OREGON CLINICAL & TRANSLATIONAL *Research Institute*

OHSU

Writing and Presenting a Scientific Conference Abstract

DATE: December 9, 21 PRESENTED BY: Allison Fryer, PhD and Cynthia Morris, PhD

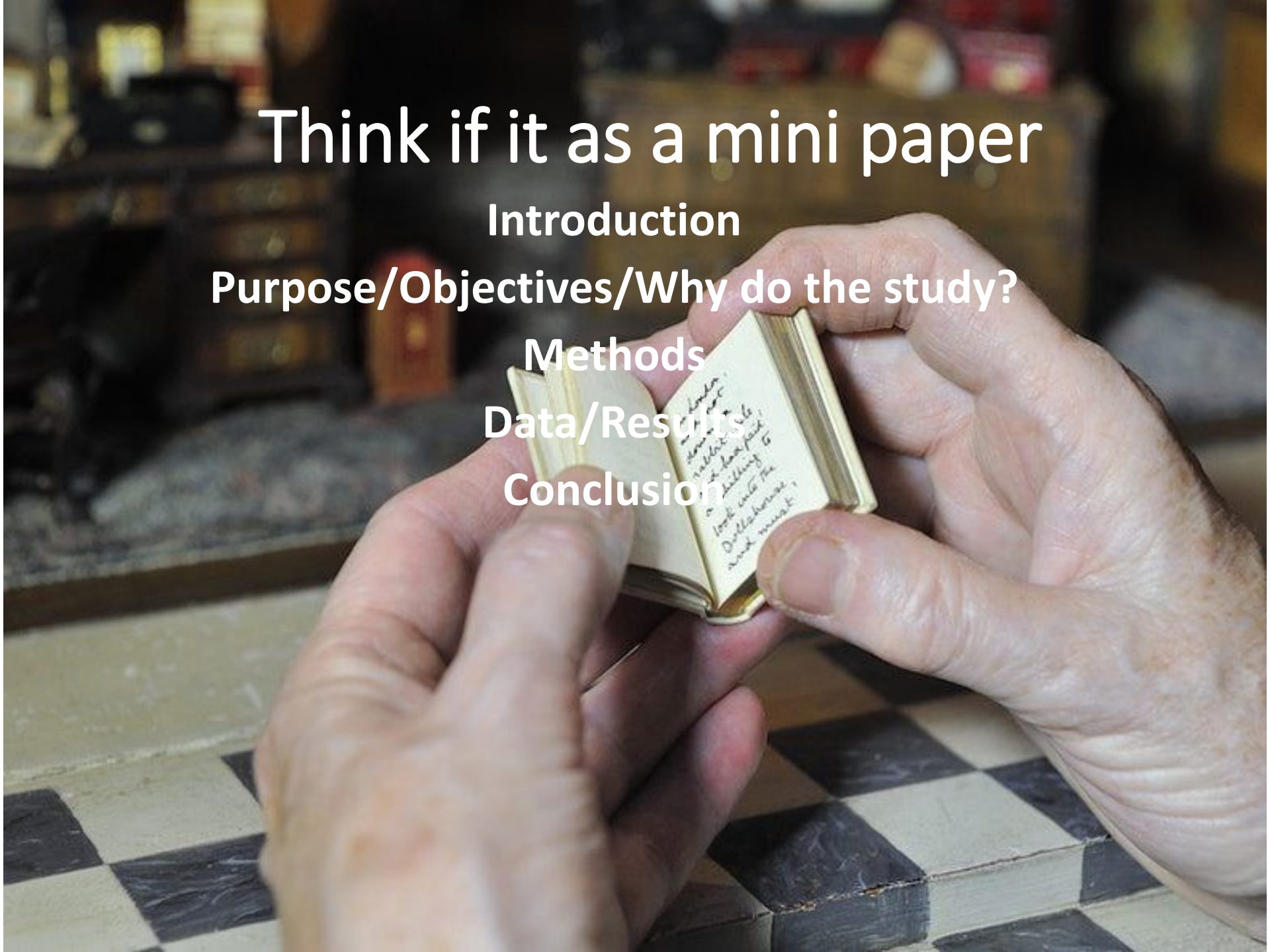
How to Write an Abstract

Allison Fryer, PhD

Associate Dean Graduate Studies


Abstracts are
IMPORTANT
INVITATION to read the rest of the paper.

After the title, the abstract is the second-most-read part of your article.


Think if it as a mini paper
Introduction
Purpose/Objectives/Why do the study?
Methods
Data/Results
Conclusion

It must stand alone

Antigen Sensitization Influences Organophosphorus Pesticide–Induced Airway Hyperreactivity

Becky J. Proskocil,[1] Donald A. Bruun,[2] Jesse K. Lorton,[1] Kirsten C. Blensly,[1] David B. Jacoby,[1,3] Pamela J. Lein,[2] and Allison D. Fryer[1]

[1]Department of Physiology and Pharmacology, [2]Center for Research on Occupational and Environmental Toxicology, and [3]Division of Pulmonary and Critical Care Medicine, Oregon Health & Science University, Portland, Oregon, USA

BACKGROUND: Recent epidemiologic studies have identified organophosphorus pesticides (OPs) as environmental factors potentially contributing to the increase in asthma prevalence over the last 25 years. In support of this hypothesis, we have demonstrated that environmentally relevant concentrations of OPs induce airway hyperreactivity in guinea pigs.

OBJECTIVES: Sensitization to allergen is a significant contributing factor in asthma, and we have shown that sensitization changes virus-induced airway hyperreactivity from an eosinophil-independent mechanism to one mediated by eosinophils. Here, we determine whether sensitization similarly influences OP-induced airway hyperreactivity.

METHODS: Nonsensitized and ovalbumin-sensitized guinea pigs were injected subcutaneously with the OP parathion (0.001–1.0 mg/kg). Twenty-four hours later, animals were anesthetized and ventilated, and bronchoconstriction was measured in response to either vagal stimulation or intravenous acetylcholine. Inflammatory cells and acetylcholinesterase activity were assessed in tissues collected immediately after physiologic measurements.

RESULTS: Ovalbumin sensitization decreased the threshold dose for parathion-induced airway hyperreactivity and exacerbated parathion effects on vagally induced bronchoconstriction. Pretreatment with antibody to interleukin (IL)-5 prevented parathion-induced hyperreactivity in sensitized but not in nonsensitized guinea pigs. Parathion did not increase the number of eosinophils in airways or the number of eosinophils associated with airway nerves nor did it alter eosinophil activation as assessed by major basic protein deposition.

CONCLUSIONS: Antigen sensitization increases vulnerability to parathion-induced airway hyperreactivity and changes the mechanism to one that is dependent on IL-5. Because sensitization to allergens is characteristic of 50% of the general population and 80% of asthmatics (including children), these findings have significant implications for OP risk assessment, intervention, and treatment strategies.

