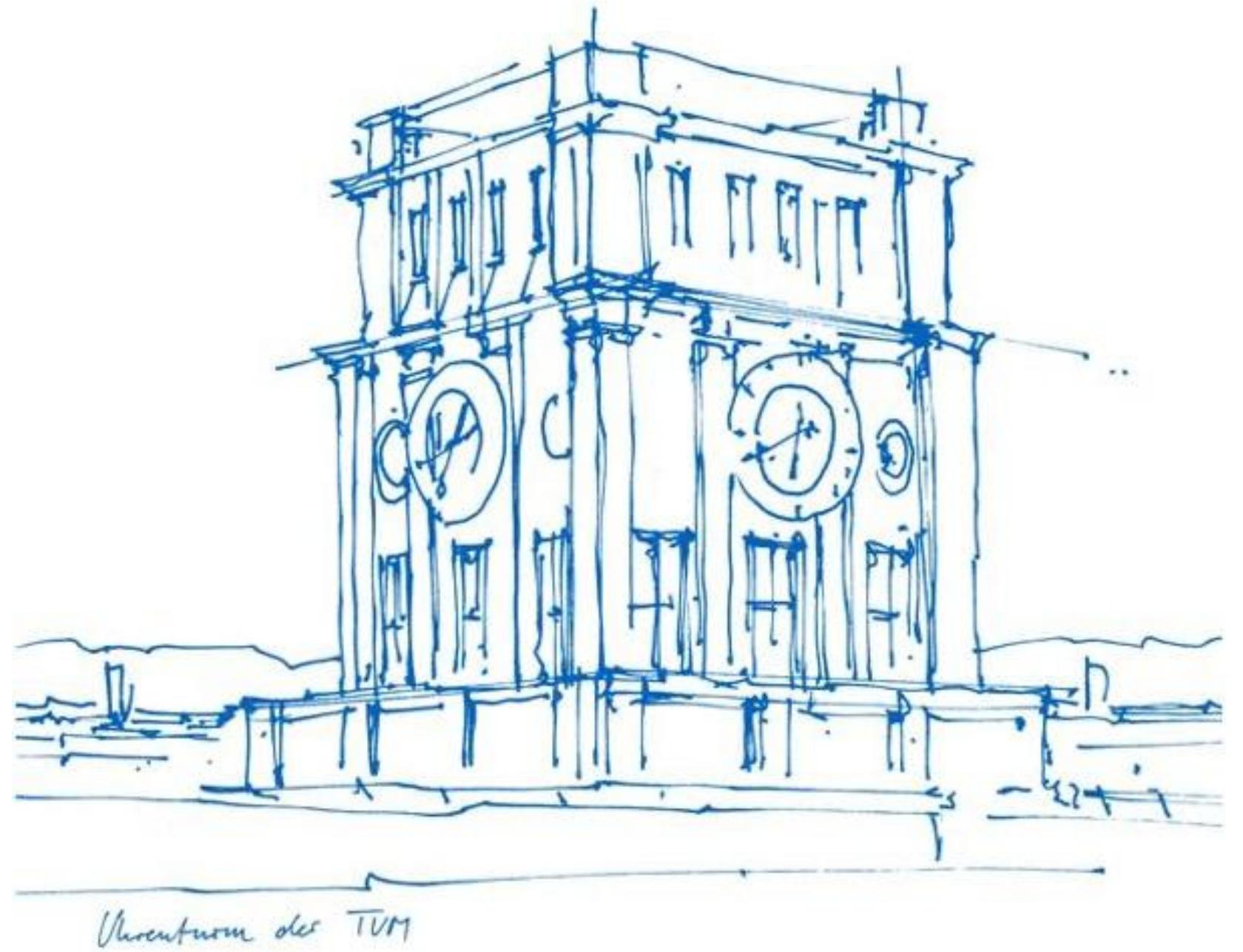


Master Seminar AI for Vision-Language Models in Medical Imaging (IN2107, IN45010)



I32 – Chair for Computational Imaging and AI in Medicine - **CompAI**
Faculty of Informatics and Institute for Advanced Study



Contents

- **Preface**
- **How to Read a Paper**
- **How to Review a Paper**
- **How to Publish a Paper**
- **How to Make a Poster**

Seminar structure (Preliminary)



Outlook

30.04		Welcome and Introduction to VLMs-L1
07.05		How to read papers make posters
14.05		VLMs-L2
21.05		
28.05		Student Presentations
04.06		Guest talk: Prof. Dr. Benedikt Wiestler
11.06		Student Presentations
18.06		Student Presentations
25.06		Guest talk:
02.07		Student Presentations
09.07		Guest talk:
16.07		Student Presentations
23.07.		Guest talk: Prof. Bernhard Kainz
30.07		Poster Session (All groups)



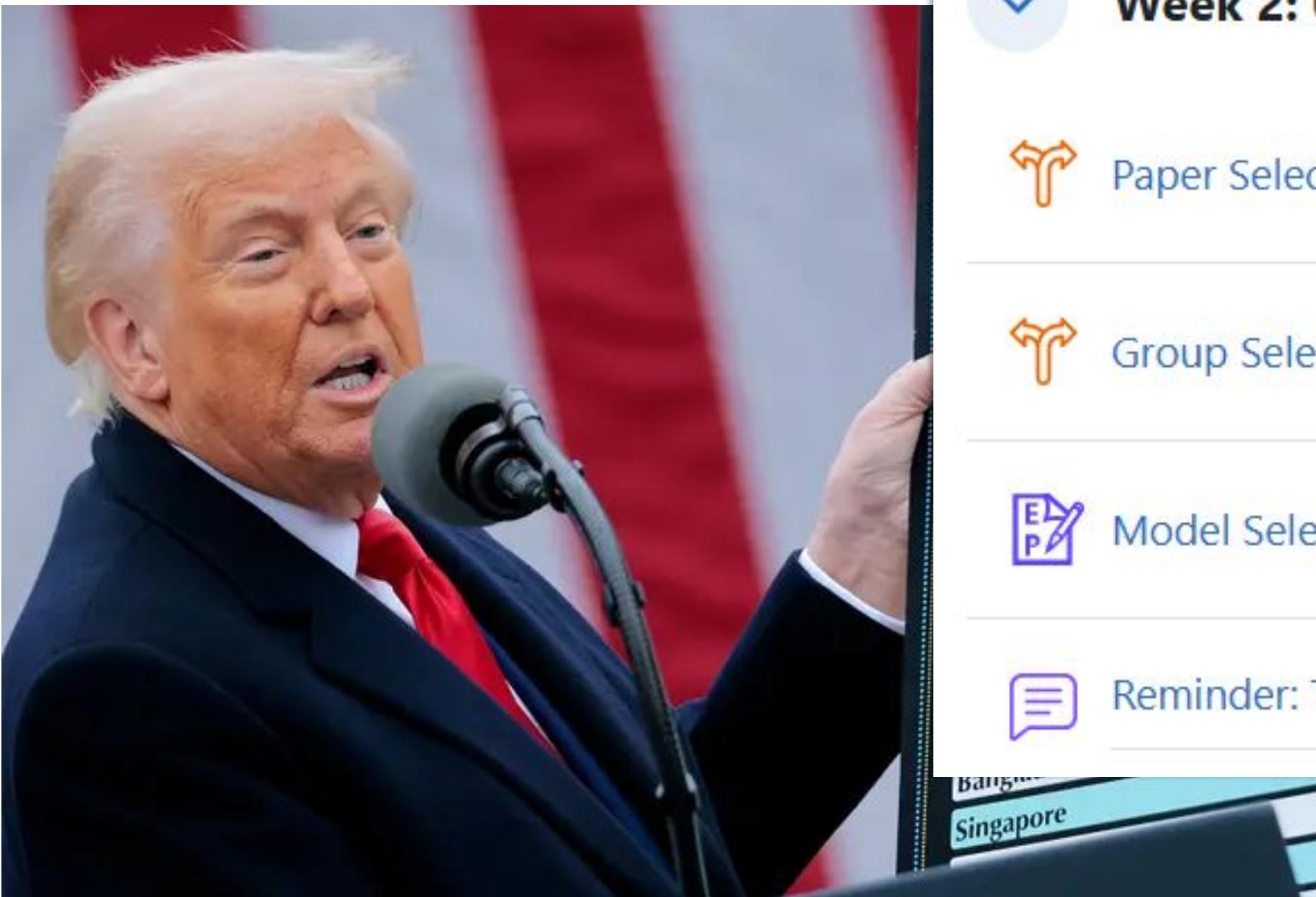
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00.08.059



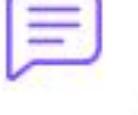
On Zoom*

Group/Paper/Model Selection

Already Open on Moodle :)



Week 2: 05 May-09 May

-  Paper Selection [VLM]
-  Group Selection [VLM]
-  Model Selection [VLM]
-  Reminder: Tomorrow's Seminar & VLM Voting Opening

Group/Paper/Model Selection



Paper Selection

This week will be the preparation time for the first group :)

Opened: Wednesday, 7 May 2025, 12:00 AM

Closes: Wednesday, 14 May 2025, 11:59 PM

30.04	🕒	Welcome and Introduction to VLMs-L1
07.05	🕒	How to read papers make posters
14.05	🕒	VLMs-L2
21.05		
28.05	🕒	Student Presentations

Title

- 1 Reason-RFT: Reinforcement Fine-Tuning for Visual Reasoning
- 2 LIMO: Less is More for Reasoning
- 3 Quantifying the Reasoning Abilities of LLMs on Real-world Clinical Cases
- 4 Demystifying Long Chain-of-Thought Reasoning in LLMs
- 5 Chain-of-Thought Prompting Elicits Reasoning in Large Language Models
- 6 Visual-RFT: Visual Reinforcement Fine-Tuning
- 7 RadVLM: A Multitask Conversational Vision-Language Model for Radiology
- 8 CheXagent: Towards a Foundation Model for Chest X-Ray Interpretation
- 9 MAIRA-2: Grounded Radiology Report Generation
- 10 Qwen2.5-VL Technical Report
- 11 LLaVA-Med: Training a Large Language-and-Vision Assistant for Biomedicine in One Day
- 12 Towards Evaluating and Building Versatile Large Language Models for Medicine
- 13 Can Modern LLMs Act as Agent Cores in Radiology Environments?
- 14 A Large Model for Non-Invasive and Personalized Management of Breast Cancer from MRI
- 15 MedRAX: Medical Reasoning Agent for Chest X-ray
- 16 MDTeamGPT: A Self-Evolving LLM-based Multi-Agent Framework for Medical Consultation
- 17 Premise Order Matters in Reasoning with Large Language Models
- 18 A Scalable Framework for Evaluating Health Language Models
- 19 Detecting hallucinations in large language models using semantic entropy

Time

Link Topics

- | | | |
|-------------|----------------------|--|
| 27 Mar 2025 | Link | Visual Reasoning, Reinforcement Learning |
| 5 Feb 2025 | Link | Efficient Reasoning, LLMs |
| 6 Mar 2025 | Link | Clinical Reasoning, Evaluation |
| 5 Feb 2025 | Link | CoT Analysis, LLMs |
| 10 Jan 2023 | Link | Prompt Engineering, Reasoning |
| 3 Mar 2025 | Link | Vision-Language, RL |
| 18 Dec 2024 | Link | Radiology, VLM |
| 22 Jan 2024 | Link | Medical VLMs, X-ray |
| 6 Jun 2024 | Link | Report Generation, Radiology |
| 19 Feb 2025 | Link | Foundation Model, VLM |
| 1 Jun 2023 | Link | Medical VLMs |
| 5 Sep 2024 | Link | Medical LLMs, Generalist Models |
| 8 Apr 2025 | Link | Agents, Radiology |
| 17 Apr 2025 | Link | Breast Cancer, MRI, Personalized AI |
| 4 Feb 2025 | Link | X-ray Reasoning, Agents |
| 18 Mar 2025 | Link | Multi-Agent, Healthcare AI |
| 14 Feb 2024 | Link | Logical Reasoning, Prompt Order |
| 30 Mar 2025 | Link | Evaluation, Health LLMs |
| 19 Jun 2024 | Link | LLM Evaluation, Hallucinations |

The presentation order **will be announced after the paper selection period.**

Group/Paper/Model Selection



Group Selection

Group Selection [VLM]

Choice

Settings

Responses

More ▾

30.04	🕒🕒🕒	Welcome and Introduction to VLMs-L1
07.05	🕒🕒🕒	How to read papers make posters
14.05	🕒🕒🕒	VLMs-L2
21.05		
28.05	🕒🕒🕒	Student Presentations
04.06	🕒🕒🕒	Guest talk: Prof. Dr. Benedikt Wiestler
11.06	🕒🕒🕒	Student Presentations

Opened: Wednesday, 7 May 2025, 12:00 AM

Closes: Wednesday, 11 June 2025, 11:59 PM

Please choose groups of 2 persons to work together for the coding and poster part.

Group 1

Responses: 1

Limit: 2

Group 2

Responses: 1

Limit: 2

Group 3

Responses: 1

Limit: 2

Group 4

Responses: 0

Limit: 2

Group 5

Responses: 0

Limit: 2

Group 6

Responses: 0

Limit: 2

Group/Paper/Model Selection



Group Selection

Master-Seminar: 950833399 (S25) / Week 2: 05 May-09 May / Model Selection [VLM]

Model Selection [VLM]

Etherpad Lite

Settings

More ▾

30.04	၂၀၁၄	Welcome and Introduction to VLMs-L1
07.05	၂၀၁၅	How to read papers make posters
14.05	၂၀၁၅	VLMs-L2
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28.05	၂၀၁၅	Student Presentations
04.06	၂၀၁၆	Guest talk: Prof. Dr. Benedikt Wiestler
11.06	၂၀၁၆	Student Presentations

Opened: Wednesday, 7 May 2025, 12:00 AM

Closes: Wednesday, 11 June 2025, 11:59 PM

Model List:

[compai-lab/Master-Seminar-AI-for-Vision-Language-Models-in-Medical-Imaging-IN2107-IN45069-: Master-Seminar: AI for Vision-Language Models in Medical Imaging \(IN2107, IN45069\)](#)

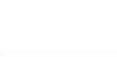
Group/Paper/Model Selection

Dataset Release

1) Datasets:

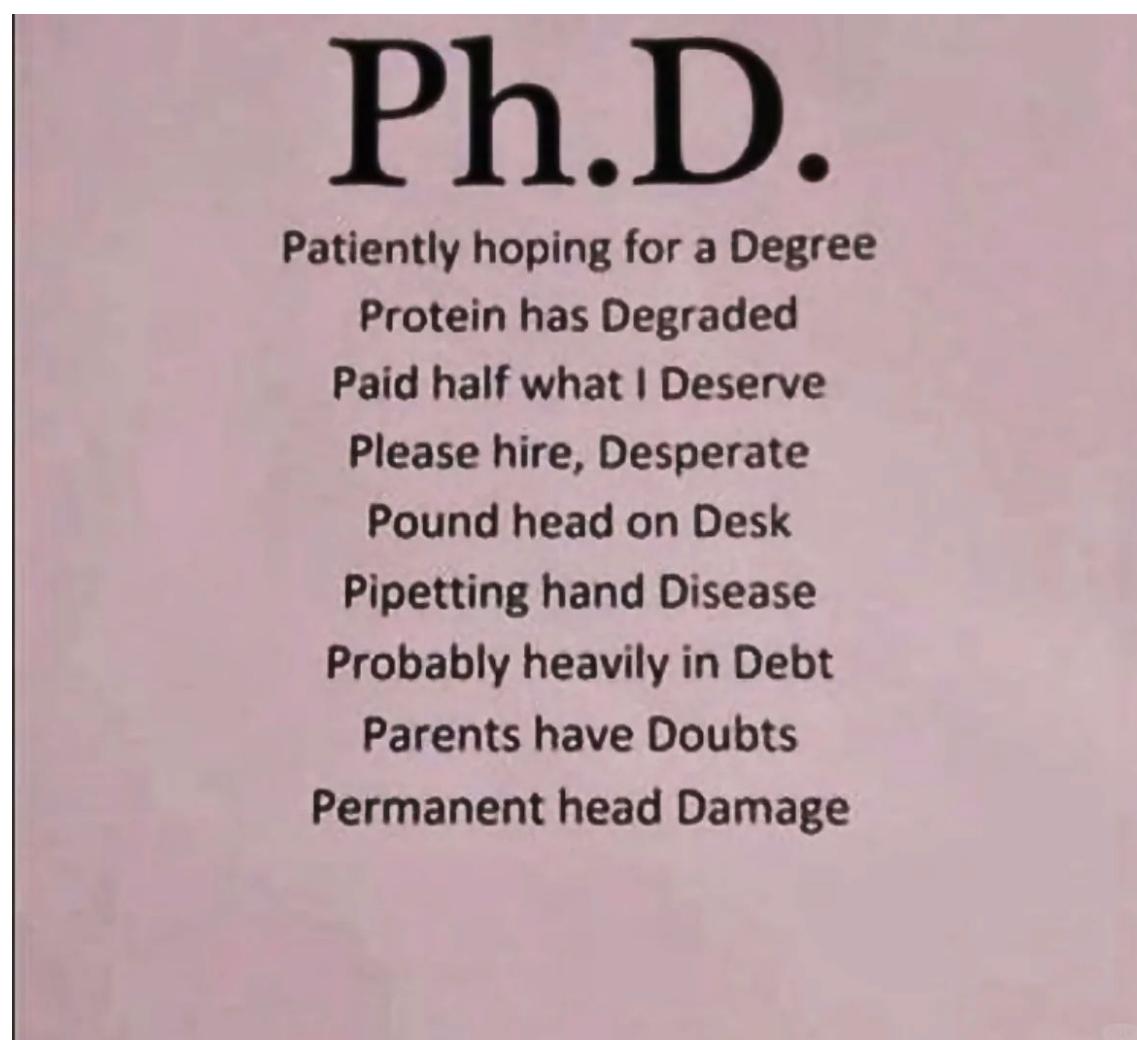
We will provide medial datasets for you, more details will come soon :)

- We will release the datasets maybe before 28.05.2025
 - Datasets are about **image diagnosis**
 - You use your chosen model to inference on the datasets.
 - Then evaluate the performance. (Will provide some simple example for evaluation)
- You can use the **API or computational resources from Google Colab** for inference.
- The datasets will be shared **via Moodle or Google Drive** (please do not share the original images online).
- The **datasets are relatively small—around 100 samples.** :)
- We encourage a deeper analysis of LLM performance, as well as exploring interesting ideas from relevant papers to improve performance or identify potential issues with LLMs.

