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در پایان این دوره آموزشی دانشجو قادر است:

- ❑ **مراحل یک کاوش علمی و اجزای آن را بشناسد.**
- ❑ **محتوای پاب مد را بشناسد.**
- ❑ **شیوه های کاوش ساده و پیشرفته در پاب مد را بشناسد.**
- ❑ **امکانات و ابزارهای گوناگون مانند:** Limits, History, Search Builder, Search History **را بشناسد.**
- ❑ **قادرباشد فرمول و استراتژی کاوش خود راذخیره نماید.**
- ❑ **شیوه های کاوش و دسترسی به مقالات دارای متن کامل را بشناسد.**

•علم پزشکی متکی بر تجربه و تحقیق است و نقش اساسی در پیشبرد این علم ایفا می کند. در این میان پزشکان با چالشی عمده مواجه هستند و باید در تنگاتنگ یک زندگی حرفه ای پرمشغله علم خود را به روز نگاهدارند

•برای آغاز هر پژوهشی شما بایستی از تحقیقات قبلی با اطلاع باشید و به بررسی متون و مقاله های موجود بپردازید.

• استناد به پژوهشهای انجام گرفته شرط لازم برای ارایه خدمات کیفی است. هر مطالعه و پژوهش جدید باید با تحقیقات گذشته در ارتباط باشد.

- امروزه جدیدترین یافته های علمی بیشتردرمقاله های مجلات تخصصی وجود دارند و هر پژوهشگری برای ارتقاء سطح دانش خودنیازمند مطالعه مستمر آنهاست.
- ولی بدلیل حجم روزافزون اطلاعات و انتشارات و نیز افزایش عناوین نشریه های منتشرشده در حوزه های مختلف علوم مانند پزشکی کمتر کسی وقت کافي براي حتی ورق زدن مجله های تخصصی حوزه خود دارد.
- امروزه با استفاده از رایانه و فناوری نوین اطلاعات پایگاههای اطلاعاتی از مقاله های موجود در مجلات تخصصی طراحی و تولید و در محیط الکترونیکی عرضه می شوند.

مراحل یک کاوش علمی؟

1. **فرموله کردن سوال یا مشکل (Problem formulation)**

2. **تعیین استراتژی جستجو (Search strategy)**
   متدولوژی جستجو مشخص شود.

3. **کاوش منابع اطلاعاتی (Literature search)**
   کاوش پژوهش های مرتبط با موضوع در انواع پایگاه های اطلاعاتی و مجموعه مجلات و منابع الکترونیکی....

❑فرموله کردن سوال چه فایده‌ای دارد؟

- **به پژوهشگر کمک می کند:**
  - واضح‌تر بفهمد که دنبال چه می‌گردد.

- **به استفاده کننده کمک می کند:**
  - آیا این سوال پاسخ او را هم می دهد؟

تعیین استراتژی جستجو/کاوش؟

- تعریفی ساده: تهیه یک دستور العمل شفاف برای جستجو
- جزئیات استراتژی جستجو در پایگاه‌های مختلف تفاوتهایی با یکدیگر دارد، اما اصول استراتژی جستجو در پایگاه‌های مختلف تقریبا مشابه است.

ادامه مراحل یک کاوش علمی؟

- **تعیین استراتژی جستجو**

قبل از شروع جستجو در مدلاین بایستی مفهوم یا مفاهیم مرتبط با موضوع مورد جستجو تعیین شوند.

موضوع تحقیق: اثرات سیگار بر ناهنجاریهای جنینی

مفاهیم :

smoking/cigarette/tobacco/nicotine/fetus/abnormalities/birth defects…

2. با تعیین مفاهیم می توانید واژه های کلیدی یعنی موضوعهای اصلی و فرعی تحقیق خود را برای جستجو تعیین کنید.

3 . سپس در مدلاین با تایپ واژه های کلیدی جستجو را آغاز کنید.

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- PubMed is a Web-based retrieval system developed by the National Center for Biotechnology Information (NCBI) at the National Library of Medicine.
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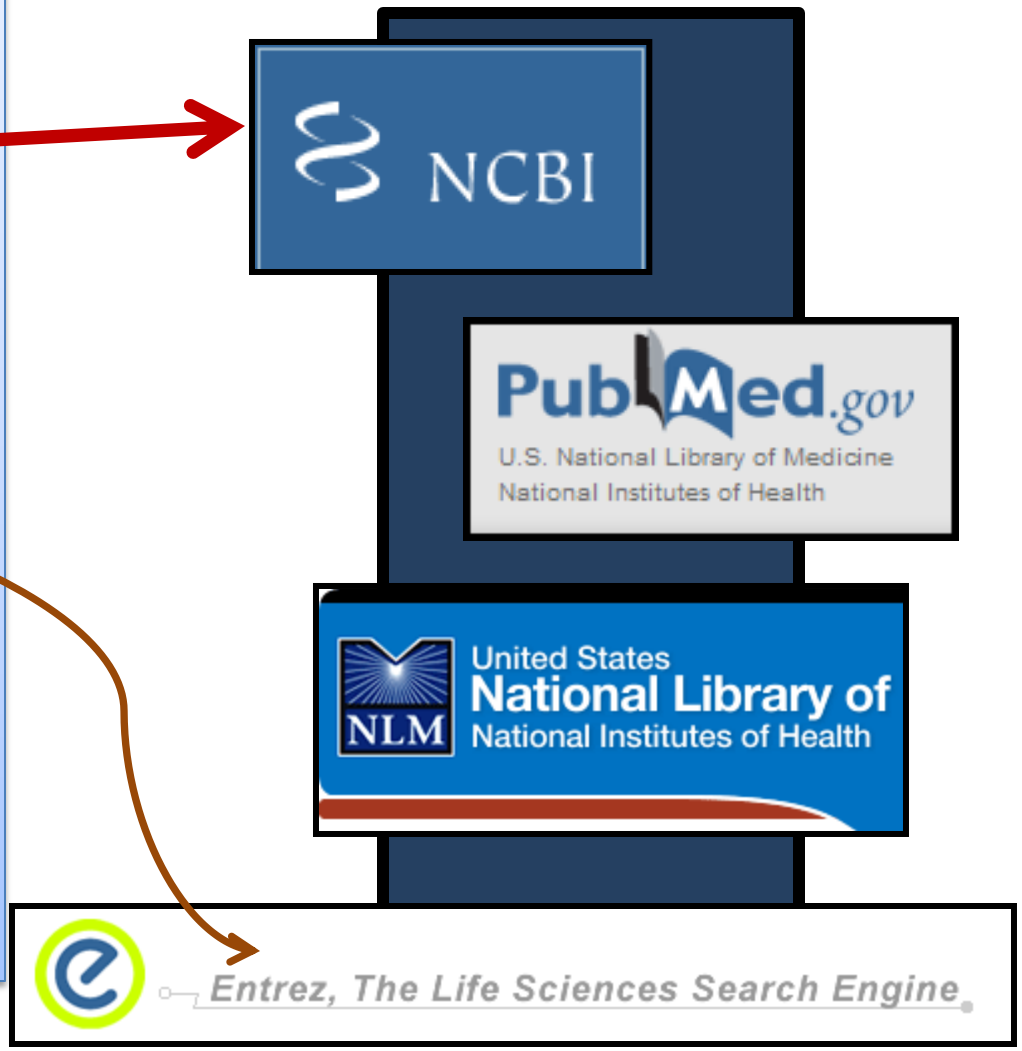

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مسير اول: تايپ نشانی کتابخانه ملی پزشکی آمریکاو سپس بر لینک PubMed کلیک کنید.