KEY WORDS: airway hyperreactivity, asthma, atopy, eosinophils, organophosphorus pesticides, parathion, sensitization. *Environ Health Perspect* 116:381–388 (2008). doi:10.1289/ehp.10694 available via *http://dx.doi.org/* [Online 2 January 2008]

Asthma and antigen challenge–induced airway hyperreactivity are characterized by airway eosinophilia (Adamko et al. 2005; Wenzel 2003). Antigen sensitization, without challenge, causes eosinophils to cluster around airway nerves in guinea pigs (Adamko et al. 2003), a finding also reported in human asthma (Costello et al. 1997). Antigen inhalation activates eosinophils, causing release of major basic protein that binds to and inhibits neuronal M2 muscarinic receptors leading to airway hyperreactivity (Fryer and Jacoby 1992; Jacoby et al. 1993). Antigen sensitization may also influence airway hyperreactivity triggered by other factors. For example, viral infection, which exacerbates asthma (Johnston [illegible] Nicholson et al. 1993), causes airway hyperreactivity by inhibiting neuronal M2 receptor function (Fryer and Jacoby 1991). In nonsensitized animals, virus-induced hyperreactivity does not require the presence of eosinophils around airway nerves (Adamko et al. 1999); however, with sensitization, the mechanism of virus-induced hyperreactivity changes to become eosinophil dependent (Adamko et al. 1999). Here we show that prior sensitization to antigen not only changes the mechanism of organophosphorus pesticide-induced airway hyperreactiv-

Introduction

Purpose/Why did you do the study?

Methods

Data

Conclusion

Dissecting an Abstract

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Airway Hyperreactivit...

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1 ɔal, one point

ɜt you did and how you did it
ıt them to remember?

500 word limit

- **A Morphological and Genetic Analysis of *Polistes versicolor*: the Paper Wasp Invading the Galápagos Islands**

  - OHSU Student, OHSU Student, Faculty member
    OHSU, Portland Oregon

- *Polistes versicolor*, a wasp native to Ecuador, has only recently invaded the Galápagos Islands. This invasion may have put us in position to explore evolution as it occurs, but only if we collect data as the invasion progresses. With preliminary evidence suggesting *P. versicolor* body characteristics vary with elevation, we gathered ecological, morphological, and genetic data during the early phase of this invasion. Individuals (n = 714) from the Ecuador mainland and six different island regions were collected in 2007 and 2008. Head, wing, and leg measurements were gathered. DNA was extracted and cataloged for each animal, PCR was performed on mainland and select island individuals, and loci were examined by polyacrylamide gel electrophoresis. We found significant morphological differences in relation to elevation. Data suggests that larger heads, smaller wings, and smaller legs are seen at higher elevations. Highly polymorphic loci have also been isolated for mainland individuals. Preliminary genetic data suggests that island-specific reductions in genetic diversity may have occurred and such limited variation supports morphological plasticity. These data will serve as a reference in morphological and genetic analyses over time to decipher whether plasticity or evolution is driving such differences.

Introduction

This covers what you were trying to achieve, e.g. to address an ongoing debate or problem, or some gap you found in the literature,

Keep in mind:

WHY should READER CARE?

Introduction: Background

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Objective

This is where you say what YOUR research covered.

This will be a narrower focus than what was covered in the background.

What specific question did YOU answer.

Objective: Why should I care?

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Methods
Results

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Results

(starts with we found, have shown…)

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Conclusion

This is what you concluded from your research, ie your ideas on what was happening or why it happened or how it relates to other research in the area.

Note: not plural conclusions

Conclusion

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nature

How to construct a *Nature* summary paragraph

Annotated example taken from *Nature* **435**, 114-118 (5 May 2005).

One or two sentences providing a ***basic introduction*** *to the field, comprehensible to a scientist in any discipline.*

Two to three sentences of ***more detailed background****, comprehensible to scientists in related disciplines.*

One sentence clearly stating the ***general problem*** *being addressed by this particular study.*

One sentence summarising the main result (with the words ***"here we show"*** *or their equivalent).*

Two or three sentences explaining what the main result reveals in direct comparison to what was thought to be the case previously, or how the main result adds to previous knowledge.

One or two sentences to put the results into a more general context.

Two or three sentences to provide a broader perspective, readily comprehensible to a scientist in any discipline, may be included in the first paragraph if the editor considers that the accessibility of the paper is significantly enhanced by their inclusion. Under these circumstances, the length of the paragraph can be up to 300 words. (The above example is 190 words without the final section, and 250 words with it).

During cell division, mitotic spindles are assembled by microtubule-based motor proteins[1,2]. The bipolar organization of spindles is essential for proper segregation of chromosomes, and requires plus-end-directed homotetrameric motor proteins of the widely conserved kinesin-5 (BimC) family[3]. Hypotheses for bipolar spindle formation include the 'push–pull mitotic muscle' model, in which kinesin-5 and opposing motor proteins act between overlapping microtubules[2,4,5]. However, the precise roles of kinesin-5 during this process are unknown. Here we show that the vertebrate kinesin-5 Eg5 drives the sliding of microtubules depending on their relative orientation. We found in controlled *in vitro* assays that Eg5 has the remarkable capability of simultaneously moving at ~20 nm s^{-1} towards the plus-ends of each of the two microtubules it crosslinks. For anti-parallel microtubules, this results in relative sliding at ~40 nm s^{-1}, comparable to spindle pole separation rates *in vivo*[6]. Furthermore, we found that Eg5 can tether microtubule plus-ends, suggesting an additional microtubule-binding mode for Eg5. Our results demonstrate how members of the kinesin-5 family are likely to function in mitosis, pushing apart interpolar microtubules as well as recruiting microtubules into bundles that are subsequently polarized by relative sliding. We anticipate our assay to be a starting point for more sophisticated *in vitro* models of mitotic spindles. For example, the individual and combined action of multiple mitotic motors could be tested, including minus-end-directed motors opposing Eg5 motility. Furthermore, Eg5 inhibition is a major target of anti-cancer drug development, and a well-defined and quantitative assay for motor function will be relevant for such developments.

Am J Respir Cell Mol Biol. 2013 Feb 28. [Epub ahead of print]

TLR2/6 and TLR9 Agonists Suppress Viral Replication but not Airway Hyperreactivity in Guinea Pigs.

Drake MG, Evans SE, Dickey BF, Fryer AD, Jacoby DB.

Pulmonary and Critical Care, Oregon Health and Science University, Portland, Oregon, United States.

Abstract

Respiratory virus infections cause airway hyperreactivity (AHR). Preventative strategies for virus-induced AHR remain limited. Toll-like receptors (TLRs) have been suggested as a therapeutic target due to their central role in triggering antiviral immune responses. Prior studies have shown concurrent treatment with TLR2/6 and TLR9 agonists reduces lethality and microbial burden in murine models of bacterial and viral pneumonia. This study investigated the affects of TLR2/6 and TLR9 agonist pretreatment on parainfluenza virus pneumonia and virus-induced AHR in guinea pigs in vivo. Synthetic TLR2/6 lipopeptide agonist Pam2CSK4 and class C oligodeoxynucleotide TLR9 agonist ODN2395 given in combination 24 hours before virus infection significantly reduced viral replication in the lung. Despite a 5-fold reduction in viral titers, concurrent TLR2/6 and TLR9 agonist pretreatment did not prevent virus-induced AHR or virus-induced inhibitory M2 muscarinic receptor dysfunction. Interestingly, the TLR agonists independently caused non-M2-dependent AHR. These data confirm the therapeutic antiviral potential of TLR agonists, while suggesting virus inhibition may be insufficient to prevent virus-induced airway pathophysiology. Furthermore, TLR agonists independently cause AHR, albeit through a distinctly different mechanism from parainfluenza virus.

Follow Directions

every journal/meeting is different

Limits by space, words, characters,
tables, figures text, references?

Is presenting author first, underlined?

Paragraphs indented, justified, headings?
Font ±serif and size?