30.04		Welcome and Introduction to VLMs-L1
07.05		How to read papers make posters
14.05		VLMs-L2
21.05		
28.05		Student Presentations
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Why reading paper ?

Reason for me



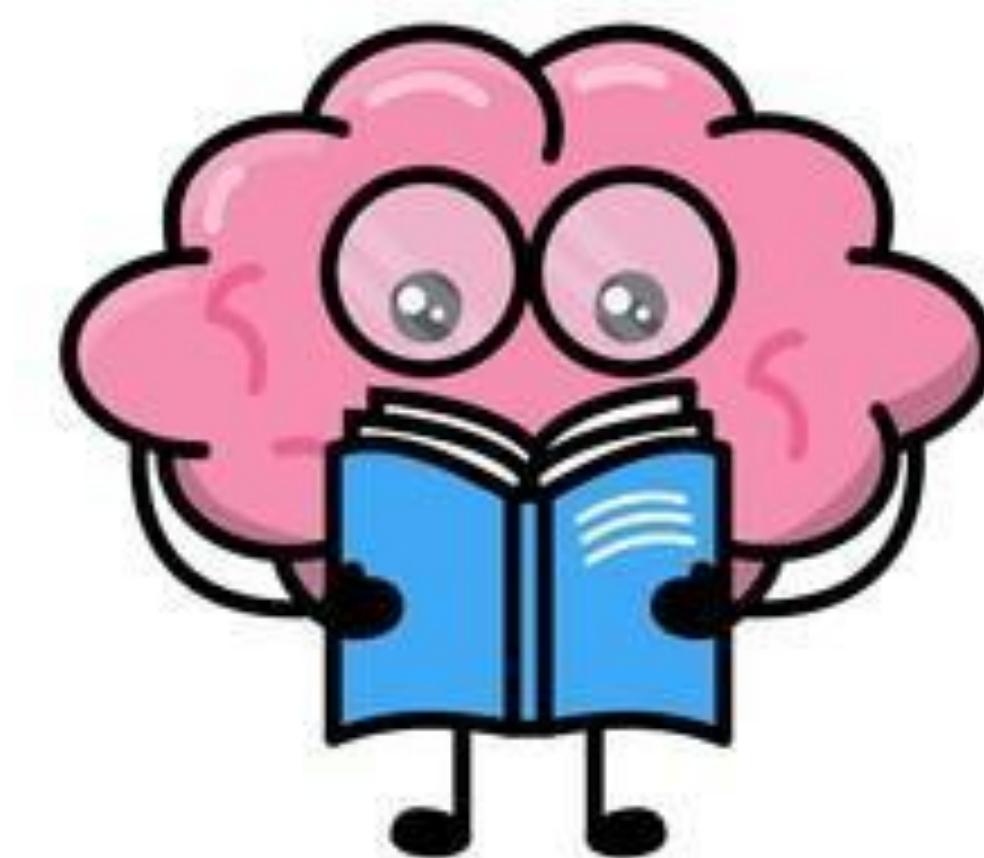
Because I am a PhD student :)

Reason maybe for you



Mandatory section in our seminar :>

Real Reason



Good for your brain !

How to read paper?



When I read paper, my brain inside

Attention Is All You Need

Ashish Vaswani*
Google Brain
avaswani@google.com **Noam Shazeer***
Google Brain
noam@google.com **Niki Parmar***
Google Research
nikip@google.com **Jakob Uszkoreit***
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aidan@cs.toronto.edu **Łukasz Kaiser***
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Illia Polosukhin* ‡
illia.polosukhin@gmail.com

Abstract

The dominant sequence transduction models are based on complex recurrent or convolutional neural networks that include an encoder and a decoder. The best performing models also connect the encoder and decoder through an attention mechanism. We propose a new simple network architecture, the Transformer, based solely on attention mechanisms, dispensing with recurrence and convolutions entirely. Experiments on two machine translation tasks show these models to be superior in quality while being more parallelizable and requiring significantly less time to train. Our model achieves 28.4 BLEU on the WMT 2014 English-to-German translation task, improving over the existing best results, including ensembles, by over 2 BLEU. On the WMT 2014 English-to-French translation task, our model establishes a new single-model state-of-the-art BLEU score of 41.8 after training for 3.5 days on eight GPUs, a small fraction of the training costs of the best models from the literature. We show that the Transformer generalizes well to other tasks by applying it successfully to English constituency parsing both with large and limited training data.

*Equal contribution. Listing order is random. Jakob proposed replacing RNNs with self-attention and started the effort to evaluate this idea. Ashish, with Illia, designed and implemented the first Transformer models and has been crucially involved in every aspect of this work. Noam proposed scaled dot-product attention, multi-head attention and the parameter-free position representation and became the other person involved in nearly every detail. Niki designed, implemented, tuned and evaluated countless model variants in our original codebase and tensor2tensor. Llion also experimented with novel model variants, was responsible for our initial codebase, and efficient inference and visualizations. Lukasz and Aidan spent countless long days designing various parts of and implementing tensor2tensor, replacing our earlier codebase, greatly improving results and massively accelerating our research.

†Work performed while at Google Brain.

‡Work performed while at Google Research.

31st Conference on Neural Information Processing Systems (NIPS 2017), Long Beach, CA, USA.

Abstract

Introduction

Method

Experiments

Conclusion

How to read paper?



Andrew Ng

Try to figure out 4 questions during reading

1. What did authors try to accomplish?
2. What were the key elements of the approach?
3. What can you use yourself?
4. What other references do you want to follow?

Attention Is All You Need

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Illia Polosukhin* ‡
illia.polosukhin@gmail.com

Abstract

The dominant sequence transduction models are based on complex recurrent or convolutional neural networks that include an encoder and a decoder. The best performing models also connect the encoder and decoder through an attention mechanism. We propose a new simple network architecture, the Transformer, based solely on attention mechanisms, dispensing with recurrence and convolutions entirely. Experiments on two machine translation tasks show these models to be superior in quality while being more parallelizable and requiring significantly less time to train. Our model achieves 28.4 BLEU on the WMT 2014 English-to-German translation task, improving over the existing best results, including ensembles, by over 2 BLEU. On the WMT 2014 English-to-French translation task, our model establishes a new single-model state-of-the-art BLEU score of 41.8 after training for 3.5 days on eight GPUs, a small fraction of the training costs of the best models from the literature. We show that the Transformer generalizes well to other tasks by applying it successfully to English constituency parsing both with large and limited training data.

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†Work performed while at Google Brain.

‡Work performed while at Google Research.

31st Conference on Neural Information Processing Systems (NIPS 2017), Long Beach, CA, USA.

How to Read Research Papers

Read Title / Abstract / Figures

Introduction / Conclusion / Figures (again) / Skim Rest

Read the paper, but skip the math

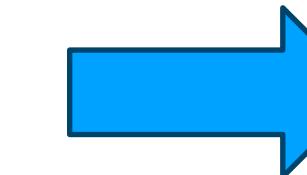
Read the whole paper but skip parts that not make sense

How to read paper?

How I read paper?

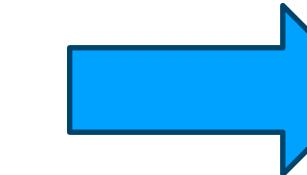


**Read Title / Abstract
/Conclusion**



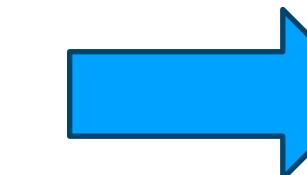
What can I get from this paper ?

Figures + Method



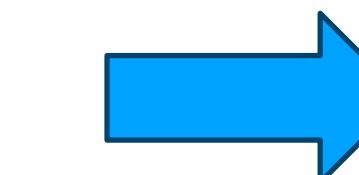
How to do it?

Experiments



Tables (Comparison Results, Ablation studies..)

Intro / Related works (if I am not familiar)



If the topic I am familiar, some time maybe skip ...



But Not Recommend directly read the paper from the first word until the last world!!

- You can have your own reading habit.
- Everyone's will be slightly different.

How to read paper?

Where to find papers?



Andrew Ng

- Twitter
- ML Subreddit: Machine Learning
- Top ML Conferences: NeurIPS, ICML, ICLR, CVPR
- ArXiv
- Friends / Online Community



- Social Media Recommendation
- Daily paper from HuggingFace
- Some famous Group / professor you like.
- Recommendation by Friends



- Train your social media recommendation algorithm to recognize that you're a PhD student.
- This is really helpful ☺

How to read paper?



Where to find papers?

Daily Papers + Submit a paper

by AK and the research community

PixelHacker: Image Inpainting with Structural and Semantic Consistency

Ziyang Xu^{1,◦}, Kangsheng Duan^{1,◦}, Xiaolei Shen², Zhipeng Ding³, Wenyu Liu¹, Xiaohu Ruan², Xiaoxin Chen², Xinggang Wang^{1,✉}
¹Huazhong University of Science and Technology ²VIVO AI Lab

[cs.CV] 30 Apr 2025



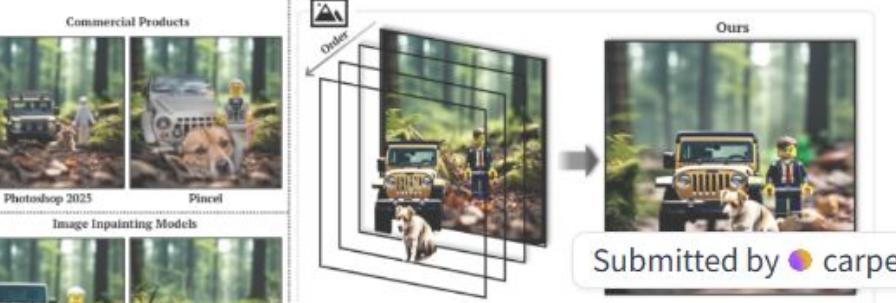
Submitted by Uyoung

PixelHacker: Image Inpainting with Structural and Semantic Consistency
17 · 8 authors

Improving Editability in Image Generation with Layer-wise Memory

Daneul Kim¹, Jaeha Lee¹, Jaesik Park¹
Seoul National University, Republic of Korea
[carpedkm, hayanz, jaesik.park@snu.ac.kr]

[cs.CV] 2 May 2025



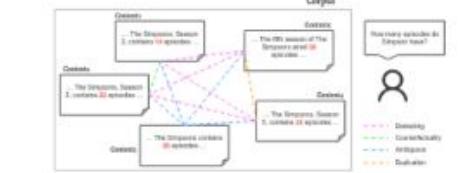
Submitted by carpedkm

Improving Editability in Image Generation with Layer-wise Memory
12 · 3 authors

CORG: Generating Answers from Complex, Interrelated Contexts

Hyunji Lee^{✉,◦}, Franck Dernoncourt^{1,◦}, Trung Bui^{1,◦}, Seunghyun Yoon^{1,◦}
¹KAIST AI ²Adobe Research
hyunji.amy.lee@kaist.ac.kr {dernonco, bui, syoon}@adobe.com

[cs.CE] 25 Apr 2025



Submitted by Franck-Dernoncourt

CORG: Generating Answers from Complex, Interrelated Contexts
4 · 4 authors

Real-World Gaps in AI Governance Research

AI safety and reliability in everyday deployments

Ilan Strauss^{1,2}, Isobel Mouré¹, Tim O'Reilly^{1,3}, and Sruly Rosenblat^{*1}
¹AI Disclosures Project, Social Science Research Council
²Institute for Innovation and Public Purpose, University College London
³O'Reilly Media

[cs.AI] 30 Apr 2025

Submitted by strauss-NYC

Abstract

TeLoGraF: Temporal Logic Planning via Graph-encoded Flow Matching

Yue Meng¹, ChuChu Fan¹

Abstract

Learning to solve complex tasks with signal temporal logic (STL) specifications is crucial to many real-world applications. However, most previous works only consider fixed or parametrized STL specifications due to the lack of a diverse STL dataset and encoders to effectively extract temporal logic information for downstream tasks. In this paper, we propose TeLoGraF, Temporal Logic Graph-encoded Flow, which utilizes Graph Neural Networks (GNN) encoder and flow-matching to learn solutions for general STL specifications. We identify four commonly used STL templates and collect a total of 200K specifications with paired demonstrations. We conduct extensive experiments in five simulation environments ranging

stop before the stop sign and may proceed only if no other cars have the right of way, etc. These cases require precise reasoning and planning to ensure safety, efficiency, and correctness. Hence, it is of utmost importance to endow agents with the ability to tackle temporal logic constraints.

Existing temporal logic specifications can be mainly categorized into linear temporal logic (LTL) (Pnueli, 1977), computation tree logic (CTL) (Clarke & Emerson, 1981), metric temporal logic (MTL) (Koymans, 1990), and signal temporal logic (STL) (Donzé & Maler, 2010; Raman et al., 2015; He et al., 2022). LTL checks a single system trace via logical operators and temporal operators, whereas CTL reasons for a tree of possible futures. Extending from LTL, MTL introduces

Submitted by yuemithucsd

X-Cross: Dynamic Integration of Language Models for Cross-Domain Sequential Recommendation

Guy Hadad¹, Haggai Roitman¹, Yotam Eshel¹
Ben-Gurion University of the Negev Ben-Gurion University of the Negev eBay Beer Sheva, Israel
Bracha Shapira¹, Lior Rokach¹
Ben-Gurion University of the Negev Ben-Gurion University of the Negev Beer Sheva, Israel

[cs.IR] 29 Apr 2025

Abstract

New products are emerging daily, recommendation systems are required to quickly adapt to possible new domains without needing extensive retraining. This work presents "X-Cross" – a novel cross-domain sequential recommendation model that recommends products in new domains by integrating several domain specific language models; each model is fine-tuned with low-rank adapters (LoRA). Given a recommendation prompt, operating layer by layer, X-Cross dynamically refines the interpretation of each source language model by integrating knowledge from all other models. These refined representations are propagated from one layer to the next, increasing the interactions from each domain adapter to another.

