The Road from Poster to Scientific Publication

CHB

Session Topics

1. Why does INBRE track posters and publications?
2. Who's creating posters and publications?
3. Differences between a poster and a publication.
4. Seven steps to turn your poster into a publication.
5. How can INBRE help you publish?

Idaho INBRE

Multi-million dollar grant for
11 institutions with the purpose to:

Increase biomedical research capacity

By mandate, more INBRE dollars go to Idaho undergraduate colleges than to Idaho research institutions

CWI, CSI, LCSC,
NIC, NNU, CoI, BYUI

UI, ISU, BSU, IVREF

~$6,000,000 ~$2,875,000

Metrics that measure research capacity

- scientific presentations
- scientific posters
- scientific publications
- funded grant proposals

Every campus reports INBRE activities and we organize, store, and analyze the information using a database.

INBRE metrics: 2014 through 2017

Presentations & Posters

1,281

Scientific Publications

262

Posters = 638

SAM

Who knows what ‘publish or perish’ means?

PUBLISH or PERISH

University of Idaho
Idaho State University
Boise State University
IVREF

Research-intensive Institutions
UI, ISU, BSU, IVREF

Posters **Publications**

472 → 247

53.2 % poster to pub conversion

Undergraduate Colleges
BYU-I, CoI, CSI, CWI, LCSC, NIC, NNU

Posters	Publications
166	15

5.7 % poster to pub conversion

Do laboratories at the colleges that create more posters, publish more?

Surprisingly, the # of posters don't increase the # of publications

Faculty lab	Posters	Publications
a	42	2
b	24	1
c	13	3
d	10	0
e	6	2
f	1	2

A scientific publication is the GOLD Standard for scholarship


TEXAS
4
14

What is an inconvenient truth in Science?

You haven’t done anything until it is published in a peer-reviewed journal.

Why aren’t faculty at undergraduate colleges publishing more?

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Why should anyone publish papers?

For Faculty

- Add to mankind's knowledge
- Get recognition from the Scientific community
- Improve your teaching skills
- Increase your students' competitiveness
- Stay current in your field
- Get funding to continue your research

For Students

- Add to mankind's knowledge
- Get recognition from the Scientific community
- Improve your training
- Get exposed to scientific rigor
- Get into graduate school
- Get into professional school
- Get a competitive job
- Impress your friends/lover
- Create something for your parent's coffee table

Posters contribute to research but they do not carry the weight of a publication

WHY?

Differences

Poster

- Scientific idea exchange
- Critical feed-back
- Preliminary data
- “testing the waters”
- Narrow audience
- No lasting imprint on the record

Publication

- Formal communication
- Peer-reviewed
- Wide audience
- Strict format
- Statistical rigor
- Editorial review
- FOREVER in the record

Publications demonstrate expertise and knowledge.

Publications are absolutely required to win competitive grant money.

Quotes from NIH grant reviewers

"A peer-reviewed paper, even a small one, would do a lot to alleviate my concerns about the quality of the preliminary data."

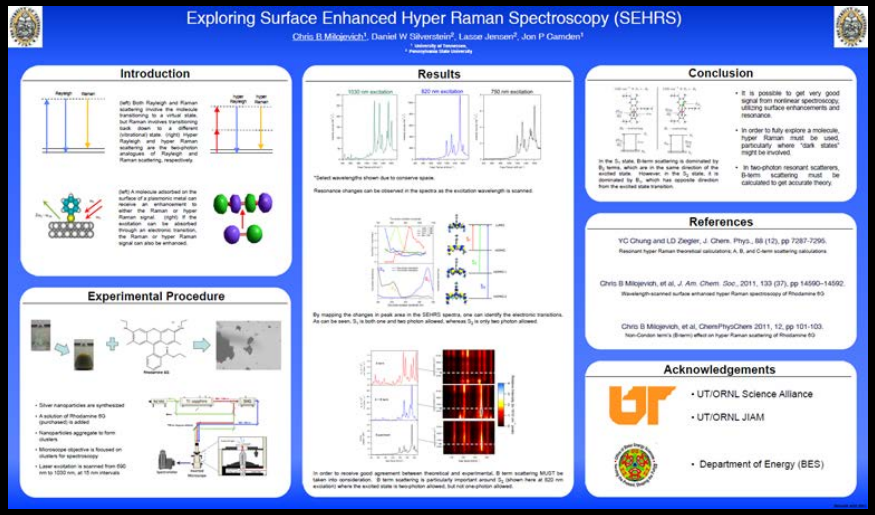
"Pubs In prep and meeting abstracts don't count as publications and are irritating!"