76

M_1-CHOLINOCEPTORS MEDIATE THE PRESSOR RESPONSES TO ELECTRICAL AND CHEMICAL STIMULATION OF AMYGDALA IN CONSCIOUS RATS

Ş. Oktay, N. Aslan, Z. Gören,U. Özkutlu, F. Onat. Departments of Pharmacology & Physiology, Marmara University, School of Medicine, Haydarpaşa, 81326, İstanbul, TURKEY.

The effects of the electrical and chemical stimulation of the central nucleus of the amygdaloid complex (CNA) on mean arterial blood pressure (MAP) were investigated in conscious, unrestraint Sprague Dawley rats. The electrical stimulation of the CNA led to reproducible increases (29.1±5.6 mm Hg) in MAP. Atropine, pirenzepine or AF-DX 116 given into CNA blocked these blood pressure changes dose-dependently. Atropine and pirenzepine were found to be equipotent in this regard (ID_{50}=1.05 and 0.23 nmol, respectively) whereas AF-DX 116 was 140.3 times less potent than pirenzepine (ID_{50}=39.5 nmol). The chemical stimulation of CNA by 5 nmol carbachol caused 21.0±3.5 mm Hg increase in MAP. Pirenzepine (icv) inhibited carbachol-induced pressor responses dose-dependently (ID_{50}=7.99 nmol) whereas AF DX 116 did not significantly alter those responses at 100 nmol. The high potency of pirenzepine compared with AF DX 116 indicates the involvement of M_1 muscarinic receptors located at CNA in the pressor responses induced by both the electrical and the chemical stimulation of this area.

This study was supported by Scientific & Technical Research Council of Turkey (Project # SBAG-1250).

77

THE EFFECTS OF MUSCARINIC CHOLINERGIC ANTAGONISTS AND AGONISTS ON PREPULSE INHIBITION OF THE ACOUSTIC STARTLE REFLEX.

C.K. Jones and H.E. Shannon. Program of Medical Neurobiology, Indiana University Medical Center and Lilly Research Laboratories, Eli Lilly and Company, Indianapolis, IN. 46285.

Prepulse Inhibition (PPI), the reduction in startle response produced by presentation of a substartling prepulse stimulus, is disrupted in schizophrenics. Lesioning of cholinergic neurons in the brainstem reticular formation has also resulted in disruption of PPI (Koch et al., 1993), suggesting a possible cholinergic involvement in both normal sensorimotor gating and the pathophysiology of schizophrenia. In the present study, male Sprague-Dawley rats were administered systemic or intracerebroventricular injections of muscarinic antagonists and agonists to determine if dose-dependent effects on startle amplitude and PPI would be observed. Test sessions consisted of counterbalanced presentations of the following four trial types: no stimulus, startle pulse alone (106 db), prepulse alone (77 db), and prepulse+pulse. Nonselective muscarinic antagonists, such as scopolamine, benztropine, and trihexyphenidyl produced a significant dose-dependent disruption of PPI with no significant effect on startle amplitude. The muscarinic agonists oxotremorine, pilocarpine, RS-86, and arecoline had no effect on PPI. However, each agonist caused a significant decrease in startle amplitude at the highest dose tested. In further experiments, the interstimulus interval (ISI) between the prepulse and startle stimuli was varied from 30 to 1000 msecs. The nonselective, muscarinic antagonists disrupt PPI in an ISI-dependent manner. The present findings suggest that the muscarinic cholinergic system may be involved in the characteristic cognitve flooding and fragmentation observed in schizophrenia.

AJS 2000
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EXPRESSION OF INTRACELLULAR ADHESION MOLECULE (ICAM) BY GUINEA PIG PARASYMPATHETIC NERVES IN CULTURE.

DA Sawatzky, B Kumaravel, RW Costello, DB Jacoby, AD Fryer. Johns Hopkins University, Baltimore MD and University of Liverpool, Liverpool, England.

In the lungs, the parasympathetic nerves provide the primary autonomic control of airway smooth muscle. In antigen challenged, but not in control guinea pigs, eosinophils are found within nerve bundles, clustered around the parasympathetic ganglia, and are associated with postganglionic nerve fibers [Am J Physiol. 1997 273:L93]. Eosinophils adhere to cells via interactions with adhesion molecules, one of which is intracellular adhesion molecule (I-CAM). Therefore, we tested whether guinea pig parasympathetic nerves can express ICAM. Guinea pigs were killed and their trachess removed. The tracheas were homogenized and incubated with collagenase to disassociate the cells. Macrophages and smooth muscles were removed by preplating on plastic, and the remaining cells grown on matrigel coated slides, in serum free medium in the presence of cytosine arabinoside to prevent cell division. One week later, nerves had adhered to the plates and were growing neurites. Some cell cultures were incubated with tumor necrosis factor (TNF-α; 2ng/ml) and interferon-γ (INF-γ; 1000U/ml) for 48 hours. Cells were then washed and incubated with AbICAM. Binding was detected using a secondary biotinylated antibody. Cells not incubated with TNF-α /INF-γ did not express labeling with the AbICAM. In contrast, parasympathetic nerves incubated with TNF-α /INF-γ did express labeling of ICAM on the cell bodies and along some of the neurites. Addition of guinea pig eosinophils, obtained by peritoneal lavage demonstrated adhesion of the eosinophils to the nerves exposed to TNF-α /INF-γ, with less adherence to the non-stimulated nerves. These results demonstrate that parasympathetic nerves are capable of expressing ICAM, and that eosinophils will adhere to the nerves, possibly through adhesion to ICAM.

American Heart Association, HL54659, HL55543, HL61013, HL10342

This abstract is funded by:

LYSOPHOSPHATIDYLCHOLINE INDUCES EOSINOPHIL RECRUITMENT TO LUNG

O. Nishiyama, H. Kume, Y. Ito, R. Suzuki, and K. Yamaki
Second Department of Internal Medicine, School of Medicine, Nagoya University, Nagoya 466-8560, Japan.

Lysophosphatidylcholine (lyso PC), a natural product of phospholipase A2 (PLA2) metabolism, is considered to be involved in pathogenesis of asthma, because previous reports have indicated that its plasma level correlated with airway sensitivity, and that its level in bronchoalveolar lavage fluid (BALF) markedly increased in patients with asthma. Moreover, lyso PC is considered to induce adhesion molecule such as ICAM-1 and VCAM-1 in arterial endothelial cells. Thus, lyso PC is likely to have important role in asthma, however, little is currently known about the machanism. In this study we examined the effects of lyso PC on airway inflammation in guinea pigs. Hartley guinea pigs (250-300g) were placed in a box for exposure to aerosolized lyso PC generated from 0.5mg/ml solution for 10 minutes, using an ultra nebulizer. BALF and histological examination were performed 6h and 24h after the exposure. On the other hand, the animals were exposed to aerosolized phosphatidylcholine (PC) in the same procedure as control. Significant increases in total number of inflammatory cells and percentage of eosinophil in BALF were observed 6h and 24h after lyso PC exposure, respectively (p<0.0001). In histological examinations, number of eosinophils surrounding the trachea, bronchi, and bronchioles significantly increased 6h and 24h after lyso PC exposure, respectively (p=0.01 to <0.0001). These results indicates that lyso PC may have important role in recruitment of eosinophils to lung. It is probable that adhesion molecules are involved in the eosinophil recruitment by lyso PC.

EliCell: A NOVEL HIGH-SENSITIVE METHOD TO DETECT CYTOKINE RELEASE BY EOSINOPHILS.