Keywords

Cross-domain recommendation, language models, natural language processing, dynamic integration, LoRA, parameter and data efficiency

ACM Reference Format:

Guy Hadad, Haggai Roitman, Yotam Eshel, Bracha Shapira, and Lior Rokach. 2025. X-Cross: Dynamic Integration of Language Models for Cross-Domain Sequential Recommendation. In *Proceedings of the 48th International ACM SIGIR Conference '25*, July 11–15. Submitted by guyhadad01
<https://doi.org/>

<https://huggingface.co/papers>

1

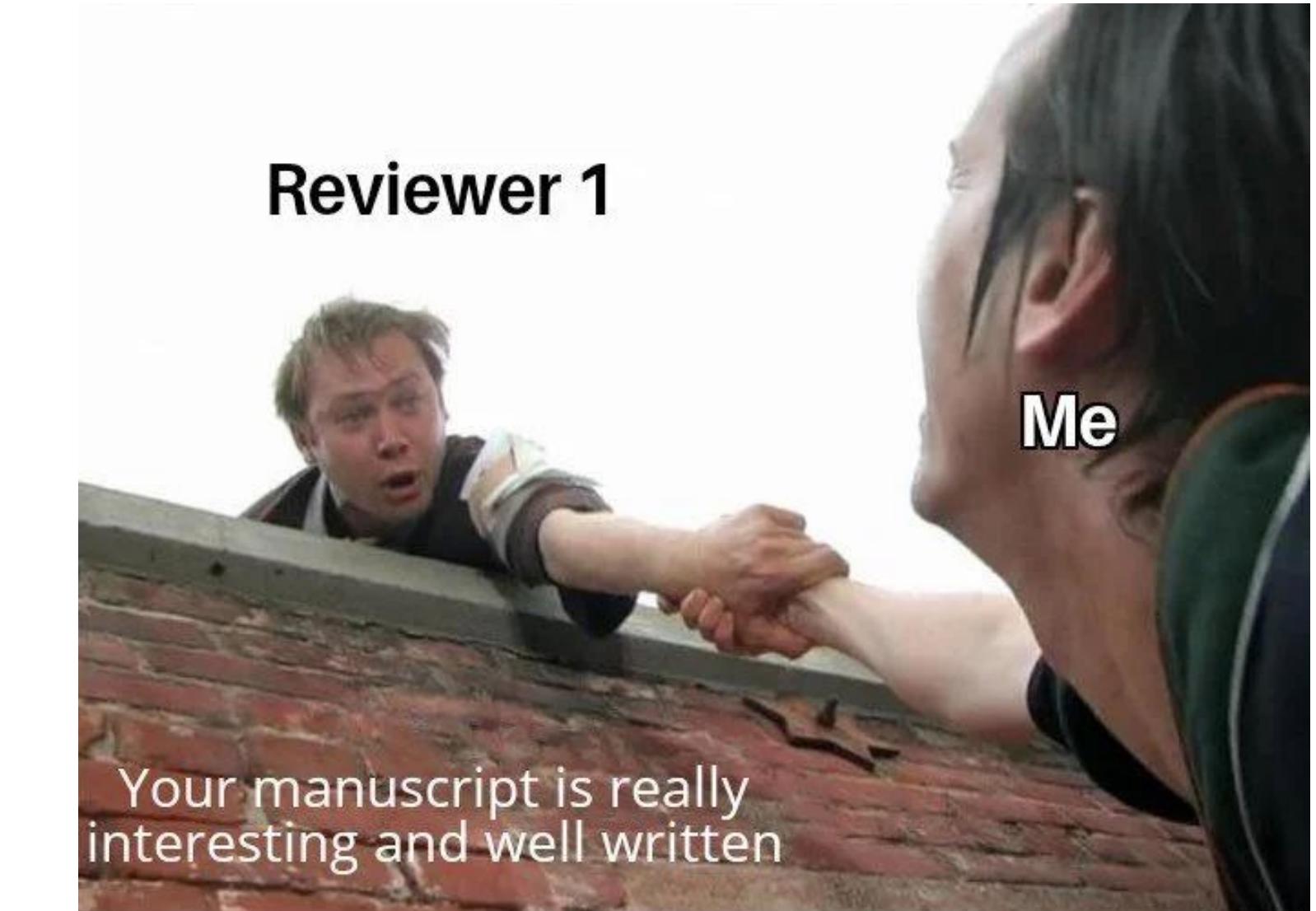
4

How to review a paper?

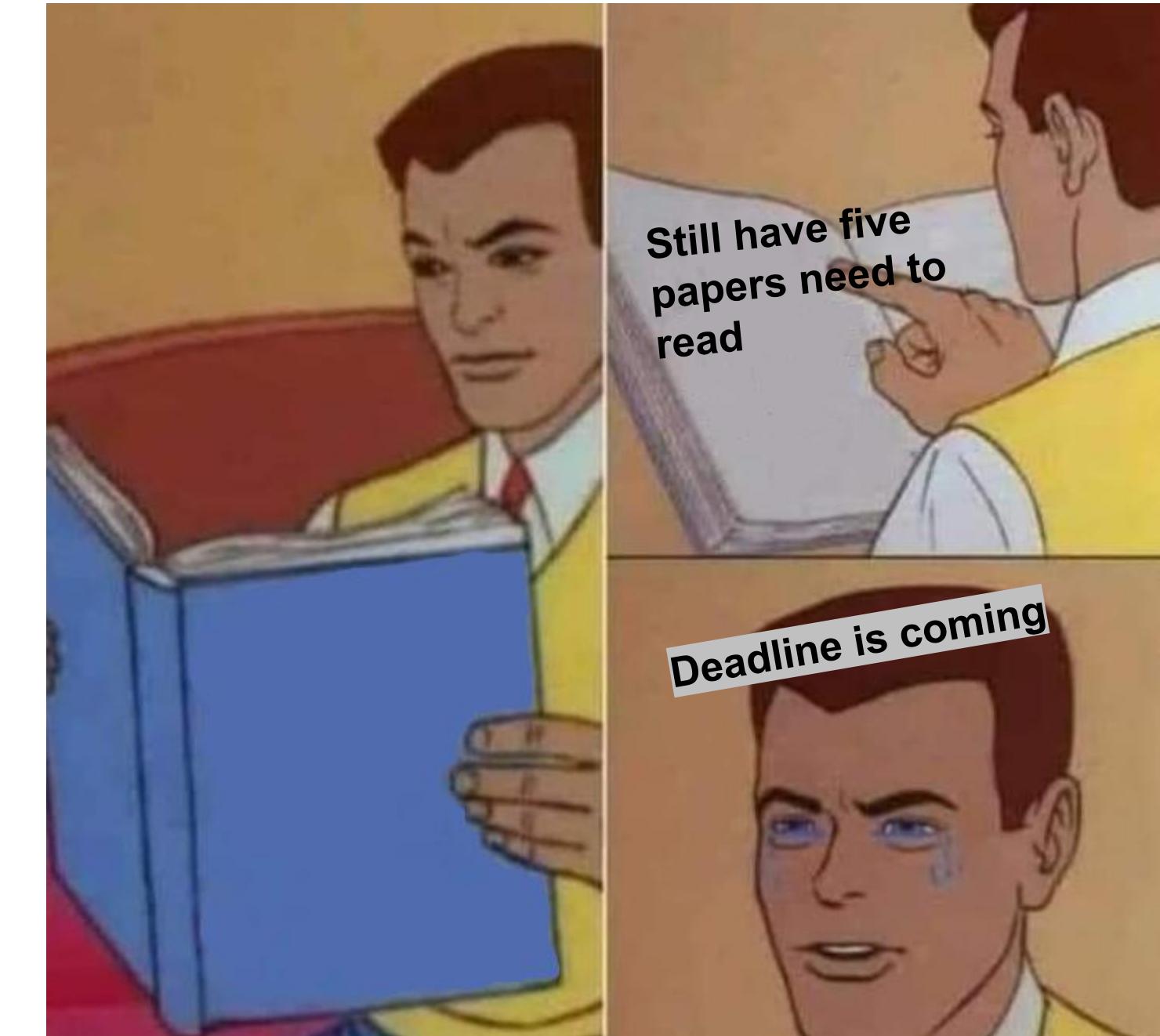
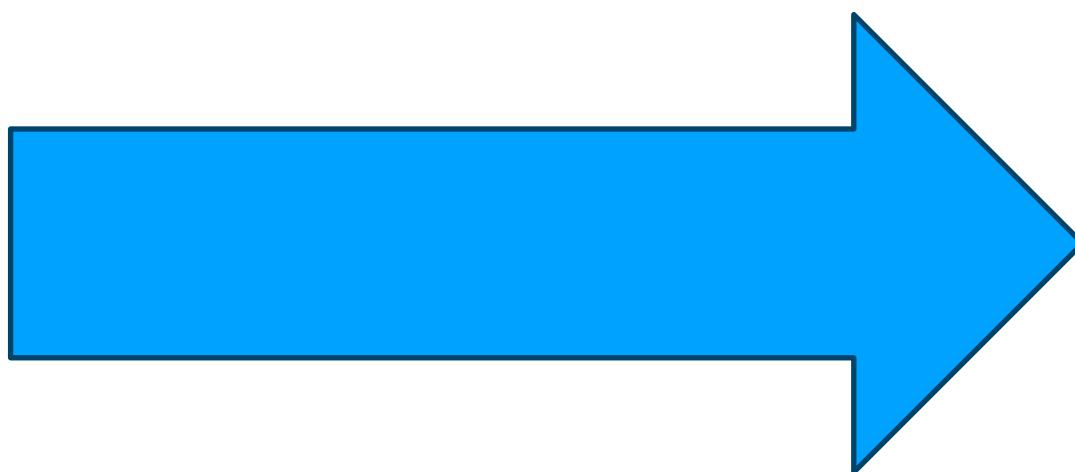


Reviewers are the quality control of a conference / journal, they

- Identify errors and flaws
- Improve the manuscript through suggestions
- Give credibility to the work



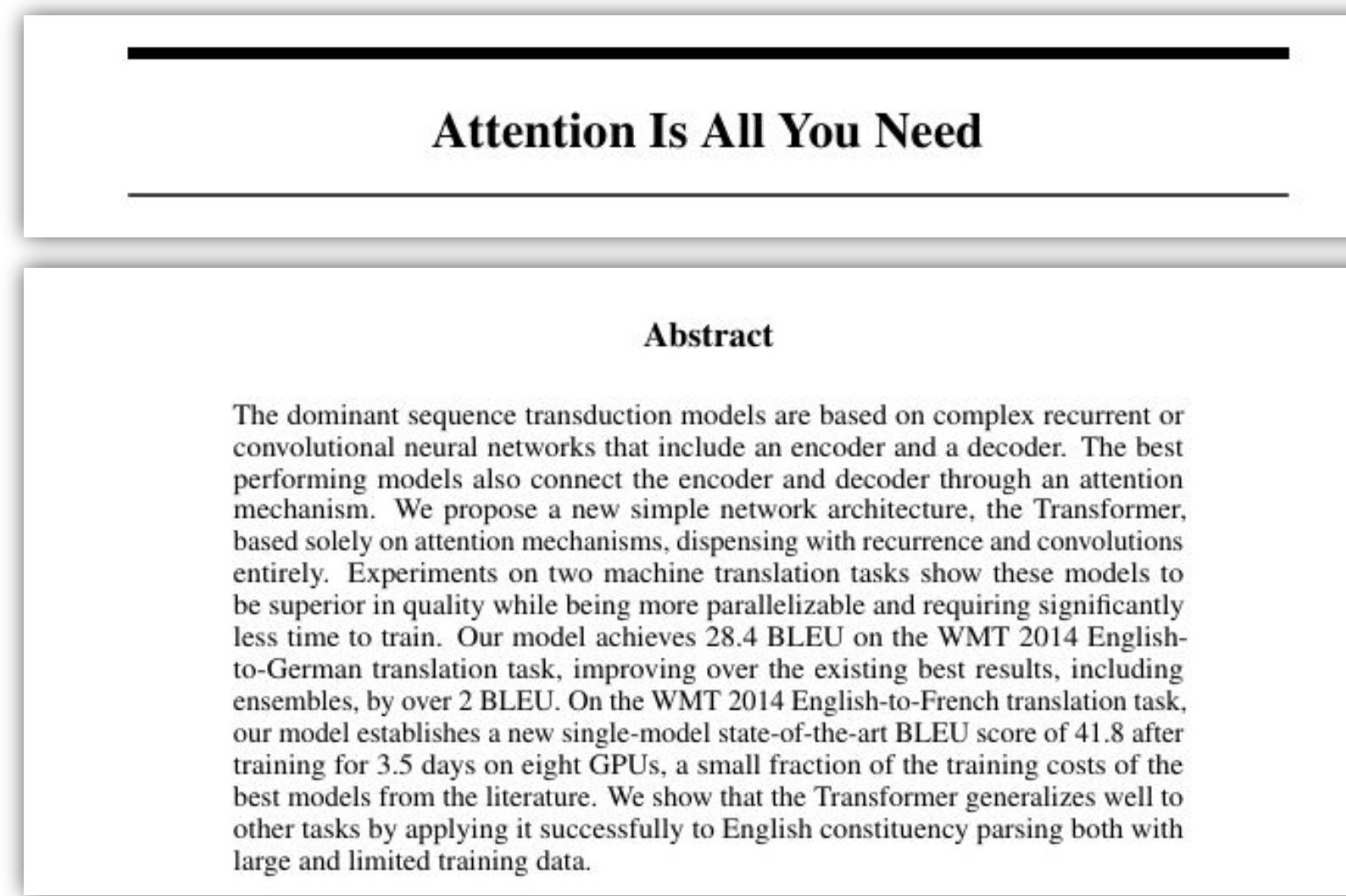
How to review a paper?



Reviewers are readers too — just readers with specific expectations who need to give feedback by a deadline.

How to review a paper?

Bidding Process



The screenshot shows a paper submission interface. At the top, a header reads "Attention Is All You Need". Below it, a section titled "Abstract" contains the following text:

The dominant sequence transduction models are based on complex recurrent or convolutional neural networks that include an encoder and a decoder. The best performing models also connect the encoder and decoder through an attention mechanism. We propose a new simple network architecture, the Transformer, based solely on attention mechanisms, dispensing with recurrence and convolutions entirely. Experiments on two machine translation tasks show these models to be superior in quality while being more parallelizable and requiring significantly less time to train. Our model achieves 28.4 BLEU on the WMT 2014 English-to-German translation task, improving over the existing best results, including ensembles, by over 2 BLEU. On the WMT 2014 English-to-French translation task, our model establishes a new single-model state-of-the-art BLEU score of 41.8 after training for 3.5 days on eight GPUs, a small fraction of the training costs of the best models from the literature. We show that the Transformer generalizes well to other tasks by applying it successfully to English constituency parsing both with large and limited training data.



The screenshot shows a search interface with the following navigation and filter options:

1 - 61 of 61 «« « **1** » »» Show: 25 50 100 All Clear All Filters Actions ▾

The Area Chair (AC) will initially assign a set of submissions to each reviewer based on their research background.

Reviewers should:

- Indicate which papers they are interested in reviewing.
- Indicate which papers they prefer not to review.

Based on this feedback, the AC will make the final paper assignments for each reviewer.

How to review a paper?

Review Process

Contact Chairs Help Center Select Your Role : Reviewer MICCAI2025 Jun Li

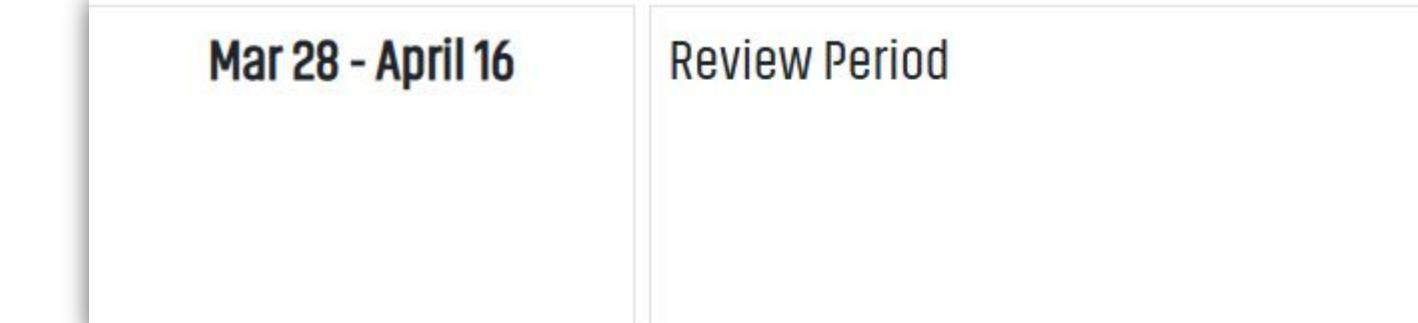
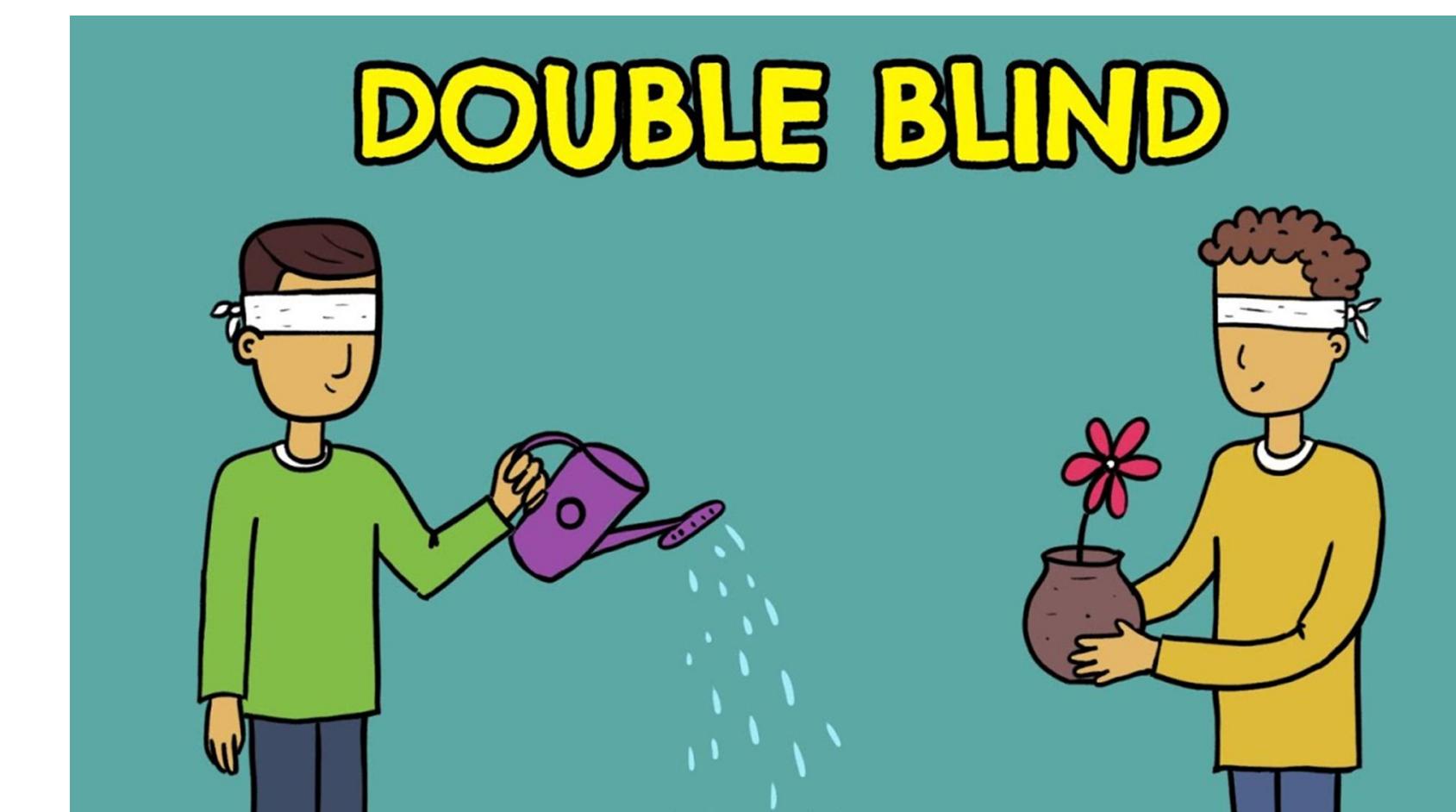
Reviewer Console

Please click [here](#) to view Welcome Message & Instructions.