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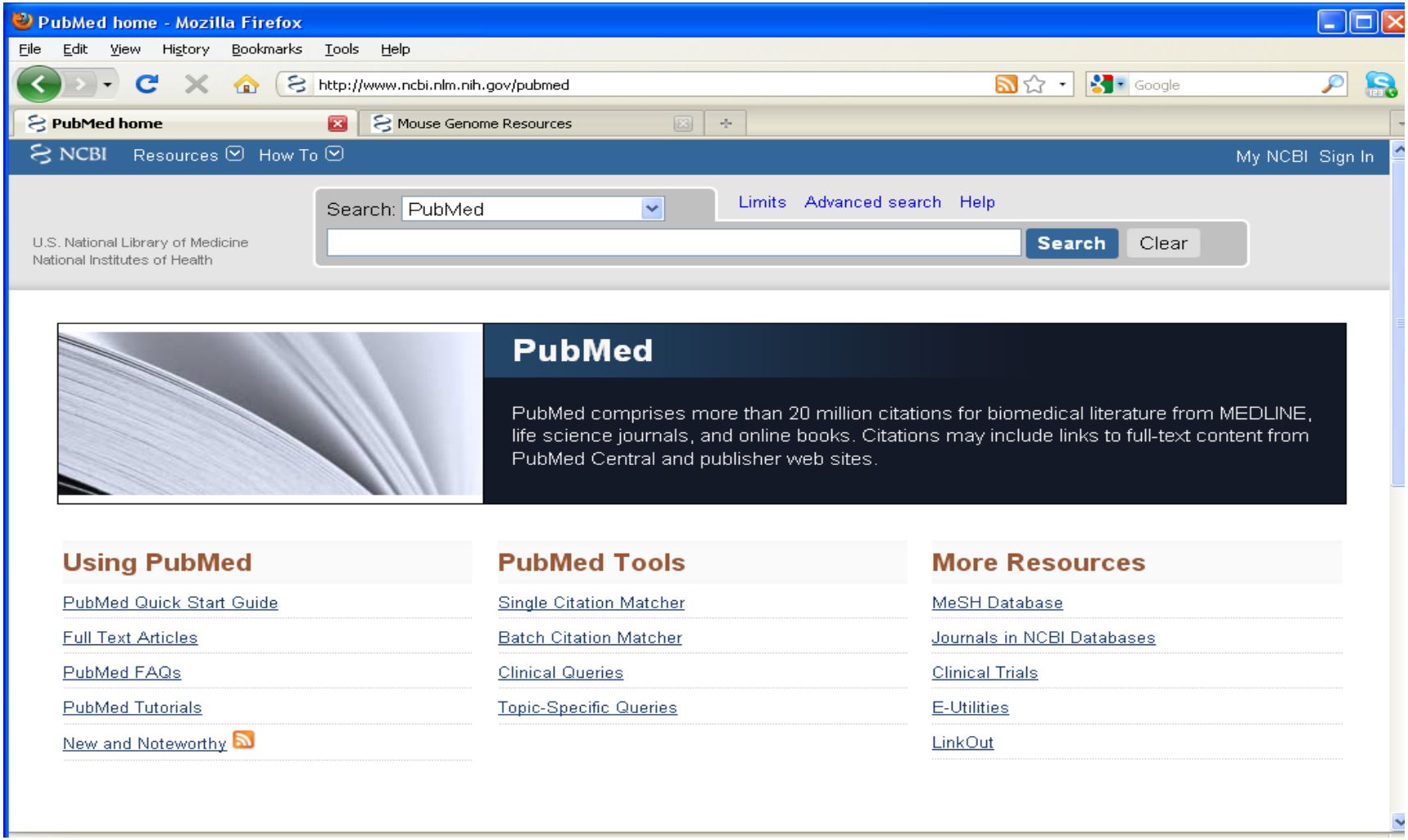
PubMed History

- NLM has been indexing the biomedical literature since 1879 .
- Old printed index : the *Index Medicus*
- PubMed as an experimental database : January 1996
- on June 26, 1997, officially announced
- PubMed Redesign: fall of 2009.
- In 1996, 600,000 searches each month. Today> one billion searches annually.

اگر مایلید کلیاتی درباره این مجموعه و امکانات آن بدانید بر لینک زیر کلیک نمایید

https://www.nlm.nih.gov/bsd/disted/pubmedtutorial/cover.html

- مدلاین معتبرترین پایگاه اطلاعاتی مقالات علمی منتشرشده ۵۶۰۰ ژورنال حوزه زیست پزشکی جهان است که از طریق محیط پاب مد PubMed عرضه می شود.
- پوشش موضوعی آن : علوم پایه و بالینی پزشکی و رشته های مختلف آن (دندانپزشکی/ داروسازی/ پرستاری و مامایی/ بهداشت و پیراپزشکی/ دامپزشکی و ....
- رکورد های جدید هر هفته از سه شنبه تا شنبه به این پایگاه افزوده می شود.
- بیش از ۹۰ درصد رکوردها (مقالات) چکیده به زبان انگلیسی دارند.
- پوشش زمانی: از حدود ۱۹۵۰ میلادی تا کنون