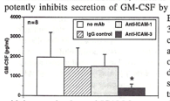
Bandeira-Melo, C., Gillard, G. & Weller, P.F. Div. Allergy and Inflammation, Dept. Medicine, Beth Israel Deaconess Medical Center, Harvard Medical School.

Eosinophils have roles in health and allergic conditions. In addition to their content of distinctive granular cationic proteins, eosinophils are also sources of multiple preformed cytokines. Thus, in clear contrast with lymphocytes, eosinophils have the potential to promptly release preformed cytokines. However, the mechanisms regulating such release are unknown and sensitive techniques for this study are not available. To suitably study the mobilization of stored eosinophil cytokines, the method should not (i) dilute low levels of released proteins; (ii) permeabilize viable cells; or (iii) require large number of eosinophils. In addition, it should be capable of detecting focal release at the cell surface. Here, using a novel strategy termed EliCell, we could detect RANTES release by human blood eosinophils at the single cell level. Briefly, highly purified viable eosinophils were embedded in a gel matrix containing capture-antibody to human RANTES and incubated with appropriate stimuli (e.g. IFN-γ) for varying durations (37°C; 5% CO_2). Thereafter, eosinophils without membrane permeabilization were incubated with fluorochrome-labeled anti-RANTES to detect the chemokine release. Analysis by fluorescence microscopy revealed that IFN-γ induced a dose- and time-dependent focal RANTES release with a punctate immunostaining pattern. In direct comparisons, EliCell assays were able to detect significant IFN-γ-induced RANTES release earlier and with lower concentrations of stimulus than ELISAs of eosinophil supernatants. Thus, EliCell is a sensitive method to evaluate cytokine release by eosinophil piecemeal degranulation, a process by which just very small amounts of granule contents are mobilized. *Financial support:* CAPES (Brazil) and NIH (AI20241, HL56386).

ICAM-3 INHIBITS GM-CSF SECRETION BY EOSINOPHILS

JM Kessel. University of Wisconsin, Madison, WI, USA

The leukocyte adhesion molecule ICAM-3 has high level and constitutive expression on the surfaces of freshly isolated and primed human eosinophils (EOS), but its biological relevance is not known. To test the hypothesis that ICAM-3 regulates cytokine secretion from human EOS, a monoclonal antibody (mAb) was used to activate this receptor. To evaluate this possibility, isolated EOS were incubated with ionomycin to stimulate GM-CSF release (10^{-6} M for 18 hours). These cells were also treated with anti-ICAM-3 or anti-ICAM-1 mAbs. An anti-ICAM-3 mAb that recognizes an epitope in the extracellular domain of ICAM-3, which is critical for recognition by the ligand LFA-1, potently inhibits secretion of GM-CSF by ionomycin-stimulated EOS (figure, +/-SD).



EOS incubated in the presence of anti-ICAM-3 mAb secrete significantly less GM-CSF compared to EOS incubated with no antibody, an isotype-matched mAb specific for ICAM-1 or non-immune IgG1 (p<0.05). We next determined whether inhibition of GM-CSF secretion was because anti-ICAM-3 mAb triggered EOS apoptosis. EOS were incubated with ionomycin plus anti-ICAM-3 or control antibodies(18 hrs). None of these treatments induced apoptosis or cell death, as determined by staining with propidium iodide or Hoerchst reagent(data not shown). These experiments suggest that engagement of ICAM-3 triggers signaling pathways that inhibit GM-CSF secretion from ionomycin-stimulated EOS. Our findings also suggest that in activated EOS, signaling via ICAM-3 may be one possible mechanism for suppressing transcription, translation, or secretion of GM-CSF, a cytokine that primes EOS and prolongs their survival. ICAM-3 may function to regulate EOS-dependent inflammation in asthma. (abstract funded by UW Graduate School)

HEPARIN-BINDING EPIDERMAL GROWTH FACTOR-LIKE GROWTH FACTOR (HB-EGF) EXPRESSION ON HUMAN EOSINOPHILS.

K. Sugiyama, Bandeira-Melo C., Jin Z. and Weller P.F. Div. Allergy and Inflammation, Dept. Med., Beth Israel Deaconess Medical Center, Harvard Medical School, Boston, MA, USA

Eosinophils release some fibrogenic cytokines, and are thought to be important in fibrosis. HB-EGF is a fibrogenic cytokine, and we studied HB-EGF expression by eosinophils. Human eosinophils were isolated by MACS system (3~5 donors for each experiment), and incubated for 0, 2, 4 or 18hrs with eotaxin (100ng/ml). By immunocyto-chemistry and FACS analysis, with and without detergent permeabilization, all freshly isolated eosinophils contained preformed intracellular HB-EGF, but did not express membrane HB-EGF. Incubations with eotaxin for 18hrs increased levels of intracellular HB-EGF and induced surface membrane HB-EGF expression (Table), but did not release free HB-EGF in supernatants (as assessed by ELISA). By RT-PCR, eotaxin also increased HB-EGF mRNA at 2hrs. In conclusion, eosinophils store HB-EGF constitutively, and synthesize HB-EGF under eotaxin stimulation. Eotaxin-stimulated eosinophils express HB-EGF on their cell membranes. It is suggested that eosinophils may contribute to fibrosis by juxtacrine contact with fibrogenic cells.

	immunocytochemistry (% of eos)				FACS (%mean fluorescence above control)			
	intracellular		membrane		intracellular		membrane	
fresh cells	33±4		0±0		30±14		1±11	
18h med. alone	28±3	**	5±2	**	69±19	*	17±22	**
18h eotaxin	62±6	**	57±8	**	153±56	*	81±15	**

mean±S.E. * : p<0.05 ** : p<0.01

This abstract is funded by: NIH: HL56386, AI20241

THE PROLIFERATION OF CULTURED HUMAN AIRWAY SMOOTH MUSCLE CELLS INDUCED BY EOSINOPHILS.

Kazuko Masu, Isao Ohno, Shinji Okada, Katsunori Kumagai, Katsutoshi Hayashi, Kunio Shirato. First Department of Internal Medicine, Tohoku University School of Medicine, Sendai, Japan.

The hypertrophy/hyperplasia of airway smooth muscle (ASM) cells is one of the characteristic features of bronchial asthma. However, the pathogenesis of this structural change has not been determined yet. Eosinophils, which play a pathogenic role in asthma, have been demonstrated to have proliferative activity for fibroblasts and vascular smooth muscle cells. To investigate the potential of eosinophils to induce the proliferation of ASM cells, we examined the effect of lysates of eosinophils purified from peripheral blood of healthy donors on human ASM cell proliferation. Eosinophil lysates significantly induced ASM cell proliferation in time-and dose-dependent manners, reaching a maximum on day 6 at 50% eosinophil lysates ($6.0\pm0.7 \times 10^4$ [mean ± SD] /well, n=5 vs $4.5\pm1.1 \times 10^4$/well, n=5; p<0.05). This proliferative activity was heat-sensitive and recovered in the soluble fraction of the eosinophil lysates. The number of cells cultured with eosinophil lysates were reduced to the level of control by anti-IGF-II antibody (88.7±6.1% (n=3) of control). Although neither anti-PDGF nor anti-TNF-α antibody had a significant effect on the proliferative activity of the eosinophil lysates, the combination of these antibodies completely abolished the activity (102.7±1.4%, n=3). These results suggest that eosinophils might contribute to the hyperplasia of ASM cells in asthmatics through the release of pre-formed protein(s) which stimulate the release of IGF-II, PDGF and TNF-α capable to induce ASM cell proliferation.