Reviewing 1 - 7 of 7 << < > >> Show: 25 50 100 All Clear All Filters Actions

Paper ID	Title	Review & Discussion
		View Review ID: Reviewer #5 <input checked="" type="checkbox"/> Email Meta-Reviewer
		View Review ID: Reviewer #2 <input checked="" type="checkbox"/> Email Meta-Reviewer
		View Review ID: Reviewer #2 <input checked="" type="checkbox"/> Email Meta-Reviewer
		View Review ID: Reviewer #4 <input checked="" type="checkbox"/> Email Meta-Reviewer
		View Review ID: Reviewer #3 <input checked="" type="checkbox"/> Email Meta-Reviewer
		View Review ID: Reviewer #3 <input checked="" type="checkbox"/> Email Meta-Reviewer
		View Review ID: Reviewer #2 <input checked="" type="checkbox"/> Email Meta-Reviewer

- Usually, each reviewer will be assigned 5 papers.
- Also each paper will typically have 5 reviewers.



Ab. Two weeks to review 5 papers

How to review a paper?



What feedback do reviewers need on each paper?

- **Summary of the work:** (*short paragraph*)
- **Strengths:** (*bullet points*)
- **Weaknesses:** (*bullet points*)
- **Suggestion to Authors:** (*short paragraph to improve the paper*)
- **Rating:** (*pull-down menu*)
 - [strong accept | weak accept | borderline | weak reject | strong reject]
- **Rating Justification (preliminary/final):** (*short paragraph*)
- **Reviewer Confidence:** (*helps to weight review score*)

How to review a paper?

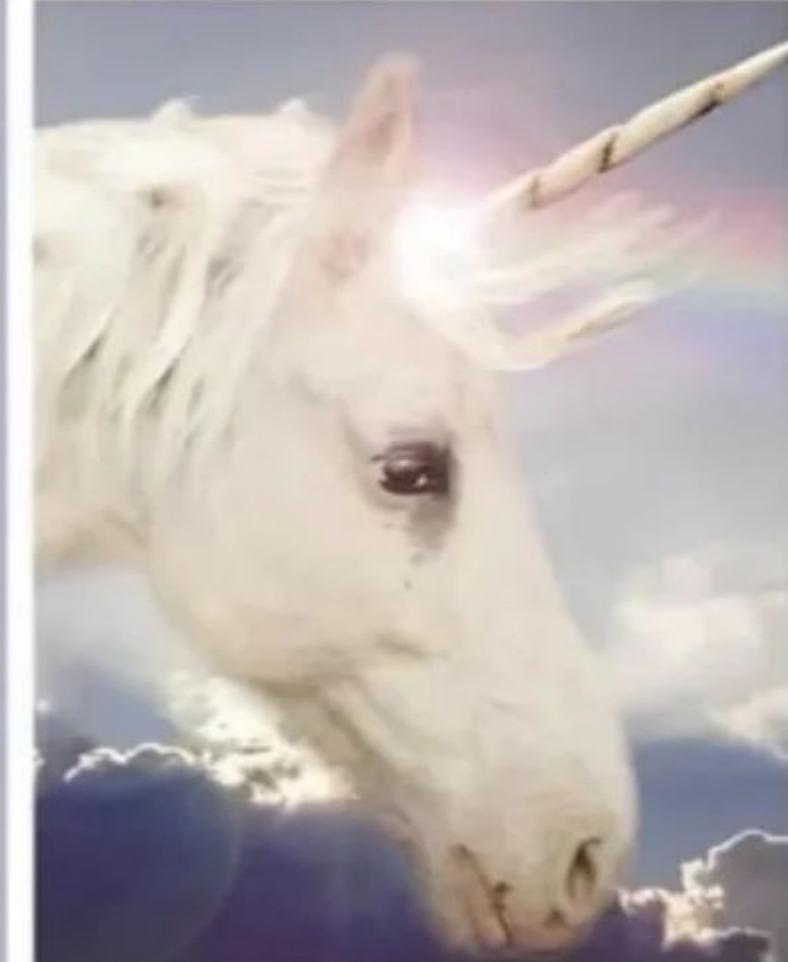
But, how to review a paper?

Sorry I will not tell you :)
You will find the answer when you
are a reviewer....



What is a good paper?

**Research
proposal**



**Actual
research**



Good Motivation

Clear Presentation

Well-designed
Experiments

Contribution to the
community

How to review a paper?

Good Motivation



- Unsolved Problem
- Gap in Existing Research
- New Meaningful Application
-

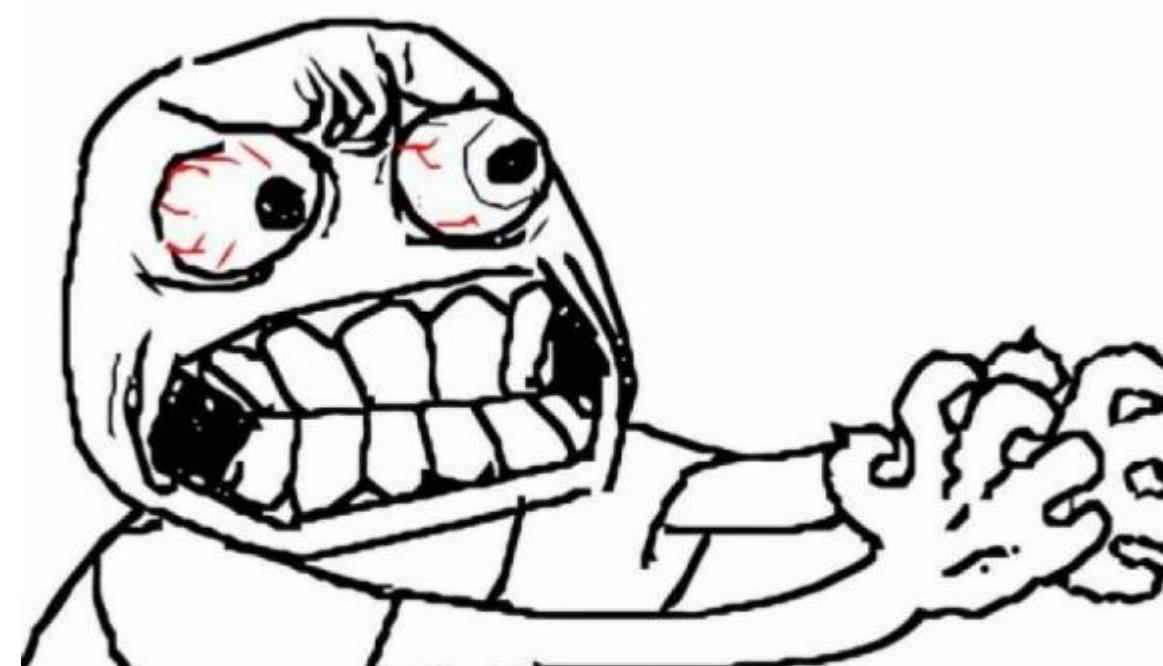
I want to research how to let
half of the people disappear



How to review a paper?

Clear Presentation

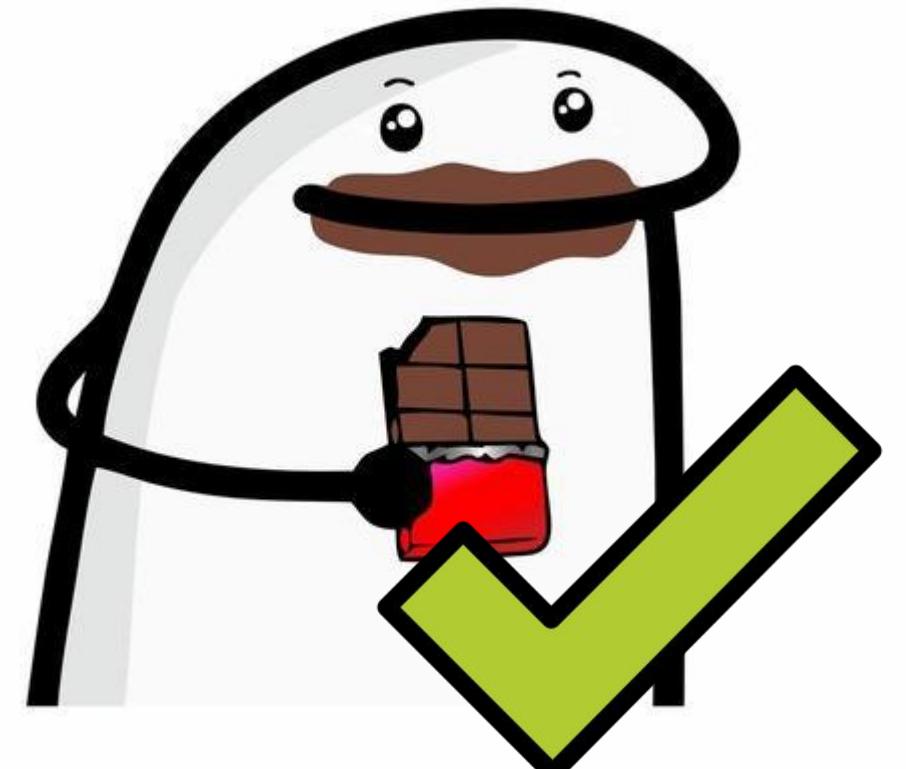
- No typos, No logical mistakes
- Clear structure
- Well-designed and informative figures
-



- This part I feel is most important for reviewer to have a basic feeling for the paper !!
- At least for me !!!

How to review a paper?

- Why clear Presentation is really important ?



Chocolate Flavored Poo



Poo Flavored Chocolate



POKER FACE



anger



bargaining



depression

How I feeling when I read a paper with logical problems, unclear motivation, unclear about the method details, experiment that not make sense

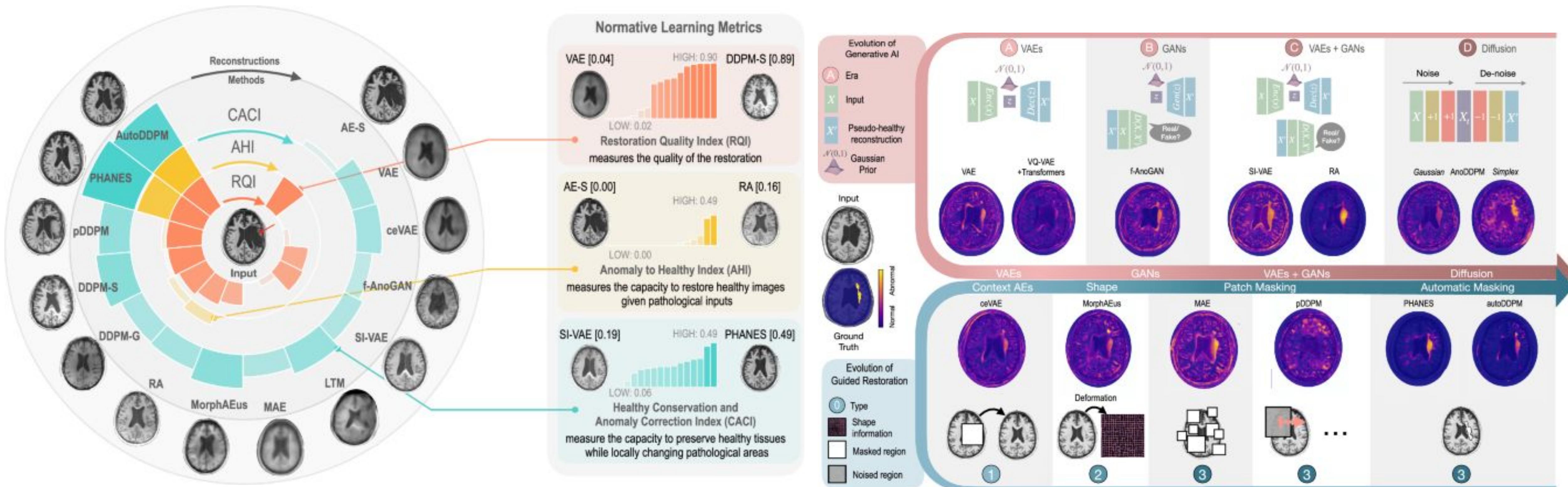
A good presentation may not guarantee your paper will be accepted

But a poor presentation almost certainly leads to rejection.

If you haven't even made an effort to get the basics right, how can reviewers trust that you put serious effort into the rest of the work?

How to review a paper?

Well-designed and informative figures



How to review a paper?

Well-designed
Experiments

Contribution to the
community

These two parts are where you
show muscle time 💪

After understanding your methods, reviewers will carefully examine your experiments, including:

- **How you test your methods** (Dataset, Implementation details)
- **Whether the results are convincing** (Have you provided enough comparison results?)
- **Whether each module in your method makes sense** (Detailed ablation studies for every component)A thorough discussion of your experimental details.

Contribution to the Community:

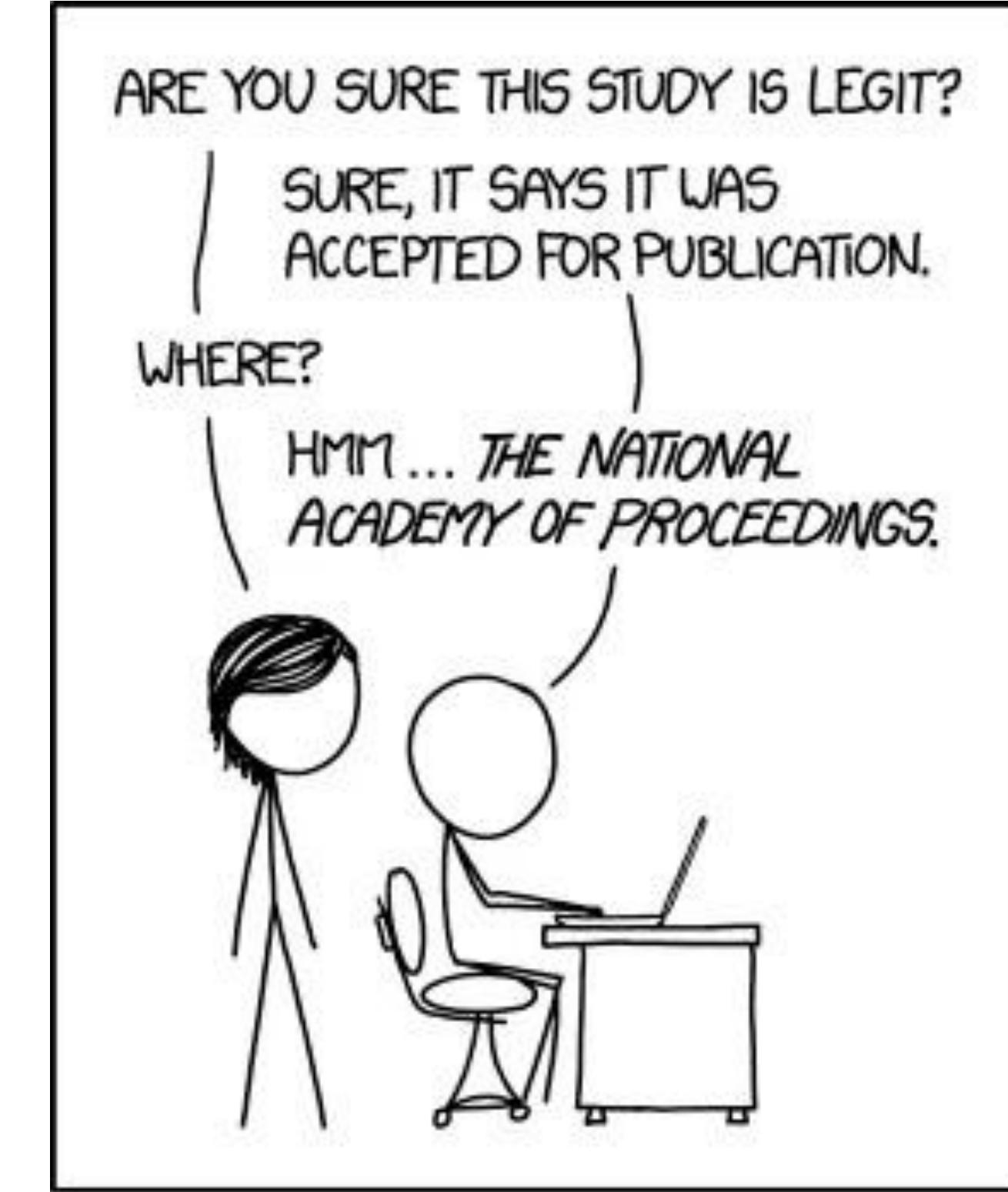
- Does the paper present an **interesting idea that hasn't been explored before?**
- **Is the paper's work reproducible?**
- Does it offer **meaningful discussion that encourages others to explore further?**
-



Publishing a paper

Why?