"Although Dr. X has a good publication record from his PhD, he hasn't published recently."

SAM

It is easier than you think to turn a Poster into a Publication.

You're just steps away!



Exploring Surface Enhanced Hyper Raman Spectroscopy (SEHRS)

Chris B Milojevich[1], Daniel W Silverstein[2], Lasse Jensen[2], Jon P Camden[1]

[1] University of Tennessee, [2] Pennsylvania State University

Introduction

(left) Both Rayleigh and Raman scattering involve the molecule transitioning to a virtual state, but Raman involves transitioning back down to a different (vibrational) state. (right) Hyper Rayleigh and hyper Raman scattering are the respective analogues of Rayleigh and Raman scattering, respectively.

(left) A molecule adsorbed on the surface of a plasmonic metal can receive an enhancement to both the incident and Raman signal. (right) If the excitation can be absorbed through an electronic transition, the Raman or hyper Raman signal can also be enhanced.

Experimental Procedure

- Silver nanoparticles are synthesized
- A solution of Rhodamine 6G (purchased) is added
- Nanoparticles aggregate to form clusters
- Microscope objective is focused on clusters for spectroscopy
- Laser excitation is scanned from 690 nm to 1200 nm, at 15 nm intervals

Results

1030 nm excitation | 820 nm excitation | 790 nm excitation

*Select wavelengths shown due to conserve space.

Resonance changes can be observed in the spectra as the excitation wavelength is scanned.

By mapping the changes in peak area in the SEHRS spectra, one can identify the electronic transitions. As can be seen, S_1 is both one and two photon allowed, whereas S_2 is only two photon allowed.

In order to resolve good agreement between theoretical and experimental, B term scattering MUST be taken into consideration. B term scattering is particularly important around S_2, shown here at 820 nm excitation, where the excited state is two-photon allowed, but not one-photon allowed.

Conclusion

- It is possible to get very good signal from nonlinear spectroscopy, utilizing surface enhancements and resonance.
- In order to fully explore a molecule, hyper Raman must be used, particularly where "dark states" might be involved.
- In two-photon resonant scatterers, β-term scattering must be calculated to get accurate theory.

In the S_1 state, B-term scattering is dominated by B_y terms, which are in the same direction of the excited state. However, in the S_2 state, it is dominated by B_x, which has opposite direction from the excited state transition.

References

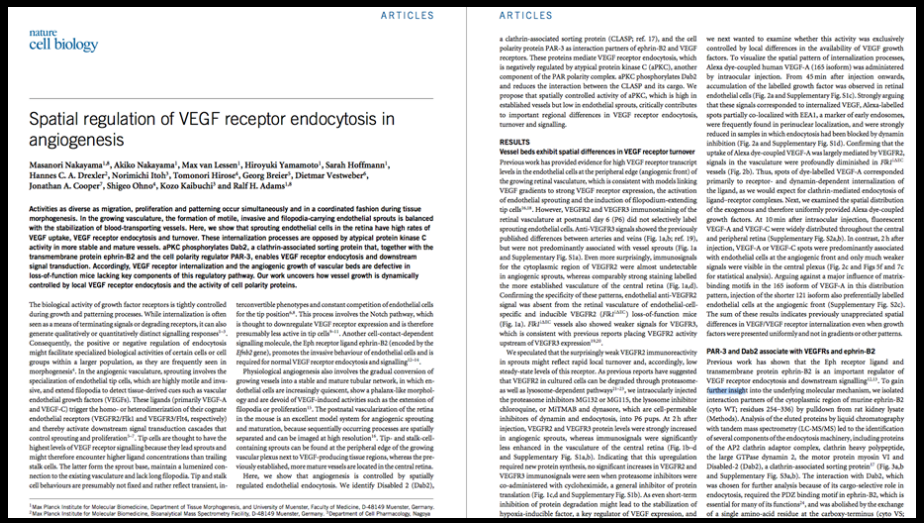
YC Chung and LD Ziegler, J. Chem. Phys., 88 (12), pp 7287-7295.
Resonant hyper Raman theoretical calculations; A, B, and C-term scattering calculations.