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Simple Subject Search

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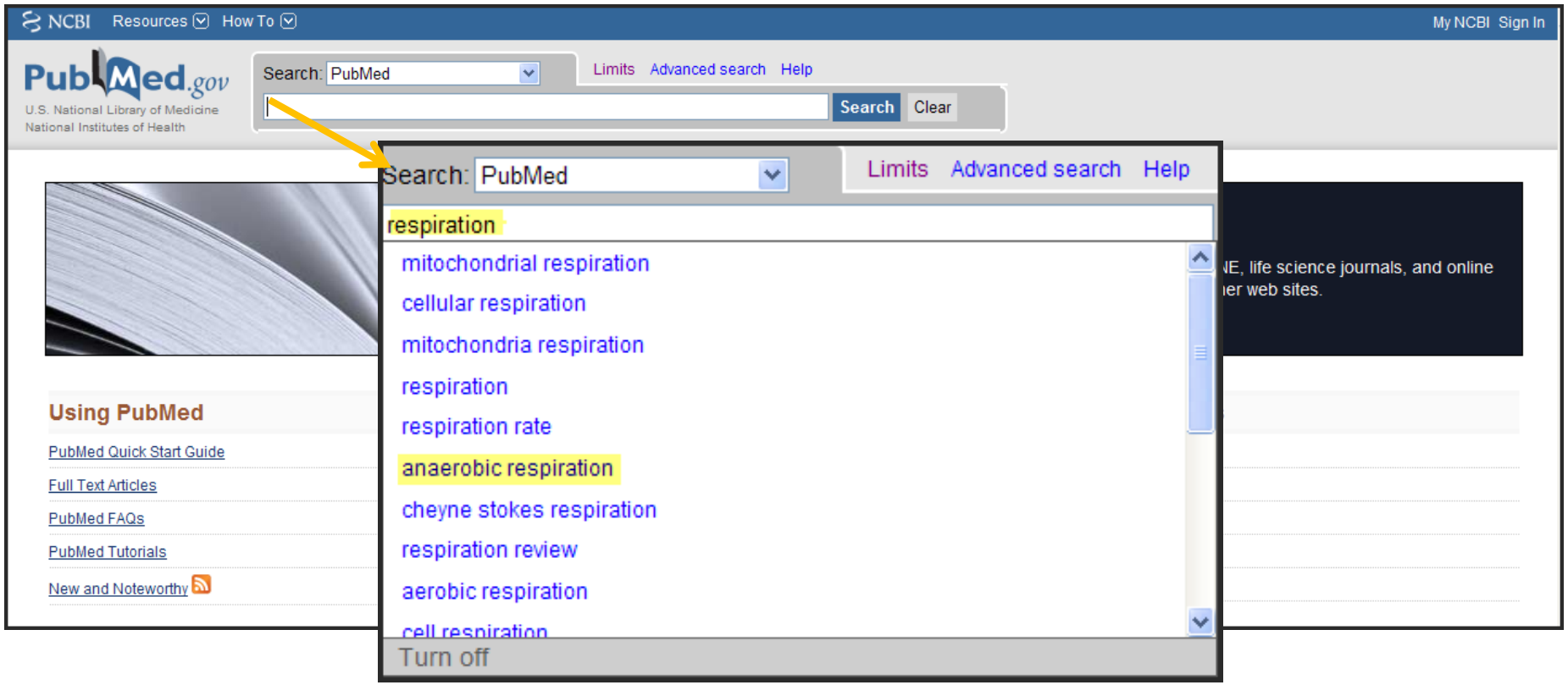

❑در کاوش ساده، صرفا واژه مورد نظرتان را تایپ کنید.

❑کادری از واژه های مشابه با واژه مورد نظر شما باز می شود. کافی است واژه خود را انتخاب کنید تا کاوش آغاز شود.

