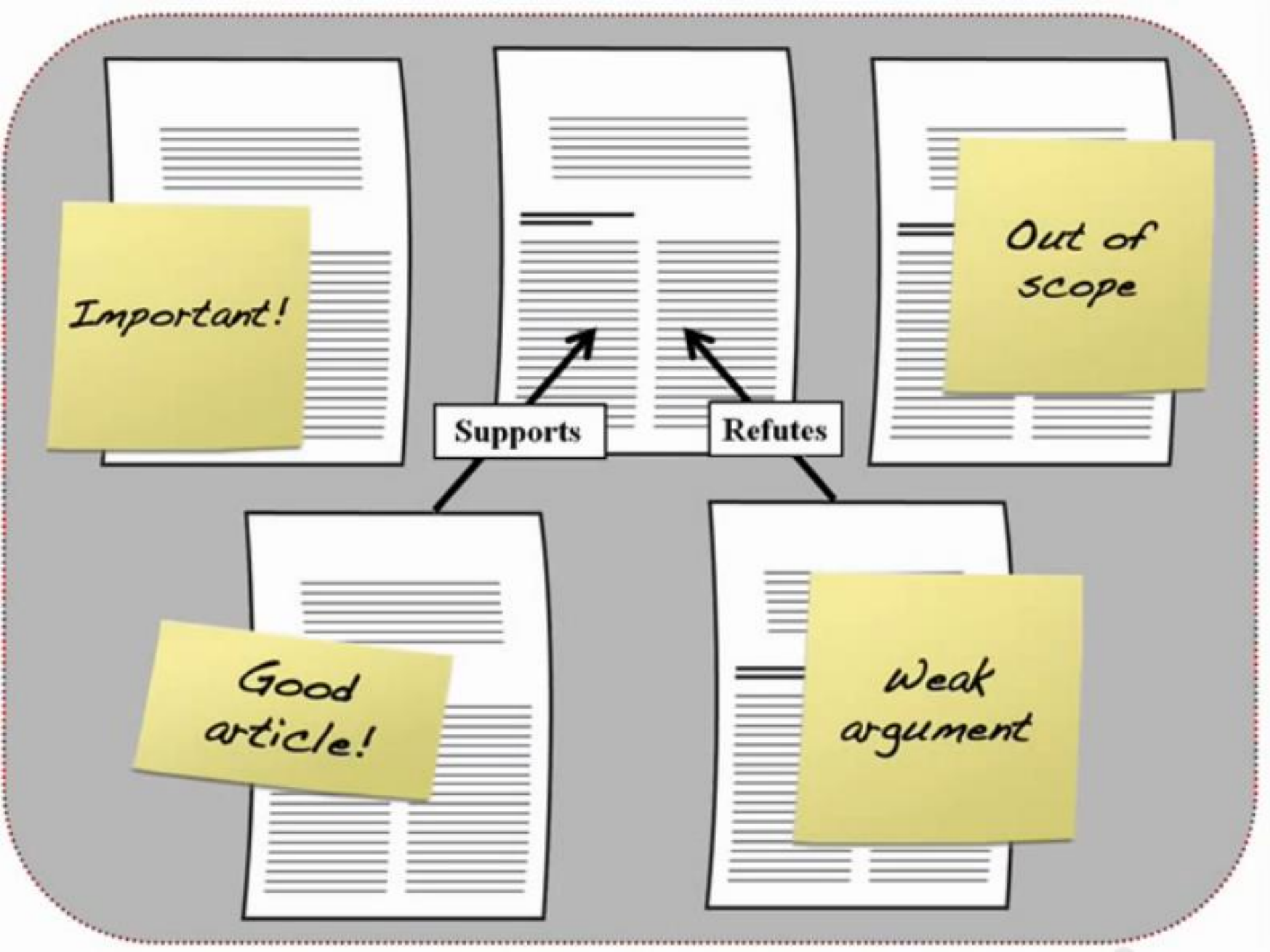
- What do scholars say about this topic?
- What are debates within this topic?
- What ideas do you agree (or disagree) with?
  - Why or why not?
- What has not been said about this topic?