Chris B Milojevich, et al, J. Am. Chem. Soc., 2011, 133 (37), pp 14590–14592.
Wavelength-scanned surface enhanced hyper Raman spectroscopy of Rhodamine 6G

Chris B Milojevich, et al, ChemPhysChem 2011, 12, pp 101-103.
Non-Condon term's (B-term) effect on hyper Raman scattering of Rhodamine 6G

Acknowledgements

- UT/ORNL Science Alliance
- UT/ORNL JIAM
- Department of Energy (BES)



ARTICLES

nature cell biology

Spatial regulation of VEGF receptor endocytosis in angiogenesis

Masanori Nakayama[1,3], Akiko Nakayama[1], Max van Lessen[1], Hiroyuki Yamamoto[1], Sarah Hoffmann[1], Hannes C. A. Drexler[2], Norimichi Itoh[1], Tomonori Hirose[1], Georg Breier[2], Dietmar Vestweber[2], Jonathan A. Cooper[1], Shigeo Ohno[4], Kozo Kaibuchi[3] and Ralf H. Adams[1,5]

Activities as diverse as migration, proliferation and patterning occur simultaneously and in a coordinated fashion during tissue morphogenesis. In the growing vasculature, the formation of motile, invasive and filopodia-carrying endothelial sprouts is balanced with the stabilization of blood-transporting vessels. Here, we show that sprouting endothelial cells in the retina have high rates of VEGF uptake, VEGF receptor endocytosis and turnover. These internalization processes are opposed by atypical protein kinase C activity in more stable and mature vessels. aPKC phosphorylates Dab2, a clathrin-associated sorting protein that, together with the transmembrane protein ephrin-B2 and the cell polarity regulator PAR-3, enables VEGF receptor endocytosis and downstream signal transduction. Accordingly, VEGF receptor internalization and the angiogenic growth of vascular beds are defective in loss-of-function mice lacking key components of this regulatory pathway. Our work uncovers how vessel growth is dynamically controlled by local VEGF receptor endocytosis and the activity of cell polarity proteins.

The biological activity of growth factor receptors is tightly controlled during growth and patterning processes. While internalization is often seen as a means of terminating signals or degrading receptors, it can also generate qualitatively or quantitatively distinct signalling responses[1–3]. Consequently, the positive or negative regulation of endocytosis might facilitate specialized biological activities of certain cells or cell groups within a larger population, as they are frequently seen in morphogenesis[4]. In the angiogenic vasculature, sprouting involves the specialization of endothelial tip cells, which are highly motile and invasive, and extend filopodia to detect tissue-derived cues such as vascular endothelial growth factors (VEGFs). These ligands (primarily VEGF-A and VEGF-C) trigger the homo- or heterodimerization of their cognate endothelial receptors (VEGFR2/Flk1 and VEGFR3/Flt4, respectively) and thereby activate downstream signal transduction cascades that control sprouting and proliferation[5–7]. Tip cells are thought to have the highest levels of VEGF receptor signalling because they lead sprouts and might therefore encounter higher ligand concentrations than trailing stalk cells. The latter form the sprout base, maintain a lumenized connection to the existing vasculature and lack long filopodia. Tip and stalk cell behaviours are presumably not fixed and rather reflect transient, interconvertible phenotypes and constant competition of endothelial cells for the tip position[8,9]. This process involves the Notch pathway, which is thought to downregulate VEGF receptor expression and is therefore presumably less active in tip cells[10–13]. Another cell-contact-dependent signalling molecule, the Eph receptor ligand ephrin-B2 (encoded by the *Efnb2* gene), promotes the invasive behaviour of endothelial cells and is required for normal VEGF receptor endocytosis and signalling[12,14].