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RESEARCH PAPER

Non-bronchodilating mechanisms of tiotropium prevent airway hyperreactivity in a guinea-pig model of allergic asthma

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Keywords
anticholinergic; tiotropium; atropine; muscarinic receptors; parasympathetic nerves; airway hyperreactivity; eosinophil; asthma

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BACKGROUND AND PURPOSE

Asthma is characterized by reversible bronchoconstriction and airway hyperreactivity. Although M_3 muscarinic receptors mediate bronchoconstriction, non-selective muscarinic receptor antagonists are not currently recommended for chronic control of asthma. We tested whether selective blockade of M_3 receptors, at the time of antigen challenge, blocks subsequent development of airway hyperreactivity in antigen-challenged guinea-pigs.

EXPERIMENTAL APPROACH

Ovalbumin-sensitized guinea-pigs were pretreated with 1 $\mu g \cdot kg^{-1}$ of a kinetically selective M_3 receptor antagonist, tiotropium, or 1 $mg \cdot kg^{-1}$ of a non-selective muscarinic receptor antagonist, atropine, and challenged with inhaled ovalbumin. Animals were anaesthetized, paralyzed, ventilated and vagotomized 24 h later. We measured vagally mediated bronchoconstriction and i.v. ACh-induced bronchoconstriction.

KEY RESULTS

Electrical stimulation of both vagus nerves induced frequency-dependent bronchoconstriction in sensitized animals that was significantly increased after antigen challenge. Antigen-induced hyperreactivity was completely blocked by tiotropium pretreatment but only partially blocked by atropine pretreatment. Surprisingly, although tiotropium blocked bronchoconstriction induced by i.v. ACh, it did not inhibit vagally-induced bronchoconstriction in sensitized controls, suggesting that tiotropium does not block hyperreactivity by blocking receptors for vagally released ACh. Rather, tiotropium may have worked through an anti-inflammatory mechanism, since it inhibited eosinophil accumulation in the lungs and around nerves.

CONCLUSIONS AND IMPLICATIONS

These data confirm that testing M_3 receptor blockade with exogenous ACh does not predict vagal blockade. Our data also suggest that selective blockade of M_3 receptors may be effective in asthma via mechanisms that are separate from inhibition of bronchoconstriction.

Abbreviations

i.s., insufflated

Introduction

In the lungs, airway smooth muscle tone is controlled by ACh released from parasympathetic nerves. ACh stimulates M_3 muscarinic receptors on airway smooth muscle to induce smooth muscle contraction, resulting in bronchoconstriction. Parasympathetic nerves not only maintain airway tone, but they also mediate reflex bronchoconstriction (Canning, 2006). Diverse chemical and physical stimuli, including inhaled antigens, histamine and cold air, activate afferent sensory nerves in the lungs to initiate a reflex that increases ACh release from parasympathetic nerves and

OPEN ACCESS Freely available online

PLoS one

Eosinophils Increase Neuron Branching in Human and Murine Skin and *In Vitro*

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Abstract

Cutaneous nerves are increased in atopic dermatitis, and itch is a prominent symptom. We studied the functional interactions between eosinophils and nerves in human and mouse skin and in culture. We demonstrated that human atopic dermatitis skin has eosinophil granule proteins present in the same region as increased nerves. Transgenic mice in which interleukin-5 (IL-5) expression is driven by a keratin-14 (K14) promoter had many eosinophils in the epidermis, and the number of nerves was also significantly increased in the epidermis. In co-cultures, eosinophils dramatically increased branching of sensory neurons isolated from the dorsal root ganglia (DRG) of mice. This effect did not occur in DRG neurons co-cultured with mast cells or with dead eosinophils. Physical contact of the eosinophils with the neurons was not required, and the effect was not blocked by an antibody to nerve growth factor. DRG neurons express eotaxin-1, ICAM-1 and VCAM-1, which may be important in the recruitment, binding, and activation of eosinophils in the region of cutaneous nerves. These data indicate a pathophysiological role for eosinophils in cutaneous nerve growth in atopic dermatitis, and suggest they may present a therapeutic target in atopic dermatitis and other eosinophilic skin conditions with neuronal symptoms such as itch.

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Introduction

Atopic dermatitis is characterized by itch, which greatly affects the quality of life of patients [Jacquet, 1904 and [1]]. The itch often begins before any lesions appear, and marks on the skin can be limited to excoriations, or scratches, made by the patient. Patients with atopic dermatitis experience itch instead of pain when tested with mechanical, electrical, low pH, or heat stimuli [2]. The sensory neurons that transmit itch are primary afferents whose cell bodies are in the dorsal root ganglia (DRG). These free nerve endings in the epidermis and upper dermis can be activated by a variety of stimuli, including proteases, neurotrophins, cytokines and other small molecules (reviewed in [3]).

The mechanisms for enhanced itch sensations in atopic dermatitis are unclear. One potential mechanism is an increase in nerve endings in atopic dermatitis skin [4]. Specifically, there are more nerve fibers in the papillary and upper dermis as disease progresses from clinically normal-appearing, or *non-lesional*, skin to active disease, or *lesional*, skin [5].

Eosinophils have been linked to atopic dermatitis for well over forty years, due to high numbers circulating in the blood of atopic dermatitis patients [6]. Serum concentrations of eosinophil granule proteins correlate with the severity of atopic dermatitis [7,8], and peripheral blood eosinophils from patients have more neurotrophin receptors and more functional activity in response to neurotrophins than eosinophils from healthy controls [9]. Intact eosinophils are not found in high numbers in atopic dermatitis biopsies, leading some to question whether these cells have a role in the pathogenesis of this disease. However, the presence of eosinophil granule proteins in lesional skin suggests that activated eosinophil are present, but not identifiable, having degranulated [10,11].

Previously, we showed that eosinophils interact with nerves in the airways of patients with asthma, in animal models of asthma, and in culture. Eosinophils are found adjacent to nerves in airway biopsies of humans who died from asthma attacks, a histological finding that is recapitulated in antigen-challenged guinea pigs and rats [12]. Primary cultures of parasympathetic neurons from guinea pig and human airways express eotaxin-1, as well as ICAM-1 and VCAM-1, and these participate in binding of eosinophils to neurons [13,14]. The association of eosinophils with airway nerves is important in the pathophysiological changes that lead to airway hyperreactivity [12,15].

Eosinophils communicate with other cell types through a variety of specific signals. They can act as antigen-presenting cells, release cytokines and chemokines after activation, or release granule proteins, such as major basic protein (MBP) and eosinophil peroxidase (EPO) [16,17]. They also constitutively synthesize specific neurotrophins, including nerve growth factor (NGF), brain-derived neurotrophic factor (BDNF), and neurotrophin-3

Special Considerations
for Meeting/Abstracts.

-limit points to…1/ONE/a single.