- Make your work more credible
- Share it with the community
- Get feedback
- Get citations (and fame)
- Get grants and jobs to publish more papers ...



IF SOMETHING IS FORMATTED LIKE A SERIOUS SCIENTIFIC PAPER, IT CAN TAKE ME A WHILE TO REALIZE IT ISN'T ONE.

xkcd.com

Where to publish?

Conference

- Present your work at a physical location in the presence of other researchers
- Strict submission deadline (usually once a year)
- Shorter papers
- Papers get published in the conference proceedings
- Usually two rounds of peer review

Journal

- No presentation
- Submit your work at any time
- Requires more rigorous analysis (often extensions of conference papers)
- Multiple rounds of peer review and resubmissions (longer time until decision)



Poster sessions in conferences

- Personal
- Informal
- Good way of connecting with people and promoting your research



Poster sessions (real - Neurips 23)



What a poster should do

- Catch attention
- Visual aid for your pitch
- Cover the gist of your work



Your poster must/should include certain elements

- **A title.** It doesn't need to be the paper title, the main conclusion does also work. The paper title should then appear at least in the subtitle
- **The authors.** In your case also the presenter (you)
- **Affiliations.** All the universities, institutes, and companies that were involved
- **The main content.** See next slide
- **Contact information.** An E-Mail address is sufficient
- **Qr-code.** To the project page, GitHub, paper, ... (Not to the poster itself!)

Your poster should tell a story of three parts

01.

What is the problem?

There is a reason why you did this research. Name it!

02.

How did you solve it?

Mention your **key contributions** to solve the problem above.

03.

Did it work?

Show your main experimental results to convince the listener.

(PS in machine learning papers, the answer to „did it work?“ Must always be „yes“)

Layout

- **Use only minimal text.** Nobody will read through blocks of text
- **Formulas only if they are a key contribution.** I know it looks nice and you feel smart but they add no information and waste space
- **Only key results and figures.** Put enough results to convince your listeners to trust the research but don't add every ablation study
- **Add visual results if possible.** They help to understand the problem a lot
- **Highlight your most important parts.** For example your key finding
- **Use visual separations** to organize your poster. Use borders, boxes, background colors, or distances to group parts together
- **Don't use more than three font sizes**
- **Use fonts without serifs**
- **Use a color palette.** For example from here: coolors.co/palettes/trending
- **Use talking headlines.** E.g. „current methods fail at xy“ instead of „Motivation“
- **Important information on the top.** In crowded poster sessions, often only the top is visible.³³

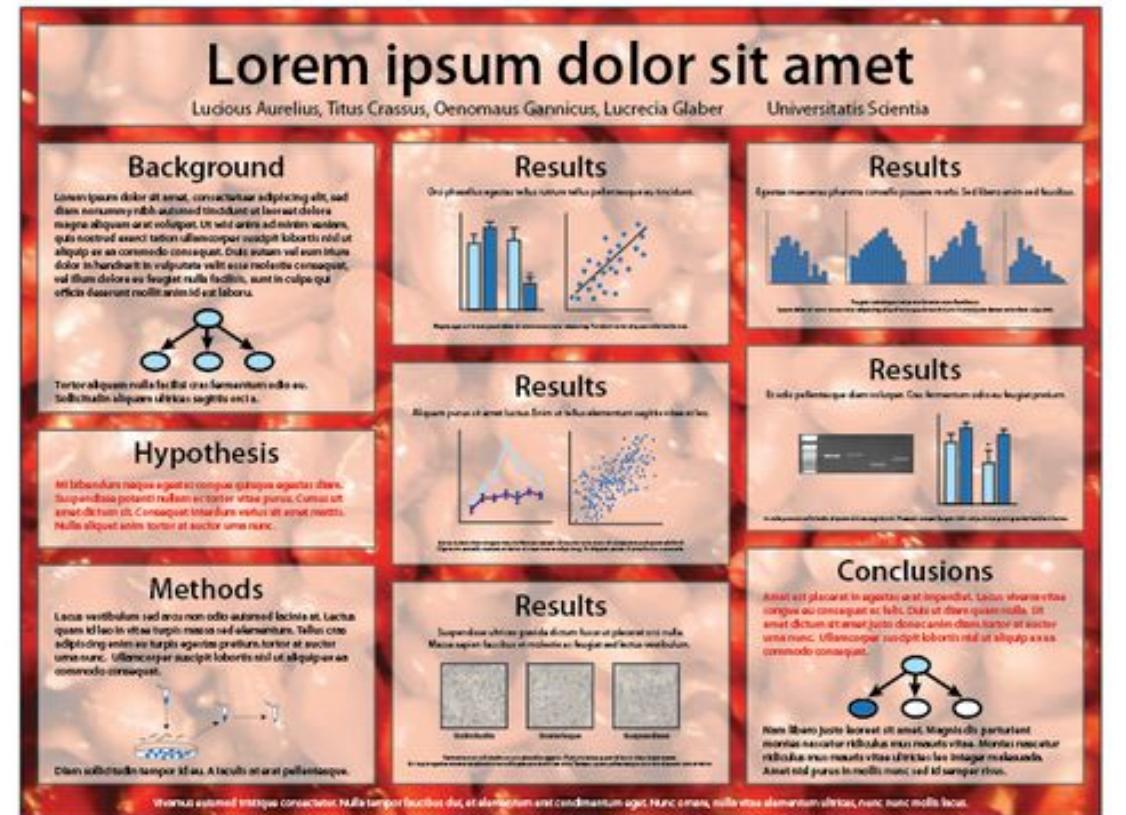
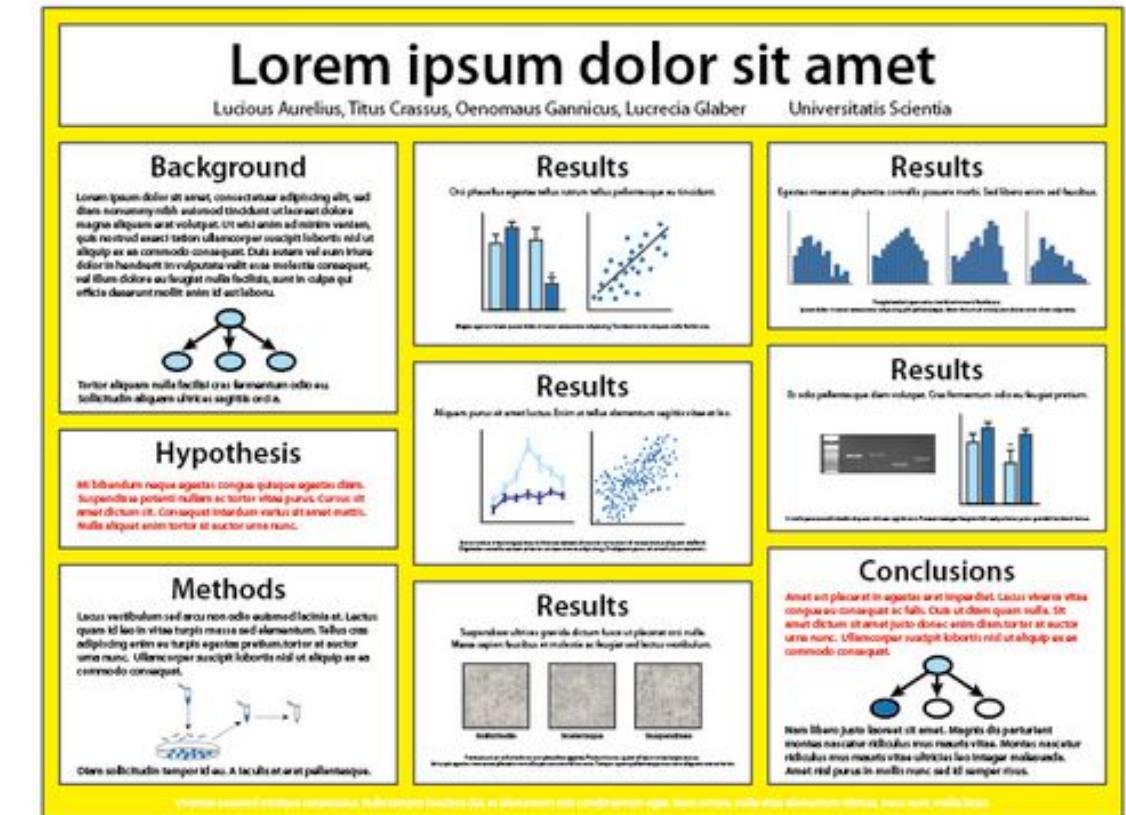
How not to make a poster

- Too many colors
- Too bright colors
- Distracting background colors

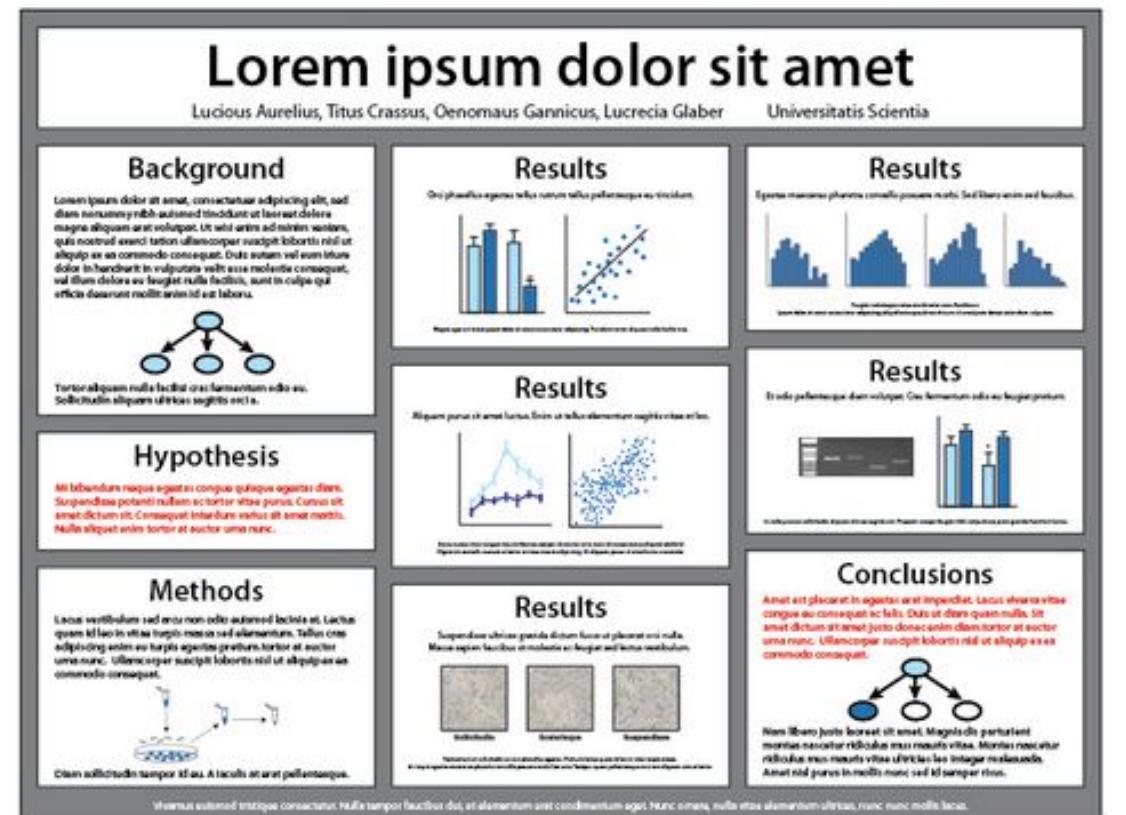
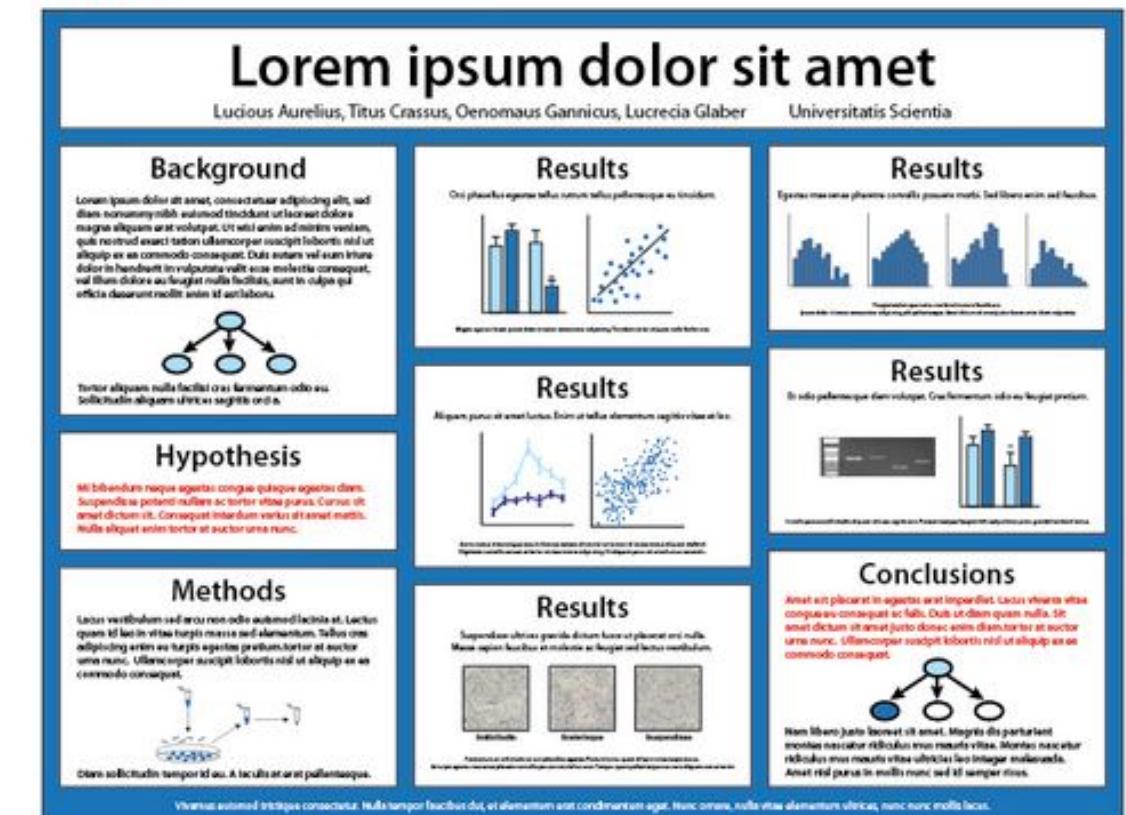
Kill me now!



Before



After



Cool colors naturally fade into the background so that your text and figures are the main attraction.

How to make a poster



T-04-046

Fast Non-Markovian Diffusion Model for Weakly Supervised Anomaly Detection in Brain MR Images

Jinpeng Li¹, Hanqun Cao¹, Jiaze Wang¹, Furui Liu¹, Qi Dou^{1,2}, Guangyong Chen³, and Pheng-Ann Heng^{1,2}

¹ Department of Computer Science and Engineering, The Chinese University of Hong Kong
² Institute of Medical Intelligence and XR, The Chinese University of Hong Kong
³ Zhejiang Lab

Introduction
Background: Deep generative model for medical image analysis plays an increasingly important role in medical anomaly detection. Deep learning methods require large amount of high-quality supervised data for accurate segmentation, while only limited exquisite amount labelled data and large amount of informativeless data are obtained in real-world application, presenting challenges for segmentation.

Challenges: Existing anomaly detection methods including GAN, NF, and Diffusion models detect anomaly by generated healthy samples. However, the performance is limited by the lack of high-fidelity training data, low image expressiveness, and ill-conditioned guidance for image translation. Ideally, generative models should take full usage of weak-annotated training data and conduct sampling with strong guidance.

Method
The Proposed Framework: We propose a novel non-Markovian hybrid conditioned diffusion model with fast samplers. Our approach utilizes hybrid image conditions integrated by coarse segmentation maps and gradient map [12], based on the non-Markovian assumption and performs high-order ODE sampler for fast generation [3,4].

Non-Markovian Training & Hybrid Condition

Illustration of proposed FNDM framework
Forward Condition vs **Reverse Condition**
Visualization Results:

Reference
[1] Li, J., et al. "Fast non-markovian diffusion model for weakly supervised anomaly detection in brain mr images." NeurIPS 2022.
[2] Chavhan, A. et al. "Diffusion model beat gans on image synthesis." NIPS 2022.
[3] Lu, Cheng, et al. "Dom-solver: A fast adic solver for diffusion probabilistic model sampling in around 10 steps." NIPS 2022.
[4] Lu, Cheng, et al. "Dom-solver++: Fast solver for guided sampling of diffusion probabilistic models." arxiv 2022.
[5] Li, J., et al. "Identifying the best machine learning algorithms for brain tumor segmentation, progression assessment, and overall survival prediction in the brats challenge." arxiv 2018.
[6] Petzschke, M., et al. "3D multi-organ magnetic resonance imaging stroke lesion segmentation dataset." arxiv 2022.