Physiological angiogenesis also involves the gradual conversion of growing vessels into a stable and mature tubular network, in which endothelial cells are increasingly quiescent, show a phalanx-like morphology and are devoid of VEGF-induced activities such as the extension of filopodia or proliferation[15]. The postnatal vascularization of the retina in the mouse is an excellent model system for angiogenic sprouting and maturation, because sequentially occurring processes are spatially separated and can be imaged at high resolution[16]. Tip- and stalk-cell-containing sprouts can be found at the peripheral edge of the growing vascular plexus next to VEGF-producing tissue regions, whereas the previously established, more mature vessels are located in the central retina.

Here, we show that angiogenesis is controlled by spatially regulated endothelial endocytosis. We identify Disabled 2 (Dab2), a clathrin-associated sorting protein (CLASP; ref. 17), and the cell polarity protein PAR-3 as interaction partners of ephrin-B2 and VEGF receptors. These proteins mediate VEGF receptor endocytosis, which is negatively regulated by atypical protein kinase C (aPKC), another component of the PAR polarity complex. aPKC phosphorylates Dab2 and reduces the interaction between the CLASP and its cargo. We propose that spatially controlled activity of aPKC, which is high in established vessels but low in endothelial sprouts, critically contributes to important regional differences in VEGF receptor endocytosis, turnover and signalling.

[1]Max Planck Institute for Molecular Biomedicine, Department of Tissue Morphogenesis, and University of Muenster, Faculty of Medicine, D-48149 Muenster, Germany. [2]Max Planck Institute for Molecular Biomedicine, Bioanalytical Mass Spectrometry Facility, D-48149 Muenster, Germany. [3]Department of Cell Pharmacology, Nagoya

RESULTS

Vessel beds exhibit spatial differences in VEGF receptor turnover

Previous work has provided evidence for high VEGF receptor transcript levels in the endothelial cells at the peripheral edge (angiogenic front) of the growing retinal vasculature, which is consistent with models linking VEGF gradients to strong VEGF receptor expression, the activation of endothelial sprouting and the induction of filopodium-extending tip cells[16,18]. However, VEGFR2 and VEGFR3 immunostaining of the retinal vasculature at postnatal day 6 (P6) did not selectively label sprouting endothelial cells. Anti-VEGFR3 signals showed the previously published differences between arteries and veins (Fig. 1a,b; ref. 19), but were not predominantly associated with vessel sprouts (Fig. 1a and Supplementary Fig. S1a). Even more surprisingly, immunosignals for the cytoplasmic region of VEGFR2 were almost undetectable in angiogenic sprouts, whereas comparably strong staining labelled the more established vasculature of the central retina (Fig. 1a,d). Confirming the specificity of these patterns, endothelial anti-VEGFR2 signal was absent from the retinal vasculature of endothelial-cell-specific and inducible VEGFR2 ($Flk1^{i\Delta EC}$) loss-of-function mice (Fig. 1a). $Flk1^{i\Delta EC}$ vessels also showed weaker signals for VEGFR3, which is consistent with previous reports placing VEGFR2 activity upstream of VEGFR3 expression[20,21].