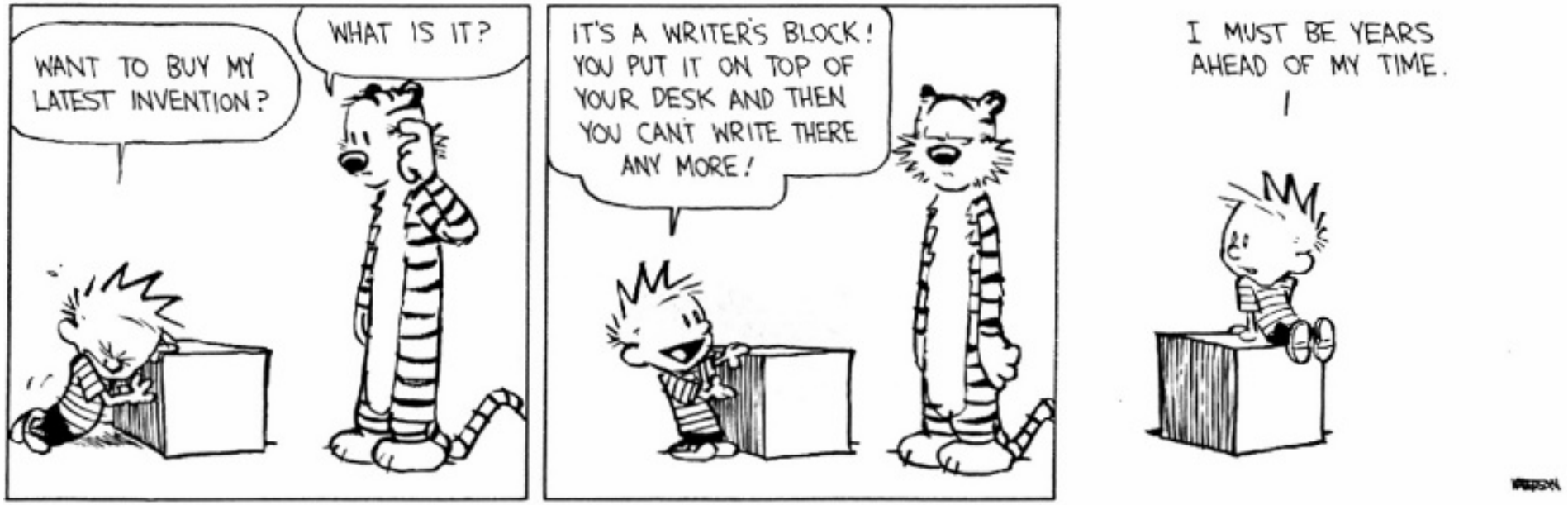
-method include enough details to allay ethical concerns (IRB approval).

3. Steps
to an Abstract

1. Write a lot

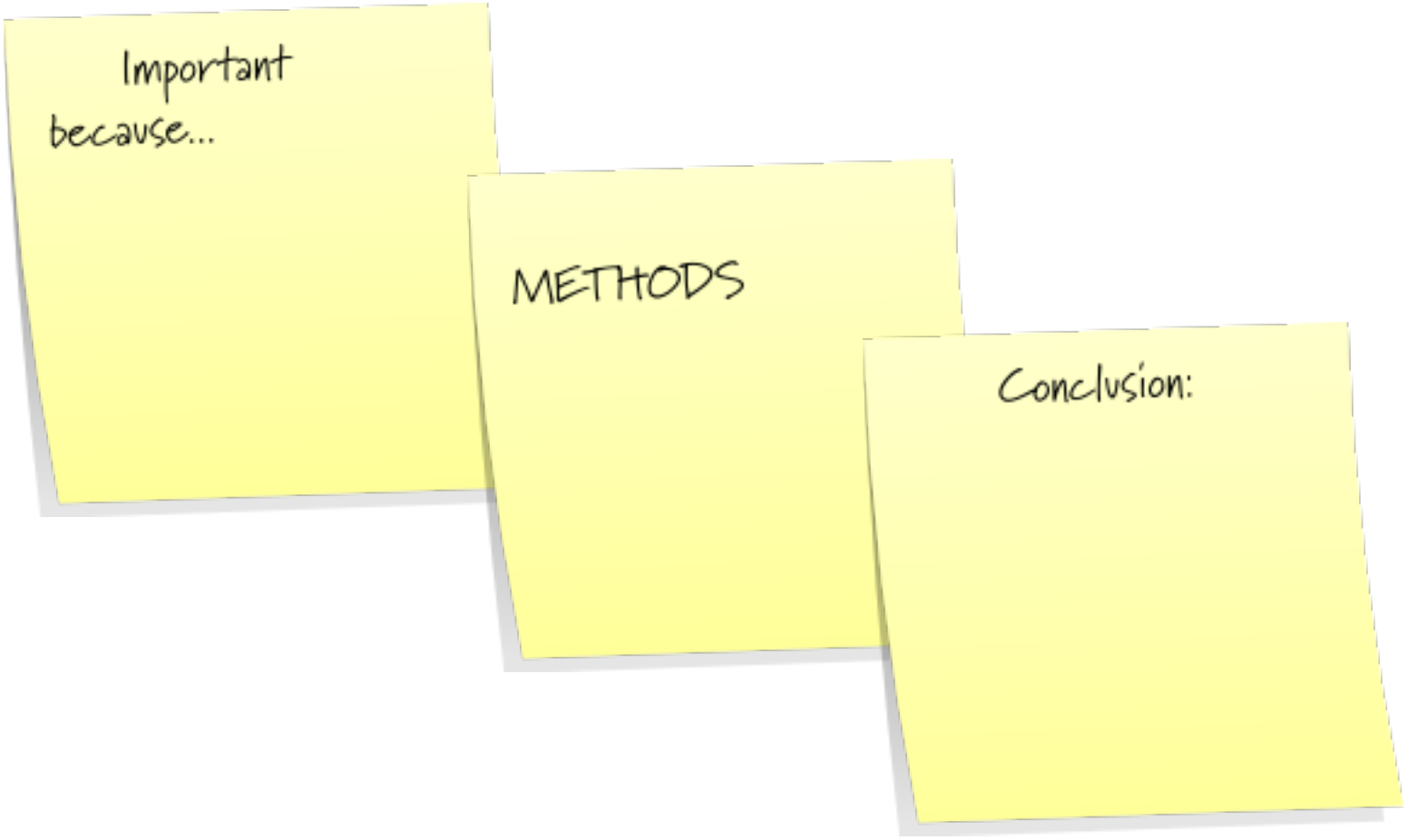
It is easiest to write a lot at the start.

Overcome ‘blank paper syndrome’ -write everything you want to say.