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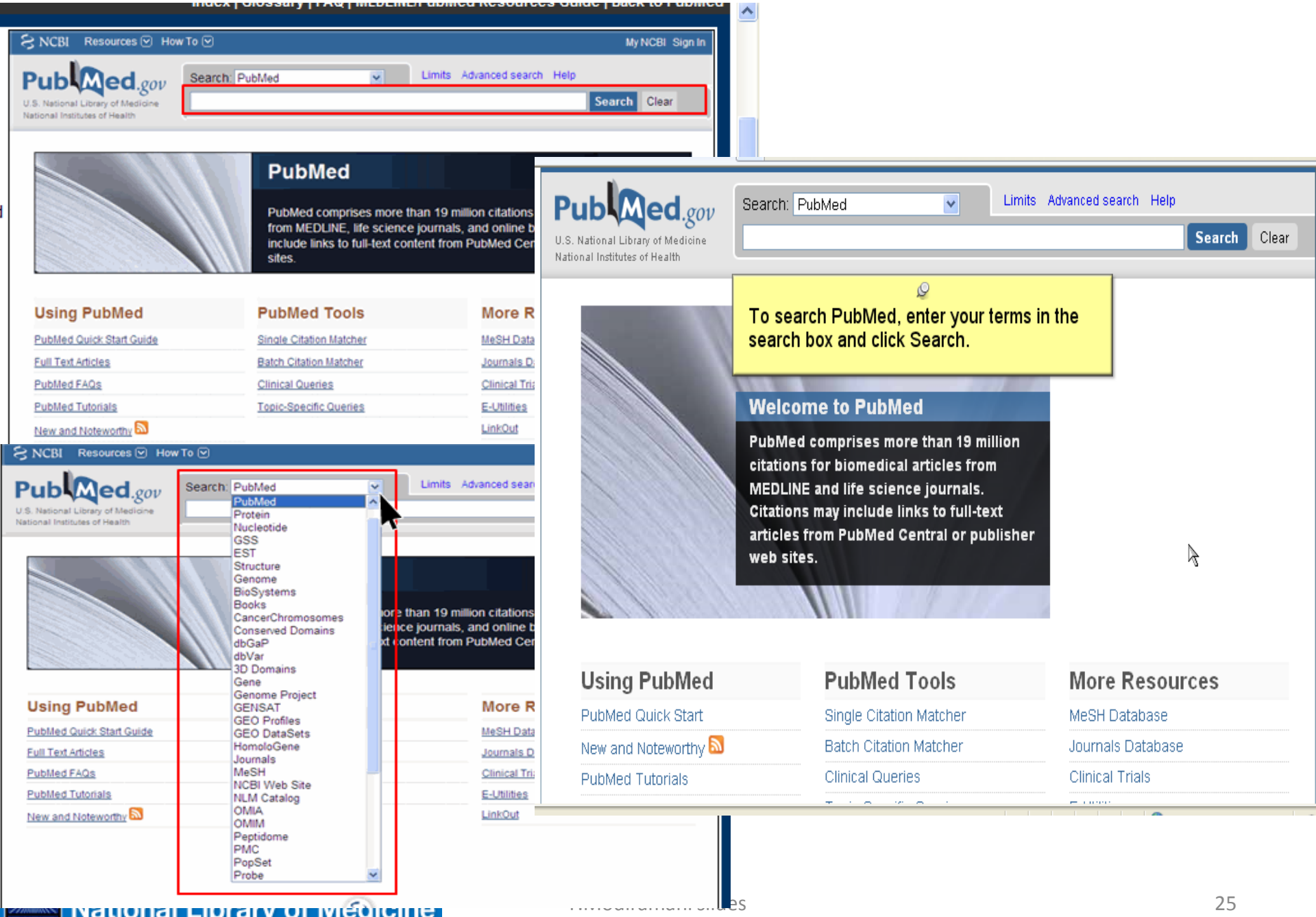
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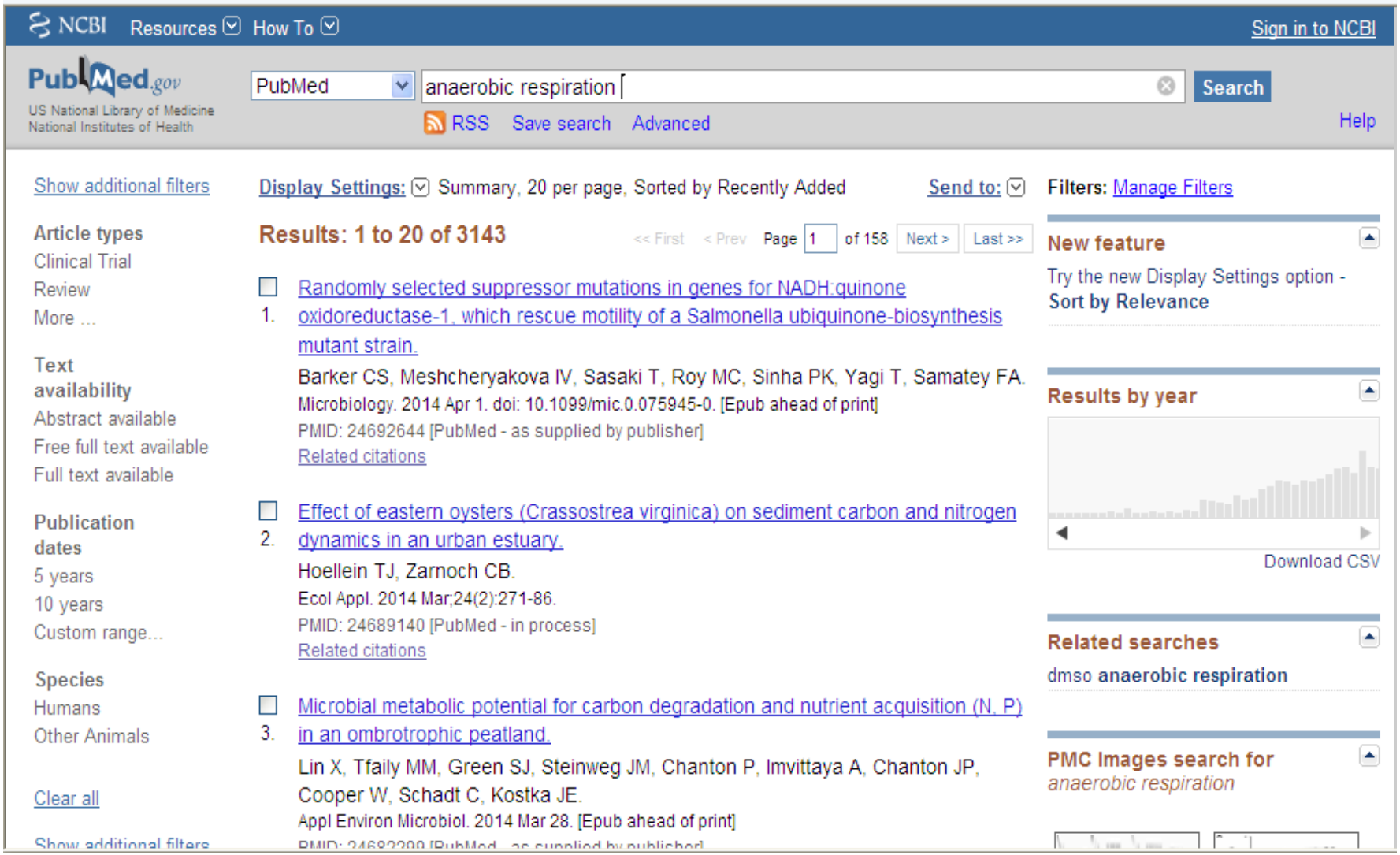
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1. Randomly selected suppressor mutations in genes for NADH:quinone oxidoreductase-1, which rescue motility of a Salmonella ubiquinone-biosynthesis mutant strain.
Barker CS, Meshcheryakova IV, Sasaki T, Roy MC, Sinha PK, Yagi T, Samatey FA.
Microbiology. 2014 Apr 1. doi: 10.1099/mic.0.075945-0. [Epub ahead of print]
PMID: 24692644 [PubMed - as supplied by publisher]
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2. Effect of eastern oysters (Crassostrea virginica) on sediment carbon and nitrogen dynamics in an urban estuary.
Hoellein TJ, Zarnoch CB.
Ecol Appl. 2014 Mar;24(2):271-86.
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Appl Environ Microbiol. 2014 Mar 28. [Epub ahead of print]
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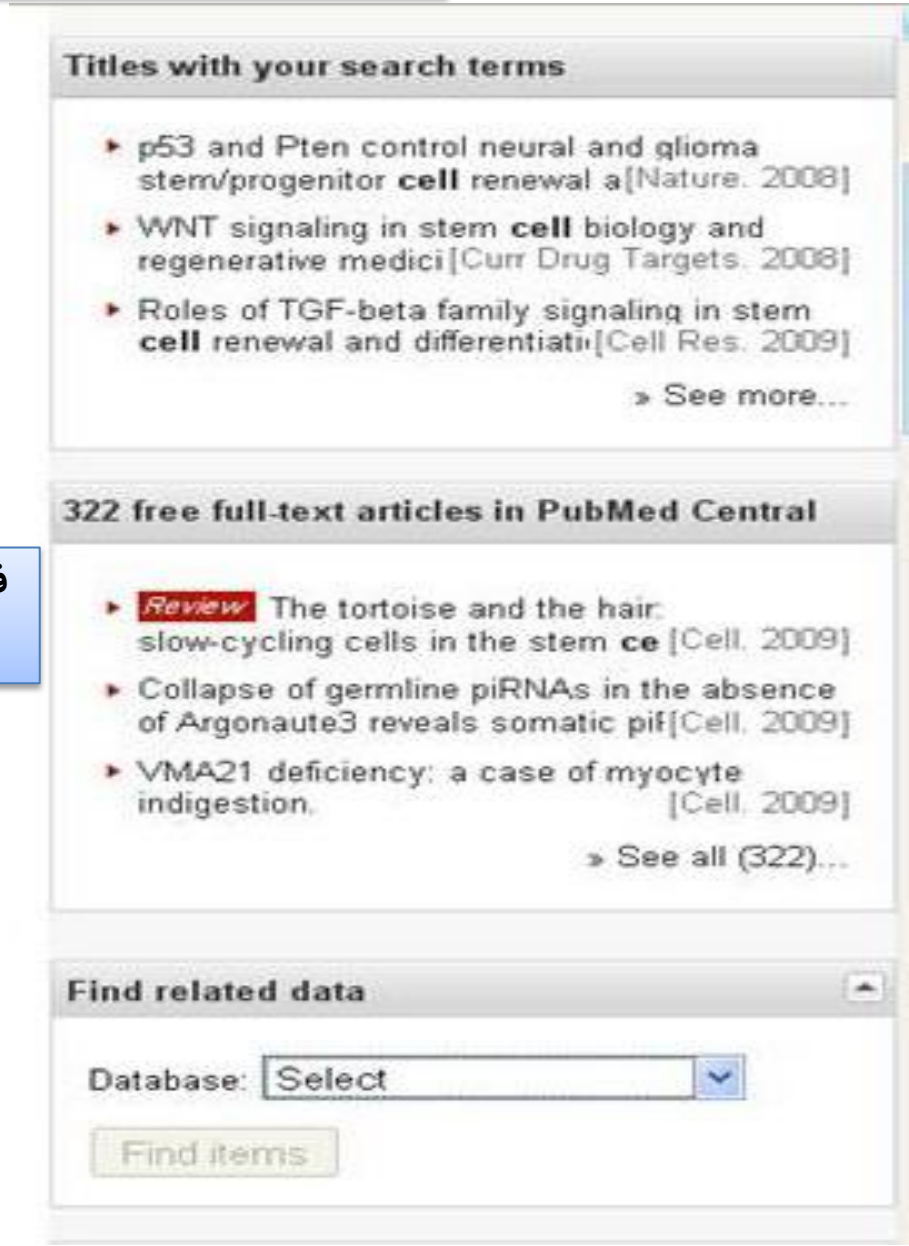

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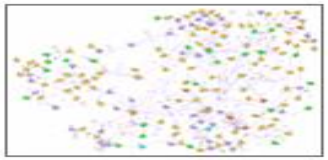