We speculated that the surprisingly weak VEGFR2 immunoreactivity in sprouts might reflect rapid local turnover and, accordingly, low steady-state levels of this receptor. As previous reports have suggested that VEGFR2 in cultured cells can be degraded through proteasome- as well as lysosome-dependent pathways[22–25], we intraocularly injected the proteasome inhibitors MG132 or MG115, the lysosome inhibitor chloroquine, or MiTMAB and dynasore, which are cell-permeable inhibitors of dynamin and endocytosis, into P6 pups. At 2 h after injection, VEGFR2 and VEGFR3 protein levels were strongly increased in angiogenic sprouts, whereas immunosignals were significantly less enhanced in the vasculature of the central retina (Fig. 1b–d and Supplementary Fig. S1a,b). Indicating that this upregulation required new protein synthesis, no significant increases in VEGFR2 and VEGFR3 immunosignals were seen when proteasome inhibitors were co-administered with cycloheximide, a general inhibitor of protein translation (Fig. 1c,d and Supplementary Fig. S1b). As even short-term inhibition of protein degradation might lead to the stabilization of hypoxia-inducible factor, a key regulator of VEGF expression, and we next wanted to examine whether this activity was exclusively controlled by local differences in the availability of VEGF growth factors. To visualize the spatial pattern of internalization processes, Alexa dye-coupled human VEGF-A (165 isoform) was administered by intraocular injection. From 45 min after injection onwards, accumulation of the labelled growth factor was observed in retinal endothelial cells (Fig. 2a and Supplementary Fig. S1c). Strongly arguing that these signals corresponded to internalized VEGF, Alexa-labelled spots partially co-localized with EEA1, a marker of early endosomes, were frequently found in perinuclear localization, and were strongly reduced in samples in which endocytosis had been blocked by dynamin inhibition (Fig. 2a and Supplementary Fig. S1d). Confirming that the uptake of Alexa dye-coupled VEGF-A was largely mediated by VEGFR2, signals in the vasculature were profoundly diminished in $Flk1^{i\Delta EC}$ vessels (Fig. 2b). Thus, spots of dye-labelled VEGF-A corresponded primarily to receptor- and dynamin-dependent internalization of the ligand, as we would expect for clathrin-mediated endocytosis of ligand–receptor complexes. Next, we examined the spatial distribution of the exogenous and therefore uniformly provided Alexa dye-coupled growth factors. At 10 min after intraocular injection, fluorescent VEGF-A and VEGF-C were widely distributed throughout the central and peripheral retina (Supplementary Fig. S2a,b). In contrast, 2 h after injection, VEGF-A or VEGF-C spots were predominantly associated with endothelial cells at the angiogenic front and only much weaker signals were visible in the central plexus (Fig. 2c and Figs 3f and 7c for statistical analysis). Arguing against a major influence of matrix-binding motifs in the 165 isoform of VEGF-A in this distribution pattern, injection of the shorter 121 isoform also preferentially labelled endothelial cells at the angiogenic front (Supplementary Fig. S2c). The sum of these results indicates previously unappreciated spatial differences in VEGF/VEGF receptor internalization even when growth factors were presented uniformly and not in gradients or other patterns.

PAR-3 and Dab2 associate with VEGFRs and ephrin-B2

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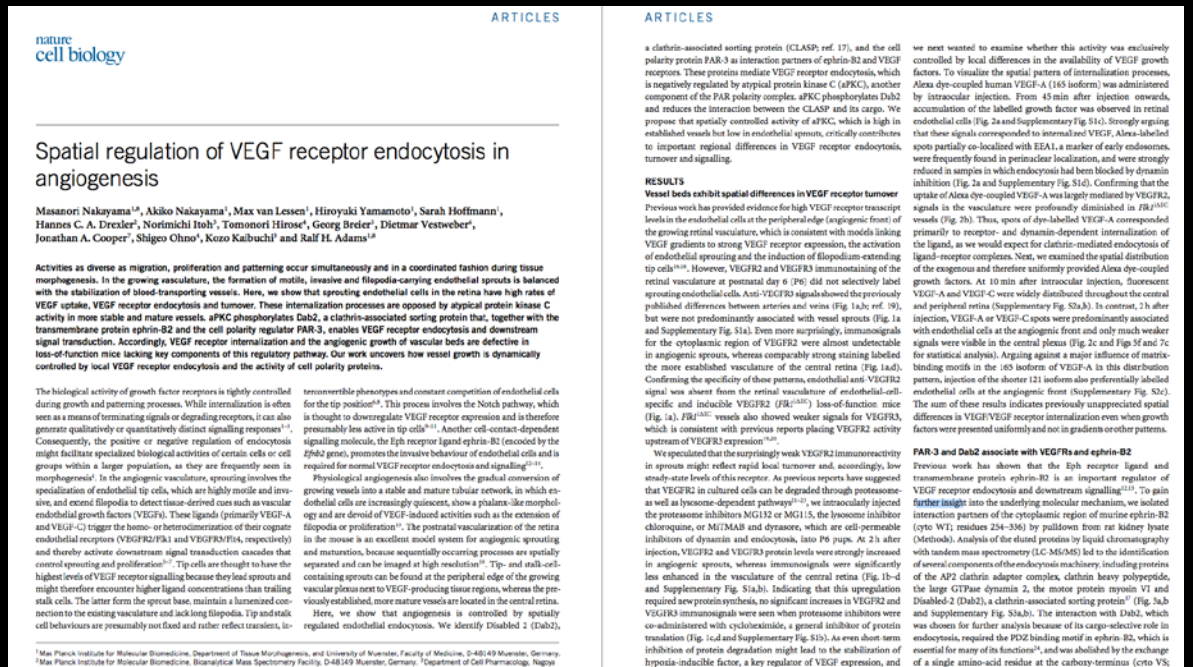
Anatomy of a Publication