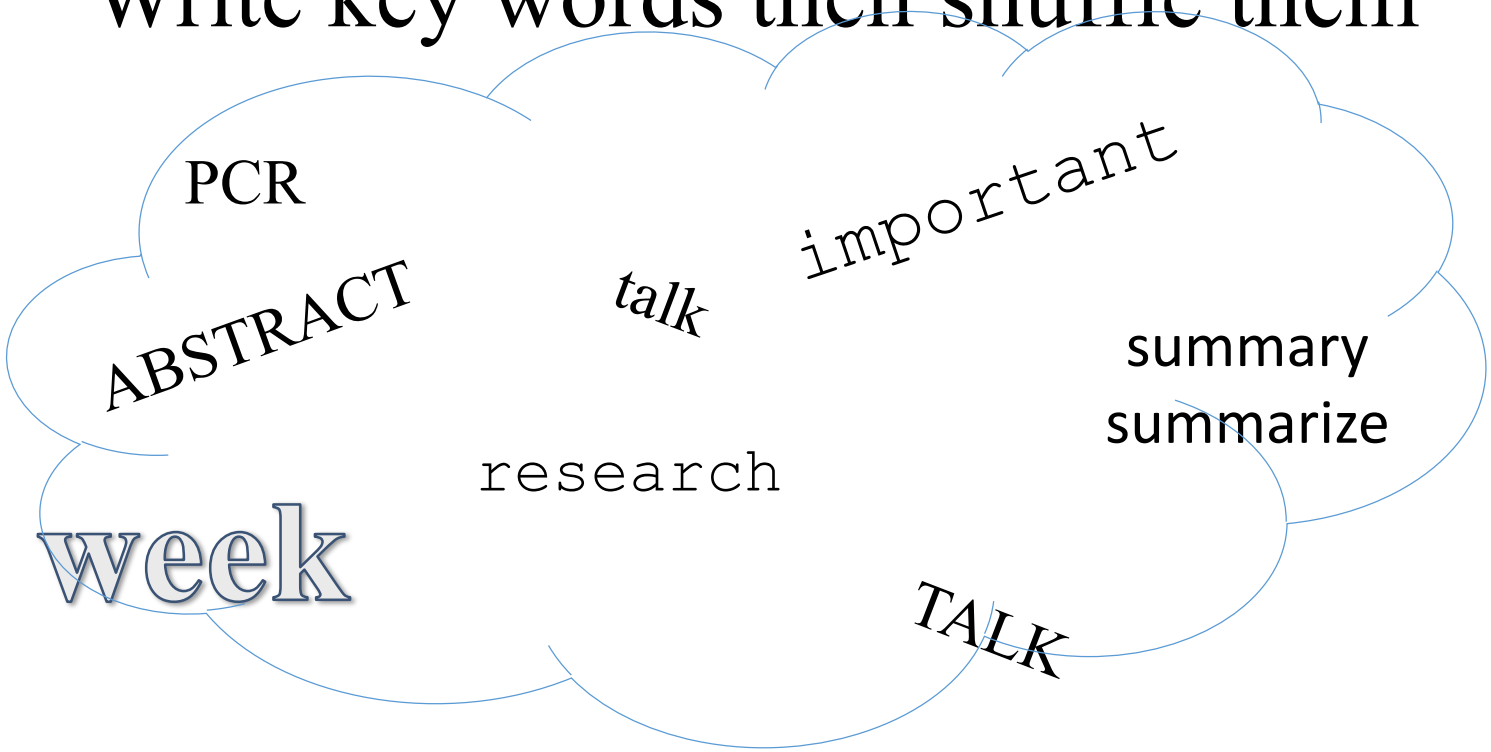


Hint: Start with Method and Data

Use sticky notes to list points you want to make, and then move them around.



Hint: TITLE
Write key words then shuffle them



How to write an Abstract: Important talk for Research Week

2. EDIT

Then edit harshly…very harshly. Do NOT be wed to your prose.

"Whatever you do, please don't edit yourself."

‘Wordiness’

O2 uptake was examined and found to vary considerably.

O2 uptake varied considerably.

Scissors were sterilized prior to use.

Scissors were sterilized, or Sterilized scissors were used.

We found a higher response in children compared to adults.

Response was higher in children than in adults.

EDIT!

‘Dear Dr. Fryer,

We loved your paper containing 11 figures and 63 references. Unfortunately we have a limit of **6,000 words/paper** and yours is currently over **13,000 words** including figure legends. If you could please shorten the text …we would be happy to publish it in the *Journal of Irreproducible Results*….’

Word Choice

- The cells were exposed to serum for 2 hours.
- After cutdown to an artery we removed the spleen.
- Clotting was changed in response to heparin.
- Serotonin was associated with an increase in the blood pressure.
- The change in CO2 was observed.

The Word Choice

- The cells were incubated with serum for 2 hours.
- After exposing an artery we removed the spleen.
- Clotting was decreased by heparin.
- Serotonin increased the blood pressure.
- The change in CO2 was measured.


NO, NO, IF YOU MAKE THE PAPER TOO EASY TO READ, EVERYONE WILL KNOW HOW YOU GOT THE RESULTS!

The power of ‘but’

BUT -implies a difference or contrast:

Cardiac output was decreased BUT blood pressure was unchanged.

Growth factors caused all cells BUT nerve cells, to divide.

The power of ‘and’

AND does not imply difference…. it could be similar

Cardiac output AND blood pressure were decreased

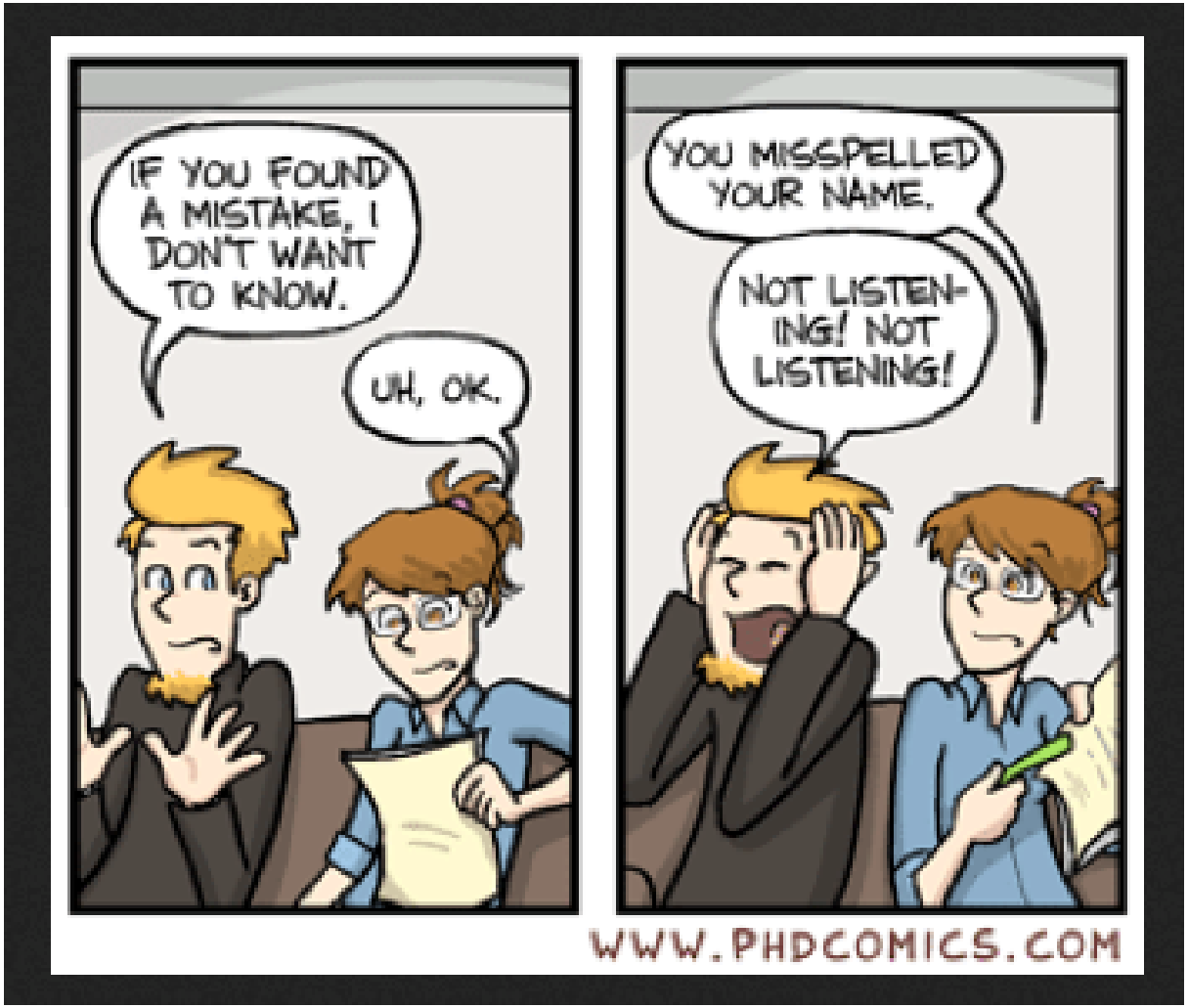
Growth factors caused fibroblasts AND nerve cells, to divide.