T-03-079

Multi-Head Multi-Loss Model Calibration
Adrian Galdran, J. Verjans, G. Carneiro, M. A. G. Ballester

Calibration: a model's predictions are aligned with the actual probability of the model being correct. State-of-the-art: Deep Ensembles (DEs), but they are expensive for training and inference. We propose: A form of simplified ensembling: replace the linear classifier at the end of a network by a set of heads supervised with different loss functions to enforce diversity on their predictions. Each head minimizes a weighted Cross-Entropy loss, but the weights are different for each branch. Results: We achieve excellent calibration without sacrificing accuracy. Multi-Head Multi-Loss classifiers are inherently well-calibrated, outperform other calibration techniques & compare to DEs.

Keywords: Model Calibration · Uncertainty Quantification · <https://arxiv.org/abs/2303.01099> · https://github.com/agaldran/mhml_calibration

Introduction
Background: We present our fast conditional sampler by modifying the forward and reverse process as a higher-order ODE scheduler [3,4] and applying hybrid conditions from predicted noise, coarse segmentation map, and original input for accelerated and high-fidelity generation. We obtain the final anomaly results by subtraction between samples and inputs.

Experimental Results
Dataset: The BRATS 2020 [5] and ISLES 2022 [6] are preprocessed MR datasets used for brain tumor and stroke lesion segmentation. They use image-level binary labels for training and Dice, Volumetric Similarity, and Hausdorff Distance Metric for evaluation.

Performance Comparison: We compare our method with existing anomaly detection methods including memory-based, normalizing flow based, distillation based and diffusion based algorithms.

Table 1. Comparison with state-of-the-art anomaly detection methods. The best performances are bolded. The second-best performances are underlined.

Method	Dice	BHSS	VSim ¹	Dice	Hausd ²	VSim ¹
PadimCore [8]	36.76	5.78	57.83	4.89	8.35	4.89
CFlow [11]	38.95	5.45	58.40	3.08	9.08	3.38
CSFlow [22]	22.83	7.05	44.00	7.73	6.06	7.23
PatchCE [13]	32.32	5.55	55.53	5.55	8.27	5.65
ResDR [4]	34.46	7.46	37.87	6.17	9.17	9.18
STPFM [21]	53.83	4.82	72.20	10.32	6.75	12.12
PatchConv [23]	51.78	4.95	63.20	6.77	7.07	7.07
DINet [14]	54.45	8.72	81.71	34.46	3.38	48.68
ENMD(Our)	76.21	<u>3.80</u>	<u>82.28</u>	<u>54.44</u>	<u>1.99</u>	<u>75.41</u>

Interpretation: If the m heads/branches return similar predictions, then the gradients at each logit z^m are equal, and updates are the same among branches p^m . Diversity of predictions making up p^i is reduced.

Multi-Head Multi Loss Models
How to promote diversity? We supervise each head with a differently weighted loss function:
 $\mathcal{L}_{MH}(\mathbf{p}, \mathbf{y}) = \mathcal{L}_{CE}(\mathbf{p}', \mathbf{y}) + \sum_{m=1}^M \mathcal{L}_{w^m - CE}(p^m, y)$ $\mathbf{p} = [p^1, \dots, p^N]$ is a stack of predictions; in the paper we show that the gradient $\nabla \mathcal{L}_{MH}(\mathbf{p}, \mathbf{y})$ results in different updates for each head iff weights w^m are different.

We use weighted Cross-Entropy with a random configuration of weights so that each branch focuses more on a different subset of categories (see paper for details).

Figure 2. Ablation study on the hybrid guidance in our method.

Figure 3. Ablation on steps. We use the same step value in encoding and sampling.

Figure 4. Ablation on steps. We use the same step value in encoding and sampling.

Experimental Results
Datasets:
1) Chaoyang dataset: 6,160 colon histopathology images unevenly distributed in 4 classes, with some label ambiguity, reflecting high aleatoric uncertainty.
2) The Kvasir dataset: 10,662 endoscopic images, challenging due to a high amount of classes (23) and high class imbalance.

Metrics:
Expected Calibration Error measures calibration alone. Accuracy quantifies discrimination but not "probabilistic quality". Finally, NLL, a PSR: it measures calibration and discrimination. It's not possible to have a low PSR value without both calibration & accuracy.

Multi-Head Multi-Loss models have better calibration, close to Deep Ensembles. In addition, they achieve good calibration without sacrificing predictive performance. This is reflected jointly by low NLL values, and a better aggregated ranking. We observe an improved performance as we increase the diversity (2HSL vs 2HML) and as we add heads (2HML vs 4HML).

T-04-111

Uncovering Heterogeneity in Alzheimer's Disease from Graphical Modeling of the Tau Spatiotemporal Topography
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Introduction
Automated tools for characterizing the heterogeneity of tau pathology will enable a more accurate understanding of Alzheimer's disease and help the development of targeted treatments.

We presents a novel Reeb graph representation that encodes the topography of tau pathology from PET imaging, and a directed-graph-based framework for uncovering spatiotemporal heterogeneity from cross-sectional PET data.

With large-scale imaging data from the ADNI and A4 studies, we obtained three subtypes with systematic spatiotemporal variations.

Both experiments on simulations and real data show our method exhibits more robust generalization performance than the state-of-art event-based model.

Results
Synthetic Datasets. The synthetic data is generated according to Eq. (4). Three seed regions are selected with corresponding peak SUVR magnitudes as shown in (a)(b). With synthetic data, we can demonstrate that our method is sufficient for disentangling correct subtypes, and its superior performance over the state-of-art Sustain method [3] as shown in (c).

Figure 1. Overview of the proposed method

Figure 2. Results on ADNI and A4 data. Using selected avoid-patches-positive (A+P+) data, we obtained three pathologically different subtypes with three stages, respectively. For subtype 1, more tau pathology distributes in the medial temporal lobe, which is consistent with the classic Braak staging for subtype 2, tau pathology primarily occurs in the occipital area and sequentially spreads to parietal and other regions; subtype 3 does not show significant differences among brain regions, and the overall SUVR is lower among all subtypes.

Figure 3. Further evidence of the clinical relevance of the subtyping results from our method is indicated by the cognitive scores of ADNI subjects

Figure 4. Generalization of proposed method. ADNI and A4 data are estimated on the models trained with the data from the other cohort. The consistency between subtyping and estimation can quantify the generalization performance. Our method focuses on the spreading patterns and is robust to intensity variations, which leads to a more stable performance across cohorts even two cohorts have different SUVR distributions.

Conclusion
In the current study, we proposed a novel directed-graph-based framework with a new spatiotemporal pattern representation for parsing tau pathology heterogeneity and demonstrated its improved performance over the state-of-art Sustain method. Application of the proposed method on large-scale PET imaging datasets successfully demonstrated three subtypes with clear relevance to previously well-described clinical subtypes with distinct spatiotemporal patterns.

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Acknowledgement: This work is supported by the National Institute of Health (NIH) under grants R01EB022744, R01AG077578, R01AG056573, R01AG064584, R21AG064776, P41EB015922, U19AG078109.

How to make a poster



O⁶-Benzylguanine Inhibits Tamoxifen Resistant Breast Cancer Cell Growth and Resensitizes Breast Cancer Cells to Anti-Estrogen Therapy

Joshua Smith¹, George C Bobustuc¹, Rafael Madero-Vishbal¹, Jimmie Colon¹, Beth Isley¹, Jonathan Ticku¹, Kalkunte S. Srivenugopal and Santhi Konduri^{1*}

¹Cancer Research Institute of M.D Anderson Cancer Center Orlando ²Texas Tech University Health Sciences Center, Amarillo, TX

MD ANDERSON CANCER CENTER ORLANDO
SUPPORTED BY THE CHARLES LEWIS INSTITUTE

Abstract

Endocrine therapies using anti-estrogens are least toxic and very effective for breast cancers, however, tumor resistance to tamoxifen remains a stumbling block for successful therapy. Based on our recent study on the involvement of the DNA repair protein MGMT in pancreatic cancer (Clin Cancer Res, 15, 6087, 2009), here, we investigated whether MGMT overexpression mediates tamoxifen resistance. Specifically, we determined whether administration of O⁶-benzylguanine (BG) at a non-toxic dose alone or in combination with the anti-estrogens (tamoxifen/fulvestrant) curtails human tamoxifen resistant breast cancer growth. Further, we also determined whether BG sensitizes breast cancers to tamoxifen using tamoxifen resistant cells.

MGMT expression was found to be increased in breast cancer cells relative to normal breast epithelial cells. Silencing of the ER- α expression using a specific siRNA resulted in augmentation of MGMT mRNA and protein levels by 2 fold. We also observed an inverse correlation between MGMT and p53 levels in breast cancer cell lines; moreover, p53 downregulation was accompanied by increased MGMT expression. Other experiments showed that BG alone or BG in combination with tamoxifen or fulvestrant decreased ER- α expression, whereas tamoxifen alone and fulvestrant alone increased and decreased the same respectively. However, all these treatments increased the p21^{WIF1} mRNA and protein expression significantly. BG inhibited tamoxifen resistant breast cancer growth in a dose-dependent manner and it also sensitized resistant breast cancer cells to anti-estrogen therapy (TAM/ICI). These combinations also enhanced the cytochrome C release and the PARP cleavage, indicative of apoptosis. In breast cancer xenografts, BG alone or a combination of BG with tamoxifen or fulvestrant caused significant tumor growth delay and immunohistochemistry revealed that BG inhibited the expression of MGMT, ER- α , ki-67 and increased p21^{WIF1} staining. These findings suggest that MGMT inhibition may provide a novel and effective approach for overcoming tamoxifen resistance.

Introduction

Recent advances in breast cancer research have identified key pathways involved in the repair of DNA damage induced by chemotherapeutic agents. The ability of cancer cells to recognize DNA damage and initiate DNA repair is an important mechanism for tumor progression. There are three main mechanisms of DNA-damaging agents: alkylating agents that attack the nucleophilic N⁷ position on guanine, forming mutagenic and highly cytotoxic interstrand DNA crosslinks. The DNA repair enzyme O⁶-benzylguanine DNA alkyltransferase (AGT), encoded by the gene MGMT, repairs alkylation at this site and is responsible for protecting both tumor and normal cells from alkylating agents. MGMT is expressed constitutively in normal cells and tissues. In breast tumors, MGMT gene expression is elevated and levels are up to 4-fold higher than in the normal breast. Interestingly, it has been shown that tamoxifen accelerates proteasomal degradation of MGMT in human cancer cells. In 1991, Pegg, Moschel, and Dolan observed that O⁶-benzylguanine (BG) inhibited AGT and potentiated the cytotoxicity of both chloroethylnitrogen mustards and alkylating agents. Moreover, BG is a substrate for MGMT and can inhibit its activity. Thus, BG is a pseudosubstrate for MGMT which results in the covalent transfer of benzyl group to the active site cysteine, the MGMT protein is degraded after each reaction. This stoichiometric reaction mechanism effectively depletes the AGT content in tumors and the associated repair of alkylation damage. BG is currently undergoing clinical trials in various cancers to increase the efficacy of alkylating agents.

Interestingly, several observations suggest an inverse correlation between the levels of MGMT and p53 tumor suppressor proteins where wild-type p53 suppresses transcription of human MGMT expression. Unfortunately, p53 function is often inactivated or suppressed in human cancers; therefore, restoration of wt-p53 activity is essential for the success of some treatments. However, whether or not this is mediated by suppression of MGMT expression has yet to be determined. To date, the cross-talk between MGMT and ER- α (and the link to p53 expression) has not been explored in drug (i.e., tamoxifen) resistant breast tumors. The anti-estrogen tamoxifen is the most commonly used treatment for patients with estrogen receptor positive breast cancer. Although many patients benefit from tamoxifen in the adjuvant and metastatic settings, resistance to this endocrine therapeutic agent is an important clinical problem. The primary goal of present study was to investigate the mechanisms of anti-estrogen drug resistance and to design new therapeutic strategies for circumventing this resistance. The results show that MGMT expression is increased in TAM-resistant breast cancers and inhibition of MGMT by BG significantly improves TAM-sensitivity.

Results

Prolonged Treatment of Tamoxifen Increases MGMT Expression: We developed a tamoxifen resistant MCF-7 cell line by using prolonged treatment of tamoxifen on the parental ER-positive breast cancer cell line, MCF-7. Tamoxifen-resistant MCF-7 cells proliferate at rates similar to the parental MCF-7. Prolonged treatment of tamoxifen on MCF-7 cells increased MGMT expression compared to parental MCF-7 cells by 2 fold (Fig.1).

Knocking Down ERA Enhances MGMT Expression in Tamoxifen Resistant Breast Cancer Cells: It is not known whether ER- α and MGMT transcriptionally regulate each other in tamoxifen resistant breast cancer cells. We therefore investigated whether down regulation of ER- α has any effect on endogenous MGMT expression in these cells. As expected, downregulation of ER- α using specific siRNA significantly reduced ER- α protein levels in these cells. Western blot analysis performed and the results showed (Fig. 2A) silencing of ER- α increases MGMT expression in these cells, and interestingly, the results in the right panel (Fig.2B) show increased MGMT mRNA levels were increased as assessed by qRT-PCR. These data suggest that ER- α -mediated signaling functions to repress MGMT gene expression in breast cancer cells.

Transcriptional Regulation Between MGMT and p53: Previously, it was reported that p53 negatively regulates MGMT in breast cancer cells. Therefore, we addressed whether or not silencing the p53 enhances endogenous MGMT transcription. Tamoxifen resistant MCF-7 cells were transfected with either p53 siRNA (p53-KD) (Fig.2C) or MGMT siRNA (MGMT-KD) (Fig.2D) along with either ER- α siRNA (NS). MGMT expression was significantly reduced in p53 knock down cells. We did experiments showing a -fold augmentation (Fig. 2A) and as expected, knocking down MGMT decreased MGMT transcription where as p53 mRNA levels were unaffected in MGMT knockdown cells (Fig.2D). These results confirms that p53 can regulate MGMT at the transcriptional level.

O⁶-Benzylguanine Modulates p53 Down-Stream Targeted Protein Expressions: Encouraged by the results reported, we investigated the effect of combination therapy on endogenous MGMT, p53, and ER- α protein expressions. As expected, BG decreased MGMT expression, while combination therapy (4-OH-TAM or ICI combined with BG) significantly decreased both MGMT and ER- α expressions. BG alone or in combination with tamoxifen or ICI decreased ER- α expression, whereas tamoxifen alone and ICI alone increased and decreased the same respectively (Fig.3A). p53 expression was slightly altered after ICI treatment. The reduction in p53 expression by ICI alone was reversed when BG was combined (Fig.3A). We investigated the effect of BG on proteins which are involved in cell cycle regulation, apoptosis in tamoxifen resistant breast cancer cells. All these treatments significantly increased the p21^{WIF1} protein expression. P21 is a tumor suppressor protein which is involved in the mitochondria, cytochrome C is released (Fig.3B), and apoptosis was triggered in these cells in presence of combination therapy. PARP cleavage is seen in BG treated cells in presence of staurosporin as an indicator of apoptosis (Fig.3C). Therefore, this data suggest that BG promotes cell cycle arrest and can induce apoptosis by modulating p53 function.

O⁶-Benzylguanine Modulates Transcriptional Targets in Tamoxifen Resistant Breast Cancer Cells: The effect of combination therapy on endogenous MGMT mRNA levels was also studied. Quantitative Real Time PCR (qRT-PCR) resulted that anti-estrogens (TAM/ICI) increased the MGMT expression while the combination therapy decreased it compared to control levels. ER- α transcription was decreased compared to controls with all these treatments (Fig.4A). Surprisingly, p21 and PUMA mRNA was significantly increased in the presence of combination treatments (Fig.4B & C). These results suggests that p53 mediated target gene transcription was affected by the drug combinations in breast cancer cells (Fig. 4A).

O⁶-Benzylguanine Enhances p21 Transcriptional Activity in Tamoxifen Resistant Breast Cancer Cells: In order to investigate the effect of BG on p53 function, we performed luciferase reporter assays. Tamoxifen resistant MCF-7 breast cancer cells were transfected with p21 luc promoter construct in presence or absence of BG (target gene of p53). These results clearly demonstrate that BG significantly enhanced p21 transcriptional activity by 4-5 fold in these cells (Fig.4D).

Figure 1. MCF-7 parental and tamoxifen resistant MCF-7 cell pellets were prepared, proteins were isolated and MGMT expression was determined by Western blot analysis. Tamoxifen resistant MCF-7 breast cancer cells significantly increased MGMT expression compared to MCF-7 parental cells.

Figure 2. MCF-7 parental and tamoxifen resistant MCF-7 cell pellets were prepared, proteins were isolated and MGMT expression was determined by Western blot analysis. Tamoxifen resistant MCF-7 cells were transfected with either p53 siRNA (p53-KD) (Fig.2C) or MGMT siRNA (MGMT-KD) (Fig.2D) along with either ER- α siRNA (NS). MGMT expression was significantly reduced in p53 knock down cells. We did experiments showing a -fold augmentation (Fig. 2A) and as expected, knocking down MGMT decreased MGMT transcription where as p53 mRNA levels were unaffected in MGMT knockdown cells (Fig.2D). These results confirms that p53 can regulate MGMT at the transcriptional level.