- Introduction 3-5 pages
- Methods 5 pages
- Results 5 pages with Figures and Tables
- Discussion 3-5 pages (conclusion)
- References 30-50



nature cell biology

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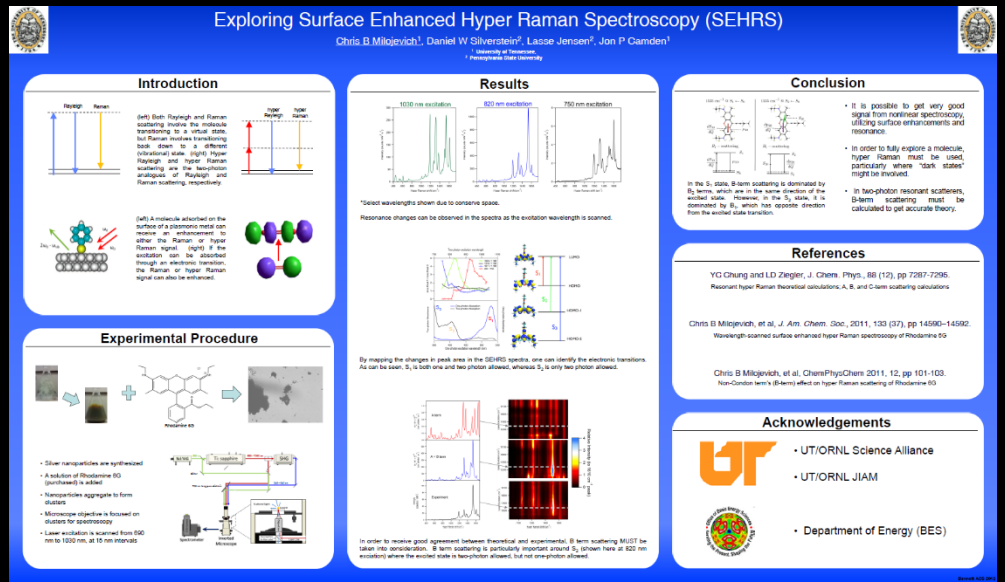
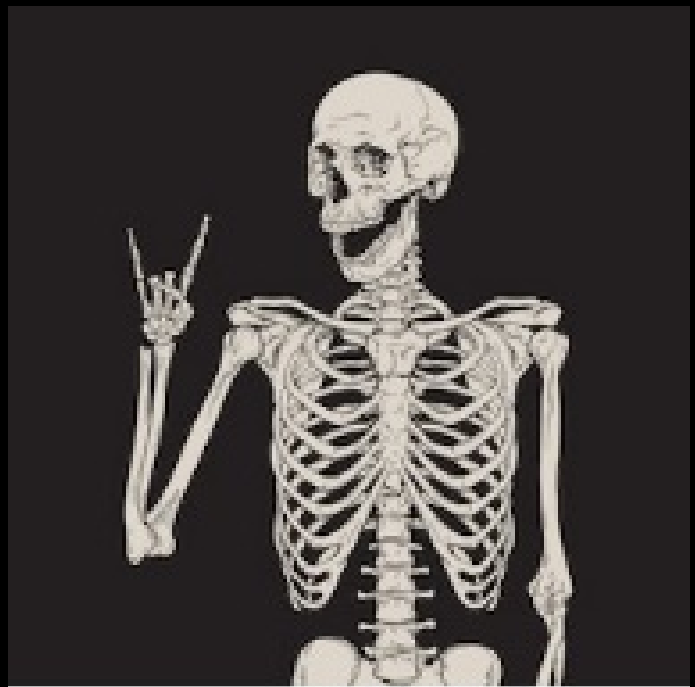
Posters have the “bones”

