

KAKO NAPISATI NAUČNI RAD?

FAZE ISTRAŽIVAČKOG RADA

Formulisanje hipoteze

Planiranje eksperimenta

Eksperimentalni rad -Prikupljanje podataka

Analiza podataka

Zaključivanje

Pisanje rada

FAZE ISTRAŽIVAČKOG RADA

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NAUČNI RAD (MORA DA) IMA

ČVRSTU STRUKTURU

- Naslov
- Apstrakt
- Uvod
- Materijal i metode
- Rezultati
- Diskusija
- Literatura-reference

ODREDJENI STIL

- Preciznost
- Jasnoća
- Ekonomičnost
- Jednostavnost
- Konciznost

ZAŠTO STRUKTURA?

Zato što ona:

- obezbedjuje jedinstveni način komunikacije u medjunarodnoj naučnoj zajednici
- omogućava čitanje na/sa više različitih nivoa

ZAŠTO STIL?

Zato što je osnovna funkcija naučnog rada da prenese informaciju jasno i nedvosmisleno.

Pisanje i mišljenje su dve tesno povezane aktivnosti.

"FUZZY WRITING REFLECTS FUZZY THINKING!"

NASLOV

SADRŽAJ

Neskrativi skup pojmova koji tačno opisuju sadržaj rada

VRSTE

- Indikativni
- Informativni

NASLOV

The Journal of
Immunology



Informativni

Sustained IL-12 Signaling Is Required for Th1 Development

Veronica Athie-Morales, Hermelijn H. Smits, Doreen A. Cantrell, and
Catharien M. U. Hilkens

J Immunol 2004 172: 61-69.

Indikativni

Effects of T Cell Frequency and Graft Size on Transplant Outcome in Mice

Chunshui He, Soren Schenk, Qiwei Zhang, Anna Valujskikh, Jörg Bayer,
Robert L. Fairchild, and Peter S. Heeger

J Immunol 2004 172: 240-247

NASLOV

NAJČEŠĆE GREŠKE

■ Preopširan

Ispitivanje efekta tiazofurina na mogućnost pasivnog prenosa eksperimentalnog autoimunskog encefalomijelitisa u Dark Agouti pacova

■ Neprecizan

Tiazofurin i eksperimentalni autoimunski encefalomijelitis

■ Sadrži neuobičajene reči, skraćenice...

TR inhibira pasivni prenos EAE u DA pacova

APSTRAKT

SADRŽAJ

Glavni aspekti rada dati u 150-200 reči po sledećem redosledu: svrha/cilj rada, eksperimentalni dizajn i metode, glavni rezultati i osnovni zaključci

STIL

Prošlo vreme

VRSTE

- Klasični (nestructurisasi)
- Strukturisasi

APSTRAKT - klasični

Rat and Human Myelin Oligodendrocyte Glycoproteins Induce Experimental Autoimmune Encephalomyelitis by Different Mechanisms in C57BL/6 Mice

Alfred R. Oliver, Geoffrey M. Lyon, and Nancy H. Ruddle

C57BL/6 mice immunized with the extracellular Ig-like domain of rat myelin oligodendrocyte glycoprotein (MOG) developed experimental autoimmune encephalomyelitis (EAE) resembling that induced by rodent MOG 35-55 in its B cell independence and predominantly mononuclear CNS infiltrate. In contrast, human MOG protein-induced EAE was B cell dependent with polymorphonuclear leukocytes. Human MOG differs from rat MOG at several residues, including a proline for serine substitution at position 42. Human MOG 35-55 was only weakly encephalitogenic, and a proline substitution in rat MOG at position 42 severely attenuated its encephalitogenicity. However, human MOG 35-55 was immunogenic, inducing proliferation and IFN- and IL-3 to human, but not rodent MOG 35-55. The B cell dependence of EAE induced by human MOG protein was not due to a requirement for Ag presentation by B cells, because spleen cells from B cell-deficient mice processed and presented human and rat MOG proteins to T cells. The different pathogenic mechanisms of human and rat MOG proteins might result from different Abs induced by these proteins. However, rat and human MOG proteins induced Abs to mouse MOG that were equivalent in titer and IgG subclass. These data demonstrate that EAE can be induced in C57BL/6 mice by two mechanisms, depending on the nature of the immunogen: an encephalitogenic T cell response to rat MOG or rodent MOG 35-55, or an encephalitogenic B cell response to epitopes on human MOG protein that most likely cross-react with mouse determinants. *The Journal of Immunology*, 2003, 171:462–468.