MY Literature Review

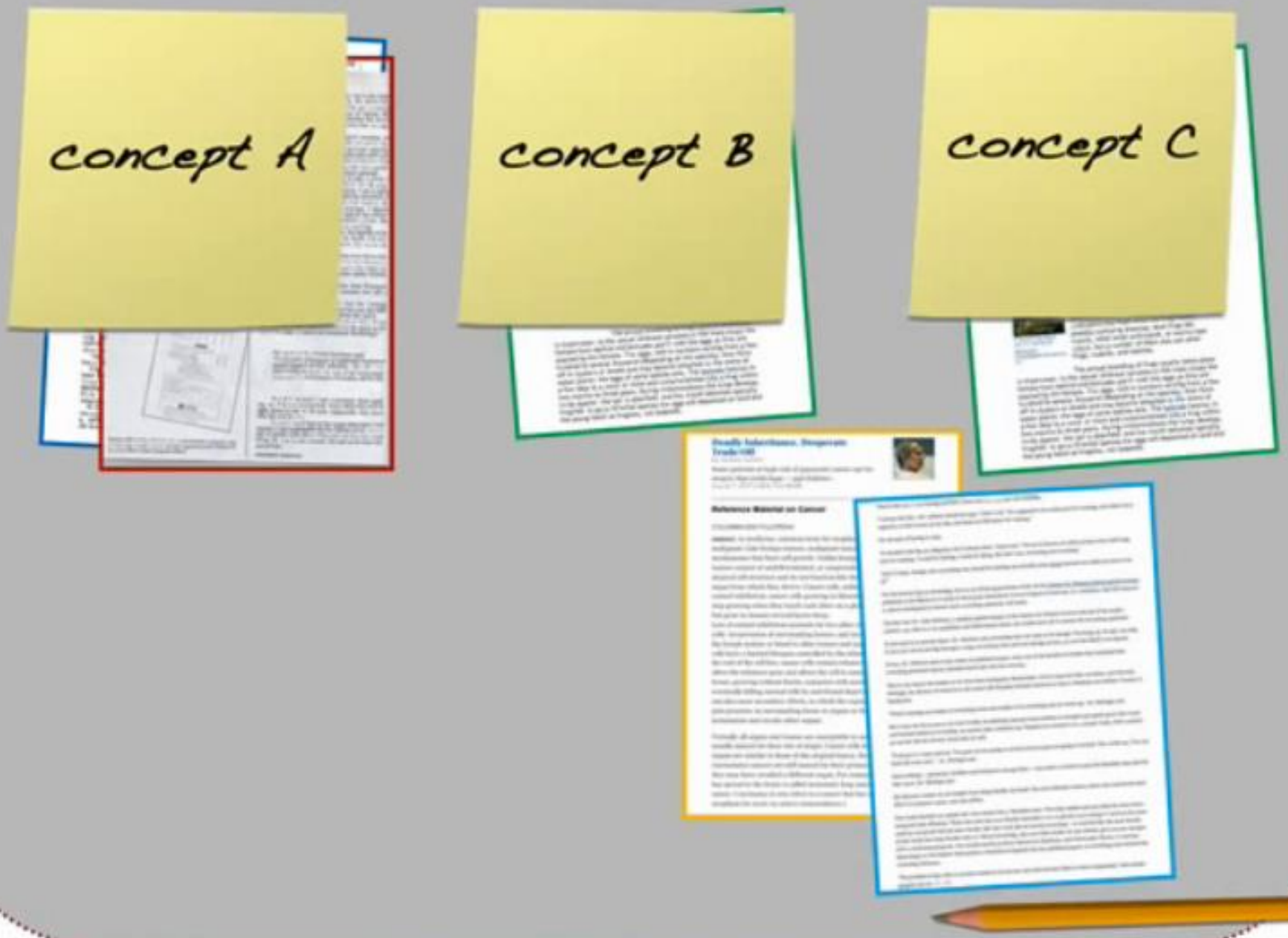
My view point


Important!
Supports
Refutes
Out of scope
Good article!
Weak argument

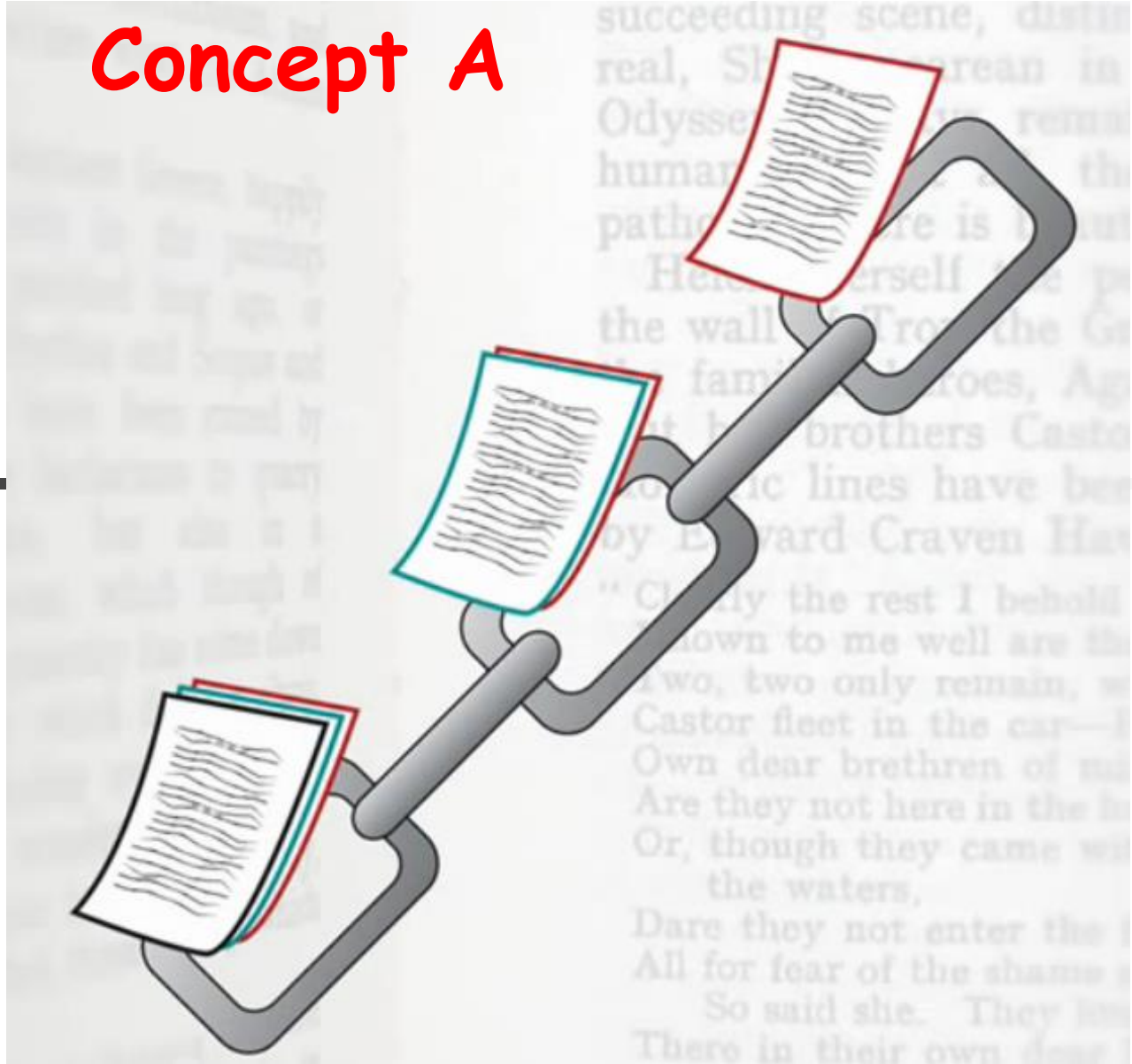
concept A

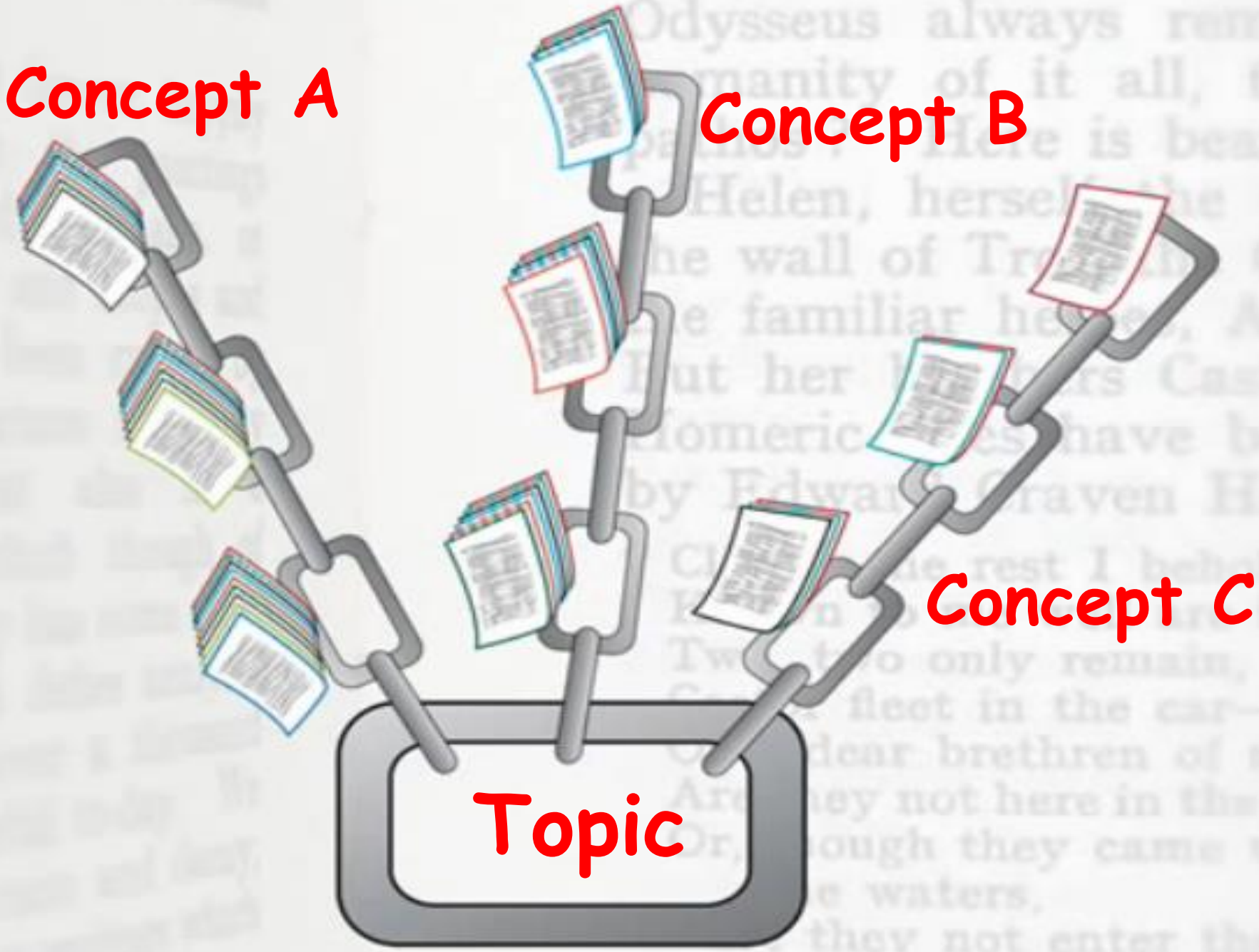
concept B

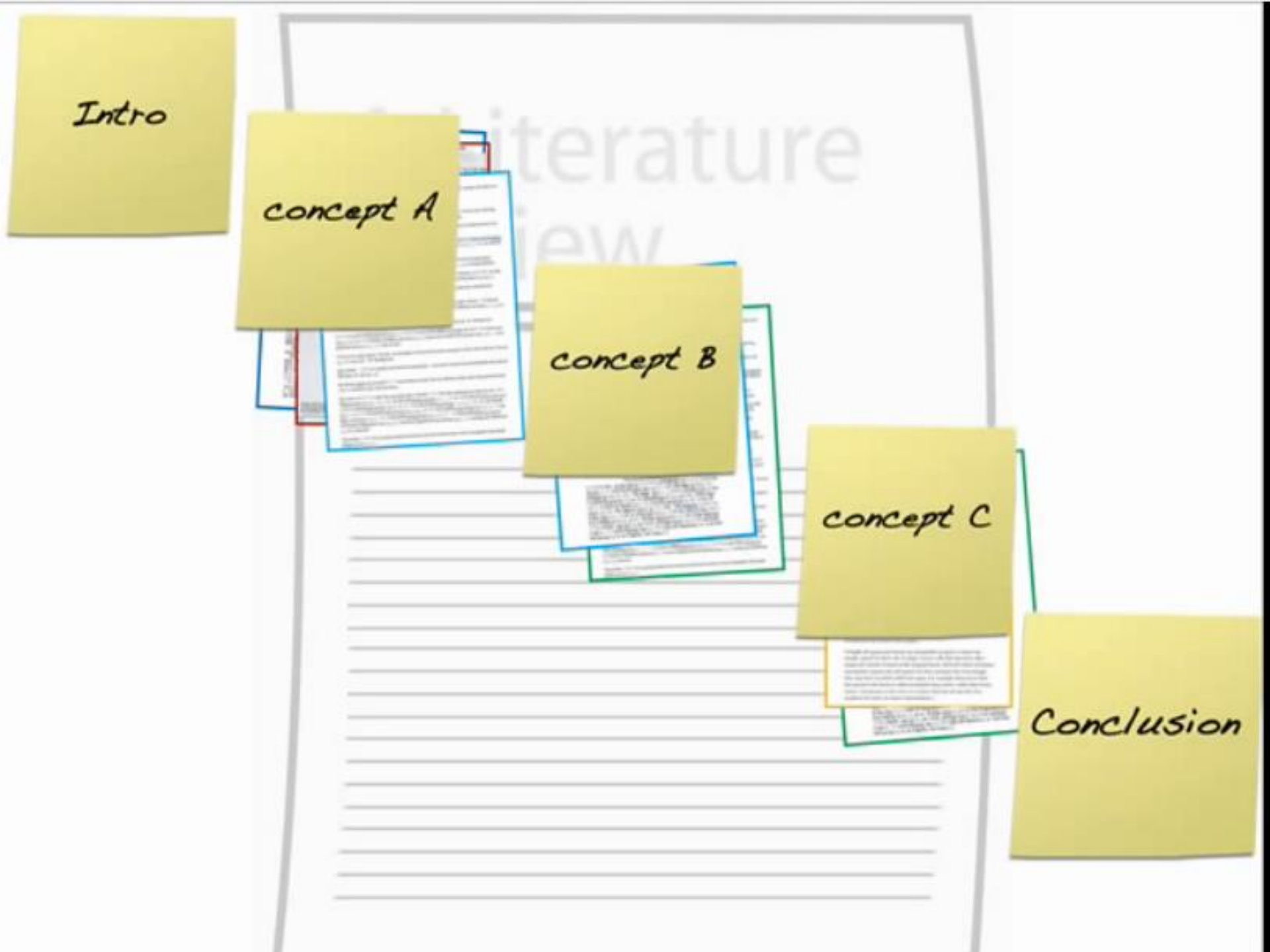
concept C


concept A
concept B
concept C

# Concept A




Concept A
Concept B
Concept C
Topic


Intro
concept A
concept B
concept C
Conclusion

# 1-3. Gap

- Overestimation of clinical benefit or underestimation of risk is potentially of major public health importance.

# Locate a Gap

- A gap or your motivation of the study
  - Describe a problem
  - Propose a hypothesis
  - Present a prediction

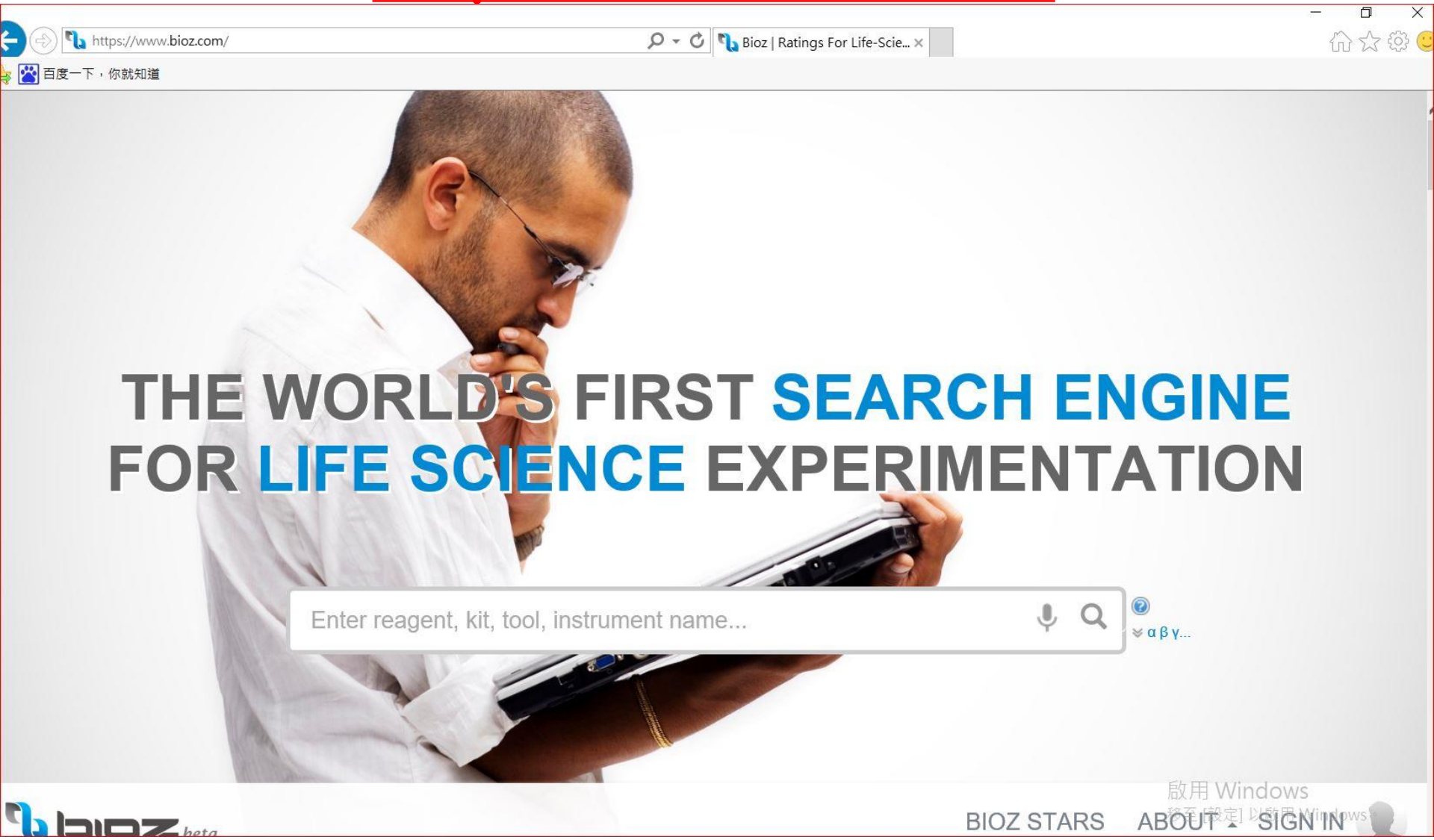
# 1-4. The present study

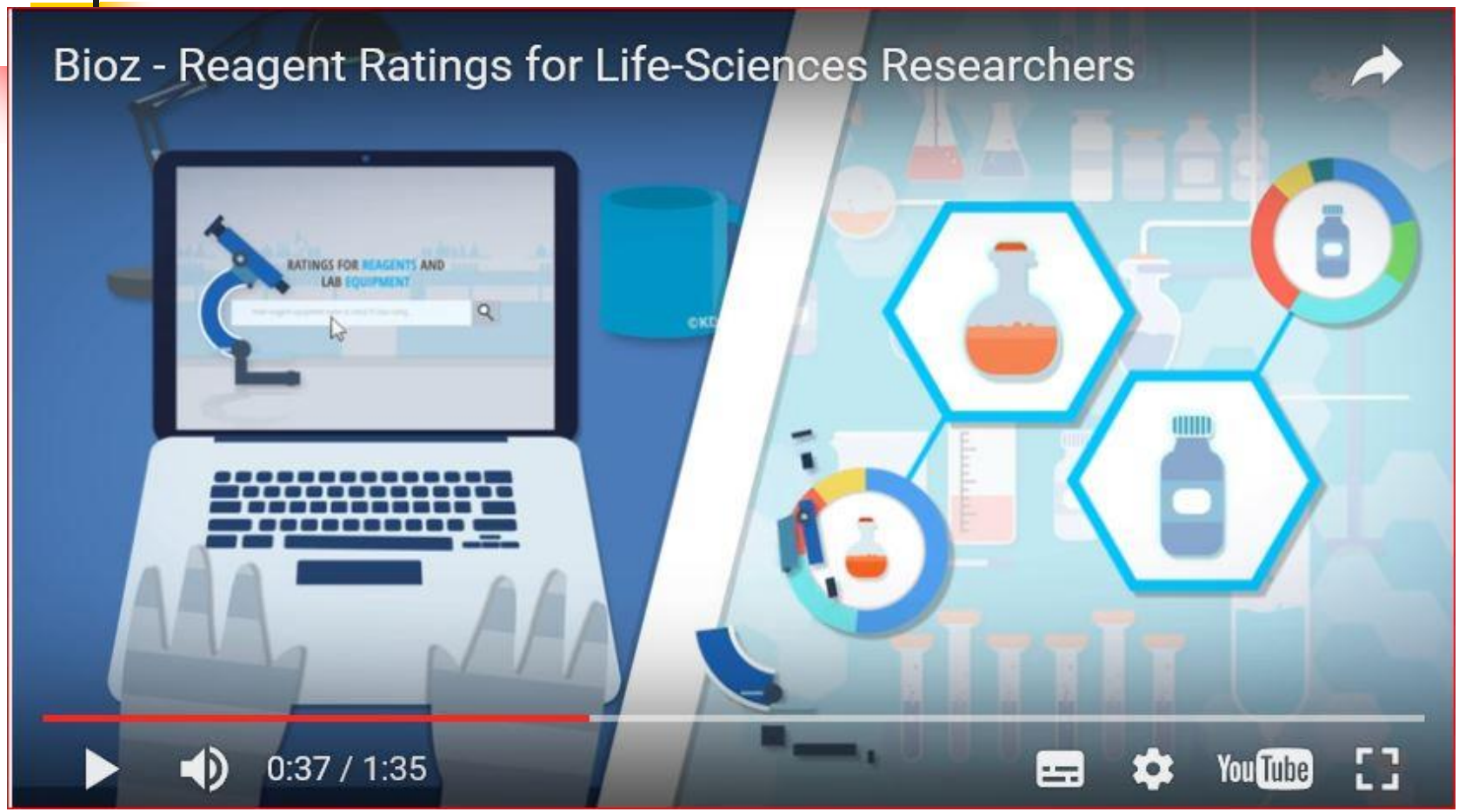
- To resolve this uncertainty, we investigated this effect by undertaking a meta-analysis of all available published and unpublished data from large placebo-controlled and standard-care-controlled statin trials,

# Materials and Methods

- Study design is selected & the subjects (patients, animals) to be studied are defined.
- Interventions (treatment) are decided on in detail.
- Measurements and other observations to be made.
- Statistical procedures/limitations for assessment of data.