- Introduction — short
- Methods — schematic
- Results — Figures and Tables
- Discussion — Brief
- Conclusion — bulleted points
- References — a few





What can you and your professor do?

Take seven steps!

Step 1. Start with the Figures and Tables

a) Create publication quality Figures with legends.

b) Create publication quality Tables with Titles and footnotes.

c) Figures and Tables ‘stand alone’.

d) Order the Figs. and Tables to tell a story.

e) Write a paragraph for each Fig. and Table telling readers what to notice. Add measurements or observations that are not in the Fig. or Table.

CHB

Now, you have the *Results* section!

It answers the question:
what did you measure and observe?

Scientific papers have 4-7 figures and tables.
You already have them in your poster
—flesh it out ---- put the flesh on your bones!

Step 2. Change your schematic methods into paragraphs

a) Write each technique as a succinct paragraph of how you made your measurements. Use enough detail so someone can repeat what you did.

b) Describe the methods in the order of their use in the *Results* section.

This is your *Methods* section.

It answers the question: how did you take the measurements?

Step 3. Expand your Introduction

a. Give background to explain why the experiments are important.
b. State the hypothesis.

This is the *Introduction* section.

It answers the question: why did you do the research?

Remember, your intro should be like a funnel, start broad and focus in to your hypothesis or question.

Step 4. Expand your discussion and conclusions

a. Explain the implications of the results.
b. DO NOT re-tell the results.
c. "The most important finding in this study was X. It was also shown that a, b, c."

This is the *Discussion* section.

It answers the question: What do the results mean? How do the results fit into the current body of knowledge?

Step 5. Add authors, references, and acknowledgements

a. Authors and their order is up to the professor.
b. 30-50 references. This is NOT a review.
c. Acknowledge intellectual contributions not made by authors, any technical help, any reagents or strains supplied as gifts, your grant support, etc.

SAM

Step 6. Create the *Abstract*

a. **Create the abstract from** sentences you have already written in the sections.
b. **This is the MOST important component of the writing, because it is the most read (often the ONLY thing read).**

Step 7. Craft the *Title*

a. Accurate
b. Descriptive
c. Specific
d. Short

Good:

A structure for deoxyribose-nucleic acid

Bad:

The dependence of cell-free protein synthesis in E. coli *upon naturally occurring or synthetic polyribonucleotides*

Breaking the genetic code with defined polyribonucleotides

Summary of 7 steps

- Figs. and Tables – Results (what?)
- Methods (how?)
- Introduction (why?)
- Discussion (so what?)
- Authors, references and acknowledgements
- Abstract
- Title

CHB

Why aren’t people publishing?

- Time
- FEAR
  - Rejection
  - Criticism
  - That your thinking and logic are inferior
- No job-related incentive
- Need help
- Lack of money
- Need to do “one more experiment”
- Not enough experienced hands in the lab
  - Undergrads
  - graduate students
  - technicians

How can INBRE help faculty move Poster to a Publication?

- Writing help?
- Pay for page charges?
- Provide a writing consultant?
- Writing workshops?
- Writing groups?
- Peer partners?

SAM

Publishing is NOT insurmountable!

The road to Hell is paved with scientific works “*In Prep*”.