APSTRAKT - strukturisani

Multiple Sclerosis with Uncommon Cerebrospinal Fluid Findings

Uroš Rot, Anton Mesec, Tomaz Pogacnik

Department of Neurology, Medical Center, Ljubljana, Slovenia

Aim. To determine the frequency and clinical and laboratory features of patients with multiple sclerosis characterized by uncommon cerebrospinal findings, ie, negative oligoclonal band or increased number of mononuclear cells in cerebrospinal fluid.

Methods. The retrospective analysis included medical records of 233 patients (158 women and 75 men) admitted to the Department of Neurology, Ljubljana Medical Center, between January 1, 1990, and December 31, 1999 and discharged with the diagnosis of multiple sclerosis. We determined clinical features and cerebrospinal fluid parameters of patients with oligoclonal band-negative multiple sclerosis and 15 mononuclear cells/mm³ in cerebrospinal fluid and compared them with patients with oligoclonal band-positive multiple sclerosis and expected number of mononuclear cells in cerebrospinal fluid, respectively. There were 26 patients with oligoclonal band-negative finding and 26 with 15 mononuclear cells/mm³ in cerebrospinal fluid. The two groups of patients did not overlap, except for one patient, who had 19 mononuclear cells/mm³ and was oligoclonal band-negative.

Results. The diagnosis was delayed in oligoclonal band-negative multiple sclerosis patients, their cerebrospinal fluid contained less leukocytes, and lower concentration of IgG. The patients with ≥ 15 leukocytes/mm³ in cerebrospinal fluid were diagnosed earlier and had increased cerebrospinal fluid protein and IgG concentrations.

Conclusion. Multiple sclerosis with negative oligoclonal band or increased count of leukocytes in cerebrospinal fluid were found in approximately 10% of patients with the disease. Because of the absence of oligoclonal band and less active cerebrospinal fluid, the diagnosis in these patients may be delayed.

Key words: *cerebrospinal fluid; immunoglobulins; leukocytes, mononuclear; multiple sclerosis*

APSTRAKT

NAJČEŠČE GREŠKE

- Previše podataka iz literature
- Navodjenje referenci
- Tabelarno i grafičko prikazivanje rezultata
- Korišćenje neuobičajenih skraćenica

UVOD

SADRŽAJ:

Kratak pregled prirode i značaja problema, pregled relevantne literature sa naglasakom na nepoznatom, pitanje koje se postavlja odnosno radna hipoteza, postupci koji su primenjeni.

(Glavni rezultati i zaključci)

STIL:

Sadašnje vreme za opšte prihvaćene istine

Prošlo vreme za sve ostalo

UVOD

Početi UVOD jasnim definisanjem polja istraživanja.
Posle kratkog i odmerenog pregleda literature
o činjenicama koje su poznate (sa citatima)
ukazati na aspekte problema koji su
nepoznati ili o kojima postoje
kontradiktorni podaci,
iza čega sledi cilj
istraživanja
u ovom
radu.

UVOD

NAJČEŠĆE GREŠKE

- Suvišna opširnost
- Definisanje opšte poznatih pojmova
- Navodjenje SVIH literaturnih podataka koji su u nekoj vezi sa predmetom rada
- Izostavljanje literaturnih podataka koji dovode u pitanje opravdanost istraživanja
- Nedostatak veze između navoda iz literature i cilja istraživanja

MATERIJAL I METODE

SADRŽAJ

Opisivanje postupaka primenjenih u istraživanju na takav način da se ono može u potpunosti reprodukovati

- Subjekti ispitivanja
- Eksperimentalni dizajn
- Eksperimentalne procedure
- Obrada podataka

MATERIJAL I METODE

STIL

- Upotreba podnaslova
- Prošlo vreme
- Koliko detaljno - Zavisi od metode:
klasične, kratko sa pozivanjem na izvor;
nove, specifične ili modifikovane, detaljno

MATERIJAL I METODE

NAJČEŠĆE GREŠKE

- Previše nepotrebnih detalja
- Premalo neophodnih podataka
- Prikazivanje rezultata

REZULTATI

SADRŽAJ:

- Prikaz ključnih rezultata istraživanja, koji sledi logiku sadržanu u hipotezi, pomoću teksta i ilustracija (tabela i slika: grafikona, fotografija, shema, etc).
- Tekst je sumarni i kritički prikaz nalaza koji prati ilustracije i poziva se na njih.
- I tekst i ilustracije moraju sadržati dovoljno podataka da se razumeju nezavisno jedni od drugih.
- Treba prikazati i negativne rezultate.