Figure 3. MCF-7 tamoxifen resistant MCF-7 breast cancer cells were treated with BG (50 μ M) for 4hrs and later 4-OH tamoxifen and ICI (50 μ M) was either alone or in combination with BG and 24hr later cells were harvested and total RNA was isolated. (A) MGMT and ER- α (B) p21 transcription (C) PUMA mRNA was determined by qRT-PCR. (D) ER- α mRNA was determined by qRT-PCR. (E) Tamoxifen resistant MCF-7 cells were transfected with p21-luc construct and 6hr later treated with BG and 24hr later cells were harvested. p21 transcriptional activity was significantly increased by BG in these cells.

Figure 4. Tamoxifen resistant MCF-7 breast cancer cells were treated with BG (50 μ M) for 4hrs and later 4-OH tamoxifen and ICI (50 μ M) was either alone or in combination with BG and 24hr later cells were harvested and total RNA was isolated. (A) MGMT and ER- α (B) p21 transcription (C) PUMA mRNA was determined by qRT-PCR. (D) ER- α mRNA was determined by qRT-PCR. (E) Tamoxifen resistant MCF-7 cells were transfected with p21-luc construct and 6hr later treated with BG and 24hr later cells were harvested. p21 transcriptional activity was significantly increased by BG in these cells.

Conclusions

- 1. In the present study, we observed that prolonged treatment with anti-estrogens causes drug resistance by inducing the DNA repair protein O⁶-benzylguanine DNA methyltransferase (MGMT).
- 2. Decreasing the expression of MGMT by exposing breast cancer cells to BG sensitized these cells to anti-estrogen therapy (tamoxifen and ICI 182,786).
- 3. We also observed that combination therapy of anti-estrogens and MGMT blockers not only overcame the MGMT derived drug (tamoxifen and ICI) resistance but also increased the efficacy of anti-estrogen therapy by decreasing estrogen receptor expression and restoration of the functional activity of p53 in tamoxifen-resistant breast cancer cells.
- 4. Combination therapy inhibited tamoxifen resistant breast tumor growth *in vivo*.

Acknowledgements

We would like to thank the Florida Department of Health, Sunshine-Caley Cancer Research Program (CPR-04-04) for their funding of this project.

Good

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Bad

- ?

36

How to make a poster



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Joshua Smith¹, George C Bobustuc¹, Rafael Madero-Vishbal¹, Jimmie Colon¹, Beth Isley¹, Jonathan Ticku¹, Kalkunte S. Srivenugopal and Santhi Konduri^{1*}

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Abstract

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MGMT expression was found to be increased in breast cancer cells relative to normal breast epithelial cells. However, MGMT levels were significantly higher in tamoxifen resistant MCF-7 compared to the parent cells. Silencing of the ER- α expression using a specific siRNA resulted in augmentation of MGMT mRNA and protein levels by 2 fold. We also observed an inverse correlation between MGMT and p53 levels in breast cancer cell lines; moreover, p53 downregulation was accompanied by increased MGMT expression. Other experiments showed that BG alone or BG in combination with tamoxifen or fulvestrant decreased ER- α expression, whereas tamoxifen alone and fulvestrant alone increased and decreased the same respectively. However, all these treatments increased the p21^{WIF1} mRNA and protein expression significantly. BG inhibited tamoxifen resistant breast cancer growth in a dose-dependent manner and it also sensitized resistant breast cancer cells to anti-estrogen therapy (TAM/ICI). These combinations also enhanced the cytochrome C release and the PARP cleavage, indicative of apoptosis. In breast cancer xenografts, BG alone or a combination of BG with tamoxifen or fulvestrant caused significant tumor growth delay and immunohistochemistry revealed that BG inhibited the expression of MGMT, ER- α , ki-67 and increased p21^{WIF1} staining. These findings suggest that MGMT inhibition may provide a novel and effective approach for overcoming tamoxifen resistance.

Introduction

Recent advances in breast cancer research have identified key pathways involved in the repair of DNA damage induced by chemotherapeutic agents. The ability of cancer cells to recognize DNA damage and initiate DNA repair is an important mechanism for tumor survival. Therefore, therapeutic agents that target the process of DNA-damaging and alkylating agents can attack the nucleophilic O⁶ position on guanine, forming mutagenic and highly cytotoxic interstrand DNA crosslinks. The DNA repair enzyme O⁶-benzylguanine DNA alkyltransferase (AGT), encoded by the gene MGMT, repairs alkylation at this site and is responsible for protecting both tumor and normal cells from alkylating agents. MGMT is expressed constitutively in normal cells and tissues. In breast tumors, MGMT gene expression is elevated and levels are up to 4-fold higher than in the normal breast. Interestingly, it has been shown that tamoxifen accelerates proteasomal degradation of MGMT in human cancer cells. In 1991, Pegg, Moschel, and Dolan observed that O⁶-benzylguanine (BG) inhibited AGT and potentiated the cytotoxicity of both chloroethylnitrogen and methyleneguanine agents. Since then, BG has been shown to inhibit the cytotoxicity of both chloroethylnitrogen and methyleneguanine agents. In addition, BG inhibits AGT, transferring the benzyl moiety to the active-site cysteine [29]. The reaction is very rapid and more potent than any other previously known AGT inhibitor. BG is not incorporated into DNA in living cells and reacts directly with both cytoplasmic and nuclear AGT. Because BG is a pseudosubstrate for MGMT which results in the covalent transfer of benzyl group to the active site cysteine, the MGMT protein is degraded after each reaction. This stoichiometric reaction mechanism effectively depletes the AGT content in tumors and the associated repair of alkylation damage. BG is currently undergoing clinical trials in various cancers to increase the efficacy of alkylating agents.

Interestingly, several observations suggest an inverse correlation between the levels of MGMT and p53 tumor suppressor proteins where wild-type p53 suppresses transcription of human MGMT expression. Unfortunately, p53 function is often inactivated or suppressed in human cancers; therefore, restoration of wt-p53 activity is essential for the success of some treatments. However, whether or not this is mediated by suppression of MGMT expression has yet to be determined. To date, the cross-talk between MGMT and ER- α (and the link to p53 expression) has not been explored in drug (i.e., tamoxifen) resistant breast tumors. The anti-estrogen tamoxifen is the most commonly used treatment for patients with estrogen receptor positive breast cancer. Although many patients benefit from tamoxifen in the adjuvant and metastatic setting, resistance to this endocrine therapeutic agent is an important clinical problem. The primary goal of present study was to investigate the mechanisms of anti-estrogen drug resistance and to design new therapeutic strategies for circumventing this resistance. The results show that MGMT expression is increased in TAM-resistant breast cancers and inhibition of MGMT by BG significantly improves TAM-sensitivity.

Results

Prolonged Treatment of Tamoxifen Increases MGMT Expression: We developed a tamoxifen resistant MCF-7 cell line by using prolonged treatment of tamoxifen on the parental ER-positive breast cancer cell line, MCF-7. Tamoxifen-resistant MCF-7 cells proliferate at rates similar to the parental MCF-7. Prolonged treatment of tamoxifen onto MCF-7 cells increased MGMT expression compared to parental MCF-7 cells by 2 fold (Fig.1).

Knocking Down ERA Enhances MGMT Expression in Tamoxifen Resistant Breast Cancer Cells: It is not known whether ER- α and MGMT transcriptionally regulate each other in tamoxifen resistant breast cancer cells. We therefore investigated whether down regulation of ER- α has any effect on endogenous MGMT expression in these cells. As expected, downregulation of ER- α using specific siRNA significantly reduced ER- α protein levels in these cells. Western blot analysis performed and the results are shown (Fig. 2A) showing a decrease of ER- α in tamoxifen. MGMT expression in these cells, and interestingly, the results in the right panel (Fig.2B) show increased MGMT mRNA levels were increased as assessed by qRT-PCR. These data suggest that ER- α -mediated signaling functions to repress MGMT gene expression in breast cancer cells.

Transcriptional Regulation Between MGMT and p53: Previously, it was reported that p53 negatively regulates MGMT in breast cancer cells. Therefore, we addressed whether or not silencing the p53 enhances endogenous MGMT transcription. Tamoxifen resistant MCF-7 cells were transfected with either p53 siRNA (p53-KD) (Fig.2C) or MGMT siRNA (MGMT-KD) (Fig.2D) along with tamoxifen and NS (NS). MGMT expression was significantly reduced in p53 knock down cells. We did experiments showing a -fold augmentation (Fig. 2A) and as expected, knocking down MGMT decreased MGMT transcription where as p53 mRNA levels were unaffected in MGMT knockdown cells (Fig.2D). These results confirms that p53 can regulate MGMT at the transcriptional level.

O⁶-Benzylguanine Modulates p53 Down-Stream Targeted Protein Expressions: Encouraged by the results reported, we investigated the effect of combination therapy on endogenous MGMT, p53, and ER- α protein expressions. As expected, BG decreased MGMT expression, while combination therapy (4-OH-TAM or ICI combined with BG) significantly decreased both MGMT and ER- α expressions. ER- α alone or in combination with tamoxifen or ICI decreased ER- α expression, whereas tamoxifen alone and ICI alone increased and decreased the same respectively (Fig.3A). p53 expression was slightly altered after ICI treatment. The reduction in p53 expression by ICI alone was reversed when BG was combined (Fig.3A). We investigated the effect of BG on proteins which are involved in cell cycle regulation, apoptosis in tamoxifen resistant breast cancer cells. All these treatments significantly increased the p21^{WIF1} protein levels in these cells. Hence, p53 may have translocated to the mitochondria, cytochrome C is released (Fig.3B), and apoptosis was triggered in these cells in presence of combination therapy. PARP cleavage is seen in BG treated cells in presence of staurosporin as an indicative of apoptosis (Fig.3C). Therefore, this data suggest that BG promotes cell cycle arrest and can induce apoptosis by modulating p53 function.

O⁶-Benzylguanine Inhibits Tamoxifen Resistant Breast Cancer Cell Growth and Increase Resistant Breast Cancer Cell Sensitivity to Anti-Estrogen Therapy (TAM/ICI): Detailed necropsy revealed that all the mice had tumors in the breast. The data summarized in Table 1 show the daily BG alone or in combination with twice weekly tamoxifen/ICI significantly decreased median tumor volume and weight as compared with that seen in tamoxifen/ICI treated and control mice. The combination of BG with tamoxifen or ICI produced the greatest tumor regression median tumor size (BG+TAM: 89.99 mm³; 9.33 mm³ (TAM+BG), respectively; p<0.0001; 0.89 mm³; 0.69 mm³ (ICI+BG), respectively; p<0.0001) and tumor weight was also significantly reduced in mice treated with combination therapy as compared with control mice (81.23 mg; 20.50 mg (TAM+BG), respectively; p<0.0005; 81.23 mg; 51.57 mg (ICI+BG), respectively; p<0.0005) (Table 1). Body weight was not changed among all treatment groups as compared with control mice. No visible liver metastases were present (enumerated with the aid of a dissecting microscope) in all treatment groups.

Histology and IHC Analysis: We next determined the in vivo effects of BG (alone or in combination) with tamoxifen/ICI. Tumors harvested from different treatment groups were processed for routine histological and IHC analysis. Tumors from mice treated with BG alone or in combination with tamoxifen/ICI exhibited a significant decrease in MGMT, ER- α , ki-67 as compared with tumors treated with tamoxifen/ICI alone or control group. p53 expression was not much altered in these treatment groups. In sharp contrast, the expression of p21 was significantly increased in tumors from mice treated with BG either alone or in combination with tamoxifen/ICI. The images are analyzed by ImageJ (NIH) and MGMT, ER- α , p53, p21 and ki-67 expressions were quantified by the ImmunoBlot plugin (Fig.5).

Figure 1. MCF-7 parental and tamoxifen resistant MCF-7 cells were transfected with ER- α cDNA (ER α KD) and NS siRNA (NS) and cells were harvested 72 h post transfection. Total proteins were isolated and ER- α and MGMT expression was determined by western blot analysis. MGMT protein was significantly increased in ER- α knock down cells (NS) compared to parental MCF-7 cells. (B) Total RNA was isolated and MGMT and ER- α mRNA expression was determined by qRT-PCR. (C) Total RNA was isolated and p53 mRNA expression was determined by qRT-PCR. (D) Total RNA was isolated and p53 mRNA expression was determined by qRT-PCR. (E) Total RNA was isolated and p21 mRNA expression was determined by qRT-PCR. There is an inverse correlation between MGMT and p53 in tamoxifen resistant breast cancer cells (A, D).

Figure 2. (A) Tamoxifen resistant MCF-7 breast cancer cells were transfected with 4-OH-TAM and NS siRNA (NS) and cells were harvested 72 h post transfection. Total proteins were isolated and ER- α and MGMT expression was determined by western blot analysis. MGMT protein was significantly increased in ER- α knock down cells (NS) compared to parental MCF-7 cells. (B) Total RNA was isolated and MGMT and ER- α mRNA expression was determined by qRT-PCR. (C) Total RNA was isolated and p53 mRNA expression was determined by qRT-PCR. (D) Total RNA was isolated and p53 mRNA expression was determined by qRT-PCR. (E) Total RNA was isolated and p21 mRNA expression was determined by qRT-PCR. There is an inverse correlation between MGMT and p53 in tamoxifen resistant breast cancer cells (A, D).

Figure 3. (A) Tamoxifen resistant MCF-7 breast cancer cells were treated in absence of BG (40 μ M) and 4-OH-TAM (1 μ M) alone or in combination with BG. 24h post treatment cells were harvested and proteins were isolated and western blot analysis was performed. (B) Total RNA was isolated and MGMT expression (C) PCNA, p21, p53 and ER- α mRNA expression was determined by qRT-PCR. (D) Total RNA was isolated and p21 mRNA expression was determined by qRT-PCR. (E) Total RNA was isolated and p21 mRNA expression was determined by qRT-PCR. There is an inverse correlation between MGMT and p53 in tamoxifen resistant breast cancer cells (A, D).

Figure 4. Tamoxifen resistant MCF-7 breast cancer cells were treated in presence or absence of BG (20 μ M) for 48h and later 4-OH tamoxifen and ICI (1 μ M) was either alone or in combination with BG and 24h later cells were harvested and total RNA was isolated. (A) MGMT and ER- α (B) p53 transcription (C) PUMA expression was determined by qRT-PCR. (D) Tamoxifen resistant MCF-7 breast cancer cells were transfected with p21-luc construct and 6h later treated with BG and 24h later cells were harvested. p21 transcriptional activity was significantly increased by BG in these cells.

Figure 5. Tumors were harvested from control mice and mice treated with tamoxifen/ICI, BG, or both tamoxifen/ICI and BG. The sections were immunostained for MGMT, ER- α , Ki-67, p53, p21 and bcl-2. Tumors from mice treated with tamoxifen/ICI alone or in combination with BG alone or in combination with tamoxifen/ICI and BG were harvested at 4 weeks post treatment. (A) Representative images of MGMT, ER- α and Ki-67. p53 expression was not much altered in these treatment groups. In sharp contrast, the expression of p21 was significantly increased in all these treatment groups compared to controls. Representative samples (g3X3) are shown.

Conclusions

1. In the present study, we observed that prolonged treatment with anti-estrogens causes drug resistance by inducing the DNA repair protein O⁶-benzylguanine DNA methyltransferase (MGMT).
 2. Decreasing the expression of MGMT by exposing breast cancer cells to BG sensitized these cells to anti-estrogen therapy (tamoxifen and ICI 182.786).
 3. We also observed that combination therapy of anti-estrogens and MGMT blockers not only overcame the MGMT derived drug (tamoxifen and ICI) resistance but also increased the efficacy of anti-estrogen therapy by decreasing estrogen receptor expression and restoration of the functional activity of p53 in tamoxifen-resistant breast cancer cells.
 4. Combination therapy inhibited tamoxifen resistant breast tumor growth *in vivo*.

Acknowledgements

We would like to thank the Florida Department of Health, Sunshine-Caley Cancer Research Program (C95-04) for their funding of this project.

Good

- Solid layout (three columns).

Bad

- Too much and too small text.
- Gray
- Too cramped
- Too many and too small graphics
- No key message
- Picture of the institute?
- No contact information

Examples

My first poster

Good

- Clean layout
- Separation with colours and headlines
- Only few colors (consistent palette)
- Teaser on top right

Bad

- Main Message could be clearer
- Too much text
- Too paper-like structure
- No coarse - fine structure
- QR-code would be nice

SHAMANN: Shared Memory Augmented Neural Networks

Cosmin I. Bercea^{3,*}, Olivier Pauly^{4,*}, Andreas Maier², and Florin C. Ghesu^{1,2}

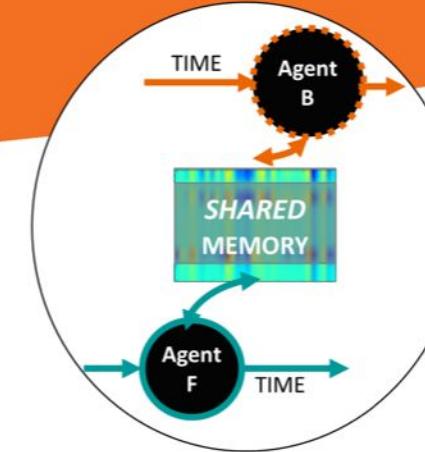
¹ Digital Technology and Innovation, Siemens Healthineers, Erlangen, Germany

² Pattern Recognition Lab, Friedrich-Alexander-Universität, Erlangen, Germany

³ Robert Bosch GmbH, Corporate Research-Computer Vision, Hildesheim, Germany

⁴ Google Cloud AI, Munich, Germany

* Contributed to this work during their time at Siemens Healthineers.