# http://www.bioz.com/




Bioz - Reagent Ratings for Life-Sciences Researchers
RATINGS FOR REAGENTS AND
LAB EQUIPMENT
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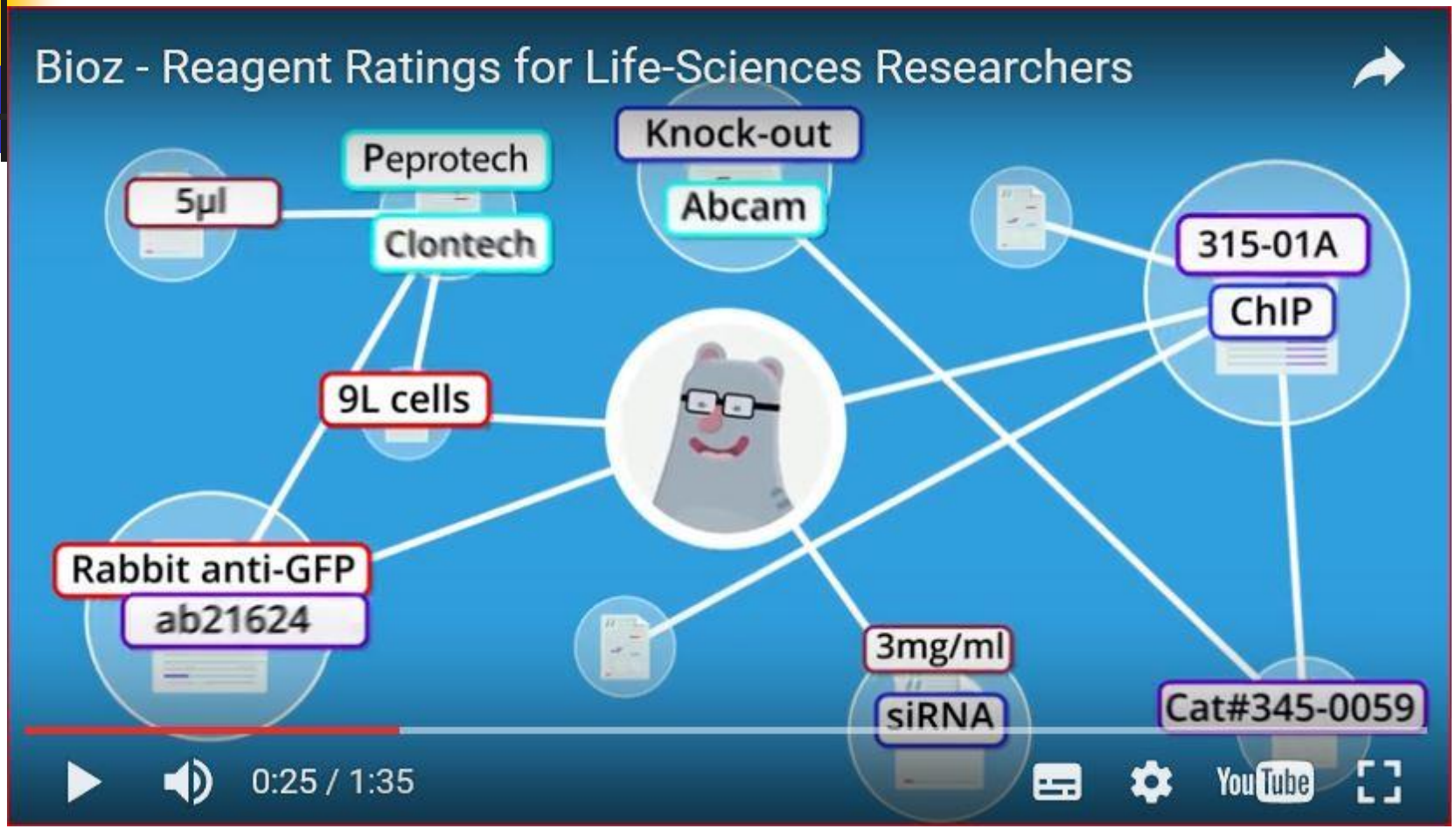
YouTube


Bioz - Reagent Ratings for Life-Sciences Researchers
Knock-out
Peprotech
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Clontech
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ChIP
9L cells
Rabbit anti-GFP
ab21624
3mg/ml
siRNA
Cat#345-0059
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YouTube

# Seraching

abcam

Anti TRPM6 antibody

Products Custom Support Events Pathways Contact us About us Careers

Products (1) Resources (0)

1 product result for Anti TRPM6 antibody

**Filters**

**Product types**

› Primary antibodies (1)

**Research areas**

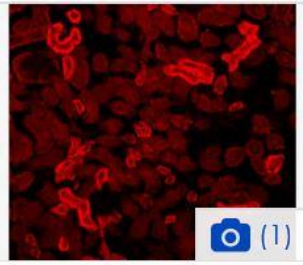
› Metabolism (1)

› Signal Transduction (1)

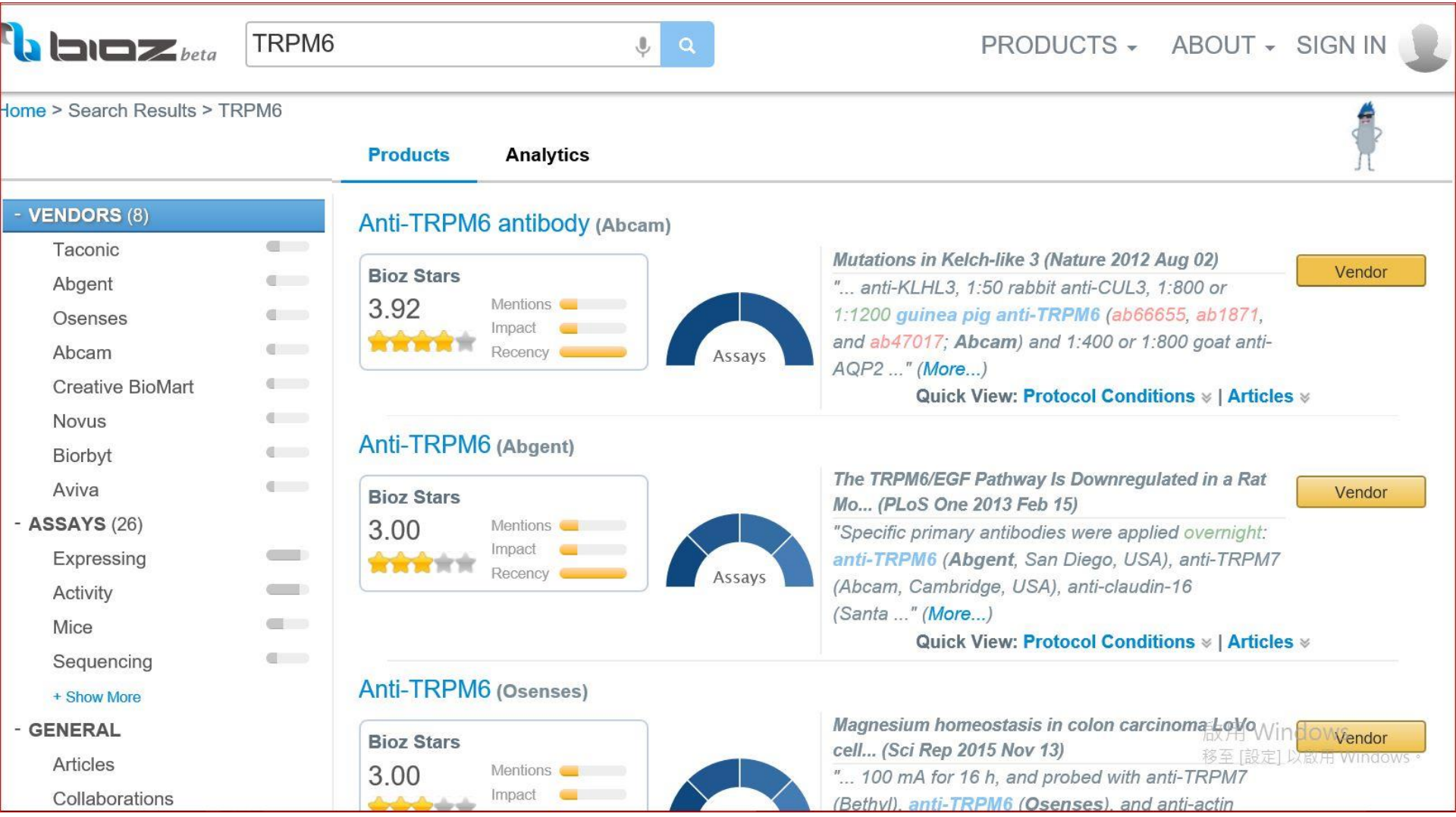
## Anti-TRPM6 antibody (ab47017)

Specific References (1)

Description: Guinea pig polyclonal to TRPM6
Application: IHC-Fr
Reactivity: Rat (predicted: Mouse)
Conjugate: Unconjugated




Compare (max 4)


bioz beta
TRPM6
PRODUCTS
ABOUT
SIGN IN
Home > Search Results > TRPM6
Products
Analytics
- VENDORS (8)
Taconic
Abgent
Osenses
Abcam
Creative BioMart
Novus
Biorbyt
Aviva
- ASSAYS (26)
Expressing
Activity
Mice
Sequencing
+ Show More
- GENERAL
Articles
Collaborations
Anti-TRPM6 antibody (Abcam)
Bioz Stars
3.92
Mentions
Impact
Recency
Assays
Mutations in Kelch-like 3 (Nature 2012 Aug 02)
"... anti-KLHL3, 1:50 rabbit anti-CUL3, 1:800 or 1:1200 guinea pig anti-TRPM6 (ab66655, ab1871, and ab47017; Abcam) and 1:400 or 1:800 goat anti-AQP2 ..." (More...)
Quick View: Protocol Conditions | Articles
Vendor
Anti-TRPM6 (Abgent)
Bioz Stars
3.00
Mentions
Impact
Recency
Assays
The TRPM6/EGF Pathway Is Downregulated in a Rat Mo... (PLoS One 2013 Feb 15)
"Specific primary antibodies were applied overnight: anti-TRPM6 (Abgent, San Diego, USA), anti-TRPM7 (Abcam, Cambridge, USA), anti-claudin-16 (Santa ..." (More...)
Quick View: Protocol Conditions | Articles
Vendor
Anti-TRPM6 (Osenses)
Bioz Stars
3.00
Mentions
Impact
Magnesium homeostasis in colon carcinoma LoVo cell... (Sci Rep 2015 Nov 13)
"... 100 mA for 16 h, and probed with anti-TRPM7 (Bethyl), anti-TRPM6 (Osenses), and anti-actin
Vendor

Products Analytics

- **VENDORS** (5)
  - KD Scientific
  - Novus
  - Creative BioMart
  - Aviva
  - Biorbyt
- **ASSAYS** (27)
  - Expressing
  - Mice
  - Concentration
  - Functional
  - + Show More
- **GENERAL**
  - Articles

## Slc41a1 knockdown (KD Scientific)

**Bioz Stars**
4.65
Mentions
Impact
Recency


***Knockdown of SLC41A1 magnesium transporter promote... (Stem Cell Res Ther 20)***

*"... catenin , and Dkk1 in control (ctrl ) and Slc41a1 knockdown (**KD** ) mMSCs with normal and high extracellular magnesium concentration ..." (More...)*

Vendor

**Quick View: Protocol Conditions | Articles**

## Slc41a1 knockdown mMSCs (KD Scientific)

**Bioz Stars**
3.68
Mentions
Impact
Recency


***Knockdown of SLC41A1 magnesium transporter promote... (Stem Cell Res Ther 20)***

*"... Mgp gene expression of control (ctrl ) mMSCs and Slc41a1 -knockdown mMSCs (**KD** ) with normal and high extracellular magnesium concentration before ..." (More...)*

Vendor

**Quick View: Protocol Conditions | Articles**

## Rabbit Polyclonal SLC41A1 Antibody (Novus)

**Bioz Stars**
N/A
Mentions
Impact

*"Rabbit Polyclonal SLC41A1 Antibody" (More...)*

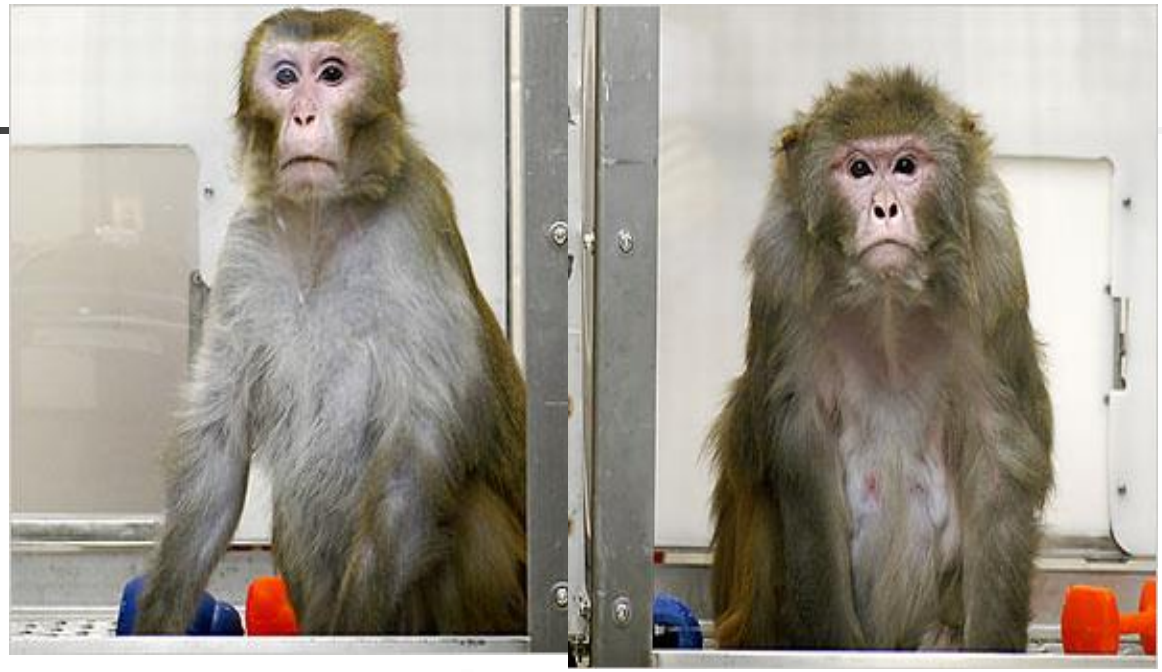
Vendor

**Assays:** Western Blot, Immunohistoche…

# Results

- Tables & Figures
- Viewing results
- Key results in detail
- Problems with results

# Caloric Restriction Delays Disease Onset and Mortality in Rhesus Monkeys



**CALORIE RESTRICTION DIET**

## Canto, 25

Although a senior citizen — the average rhesus monkey lifespan in captivity is 27 — Canto, above, is aging fairly well. Outwardly, he has a nice coat, elastic skin, a smooth gait, upright posture and an energetic demeanor. His bloodwork shows he is as healthy as he looks.

MONKEY MENU

Daily calories

445 885

Monkeys also receive an apple each day.

**NORMAL DIET**

## Owen, 26

He gets more food, but Owen, above, isn't aging as well. His posture has been affected by arthritis. His skin is wrinkled and his hair is falling out. Owen is frail and moves slowly. His bloodwork shows unhealthy levels of glucose and triglycerides.