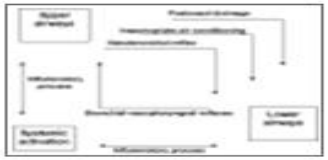

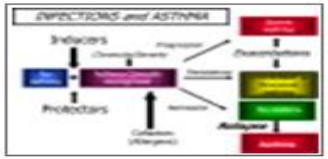

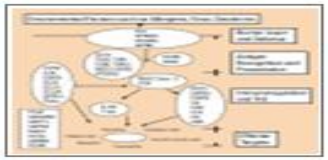

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نمایش نتایج بر حسب سال انتشار


Title
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1.
Authors
Cummings RD, McEver RP.
In: Varki A, Cummings RD, Esko JD, Freeze HH, Stanley P, Bertozzi CR, Hart GW, Etzler ME, editors. Essentials of Glycobiology. 2nd edition, Cold Spring Harbor (NY): Cold Spring Harbor Laboratory Press; 2009. Chapter 31.
PMID: 20301263 [PubMed] Books & Documents Free text
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Teaching medical students about chronic disease: patient-led teaching in rheumatoid arthritis.
2.
Journal Abbreviation
Phillpotts C, Creamer P, Andrews T.
Musculoskeletal Care. 2010 Mar 19. [Epub ahead of print]
PMID: 20301228 [PubMed - as supplied by publisher]
miR-125b-2 is a potential oncomiR on human chromosome 21 in megakaryoblastic leukemia.
3.
Klusmann JH, Li Z, Böhmer K, Maroz A, Koch ML, Emmrich S, Godinho FJ, Orkin SH, Reinhardt D.
Publication Date
Genes Dev. 2010 Mar 1;24(5):478-90.
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PMID: 20194440 [PubMed - indexed for MEDLINE] Free PMC Article Free text
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Ther Adv Neurol Disord. 2010 Nov;3(6):379-89.

Aminoglycoside-induced mutation suppression (stop codon readthrough) as a therapeutic strategy for Duchenne muscular dystrophy.

Malik V, Rodino-Klapac LR, Viollet L, Mendell JR.

The Research Institute at Nationwide Children's Hospital and Department of Pediatrics at The Ohio State University College of Medicine, Columbus, OH, USA.

Abstract

Duchenne muscular dystrophy (DMD) is the most common, lethal, X-linked genetic disease, affecting 1 in 3500 newborn males. It is caused by mutations in the DMD gene. Owing to the large size of the gene, the mutation rate in both germline and somatic cells is very high. Nearly 13-15% of DMD cases are caused by nonsense mutations leading to premature termination codons in the reading frame that results in truncated dystrophin protein. Currently there is no cure for DMD. The only available treatment is the use of glucocorticoids that have modest beneficial effects accompanied by significant side effects. Different therapeutic strategies have been developed ranging from gene therapy to exon skipping and nonsense mutation suppression to produce the full-length protein. These strategies have shown promise in the mdx mouse model of muscular dystrophy where they have been reported to ameliorate the dystrophic phenotype and correct the physiological defects in the membrane. Each of these molecular approaches are being investigated in clinical trials. Here we review nonsense mutation suppression by aminoglycosides as a therapeutic strategy to treat DMD with special emphasis on gentamicin-induced readthrough of disease-causing premature termination codons.

PMID: 21179598 [PubMed - in process] PMCID: PMC3002642 **Free PMC Article**

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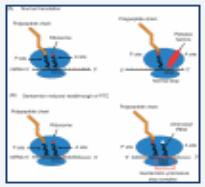
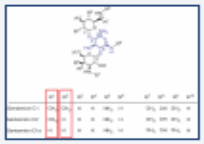
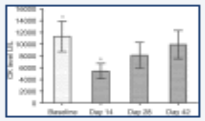
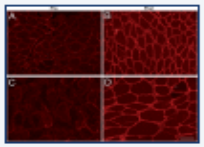


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Gentamicin-induced readthrough of stop codons in Duchenne muscul [Ann Neurol. 2010]

A duchenne muscular dystrophy gene hot spot mutation in dystrophin-deficier [PLoS One. 2010]

Sequence specificity of aminoglycoside-induced stop condon readthrough: pol [Ann Neurol. 2000]

Review [Mutation-specific treatments for Duchenne muscular dystropl [Brain Nerve. 2009]

Review Readthrough strategies for stop codons in Duchenne muscular [Acta Myol. 2006]

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J Endocrinol Invest. 2005 Nov;28(10):928-34.

Plasma total homocysteine level and its association with carotid intima-media thickness in obesity.

Uysal O, Arikan E, Cakir B.

Internal Medicine, Medical Faculty, Trakya University, Edirne, Turkey.

Comment in:

J Endocrinol Invest. 2006 Jun;29(6):573-4.

Abstract

BACKGROUND: Elevated plasma concentrations of total homocysteine (tHcy) and obesity are risk factors for cardiovascular disease. The relationship between hyperhomocysteinemia and obesity has not been totally elucidated.

OBJECTIVE: The first aim of the study was to investigate whether anthropometric measurements and insulin resistance contribute to the variation in homocysteine levels in obese adults. Our second aim was to determine if any relationship exists between the carotid intima-media thickness (IMT) and plasma tHcy levels in obese subjects without traditional cardiovascular risk factors.

MATERIAL AND METHODS: Fifty-five obese (15 male, 40 female) and 30 (11 male, 19 female) age- and sex-matched apparently healthy volunteers were included. Exclusion criteria were smoking, hypertension, diabetes, vitamin ingestion, hyperlipidemia, renal failure, liver disease, pregnancy, menopause and secondary obesity such as Cushing's syndrome, hypothyroidism. tHcy, folate, vitamin B12 levels, fasting insulin, glucose, total cholesterol, triglycerides, HDL, LDL particles, uric acid, creatinine and creatinine clearance were measured. Non-invasive ultrasound measurements of carotid IMT were performed.

RESULTS: tHcy levels and carotid IMT were comparable between obese and non-obese subjects. Waist/hip ratio (WHR) was related to tHcy and carotid IMT. Hyperhomocysteinemic subjects (tHcy >19.2 micromol/l) had greater WHR than normo-homocysteinemic subjects. Both tHcy levels and carotid IMT were higher in male subjects both in obese and non-obese subjects. No association was observed between insulin resistance and tHcy and carotid IMT. Renal function and abdominal obesity were significant predictors of plasma tHcy levels.

CONCLUSIONS: We concluded that, in obese subjects who are free from atherosclerosis and impaired renal function, plasma tHcy levels do not differ from healthy subjects. Plasma tHcy concentrations are not related to carotid IMT in obese subjects during the non-atherogenic stage. Although no significant difference was observed between insulin-resistant and insulin-sensitive subjects compared to the plasma tHcy levels, the relationship between tHcy levels and some components of the insulin resistance syndrome may support the opinion that tHcy may be considered a component of the insulin resistance syndrome.

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Solution structure of the parvulin-type PPIase domain of Staphylococcus aureus PrsA--implications for the catalytic mechanism of parvulins.

Heikkinen O, Seppala R, Tossavainen H, Heikkinen S, Koskela H, Permi P, Kilpeläinen I.