Avoid Writing in C@DΣ!

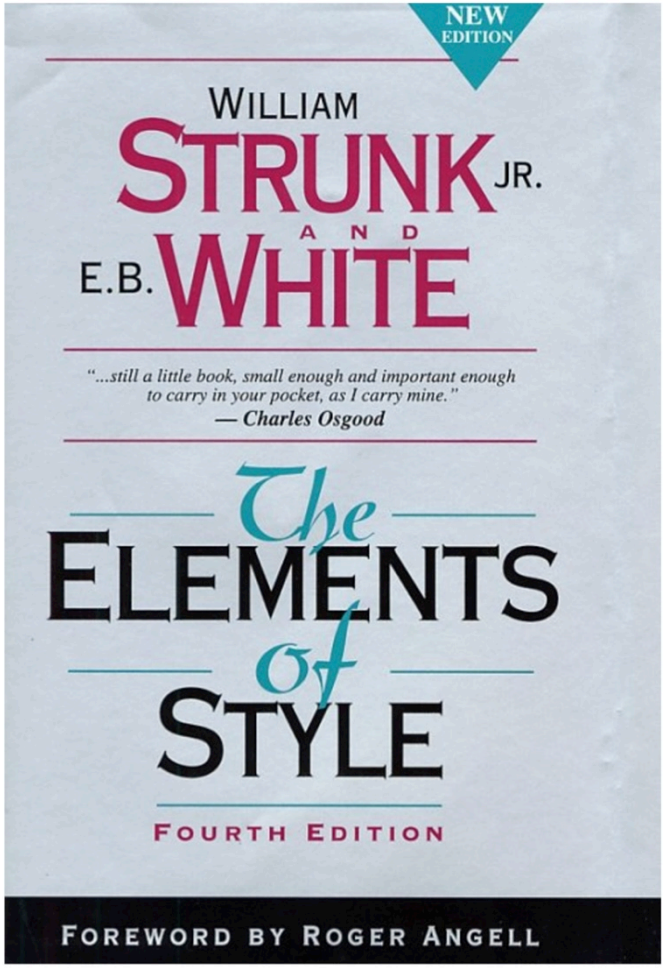
Avoid abbreviations-do not make reader work to understand content.

"In SCID and RAG-2-/- mice NK cells mediate a CHS response. Cutaneous hapten sensitization primes 2 distinct populations of CD8 T cells, to produce IFN-γ and IL-17, and elicit CHS. DNFB and Ox requires activation of both CD8 T cell populations within the AG challenge site. CD8 prime hapten-specific T cells in the vasculature of challenged sites are followed by activation that produces IL-17 and IFN-γ. Endo cells that have acquired and present hapten/MHC, activate CD8 T cells to produce cytokines after AG challenge, but it is important to note CD8 T cells do not penetrate the vascular barrier to infiltrate parenchymal tissue 1 and 8 hrs later. Both IL-17 and IFN-γ are required to stimulate Endo cells to produce CXCL1 and CXCL2, that direct

3.Seek an Outside Reader



Suggested Reading



Questions and Comments

You have 10 minutes…..

don’t screw it up

The challenge…

- This presentation is the culmination of years of hard work
- You represent your research group, department, university, practice, and most importantly, yourself.
- Don’t leave your presentation until you are on the plane en route to the meeting.

Step 1: Know your audience

- What do they know?
- What do you need to tell them?
- What do they expect?
- What will be interesting?
- What will keep them focused?

Step 2: Know your time limit

- 10 minutes with 5 minutes for questions
- Does not mean 15 minutes

Step 3: Know your message

- Every presentation should have one main message
- Be able to state your message concisely
- Know the exact take home point you want to get across

Step 4: Know yourself

- Ask yourself the tough questions
  - Does speaking in public make me nervous?
  - Do questions fluster me?
  - Am I easily distracted?

Preparation will help.

Tell the story of your data

- Every good story has a beginning (background, hypothesis), middle (methods), and end (results, conclusion).
- Do not take the audience down the same tortuous path your research has taken.
- Keep it simple.

Presentation philosophy

- Your slides should support your talk
- Your slides are not your talk

How many slides? One slide per minute

- Introduction - 1
- Hypothesis - 1
- Methods – 1-2
- Results – 4
- Conclusion – 1
- Limitations or future direction – 1

Title slide?

- On screen during your introduction
- Most meetings require disclaimers or conflict of interest slide

Introduction

- Your audience has general subject knowledge but summarize your area of research.
- Do not regurgitate everything you know about the topic.
- Reference other studies to place this in proper context.

Methods

- A schematic of your study is helpful.
- Give sufficient details to allow understanding of results.
- Define your population or model.

Results

- Show baseline or descriptive data.
- Make tables simple, highlight data
- Bar graphs, pie charts, scatterplots are useful. Use basic colors.
- If you present numeric data in a bar graph, either state the means or note on bar.

Conclusions

- What message do you most want the audience to remember after the meeting?
- List 3 major conclusions at most

What next?

- Fend off your critics. Acknowledge the major limitations of the study.
- What is the likely consequence of this study?
- Make sure your audience knows when you are done.

Answering questions

- Be prepared for questions you anticipate
- Ask for clarification
- If you don’t know, be honest
- Answers should be short, to the point
- Inconspicuously jot down the question topic if you forget under stress.

Avoid audiovisual disasters

- Compose your slides
- Do not let design overpower message
- Do not copy/paste slides from different presentations
- Use a common simple background, common font
- Minimize animation

- Solid background, avoid the rainbow.
- Maximize contrast
  - Tiny print cannot be read.
- Use no smaller than 32 pt font.
- Splllcheck.
- Use the 6x6 rule. No more than 6 lines, 6 words per line, 6 lines or bars on a graph.
- Create simple tables, graphs

Other suggestions

- No full sentences!
- Use bullet points
- Do not read slides
- Minimize UOA
- Do not crowd the slide

Write your talk, make your slides, practice

- Finish slides 2 weeks in advance.
- Write presentation word for word, craft concise sentences.
- Practice in front of colleagues who are not familiar with your study.
- Take criticism to heart and revise.
- Keep practicing. You cannot overprepare.

Get your timing down

- If you run overtime, your message is lost.
- Speak slowly. Pause on every slide to orient the audience.
- Too many words detract from your slide.
- Nothing is more distracting than gesturing wildly and talking rapidly.

On the day of your talk…

- Get to the meeting site early.
- Beware of software version mismatch, file size
- Familiarize yourself with the equipment.
- Arrive at the meeting room early, introduce yourself to the moderators, and watch the speakers before you.

Relax!

- You are well prepared and you know your study better than anyone in the audience.
- Talk to the audience, not your slides.
- Make eye contact
- If you are nervous, limit your use of the pointer.