**Human equivalent** Meals prepared by Mike Linksvayer, 36

**Breakfast** fermented soybeans and garlic

**Lunch** tofu, konyakku and carrots

**Dinner** vegan sausage, kale, tomato sauce and salad

HUMAN MENU

Daily calories

2,000 3,000

Beverages, snacks and desserts not shown. Diet varies according to body type, sex and activity level.

Diet of an average, active human male of 36

Photos by Jim Wilson and Tony Cenicola/The New York Times and Lars Klove for The New York Times

Table 1. Clinical Features, Echocardiographic Measurements, and Biochemical Values During Acute Hepatitis

| | 1st Day | 7th Day |
|---|---|---|
| Systolic arterial pressure (mm Hg) | 121.6 ± 5.6 | 117.5 ± 2.9 |
| Diastolic arterial pressure (mm Hg) | 73.7 ± 2.5 | 72.7 ± 2.1 |
| Heart rates (beats/min) | 76.0 ± 3.9 | 77.3 ± 3.3 |
| AST (IU/L) | 663.7 ± 168.5 | 397.9 ± 166.4 |
| ALT (IU/L) | 1025.8 ± 182.3 | 559.4 ± 156.8* |
| Total bilirubin (mg/dL) | 6.1 ± 1.2 | 4.3 ± 1.0 |
| ALB (g/dL) | 3.7 ± 0.1 | 3.3 ± 0.2 |
| Prothrombin time (second) | 13.0 ± 1.0 | 12.4 ± 0.3 |
| Respiration rate (times/min) | 18.2 ± 0.8 | 18.7 ± 0.7 |

*ALB, albumin; ALT, alanine aminotransferase; AST, aspartate aminotransferase.*

** $P < 0.05$, upon admission vs. the 7th day. Results are expressed as mean ± SEM.*



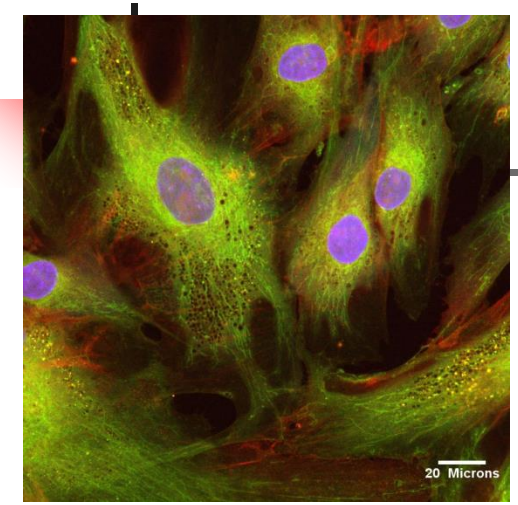
EPS
PDF

TIFF
JPEG
EPS



TIFF
EPS

# 解析度的要求(dpi)

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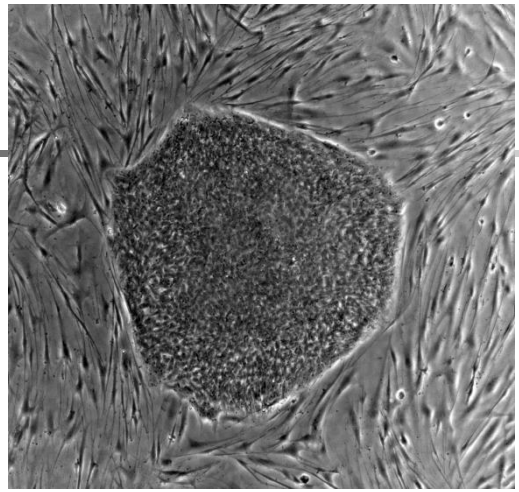

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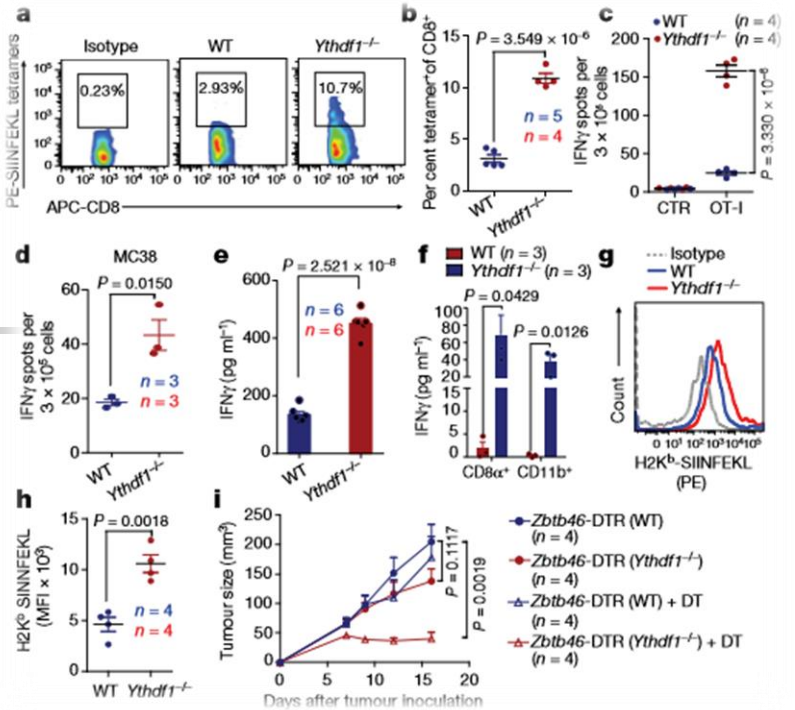



500 dpi

## Figure sizing

- Provide files at about the size they are to be printed

Academic Journals standard figure sizes are 86mm (single column) and 178mm (double column). The full depth of the page is 210mm.

## Line weights

- Lines and strokes should be set between 0.25 and 1 pt
- Do not rasterize or outline these lines if possible

0.25 pt — 1 pt

Single column: 86 mm

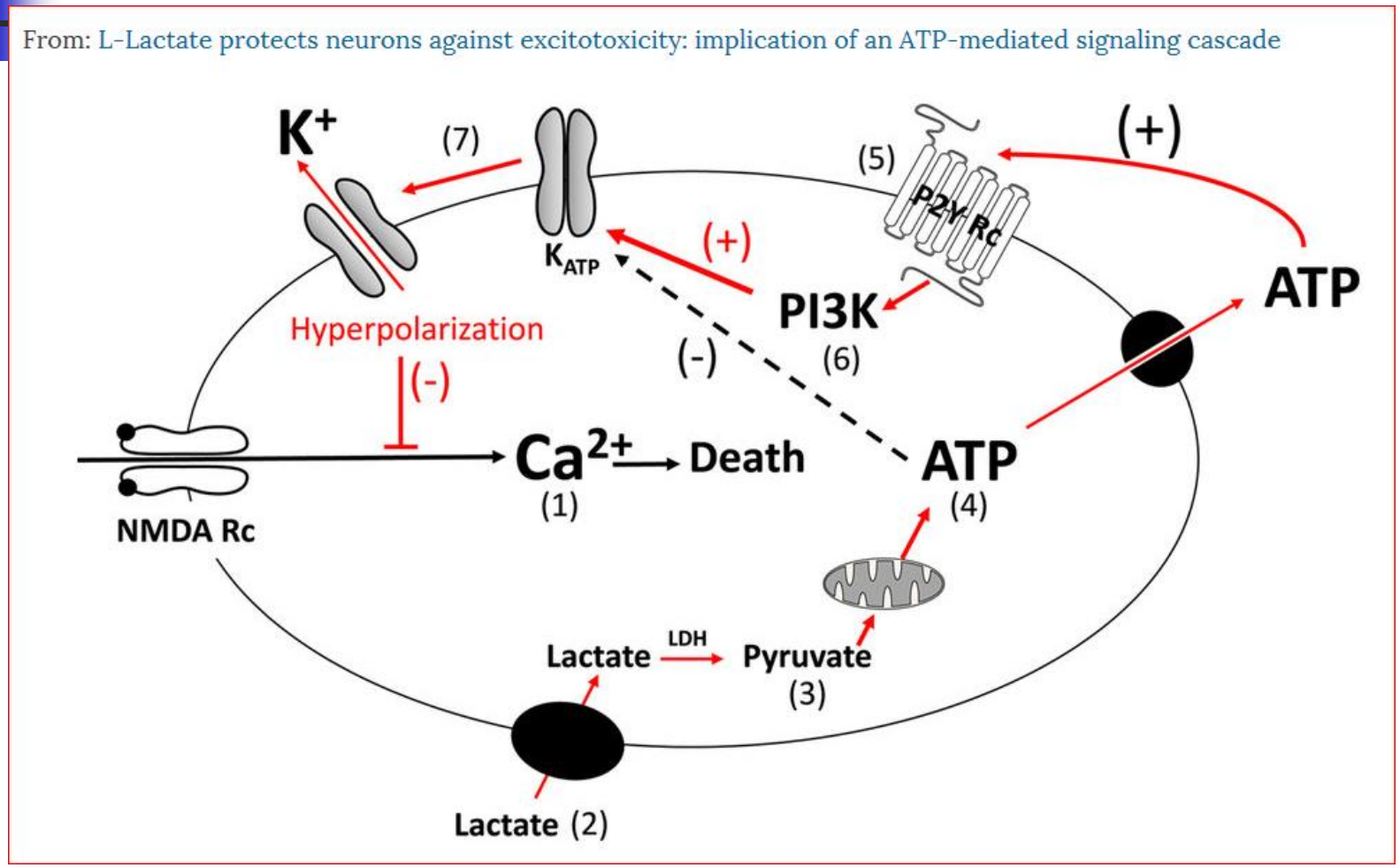
Double column: 178mm

# Discussion


- Summarizing key results
- Mapping (relationship to existing research)
- Achievement/Contribution
- Limitations/current and future works/applications

# Schematic representation of the mechanisms involved in the neuroprotective effect of L-lactate against excitotoxicity

From: L-Lactate protects neurons against excitotoxicity: implication of an ATP-mediated signaling cascade



# 4-1 summarizing key results

- Initial phases of cellular death triggered by an excessive glutamate stimulation are characterized by a massive ionic and water inflows.
- Taking advantage of the QP-DHM technique to monitor transmembrane water fluxes.., we demonstrate that L-Lactate acts as a signaling molecule conferring neuroprotection against excitotoxic insults through well-coordinated mechanisms based on an increase neuronal energy substrates availability.

# 4-2a Mapping (relationship to existing research)

- Until now, the few in vitro studies exploring the neuroprotective properties of L-Lactate have suggested a mechanism of action involving the maintenance of the cellular energy charge (18).
- Indeed, excitotoxicity is classically associated with inhibition of oxidative phosphorylation resulting in a loss of ATP to fuel ion pumps to re-establish the ionic homeostasis (9,10).
- In agreement with that, the involvement of the L-Lactate/Pyruvate pathway and the mitochondrial activity was also observed in this study.
- ….

# 4-2b Mapping (relationship to existing research)

- Another important data reported in the present study indicate the existence of an additional mechanism independent of an energetic role of L-Lactate linked to the formation of ATP ...
- Indeed data indicate that ATP produced by the L-Lactate/Pyruvate neuroenergetic pathway acts as signaling molecule following its release through the ATP channels pannexins, a mode of release in agreement with the biophysical properties of the pannexins known to be a mechanosensitive conduits for ATP sensitive to swelling (46,61).
- Interestingly, ATP released by neurons acts in autocrine/paracrine manner triggering an apyrase-sensitive purinergic signaling (Fig. 5).
- ...

# 4-3 Achievement/contribution

- Our study provides new evidences that L-Lactate can also act as a signaling molecule in pathological contexts such as excitotoxic processes.
- Considering that astrocytes are the main producers of L-Lactate in brain, our observations point to astrocytes as pivotal cellular elements for neuronal protection against excitotoxicity.
- …
- Therefore, during an excitotoxic situation, the pathological release of glutamate from neurons would strongly activate L-Lactate production and the release from astrocytes which, in turn, would provide neuroprotection by opening KATP channels, through the P2Y2/PI3K pathway.

# 4-4 Limitations/current and future work/applications

- In this general context, it would be of interest to determine whether differences in terms of levels of expression and activation of the key elements involved in the neuroprotective cascade induced by L-Lactate as described in this study..
- … could explain (or be involved in) the differential physiological response of neurons to L-Lactate.
- …
- The present results indicate that L-Lactate can be an attractive candidate as a neuroprotective compound, providing the opportunity to develop neuroprotective strategies aimed at increasing the production of L-Lactate by astrocytes.