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Abstract

BACKGROUND: Staphylococcus aureus is a Gram-positive pathogenic bacterium causing many kinds of infections from mild respiratory tract infections to life-threatening states as sepsis. Recent emergence of S. aureus strains resistant to numerous antibiotics has created a need for new antimicrobial agents and novel drug targets. S. aureus PrsA is a membrane associated extra-cytoplasmic lipoprotein which contains a parvulin-type peptidyl-prolyl cis-trans isomerase domain. PrsA is known to act as an essential folding factor for secreted proteins in Gram-positive bacteria and thus it is a potential target for antimicrobial drugs against S. aureus.

RESULTS: We have solved a high-resolution solution structure of the parvulin-type peptidyl-prolyl cis-trans isomerase domain of S. aureus PrsA (PrsA-PPIase). The results of substrate peptide titrations pinpoint the active site and demonstrate the substrate preference of the enzyme. With detailed NMR spectroscopic investigation of the orientation and tautomeric state of the active site histidines we are able to give further insight into the structure of the catalytic site. NMR relaxation analysis gives information on the dynamic behaviour of PrsA-PPIase.

CONCLUSION: Detailed structural description of the S. aureus PrsA-PPIase lays the foundation for structure-based design of enzyme inhibitors. The structure resembles hPin1-type parvulins both structurally and regarding substrate preference. Even though a wealth of structural data is available on parvulins, the catalytic mechanism has yet to be resolved. The structure of S. aureus PrsA-PPIase and our findings on the role of the conserved active site histidines help in designing further experiments to solve the detailed catalytic mechanism.

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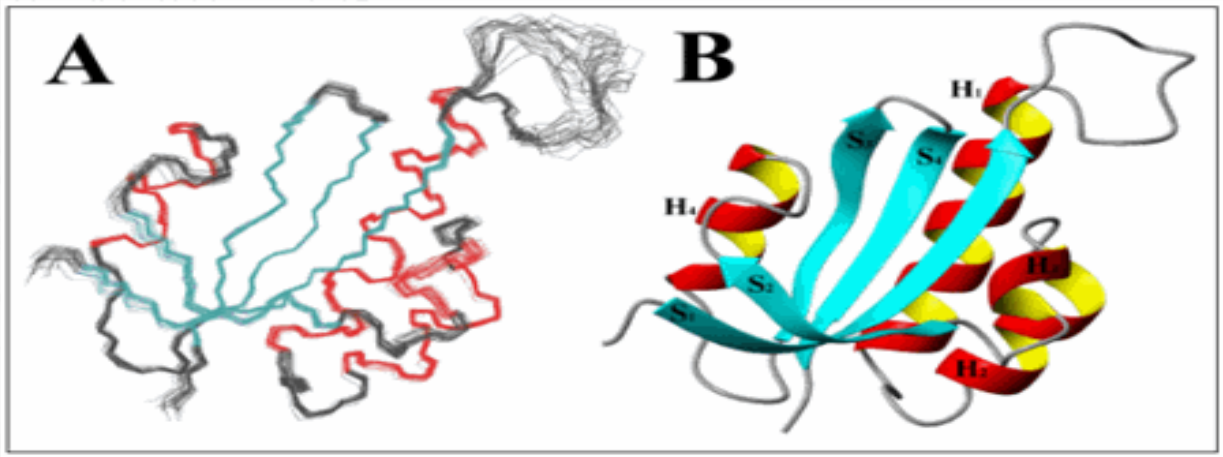

Figure 4

Solution structure of S. aureus PrsA-PPIase. The protein construct used in the study comprised residues 140–245 of the S. aureus PrsA [Swiss-Prot: P60747]. (A) Superimposed backbone traces of the 25 structures in the structure ensemble. Secondary structure elements are colour coded as: red – helix; cyan – strand; grey – coil. (B) A ribbon model of the averag...

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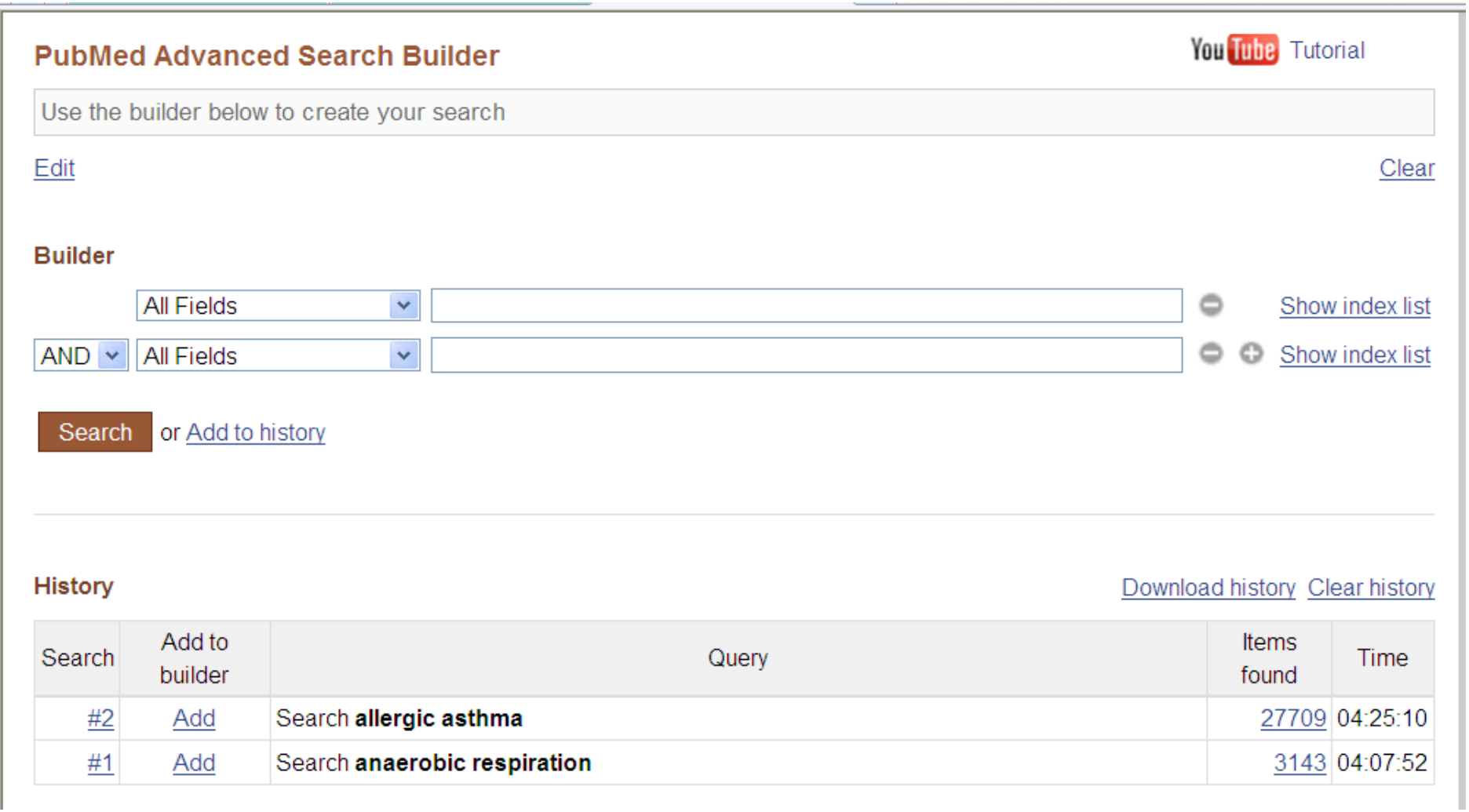