REZULTATI

Stil

- Prošlo vreme
- Upotreba podnaslova
- Tabele i slike moraju imati naslov (tabele iznad, slike ispod) i legendu
- Tabele i slike se obeležavaju posebno i to redom kojim se pojavljuju u tekstu

naslov tabele

Table 1. Clinical EAE in DA rats immunized with RSCH-PBS

Exp No	Incidence ^a	Day of onset ^b	Mean max score ^c
1	6/6	10.5±0.9	2.7±0.2
2	12/19	10.7±0.7	2.3±0.3
3	5/5	10.8±0.5	2.6±0.2
4	4/4	9.2±0.2	3±0
5	6/6	11±0.5	2.7±0.4
6	10/10	10.1±0.6	2.6±0.2
7	7/10	15.3±1	1.2±0.2
8	6/7	9.2±0.3	2.7±0.1

oznake kolona

podaci

fusnote

^a Number of rats with clinical signs of EAE/total number of rats

^b Mean day ± S.E.M. when first signs of EAE appeared

^c Mean maximal clinical score ± S.E.M. in diseased rats

oznaka y ose

oznaka x ose

naslov i
legenda

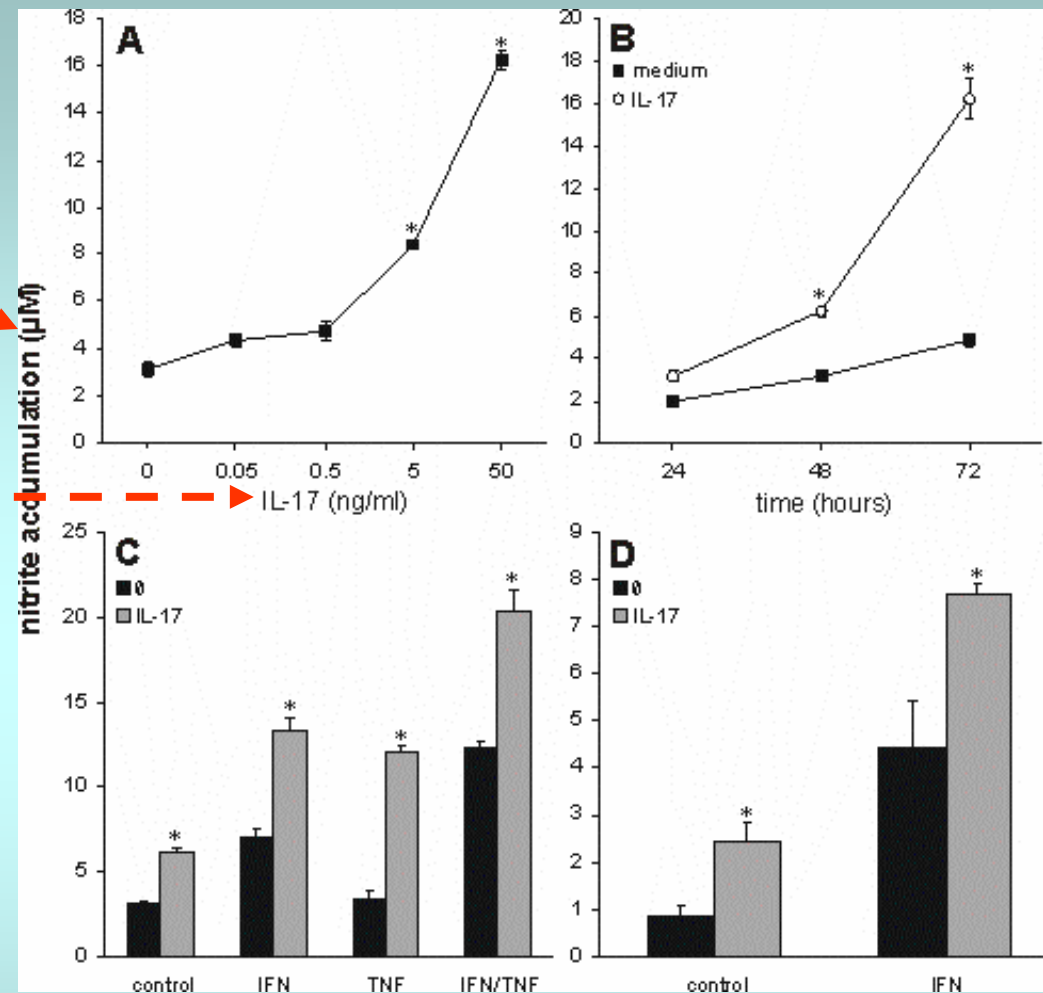


Fig. 1. IL-17 induces NO production in endothelial cells. Mouse VEC (1×10^5 /well) were incubated (A) with various concentrations of IL-17 for 72 h, or (B) or with IL-17 (50 ng/ml) for various time periods. (C) Mouse VEC (1×10^5 /well) were incubated for 48 h with or without IL-17 (50 ng/ml), in the absence (control) or presence of IFN- γ (200 U/ml), and/or TNF- α (100 U/ml). (D) Rat VEC (1×10^5 /well) were incubated for 48 hours with or without IL-17 (50 ng/ml) and/or IFN- γ (200 U/ml); * $p < 0.05$ refers to corresponding cultures without IL-17.

REZULTATI

NAJČEŠĆE GREŠKE

- Ilustracije nisu praćene tekstom
- Prikazivanje istih rezultata u tabeli i grafikonu
- Tabele koje sadrže isključivo statističke podatke
- Ilustracije nisu adekvatno obeležene
- Prikazivanje pojedinačnih, neobradjenih podataka
- Previše podataka u tabeli

DISKUSIJA

- Komentar svojih istraživanja bez ponavljanja pojedinačnih rezultata
- Interpretacija rezultata u svetlu poznatih činjenica
- Povezivanje svih pojedinačnih zaključaka u celinu
- Razmatranje teorijskih i praktičnih posledica novog saznanja
- Na kraju izvuci opšti zaključak o značenju rezultata

DISKUSIJA

Posle

sažimanja rezultata

uporedite ih sa nalazima drugih i

raspravite posledice vaših istraživanja;

pokažite šta je novo i kako se vaši rezultati uklapaju

u širu oblast; izvedite zaključke; predložite nova istraživanja.

DISKUSIJA

NAJČEŠĆE GREŠKE

- Zaključivanje koje nije zasnovano na rezultatima
- Neumerenost u isticanju značaja rezultata
- Ignorisanje ili obezvređivanje suprotnih rezultata