Title

Abstract

Introduction
(1) (2) (3) (4)

Methods
(1,2,3,4)

Results

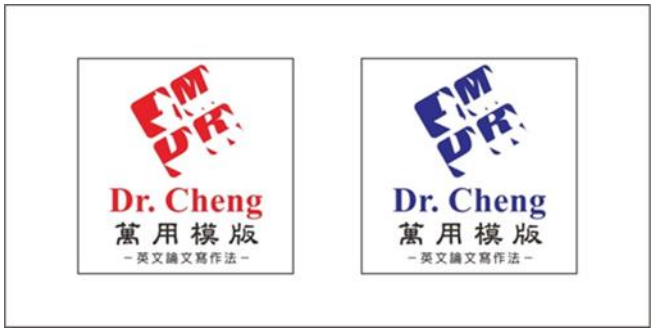
Discussion
(1) (2) (3) (4)

Point

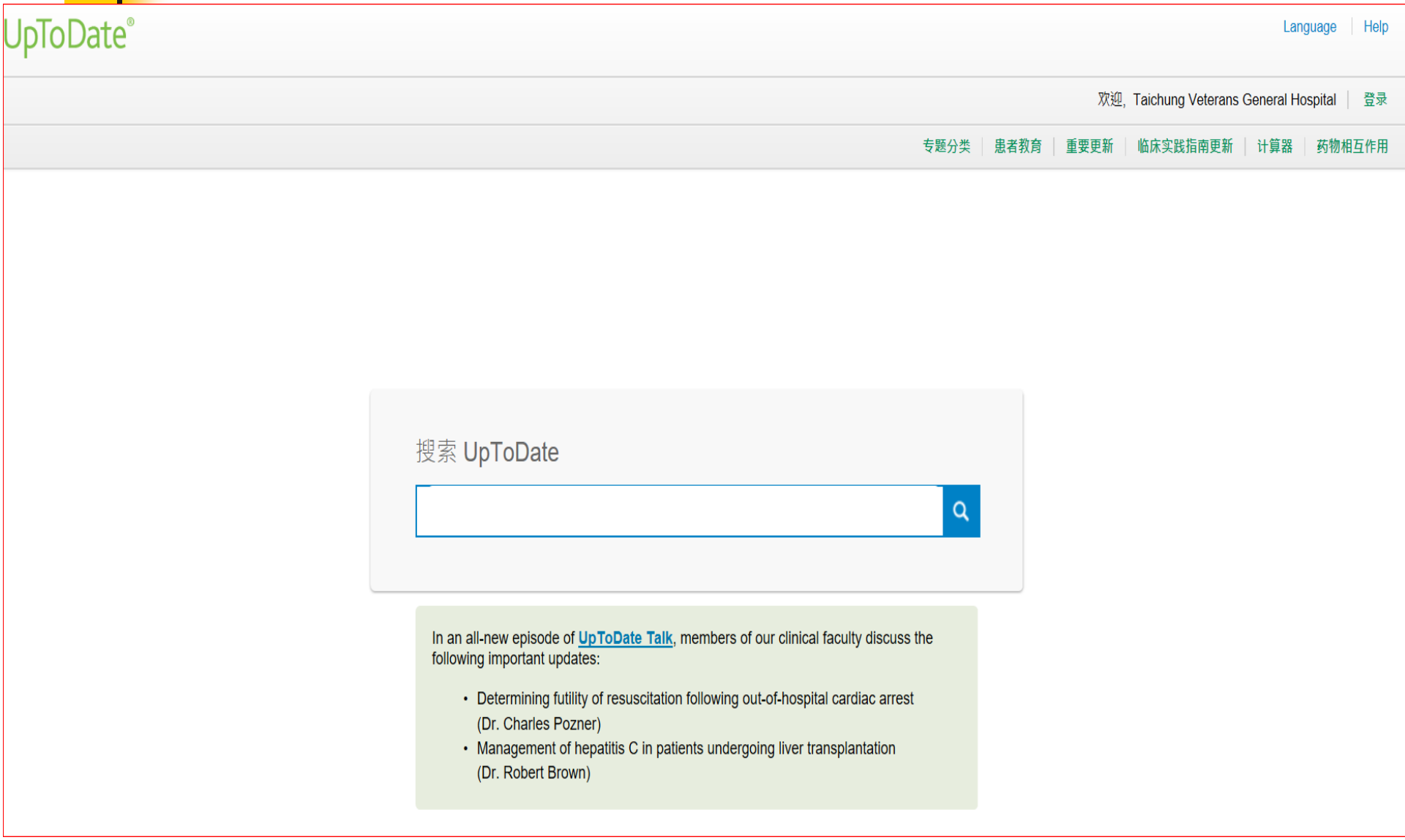
Problems;
Gap

Core;
Key results

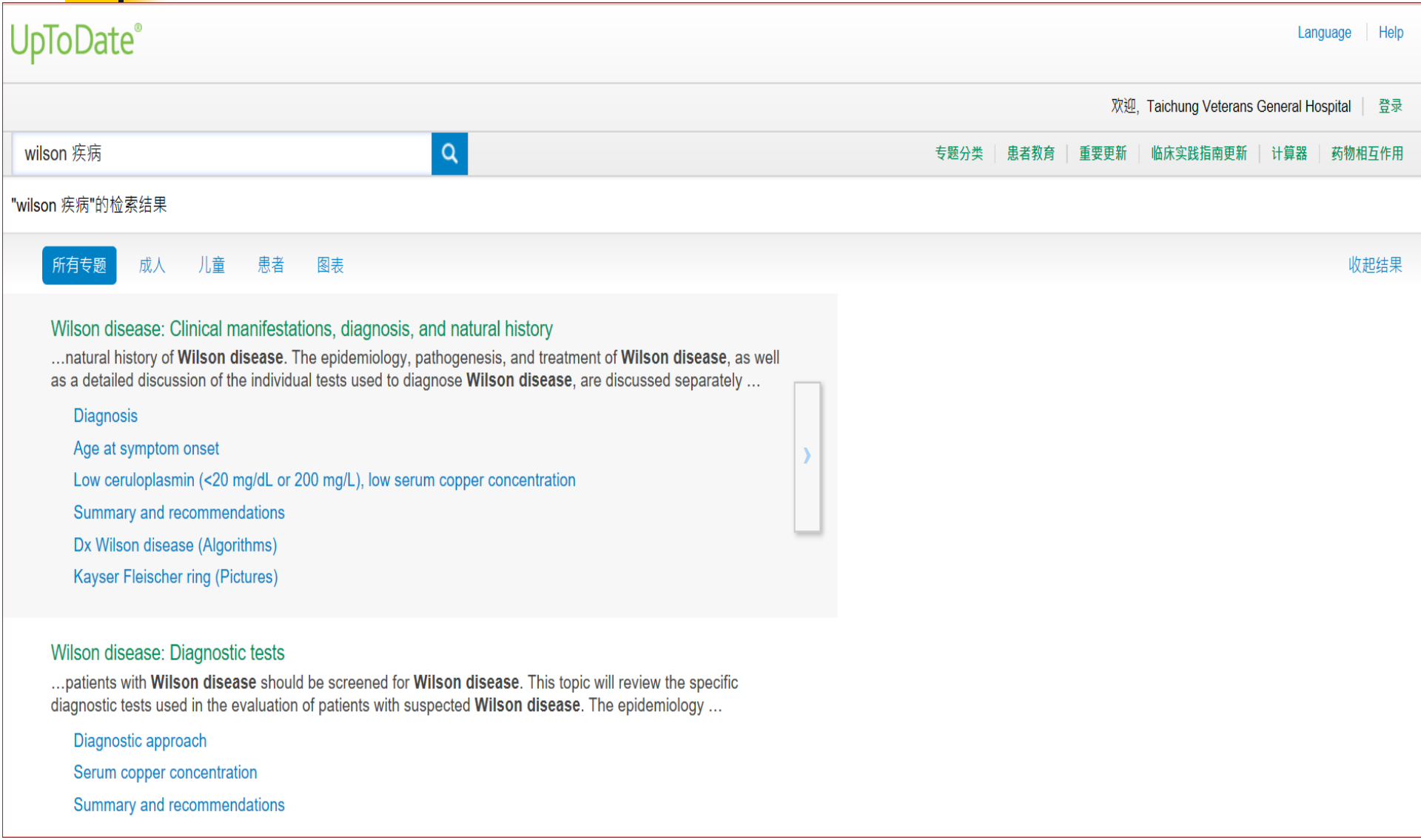
Heart



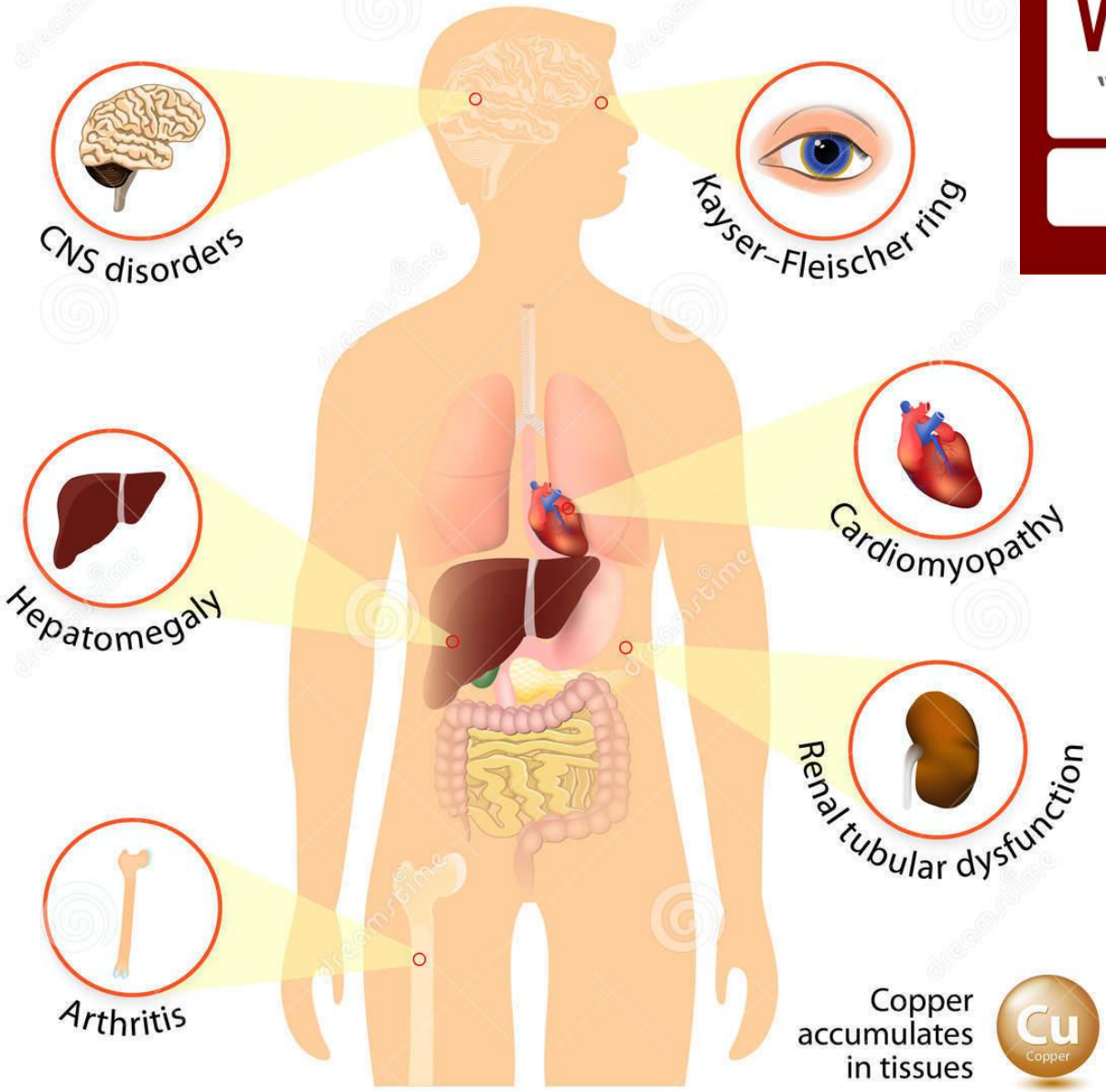
# Uptodate (https://www.uptodate.com/)



# Wilson's disease



# WILSON'S DISEASE




# Wilson's Disease

"Hepatolenticular Degeneration"

*A rare inherited disorder...*

World Journal of Hepatology

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# Wilson's disease: A review of what we ha

Kryssia Isabel Rodriguez-Castro, Francisco Javier Hevia-Urrutia, Giacomo Ca

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## Abstract

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Key words: Wilson's
agents; Penicillamin
Liver transplantation



Core tip: A centur
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and the use of pha
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Prompt recognition
life-saving.

J Nat Sci Biol Med. 2015 Jan-Jun; 6(1): 248–252. PMCID: PMC4367052
doi: 10.4103/0976-9668.149210

## Wilson's disease: A Clinical autopsy case report with review of literature

Kalyani Raju, Gayathri Nagaraj Bangalore, Suresh Nagaraj Thuruvekere, and Venkatarathnamma Narayanappa Pathavanalli[1]

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### Abstract

Go to:

Wilson's disease is an autosomal recessive disease resulting in defective copper metabolism, which is usually seen in young adults, predominantly affecting liver and brain. Although it is not uncommon in India, variation in epidemiology, clinical presentation and course are reported. However, community-based incidence and prevalence rates are not available in India and incidences are limited to hospital based reports. Most often, the diagnosis is delayed. We present a clinical autopsy case in a 39 year-old female who had presented with clinical symptoms at 18 years of age. The duration of illness was 21 years. Patient's parent had consanguineous marriage and the younger sibling had died at 5 years of age with similar complaints.

**Keywords:** Clinical autopsy, Wilson's disease, autopsy, autosomal recessive disease

### INTRODUCTION

Go to:

Wilson's disease (WD) is an autosomal recessive disease involving brain and liver secondary to altered copper metabolism. About 47% and 55% of cases reported have positive family history and consanguinity, respectively.[1] The symptoms are nonspecific and the disease may present as hepatic disease or progressive neurological disorder (hepatic dysfunction being less apparent or occasionally absent) or as psychiatric illness with liver disease. The liver disease may be asymptomatic, with only biochemical abnormalities of cirrhosis.[1,2] A patient (5-40 years old) presenting with liver disease, with a decrease in

European Review for Medical and Pharmacological Sciences 2016; 20: 1845-1851

## Impairment of time-based prospective memory in patients with Wilson's disease

T. DONG[1,2], J. QIU[3], H.-D. CHENG[4], W.-W. DONG[2], P. HUANG[2], C.-S. XU[5], K. WANG[1], W.-M. YANG[2]

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**Abstract. – OBJECTIVE:** The aim of this study was to investigate the effect of basal ganglia lesion of Wilson's disease (WD) patients on event-based prospective memory (EBPM) and time-based prospective memory (TBPM).

**PATIENTS AND METHODS:** A total of 30 WD patients and 30 age and education level matched healthy controls were included. EBPM (an action whenever particular words were presented) and TBPM (an action at certain times) were performed to test the involvement of the prospective memory in WD.

**RESULTS:** A significant difference was found in the performance of TBPM (2.9±1.1 vs. 5.8±0.4, $p<0.05$), but not EBPM (5.4±0.7 vs. 5.5±0.7, $p>0.05$) in patients with WD compared with the healthy controls.

**CONCLUSIONS:** Our results demonstrated that basal ganglia are involved in the prospective memory in patients with WD.

*Key Words:*
Wilson's disease, Event-based prospective memory, Time-based prospective memory, Prospective memory.

### Introduction

Wilson's disease (WD), a rare autosomal recessive disorder of copper metabolism, is characterized by copper accumulation in the liver, brain and kidney[1]. Typically, WD begins with a presymptomatic period, during which copper accumulation in the liver causes subclinical hepatitis, and progresses to liver cirrhosis and development of neuropsychiatricsymptoms[2,3]. With neuropsychiatric symptoms, WD patients often manifest behavior or emotional disorders (showing impulsive, instinctive behaviors, or depression), and mild cognitive deficit[4,5].