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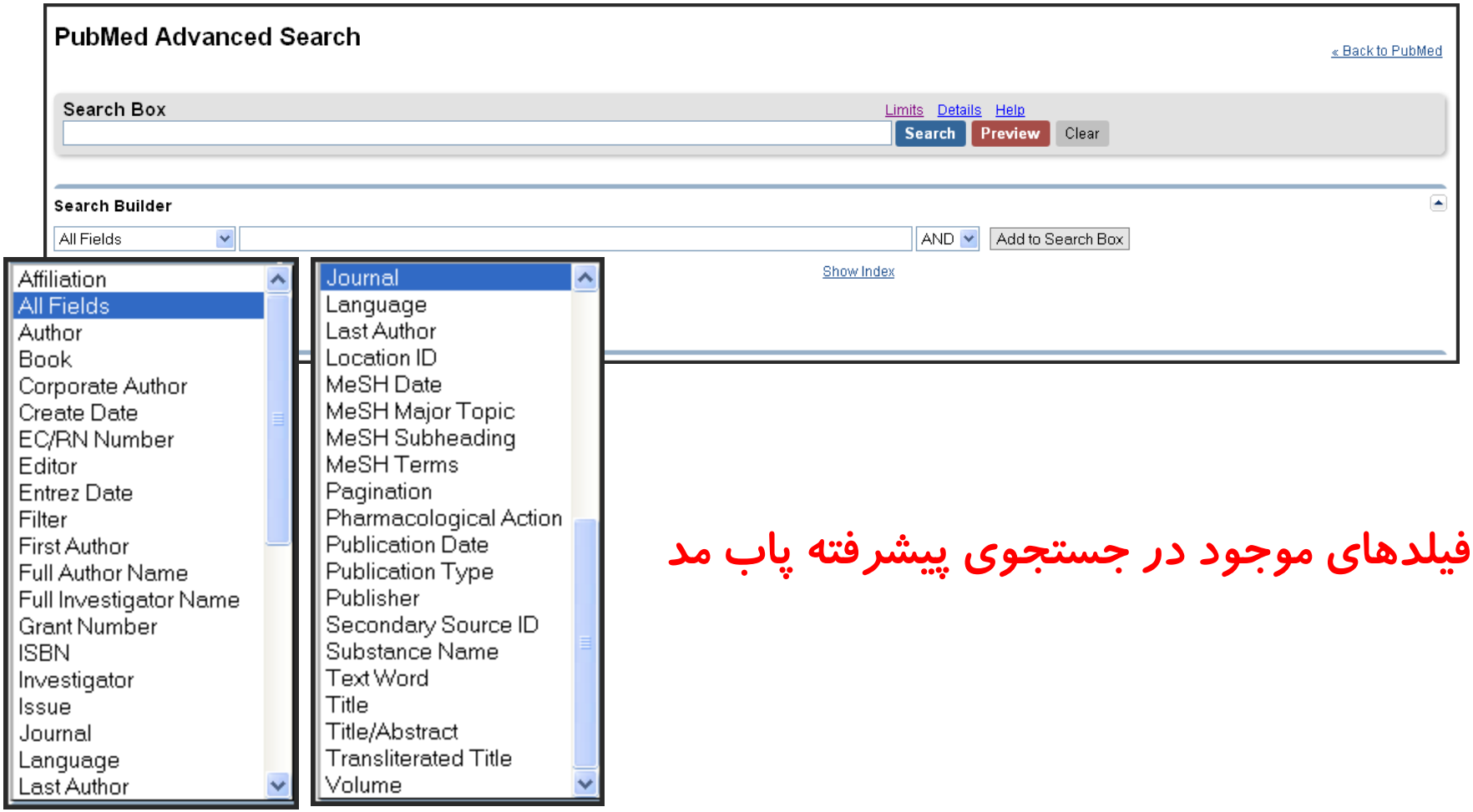

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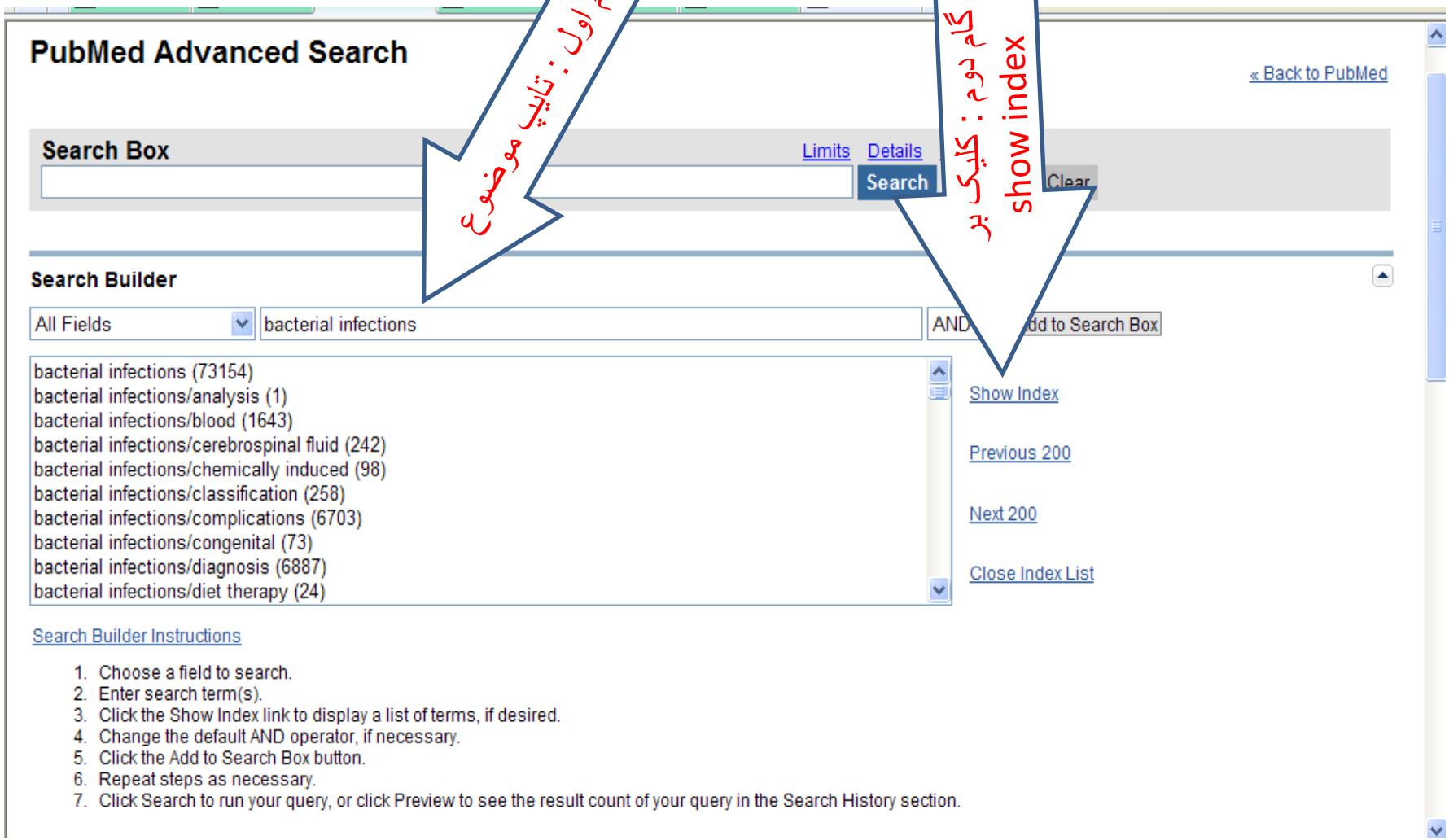