- Uvodjenje novih rezultata
- Preterana opširnost

LITERATURA - REFERENCE

Sistemi navodjenja literature:

- Vankuverski
- Harvardski
- kombinovani

LITERATURA

Vankuverski sistem

Cell death is part of normal development and maturation cycle, and is the component of many response patterns of living tissues to xenobiotic agents (i.e. micro organisms and chemicals) and to endogenous modulations, such as inflammation and disturbed blood supply (1,2). Cell death is an important variable in cancer development, cancer prevention and cancer therapy (3-5). In the treatment of cancer, the major approach is the removal, by surgery, of the neoplasm and/or the induction of cell death in neoplastic cells by radiation, toxic chemicals, antibodies and/or cells of the immune system (6-9). On the other hand, this pathobiological process remains poorly understood and the physiological and biochemical factors that lead to cell death are still not clear... ..

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LITERATURA

Harvardski sistem

Although data on TNF α levels in serum and CSF of MS patients are not consistent, a number of reports demonstrated cytokine increase during the phases of disease activity (Spuler et al., 1996; Drulovic et al., 1998) implying the significance of therapeutic approaches which would downregulate TNF α secretion or inactivate circulating TNF α . However, a pilot study in two rapidly progressive MS patients with a monoclonal anti-TNF antibody (cA2) unexpectedly revealed transiently increased magnetic resonance imaging activity with the increase of cells and immunoglobulins in CSF and no clinically significant neurologic changes (van Oosten et al., 1996). Furthermore, a phase III trial of lenercept, another TNF α inactivating agent was discontinued because of lack of efficacy (Weilbach and Gold, 1999). It has been also shown that inhibitor of TNF α synthesis, pentoxifylline lead to worsening of the disease in 12 of the 14 MS patients while the production of TNF α by monocytes was reduced (Myers et al., 1998).

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Frei, K., Eugster, H-P., Bopst, M., Constantinescu, C.S., Lavi, E. and Fontana, A., Tumor necrosis factor α and lymphotoxin α are not required for induction of acute experimental autoimmune encephalomyelitis, J. Exp. Med., 185 (1997) 2177-2182.