Memory, an important cognitive function, refers to the mental process in which individual experience is accumulated and preserved; it plays an important role in the entire mental activity. Currently, two main types – retrospective memory (RM) and prospective memory (PM) – are used to assess memory in a quantitative manner[6]. RM refers to the memory of things or actions that have occurred in the past, and PM refers to the memory of completing a certain activity at the appropriate time in the future, which can be further divided into time-based prospective memory (TBPM) and event-based prospective memory (EBPM)[7,8]. TBPM refers to the memory of the execution of an action at a target time, such as remembering to call a friend in 1 h. EBPM refers to the memory of performing an action when a specific target event occurs, such as remembering to buy some fruits when passing by a fruit stand. PM has important practical significance for the elderly for maintaining the normal activities of daily life, such as taking their medication at a specific time.

Recent studies suggested that EBPM and TBPM tasks may be mediated by different neural networks. By using positron emission tomography technology, Okuda et al[9] showed that the

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## Phenotype-Genotype Correlation in Wilson Disease in a Large Lebanese Family: Association of c.2299insC with Hepatic and of p. Ala1003Thr with Neurologic Phenotype

Julnar Usta[1], Antonios Wehbeh[2], Khaled Rida[1], Omar El-Rifai[1], Theresa Alicia Estiphan[2], Tamar Majarian[1], Kassem Barada[3]*

1 Department of Biochemistry and Molecular Genetics; Faculty of Medicine, American University of Beirut, Beirut, Lebanon, 2 Faculty of Medicine, American University of Beirut Medical Center, Beirut, Lebanon, 3 Division of Gastroenterology, Department of Internal Medicine, American University of Beirut Medical Center, Faculty of Medicine, Beirut, Lebanon

**Abstract**

Genotype phenotype correlations in Wilson disease (WD) are best established in homozygous patients or in compound heterozygous patients carrying the same set of mutations. We determined the clinical phenotype of patients with WD carrying the c.2298_2299insC in Exon 8 (c.2299insC) or the p. Ala1003Thr missense substitution in Exon 13 mutations in the homozygous or compound heterozygous state. We investigated 76 members of a single large Lebanese family. Their genotypes were determined, and clinical assessments were carried out for affected subjects. We also performed a literature search retrieving the phenotypes of patients carrying the same mutations of our patients in the homozygous or compound heterozygous state. There were 7 consanguineous marriages in this family and the prevalence of WD was 8.9% and of carriers of *ATP7B* mutation 44.7%. WD was confirmed in 9 out of 76 subjects. All 9 had the c.2299insC mutation, 3 homozygous and 4-compound heterozygous with p. Ala1003Thr. Six of our patients had hepatic, 2 had neurologic and 1 had asymptomatic phenotype. Based on our data and a literature review, clear phenotypes were reported for 38 patients worldwide carrying the c.2299insC mutation. About 53% of those have hepatic and 29% have neurologic phenotype. Furthermore, there were 10 compound heterozygous patients carrying the p. Ala1003Thr mutation. Among those, 80% having c.2299insC as the second mutation had hepatic phenotype, and all others had neurologic phenotype. We hereby report an association between the c.2299insC mutation and hepatic phenotype and between the p. Ala1003Thr mutation and neurologic phenotype.

**Citation:** Usta J, Wehbeh A, Rida K, El-Rifai O, Estiphan TA, et al. (2014) Phenotype-Genotype Correlation in Wilson Disease in a Large Lebanese Family: Association of c.2299insC with Hepatic and of p. Ala1003Thr with Neurologic Phenotype. PLoS ONE 9(11): e109727. doi:10.1371/journal.pone.0109727

**Editor:** Oleg Y. Dmitriev, University of Saskatchewan, Canada

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**Data Availability:** The authors confirm that all data underlying the findings are fully available without restriction. All relevant data are within the paper. Relevant clinical data have been disclosed in Tables 1 and 2 and in the text using subjects codes (5n).

**Funding:** The authors thank The Medical Practice Plan of AUB-MC and the University Research Board of the American University of Beirut for supporting the study by research grants to J. Usta. The funders had no role in study design, data collection and analysis, decision to publish or preparation of the manuscript.

**Competing Interests:** The authors have declared that no competing interests exist.

* Email: kbd32@aub.edu.lb

### Introduction

Wilson disease (WD; MIM# 277900) is an autosomal recessive, copper transport disorder characterized by extensive phenotypic diversity [1,2]. Patients may present at any age with hepatic, neurologic, or mixed symptoms. Yet some may be asymptomatic [3]. WD is due to a defective *ATP7B* gene (OMIM*606882; Ref seq accession #: NM_000053.3); that is located on chromosome 13 (Gene map locus: 13q 14.3-q21.1) that encodes a copper

phenotype with one mutant allele. Furthermore occupational exposure to copper has been shown to cause genomic alterations and DNA damage [7]. This in combination with epigenetic modulators and environmental factors may play a role in the phenotypic heterogeneity of WD patients [8]. These difficulties may be partially overcome by studying WD in homozygous patients [9].

Specific mutations in the ATP7B gene are more frequent in

Review Article

## Currently Clinical Views on Genetics of Wilson's Disease

Chen Chen, Bo Shen, Jia-Jia Xiao, Rong Wu, Sarah Jane Duff Canning , Xiao-Ping Wang
Department of Neurology, Shanghai General Hospital, Shanghai Jiao-Tong University, Shanghai 200080, China

**Abstract**

**Objective:** The objective of this study was to review the research on clinical genetics of Wilson's disease (WD).
**Data Sources:** We searched documents from PubMed and Wanfang databases both in English and Chinese up to 2014 using the keywords WD in combination with genetic, *ATP7B* gene, gene mutation, genotype, phenotype.
**Study Selection:** Publications about the *ATP7B* gene and protein function associated with clinical features were selected.
**Results:** Wilson's disease, also named hepatolenticular degeneration, is an autosomal recessive genetic disorder characterized by abnormal copper metabolism caused by mutations to the copper-transporting gene *ATP7B*. Decreased biliary copper excretion and reduced incorporation of copper into apoceruloplasmin caused by defunctionalization of ATP7B protein lead to accumulation of copper in many tissues and organs, including liver, brain, and cornea, finally resulting in liver disease and extrapyramidal symptoms. It is the most common genetic neurological disorder in the onset of adolescents, second to muscular dystrophy in China. Early diagnosis and medical therapy are of great significance for improving the prognosis of WD patients. However, diagnosis of this disease is usually difficult because of its complicated phenotypes. In the last 10 years, an increasing number of clinical studies have used molecular genetics techniques. Improved diagnosis and prediction of the progression of this disease at the molecular level will aid in the development of more individualized and effective interventions, which is a key to transition from molecular genetic research to the clinical study.
**Conclusions:** Clinical genetics studies are necessary to understand the mechanism underlying WD at the molecular level from the genotype to the phenotype. Clinical genetics research benefits newly emerging medical treatments including stem cell transplantation and gene therapy for WD patients.

**Key words:** *ATP7B* Gene; Clinic; Gene Mutation; Genetic; Hepatolenticular Degeneration; Phenotype; Wilson's Disease

### Introduction

Wilson's disease (WD), also named hepatolenticular degeneration, is an autosomal recessive genetic disorder caused by defects of *ATP7B* gene.[1] This disease occurs sporadically all over the world. It is found in individuals aged 3–80 years, but mainly in children and adolescents. Males have a slightly higher risk of developing WD than females, possibly because of differences in estrogen level and iron metabolism.[1] Worldwide prevalence of WD is around 1:30,000,[2] carrier rate is about 0.011, and the gene frequency is about 0.56. The prevalence of WD has been re-evaluated in a recent clinical study.[3] In this study, 6,384 individuals were randomly selected and the full-length *ATP7B* gene (1008 individuals, 1 k data) and exons 8, 14, 18 (5376 individuals, 5 k data) were tested, respectively, by gene sequencing. The frequency of the 1 k data is 0.040–0.056 and for the 5 k data, the frequency is 0.0044–0.0057. The result indicates that theoretically the prevalence of WD should be higher than 1:30,000. The difference between the number of diagnosed cases and the theoretical value may be due to declined penetrance and diagnostic limitations. The clinical presentations of WD are highly varied, mainly consisting of hepatic and neurological symptoms. Hepatic symptoms include acute and chronic liver diseases, for example, fulminant hepatic failure (also named as abdominal Wilsonian disease) and liver cirrhosis. Neurological symptoms mainly include extrapyramidal symptoms and neuropsychiatric symptoms. The extrapyramidal symptoms are dystonia and tremor while the psychiatric symptoms of WD are often accompanied by cognitive and mood disorder.[4] Hemolytic anemia and skeletal muscle disorder are also presented in a few patients.

Once diagnosed with WD, the patient should have a low-copper diet and receive anticopper treatment for the rest of their life. Western medicines for WD patients are d-penicillamine, sodium dimercaptosuccinate, dimercaptosuccinic acid, trientine, zinc preparation, tetrathiomolybdate, etc.[5] Traditional Chinese medicine has also shown to be associated with significant positive



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E-Mail: wangxp@sustc.edu

# first sentences of 5 references

- Wilson's disease (WD), a rare autosomal recessive disorder of copper metabolism, is characterized by copper accumulation in the liver, brain and kidney.
- Initially described by Kinnear Wilson[1] in 1912, Wilson's disease (WD), is the clinical condition resulting from mutations in the chromosome 13q14 in the region coding for the protein product ATP7B, and occurs in a sporadic fashion as well as inherited as an autosomal recessive disease.
- Wilson's disease (WD), also named hepatolenticular degeneration, is an autosomal recessive genetic disorder caused by defects of ATP7B gene.
- Wilson's disease (WD) is an autosomal recessive disease involving brain and liver secondary to altered copper metabolism.
- Wilson disease (WD; MIM# 277900) is an autosomal recessive, copper transport disorder characterized by extensive phenotypic diversity.

# 翻譯五篇參考文獻的第一句

- 威爾森氏病（WD），銅代謝的一種罕見的常染色體隱性遺傳疾病，其特點是在肝，腦和腎的銅積累。
- 最初由金尼爾威爾遜[1]1912年描述的，威爾森氏病（WD），是由在該蛋白質產物ATP7B的編碼區的染色體13q14突變引起的臨床病症，並且發生在零星的方式以及繼承作為常染色體隱性遺傳病。
- Wilson病（WD），又稱肝變性，是由基因ATP7B缺陷為常染色體隱性遺傳疾病。
- Wilson病（WD）是一個涉及大腦和肝臟繼發改變銅代謝常染色體隱性遺傳病。
- Wilson病（WD; MIM＃277900）是一種常染色體隱性遺傳，銅傳輸障礙的特點是廣泛的遺傳多樣性。

# 組合第一句改寫的主題句

- 威爾遜疾病是由許多的ATP7B基因的突變，造成銅的膽汁排泄的異常的一種常染色體隱性遺傳疾病。
- 威爾森氏病（WD）是銅代謝的一種罕見的染色體隱性遺傳疾病，其特點基因ATP7B的編碼區的染色體13q14突變所引起的臨床病症。
- 或其他....

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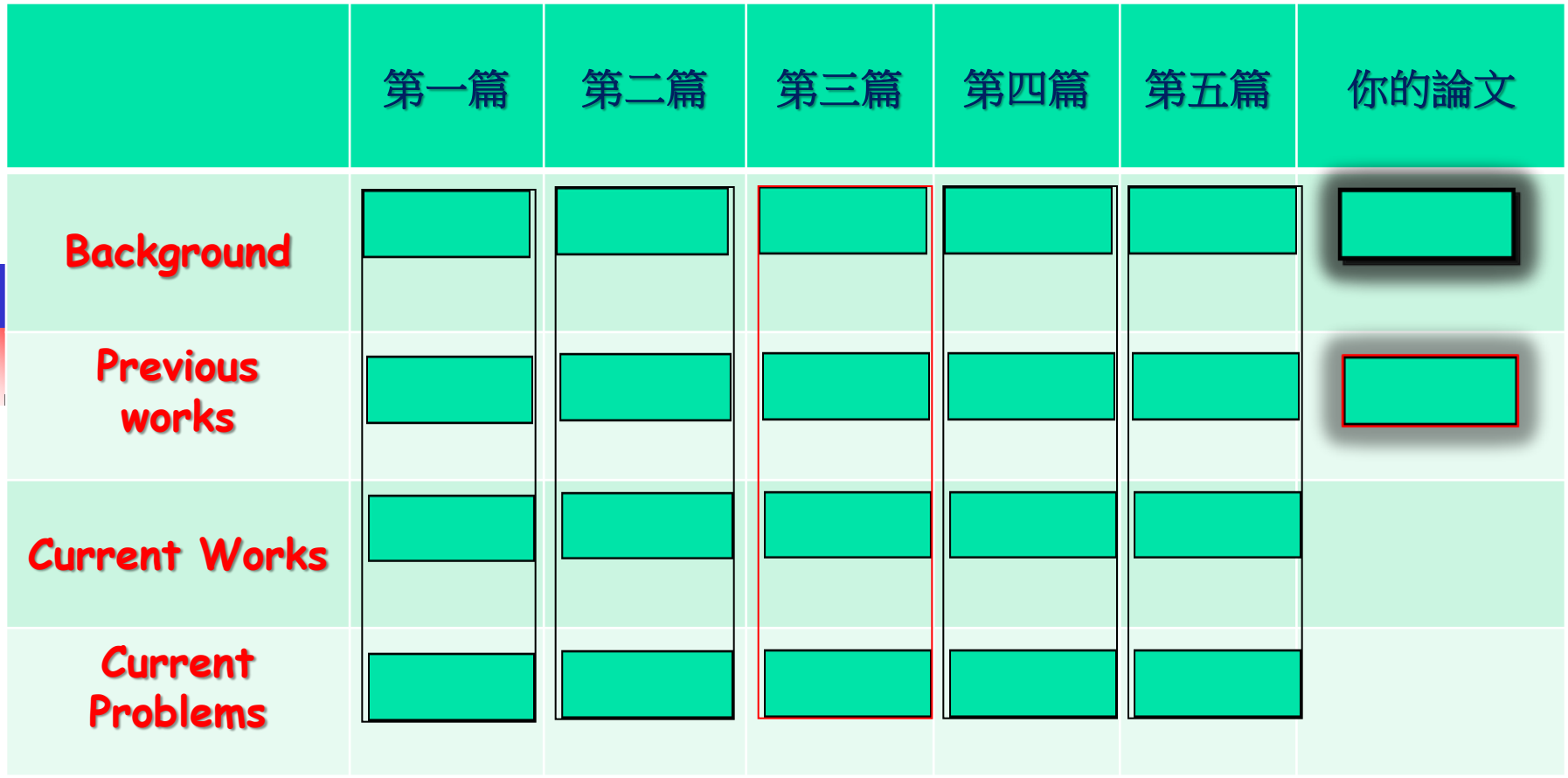
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Previous works
Current Works
Current Problems

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# 第三段如何下筆

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| Current Focus | | | | | | |

# 第一、二、三、四段如何下筆

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| Facts/Problems | | | | | | |
| Mini Review | | | | | | |
| Gap | | | | | | |
| The present work | | | | | | |

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a systematic review was undertaken exploring the experiences of hypoglycaemia in
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community-dwelling people with diabetes mellitus.
Objective This review aimed to synthesise evidence on the
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TRPM7 regulates sinoatrial node fibrosis in sick sinus syndrome rat by AngII/ Smads signaling pathway
Objective: To monitor the changes in the Ang II/TRPM7/Smads signaling pathway in sinoatrial node (SAN) tissue and its correlation with collagen synthesis; to study the role of TRPM7/Smads signaling pathway on Ang II-induced collagen synthesis in cardiac fibroblasts (CFs) and investigate the possible regulatory mechanism of TRPM7/Smads on the fibrosis of SAN in rat with sick sinus syndrome (SSS).