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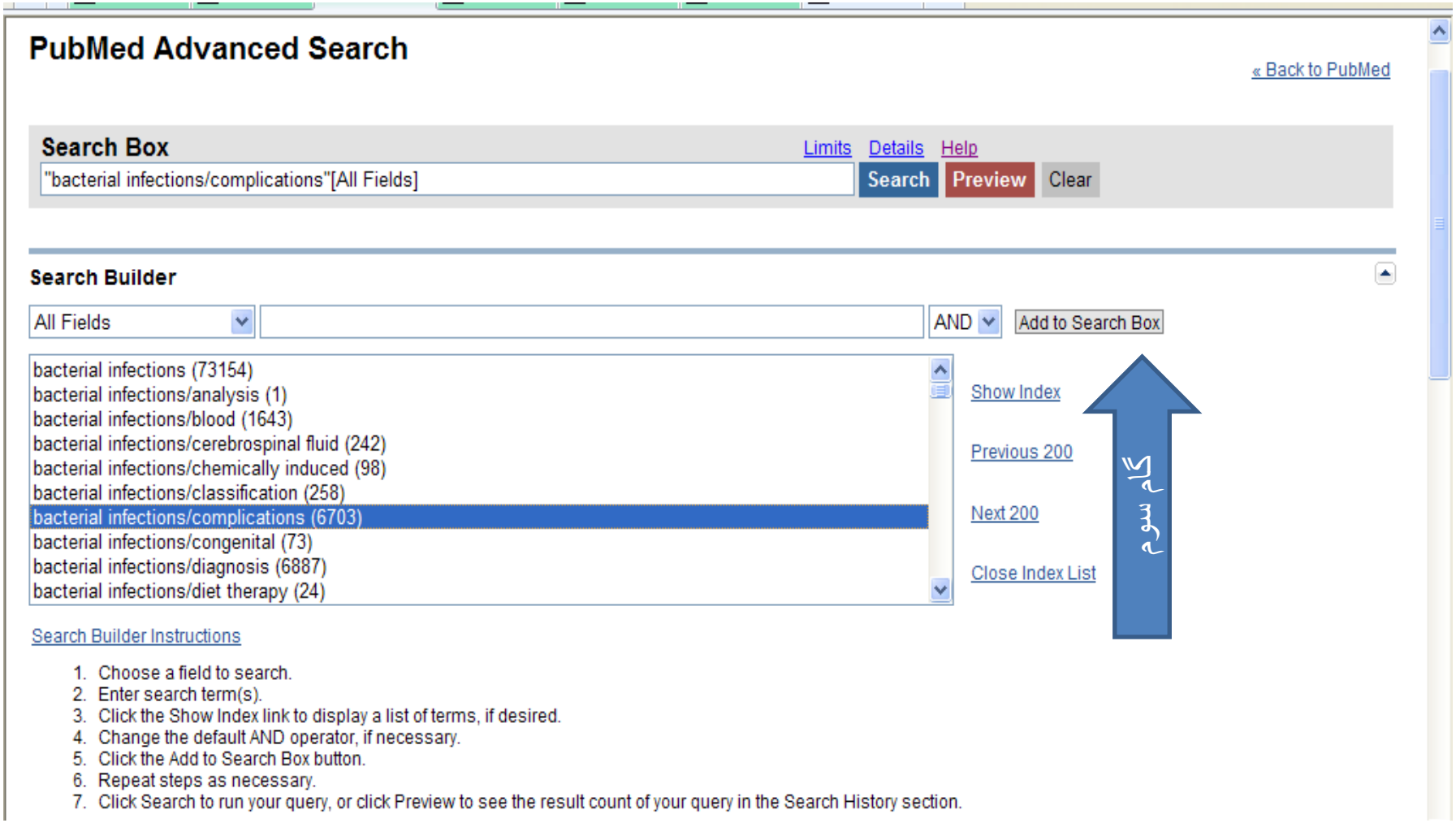

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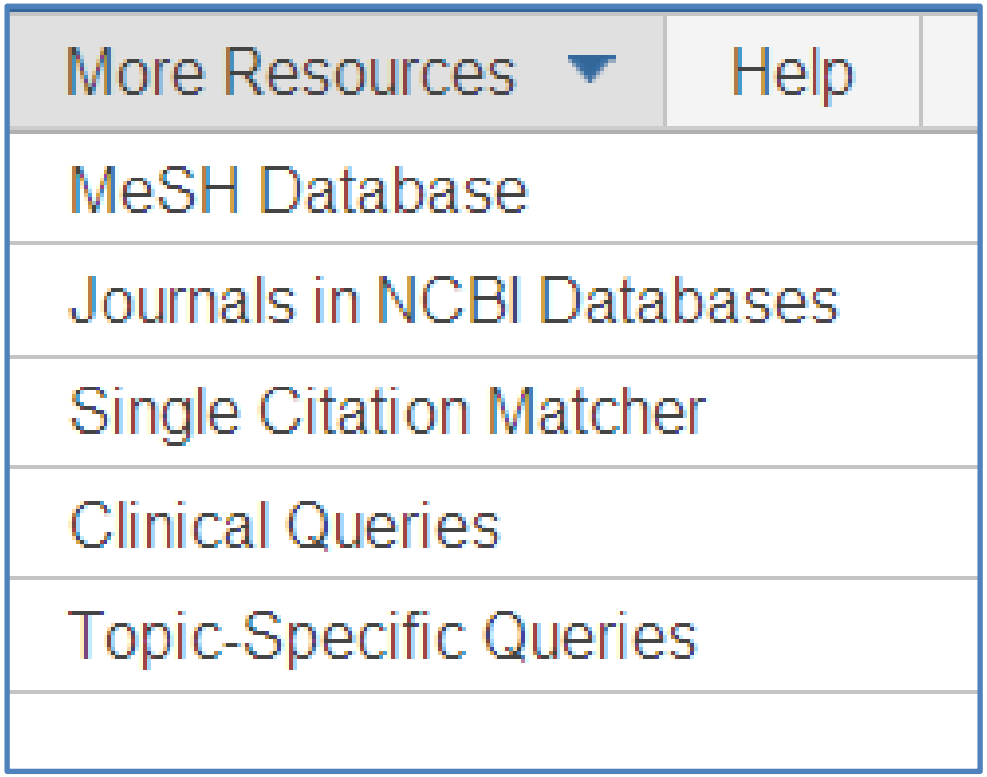

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