Methods: In the in vivo experiments of this study used 20% sodium hydroxide pinpoint pressing permeation method to establish SSS rat model. Forty-eight 12-week male Sprague Dawley (SD) rats were divided into six groups: normal control (control, n=8); sham operation (sham, n=8); postoperative 1-week SSS (SSS1, n=8); postoperative 2-week SSS (SSS2, n=8); postoperative 3-week SSS (SSS3, n=8); and postoperative 4-week SSS (SSS4, n=8) groups. Distribution and content of collagen in the myocardium of all rats was assessed using PASM-Masson staining. Ang II, Col I, and Col III levels in Serum and SAN tissue of all rats were determined by ELISA. TRPM7 levels in SAN tissue in the rats were determined by immunohistochemistry. TRPM7 mRNA expression was assessed by real-time PCR (RT-PCR); TRPM7, Smad2/p-Smad2 protein levels were determined by Western blot. In the in vitro experiments, the tissue explant culture method was used to culture CFs from rat SAN tissues, followed by immunocytochemistry and immunofluorescence staining to identify the CFs. Supernatant collagen levels in CFs were measured by ELISA, and TRPM7 mRNA expression in CFs was determined by RT-PCR. TRPM7 and Smad2/p-Smad2 protein levels were determined by Western blot. After transfecting small interfering RNA (siRNA) to downregulate TRPM7 expression and transfecting plasmid DNA vector to overexpress TRPM7, the effect of TRPM7/Smad2 signaling protein on Ang II-induced CFs collagen synthesis was monitored.

Results: In the in vivo experiments, Ang II levels in serum and Ang II, Col I, and Col III expression in SAN tissues of the SSS1 group were significantly higher than in the sham group (P<0.05), and Ang II levels in serum and SAN tissues were peaked in SSS rats at the third week after the operation. No significant differences in Ang II, Col I, or Col III levels in serum or SAN tissues were found between the sham and control groups (P>0.05); and no significant differences Col I and Col III levels in serum were found among different groups (P>0.05). TRPM7 immunohistochemistry showed significantly higher levels of TRPM7 expression in SAN tissues in SSS1 group than in Sham group (P<0.05), and TRPM7 expression in SAN tissues were further increased in SSS2, SSS3, and SSS4 groups (P<0.01). RT-PCR showed TRPM7 mRNA expression in SAN tissues of the SSS1 group were significantly higher than in the sham group (P<0.01), and TRPM7 mRNA expression in SAN tissues were still higher in SSS2, SSS3, and SSS4 groups than in sham group (P<0.01). Western blot analysis showed there were significantly more TRPM7 and p-Smad2 expression in SAN tissues of the SSS1 group than in control group (P<0.01), and there was still more TRPM7 and p-Smad2 expression in SAN tissues in SSS2, SSS3, and SSS4 groups (P<0.01). No significant difference in TRPM7 expression in SAN tissues was found between the sham and control groups (P>0.05). In the in vitro experiment showed that Ang II promoted CFs collagen synthesis in dose-dependent manner. It also promoted TRPM7 and p-Smad2 expression in CFs. Reduced TRPM7 gene expression inhibited Ang II-induced p-Smad2 protein expression and reduced Ang II-induced collagen synthesis in CFs. Upregulated TRPM7 expression elevated Ang II-induced p-Smad2 protein expression and promoted Ang II-induced collagen synthesis in CFs.
Conclusions: SSS promoted fibrosis of SAN tissues and enhanced Ang II and TRPM7/Smad2 signal protein expression in SAN tissues. Ang II-induced CFs collagen synthesis promoted TRPM7/Smad2 signal protein expression. Fibrosis of SAN tissues in SSS rats might be taken place through the TRPM7/Smad2 signaling pathway.
Keywords: Sick sinus syndrome; Sinoatrial node; angiotensin II; collagen type I, collagen type III, TRPM7, Smad2, p-Smad2.

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English | Chinese (Traditional) | 翻譯

TRPM7通過AngII / Smads信號通路調節病竇綜合徵大鼠的竇房結纖維化
目的：監測竇房結（SAN）組織中Ang II / TRPM7 / Smads信號通路的變化及其與膠原合成的相關性;研究TRPM7 / Smads信號通路對心臟成纖維細胞（CFs）中Ang II誘導的膠原合成的作用，並研究TRPM7 / Smads對病態竇綜合徵（SSS）大鼠SAN纖維化的可能調節機制。

方法：本研究體內實驗採用20%氫氧化鈉精確按壓滲透法建立SSS大鼠模型。將48只12週齡的Sprague Dawley（SD）大鼠分為6組：正常對照組（對照組，n = 8）;假手術（假手術，n = 8）;術後1週SSS（SSS1，n = 8）;術後2週SSS（SSS2，n = 8）;術後3週SSS（SSS3，n = 8）;和術後4週SSS（SSS4，n = 8）組。使用PASM-Masson染色評估所有大鼠心肌中膠原蛋白的分佈和含量。通過ELISA測定所有大鼠的血清和SAN組織中的Ang II，Col I和Col III水平。通過免疫組織化學測定大鼠SAN組織中的TRPM7水平。通過實時定量PCR（RT-PCR）評價TRPM7 mRNA表達;通過蛋白質印跡測定TRPM7，Smad2 / p-Smad2蛋白水平。在體外實驗中，組織外植體培養法用於培養來自大鼠SAN組織的CF，然後進行免疫細胞化學和免疫熒光染色以鑑定CF。通過ELISA測量CF中的上清膠原水平，並通過RT-PCR測定CF中的TRPM7 mRNA表達。通過Western印跡測定TRPM7和Smad2 / p-Smad2蛋白水平。在轉染小干擾RNA（siRNA）以下調TRPM7表達並轉染質粒DNA載體以過表達TRPM7後，監測TRPM7 / Smad2信號蛋白對Ang II誘導的CFs膠原合成的影響。

結果：在體內實驗中，SSS1組SAN組織血清Ang II，Col I，Col III表達水平均顯著高於假手術組（P <0.05），Ang II水平升高血清和SAN組織在手術後第三週在SSS大鼠中達到峰值。假手術組和對照組血清或SAN組織Ang II，Col I或Col III水平無明顯差異（P> 0.05）。差異無統計學意義（P> 0.05）。 TRPM7免疫組化顯示SSS1組SAN組織TRPM7表達水平明顯高於Sham組（P <0.05），SSS2，SSS3和SSS4組SAN組織TRPM7表達進一步增加（P <0.01）。 RT-PCR顯示SSS1組SAN組織TRPM7 mRNA表達明顯高於假手術組（P <0.01），SSS2，SSS3，SSS4組TRPM7 mRNA表達明顯高於假手術組（P <0.01）。蛋白質印跡分析顯示SSS1組SAN組織TRPM7和p-Smad2表達明顯高於對照組（P <0.01），SSS2，SSS3組織中TRPM7和p-Smad2表達仍較多，和SSS4組（P <0.01）。假手術組和對照組之間TRPM7表達無明顯差異（P> 0.05）。在體外實驗表明，Ang II以劑量依賴的方式促進CFs膠原合成。它還促進了CFs中的TRPM7和p-Smad2表達。減少的TRPM7基因表達抑制Ang II誘導的p-Smad2蛋白表達並減少CF中的Ang II誘導的膠原合成。上調TRPM7表達升高血管緊張素II誘導的p-Smad2蛋白表達，並促進血管緊張素II誘導的CF中的膠原合成。
結論：SSS促進SAN組織纖維化，增強SAN組織中Ang II和TRPM7 / Smad2信號蛋白表達。 Ang II誘導的CFs膠原合成促進TRPM7 / Smad2信號蛋白表達。 SSS大鼠的SAN組織纖維化可能通過TRPM7 / Smad2信號通路發生。
關鍵詞：病態竇綜合徵;中間節點血管緊張素II I型膠原，III型膠原，TRPM7，Smad2，p-Smad2。

介紹
病態竇綜合徵（SSS）是一種常見的臨床心律失常，表現為心動過緩，猝停，竇房阻塞或心動過緩 - 心動過緩綜合徵，可能造成很大的傷害[1,2]。在竇房結（SAN）及其周圍組織中的異常脈衝形成和脈衝傳導被認為是SSS的主要病理生理變化[3]，機制不清楚。 SAN解剖形態的研究表明，SAN中的纖維組織在維持正常起搏和傳導SAN中起重要作用。 SAN組織的纖維化影響SAN動作電位和傳導的產生，導致SSS [4-6]。心臟結構的機制研究



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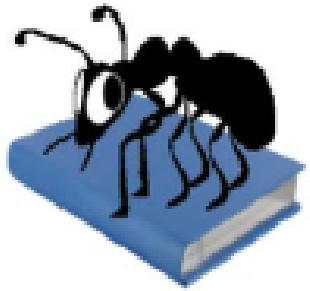

# AntConc (Windows, Macintosh OS X, and Linux)

Build 3.2.4 http://www.laurenceanthony.net/software.html

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## Introduction

*AntConc* is a freeware, multiplatform tool for carrying out corpus linguistics research and data-driven learning. It runs on any computer running Microsoft Windows (tested on Win 98/Me/2000/NT, XP, Vista, Win 7), Macintosh OS X (tested on 10.4.x, 10.5.x, 10.6.x), and Linux (tested on Ubuntu 10). It is developed in Perl using ActiveState's PerlApp compiler to generate executables for the different operating systems.

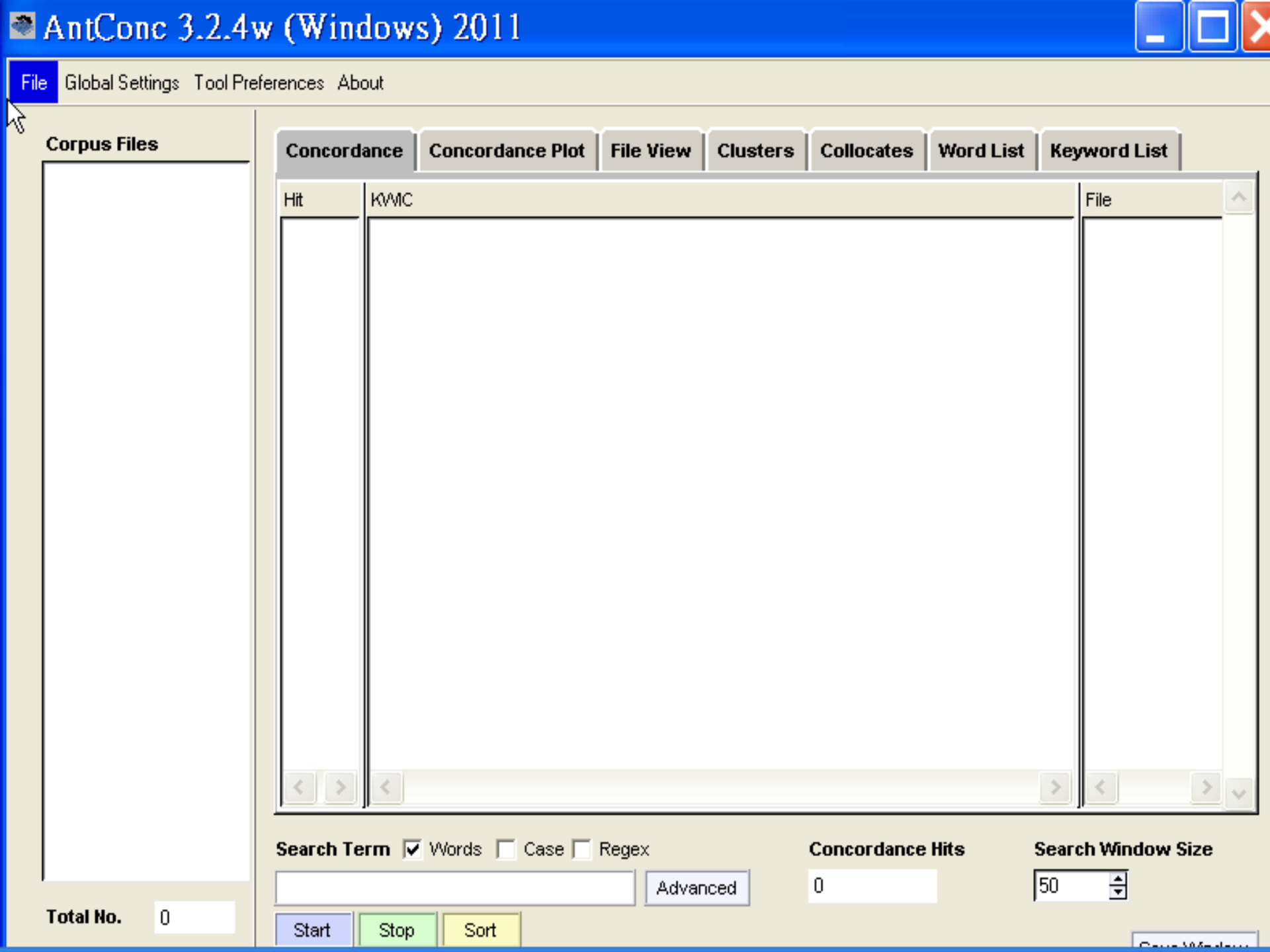
## Installation

### Windows

On Windows systems, simply double click the *AntConc* icon and this will launch the program. No installation is necessary.

### Macintosh OS X

On Macintosh systems, first install and launch X11. X11 is a graphical toolkit that is available on the disks included with the computer or via the Apple website. Next, double click double click the *AntConc* icon and this will launch the program. No installation is necessary.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| 123 | ne-Delalande B. Loss of MAGT1 abrogates the Mg2+ flux | Loss of MAGT |
| 124 | of the Mg2+ transporter MAGT1 as the cause of this d | Loss of MAGT |
| 125 | d an Mg2+ flux through MAGT1 that was important for | Loss of MAGT |
| 126 | we ruled out large MAGT1 in T cell signaling + | Loss of MAGT |
| 127 | nt in both patients in MAGT1, a gene coding for a M | Loss of MAGT |
| 128 | on of the exon 7 of MAGT1 and induced the splicing | Loss of MAGT |
| 129 | led to a decrease of MAGT1 mRNA of .80 % by nonse | Loss of MAGT |
| 130 | leading to the loss of MAGT1 protein expression in th | Loss of MAGT |
| 131 | sion in the patients. MAGT1 deficiency impairs TCR-in | Loss of MAGT |
| 132 | about the function of MAGT1 in lymphocytes prior to | Loss of MAGT |
| 133 | AL. 293T) showed that MAGT1 induces a highly selecti | Loss of MAGT |
| 134 | ich was consistent with MAGT1 as a Mg2+ transporter i | Loss of MAGT |
| 135 | defect and the loss of MAGT1 in the patients, we loo | Loss of MAGT |
| 136 | h ectopic expression of MAGT1, restoring the TCR-induce | Loss of MAGT |
| 137 | results established that MAGT1 mediated the Mg2+ influx | Loss of MAGT |
| 138 | cell activation. Thus, MAGT1 deficiency was the proba | Loss of MAGT |
| 139 | of PLC 1 activation in MAGT1-deficient patients Sinc | Loss of MAGT |
| 140 | Mg2+ influx mediated by MAGT1 after TCR stimulation wa | Loss of MAGT |
| 141 | d for by overexpressing MAGT1 in the B cell line [21 | Loss of MAGT |

**Search Term** ☑ Words ☐ Case ☐ Regex

MagT1 Advanced

**Concordance Hits** 263

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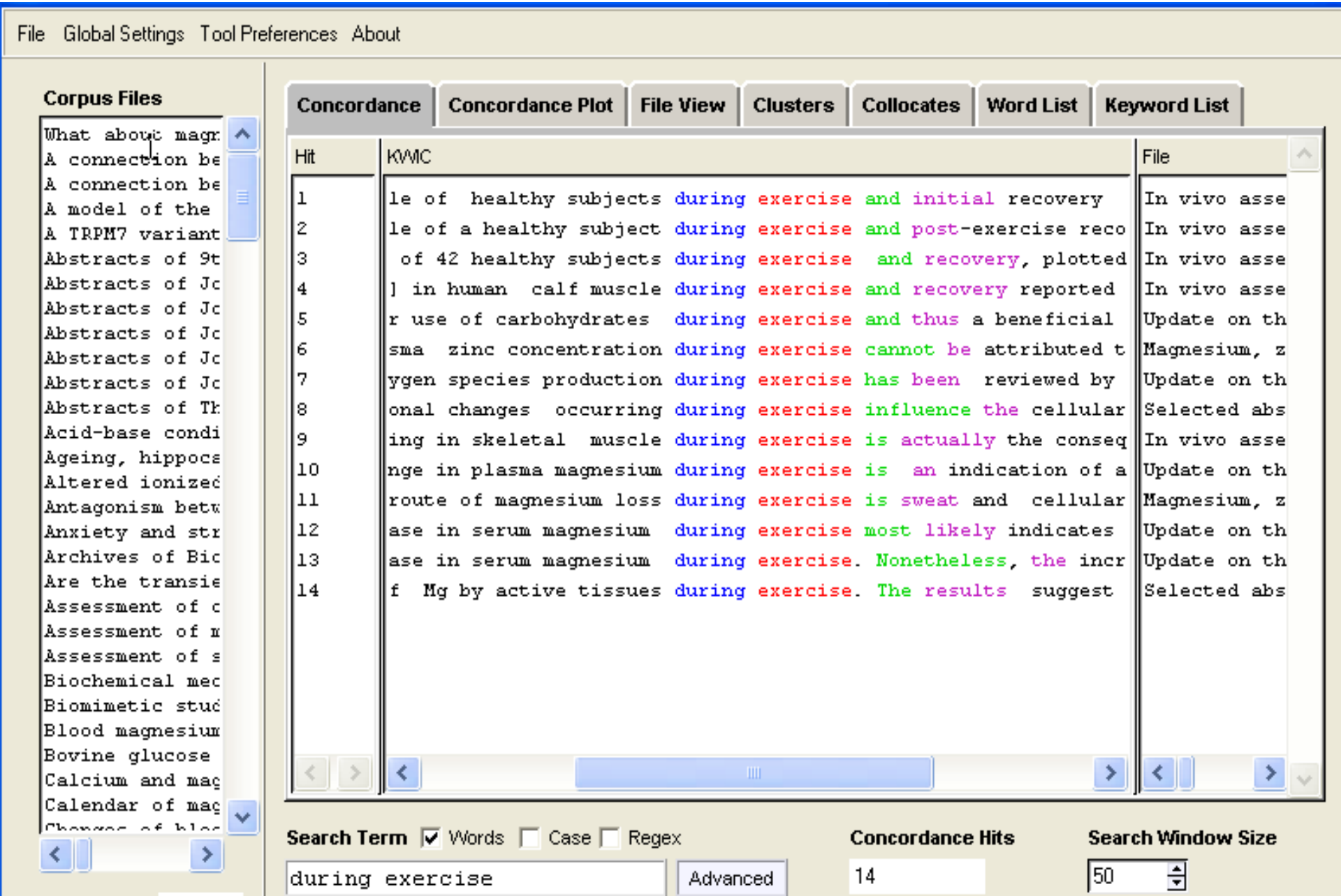
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1 le of healthy subjects during exercise and initial recovery In vivo asse
2 le of a healthy subject during exercise and post-exercise reco In vivo asse
3 of 42 healthy subjects during exercise and recovery, plotted In vivo asse
4 ] in human calf muscle during exercise and recovery reported In vivo asse
5 r use of carbohydrates during exercise and thus a beneficial Update on th
6 sma zinc concentration during exercise cannot be attributed t Magnesium, z
7 ygen species production during exercise has been reviewed by Update on th
8 onal changes occurring during exercise influence the cellular Selected abs
9 ing in skeletal muscle during exercise is actually the conseq In vivo asse
10 nge in plasma magnesium during exercise is an indication of a Update on th
11 route of magnesium loss during exercise is sweat and cellular Magnesium, z
12 ase in serum magnesium during exercise most likely indicates Update on th
13 ase in serum magnesium during exercise. Nonetheless, the incr Update on th
14 f Mg by active tissues during exercise. The results suggest Selected abs
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# Improve

KWIC

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| of other minerals like Mg, Fe, and Zn may also | improve | haematopoiesis and oxidative stress because b |
| (NA) may reduce cardiac arrhythmias and | improve | the prognosis of heart failure. It is |
| elet effect, oral Mg therapy has been shown to | improve | endothelial function significantly in patient |
| water. may be a useful natural drink to | improve | lipid metabolism and to prevent atherosc |
| minerals like Mg, Fe, and Zn may also | improve | haematopoiesis and oxidative stress becau |
| UDRAY, ET AL. verify this hypothesis and | improve | our understanding of the significance of |
| Postnatal MgSO4 infusion is safe and can | improve | short-term outcome in infants with sever |
| r brain damage, enter injured tissue and | improve | neurologic outcomes [29]. These results |
| minerals like Mg, Fe, and Zn may also | improve | haematopoiesis and oxidative stress becau |
| ll against damage caused by ischemia and | improve | its ability to resist the effects of c |
| ar disease [39]. Moreover, Mg supplements | improve | diabetic state. Eight elderly, moderately |
| lism Some diabetic treatments appear to | improve | Mg metabolism. Ewis showed a state of |
| ;¦Onofrio F. Dietary magnesium supplements | improve | B-cell response to glucose and arginine |
| Two polyol, low digestible carbohydrates | improve | the apparent absorption of magnesium but |
| sium therapy (magnesium polygalacturonate) may | improve | the function of the antioxidant system in hyp |
| values (11/19), while only 8/19 did not | improve | or improved with a decrease in Erc-Mg |
| s and magnesium salt administration could | improve | motor outcome [11]. One of the most im |
| was found, psycho- stimulants are used to | improve | mental health, probably through increasin |
| supplementation during pregnancy did not | improve | pregnancy outcome. Between 13 and 24 we |
| drug mixture, among other things, could | improve | the absorption of magnesium [22]. Furthe |
| ure elevation and cardiovascular disease. Mg++ | improve | lipid metabolism through the activation of LC |
| of combined magnesium/mild hypothermia to | improve | patient outcome following cerebral ischae |
| of particular importance to significantly | improve | our understanding of the cellular basis |
| atment could easily be applied either to | improve | the recovery after noise induced hearing |
| ogical states including hypomagnesemia. To | improve | the general health state, it is necessa |
| 2+ supplementation has been documented to | improve | flow mediated brachial vasodilation (endo |
| homeostasis in large populations [5]. To | improve | the understanding of the genetic factors |

# Perform

KWIC

| | | |
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| Analysis System (SAS) system was used to | perform | the statistical analyses. A conditional |
| metric assay (i-Ca) (Bayer Diagnostics). To | perform | Erc-Mg measurements, red blood cells (R |
| ts indicate that the choline exchanger can | perform | Mg2+ efflux via choline/Mg2+ exchange. |
| alleviated muscle spasms occurring with intense | perform | vigorous physical activity, and physical exer |
| hem. Fertilization also uses micro-elements that | perform | a lot of important physiological functions a |
| ould be expected that deficient mice would | perform | poorly in this task. The purpose of |
| rence (PLSD) test (one-tailed) was used to | perform | individual group comparisons when a sta |
| nt data on ionized calcium and albumin to | perform | additional analyses. We calculated mea |
| The function of Na+/Mg2+ antiport is to | perform | efflux of Mg2+, to establish a low an |
| efflux [2]. The Na+/Mg2+ anti- porter can | perform | 28Mg2+/24Mg2+ exchange [2]. Net uptake |
| S significantly. It would be also of interest to | perform | other tests of nociception regarding the inf |
| olyte balance. Consequently, the ability to | perform | physical work may be compromised. Many |
| neurological outcomes. Moreover, we did not | perform | pre-and postoperative neuropsychological |
| f the present study is that we could not | perform | the assessment of total intracellular M |
| participant, all the women were advised to | perform | mild to moderate physical activity with |
| metabolism. It is therefore appropriate to | perform | regression analysis, both with S and M |
| their relative paucity in the diet and the body, | perform | important roles in regulating whole-body met |
| of the influence of mineral status on ability to | perform | under conditions that a physically active ind |
| nevertheless, it is evident that it could | perform | a physiological function within cells t |

# Suggest

| | | |
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| n the sward samples from the area investigated | suggest | that fertilization and liming treatments on th |
| Recently, it has been Clinical findings | suggest | that magnesium depletion demonstrated that |
| weight and energy metabolism, some data | suggest | that this hormone could be involved in |
| atelet aggregation and arterial thrombosis | suggest | a mechanism for atherothrombotic disease |
| o an insufficient medical survey. But we | suggest | that the decrease in melatonin levels at |
| itability, respectively. This leads us to | suggest | the measurement of melatonin, extra-and i: |
| to mag- Such geographical distribution may | suggest | an envi-nesium depletion which may not b |
| ing quantitative pharmacokinetic models to | suggest | how it would be possible to become subs: |
| nase activity. Taken together, these data | suggest | that TRPM7¡¦s role in cellular ion homeo |
| ces antidepressant effects in rodents and | suggest | the involvement of this ion in human de: |
| ptoms of migraine and postspinal headache | suggest | a common pathophysiological correlate. Mag |
| IMVP may predispose to complications. The data | suggest | an autosomal dominant inheritance of IMVP that |
| d cardiac skeleton. Recently published reports | suggest | an autosomal dominant inheritance of the trait |
| yperlactataemia [27, 51]. Cohen et al. [28] | suggest | that metabolism in IMVP is associated with inc |
| sma in the two groups. These results strongly | suggest | a MD in MVPS and indicate the low level of use |
| ol group who received a placebo. These results | suggest | that the lessening of symptoms could be due to |
| ients. Certain clinical features in both groups | suggest | a relationship between these two pathological |
| lled water (control group). These results | suggest | that .deep sea water. may be a useful : |
| ween [Mg2+]c and Mg2+ flux thus obtained | suggest | that Mg2+ extrusion is steeply regulated |
| tered Ca EP metabolism. Our results also | suggest | that higher levels of Mg may prevent th |
| ts of bone loss [13]. These observations | suggest | that Mg bone, and plays important roles |
| high P diet. The results of this study | suggest | that increased dietary Mg supplementation |
| n the intestinal lumen [18]. Our results | suggest | that dietary Mg supplementation decreased |
| less of circulating PTH concentration. We | suggest | that suppression of bone resorption with |
| h P diet. In other words, these results | suggest | that bone resorption induced by a high |
| group. The results in the present study | suggest | that high Ca intake had no preventive e |

# Create new sentences

- Magnesium supplements improve lipid metabolism and prevent osteoporosis in patients with DM.
- We could not perform the statistical analyses in pre- and post-operative neuropsychological changes.
- Our data suggest that the increased dietary magnesium may be a useful…

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|  | journal of dairy science | 相似文章 |  | 活跃可见 | 月刊 | 活跃可见 | 介绍 \| 经验 \|Elsevi |

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- 4: Okay
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- Accept if major or minor revised
- Reconsider if revised
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  - Correction for spelling and grammatical errors
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- Read comments between the lines
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  - **Answer to all comments**
  - **Answer to the point**
  - **Point-by-point**
- Do not be frustrated by English problem

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