A dynamically scalable alternative for image segmentation using multiple cooperative neural networks

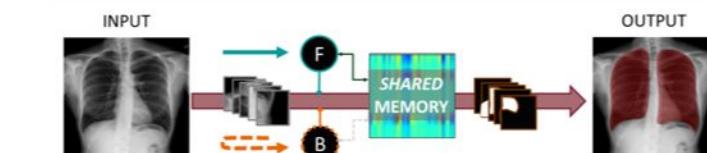
- Memory efficient and dynamically scalable
- Complex shape information through external memory
- Global context through shared memory

Motivation

State-of-the-art Deep Learning for Image Segmentation:

- **Memory issues:** when processing high-volumetric 3D/4D images, videos, etc.
- Complex learning task: need to capture complete variability in object shape, appearance and shifts

SHAMANN for Image Segmentation



Proposed Method

- **Memory efficient and dynamically scalable:**
 - decomposition of the original image in a variable length sequence of image subregions, each processed by different SHAMANN actors
 - flexible external memory size independent from the number of parameters of the network
- **Complex shape information** is captured through the memory component that encodes the sequences of local image patches to a compact global representation
- **Shared memory enables an implicit communication mechanism** between actors to better capture global image

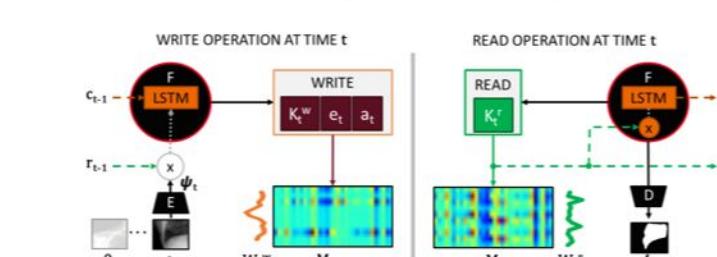
External Memory Module

Access and Sharing

- Key vectors to determine **where** to accesss the memory
- Add and erase vectors to determine **what** to write in the memory
- Actors **iteratively** access the shared memory to make predictions:

$$g_t = W(h_t \oplus r_t(M)) + b$$

Output LSTM Shared Memory Read



Disclaimer: This feature is based on research, and is not commercially available. Due to regulatory reasons its future availability cannot be guaranteed

Slightly better

TOWARDS UNBIASED PATHOLOGY SCREENING

Cosmin I. Bercea, Benedikt Wiestler, Daniel Rueckert, Julia A. Schnabel

Why?

Current methods are:

- Biased to detect hyper-intense lesions
- Biased to detect anomalies similar to the trained noise model

Good

- Text only where necessary and **highlighted keywords**
- Clean layout
- Story is easy to follow (Why, How, Results)
- Separation is clear with nicer boxes

Bad

- No different layers of information
- Main message not clear
- Could still look more professional

References:

[1] Kascenas, Antanas, et al. "Denoising Autoencoders for Unsupervised Anomaly Detection in Brain MRI". MIDL, 2022.

[2] Daniel, Tal, and Aviv Tamar. "Soft-introvae: Analyzing and improving the introspective variational autoencoder". CVPR, 2021.

HELMHOLTZ MUNICH



Improved?

Good

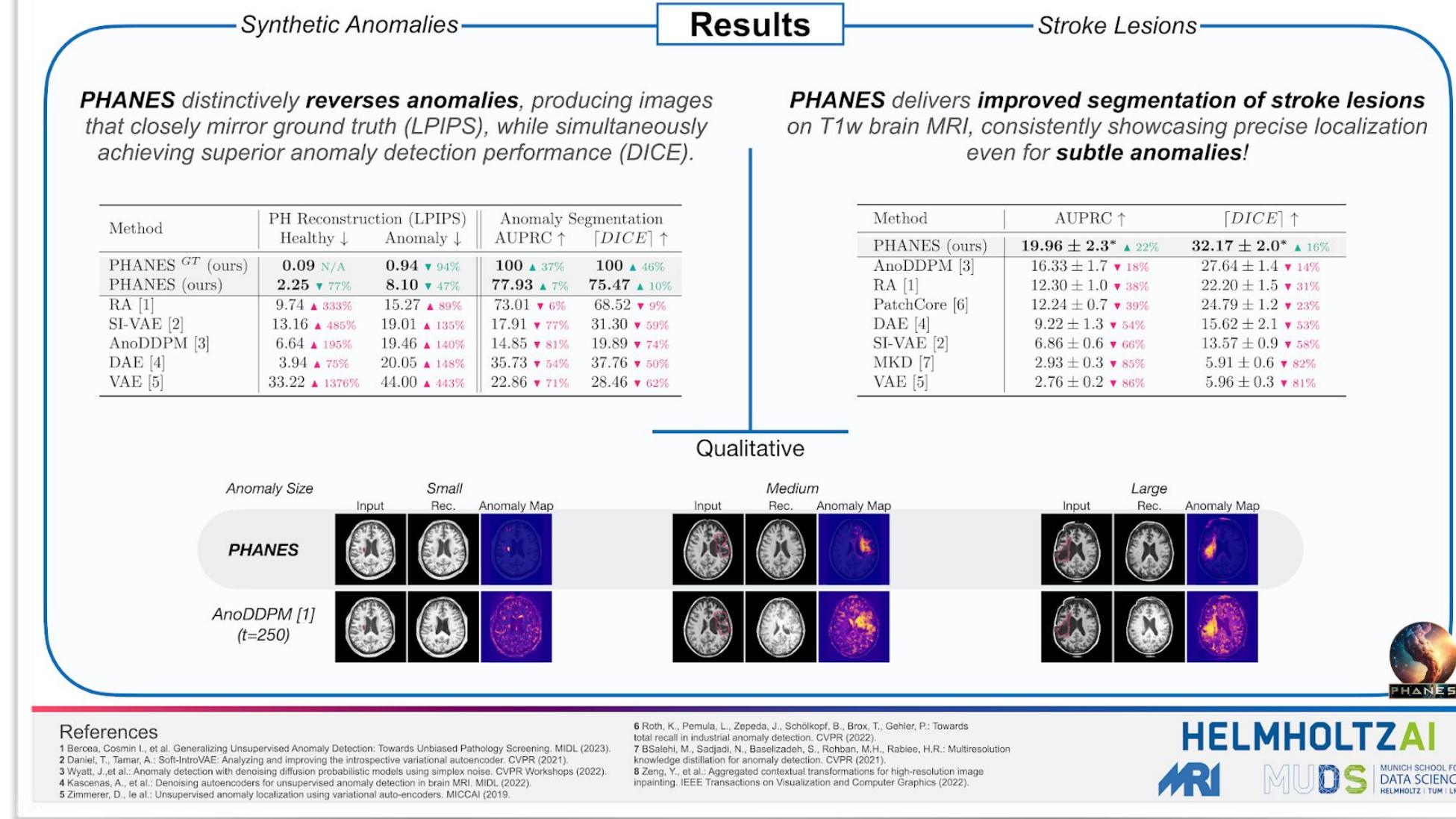
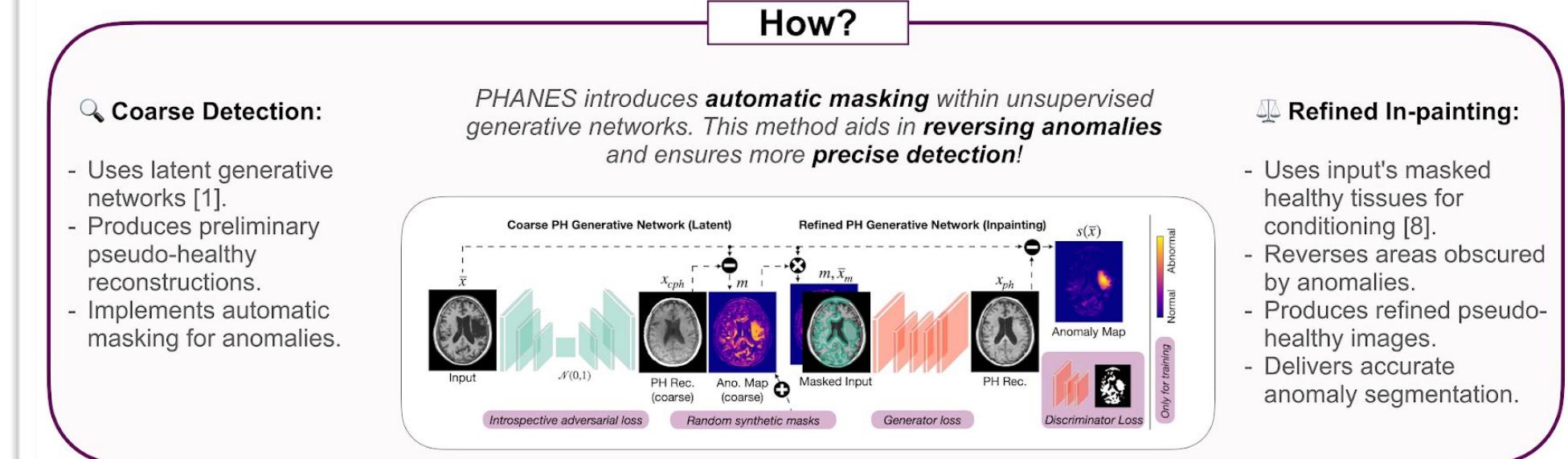
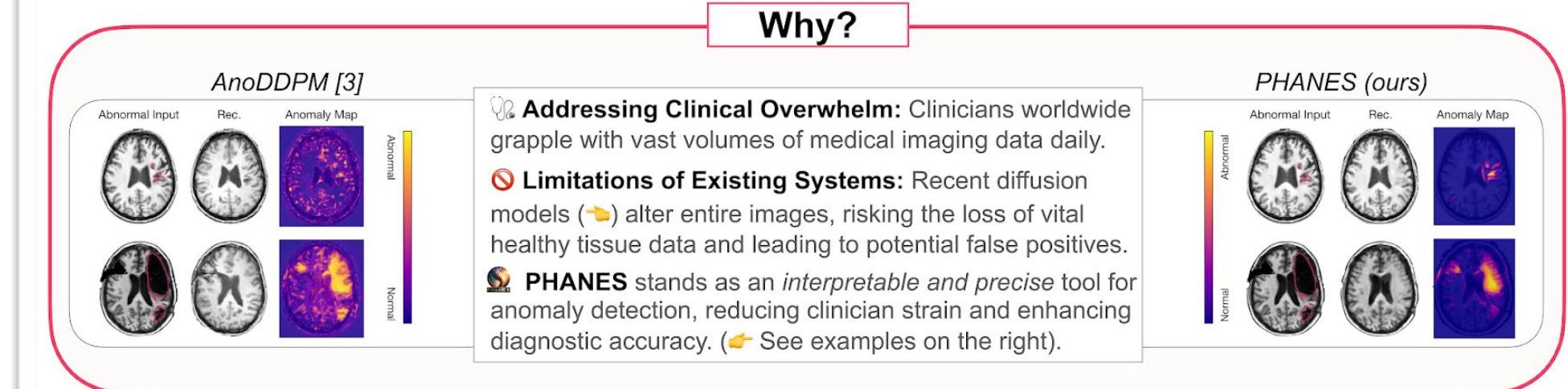
- Clean layout
 - Separation with colours and headlines
 - Color coded story
 - Visual Teaser on top bar
 - Quite visual / easy to follow narrative
 - QR-code
- ## Bad
- Main finding (text) could be clearer
 - Too much text / Same level of information

Reversing the Abnormal: Pseudo-healthy Generative Networks for Anomaly Detection

Cosmin I. Bercea^{1,2}, Benedikt Wiestler³, Daniel Rueckert^{1,3,4}, Julia A. Schnabel^{1,2,5}
 1: Technical University of Munich, Germany 2: Helmholtz AI and Helmholtz Center Munich, Germany, 3: Klinikum Rechts der Isar, Munich, Germany
 4: Imperial College London, UK, 5: King's College London, UK

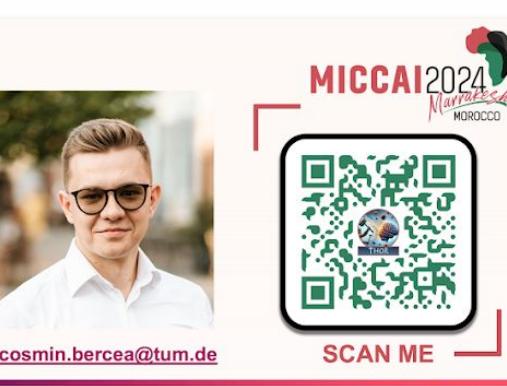


MICCAI 2023
Vancouver, Canada

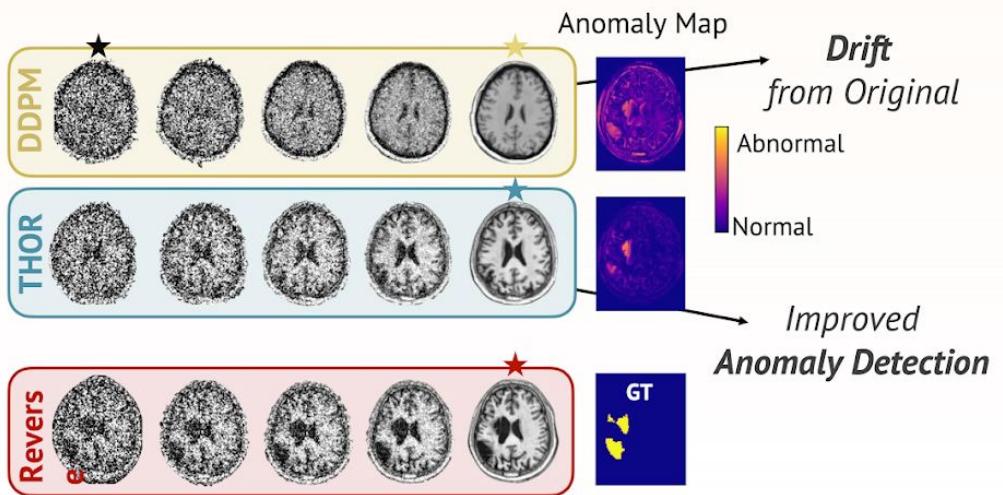
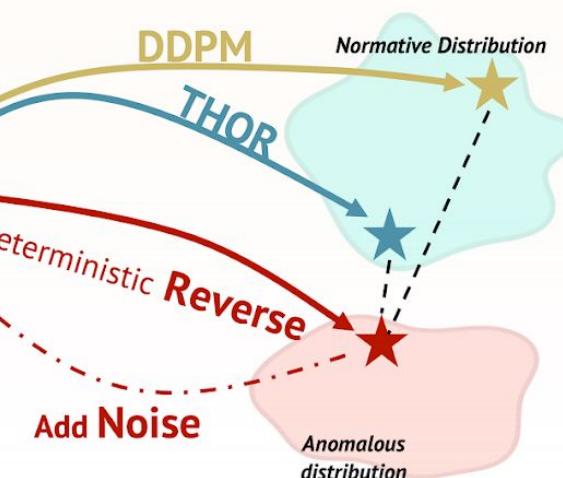


Diffusion Models with Implicit Guidance For Medical Anomaly Detection

Cosmin I. Bercea^{1,2}, Benedikt Wiestler^{1,3}, Daniel Rueckert^{1,3,4}, Julia A. Schnabel^{1,2,5}



Refine the Backward Diffusion Process for Enhanced Healthy Counterfactuals!



WH[!]?

Discover Unknown Diseases
Without Labels!

Supervised Methods
Known Diseases

Normative Representation Learning
Rare
Unknown Unknown Diseases

Rare is not rare: > 10,000 rare diseases affect 1 in 10 in the US!

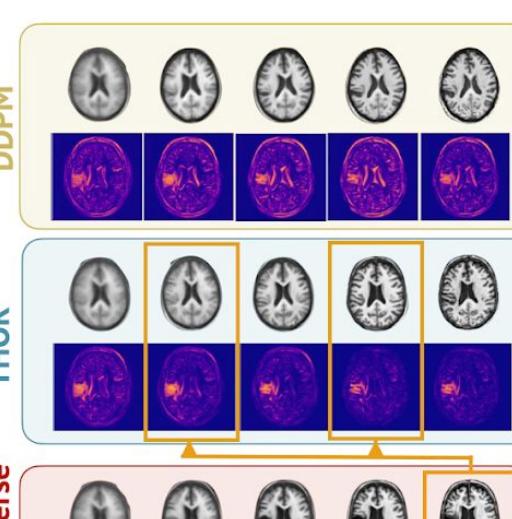
High noise levels in DDPMs ->
drift from original inputs!
(noise paradox [4])

RESULTS↑

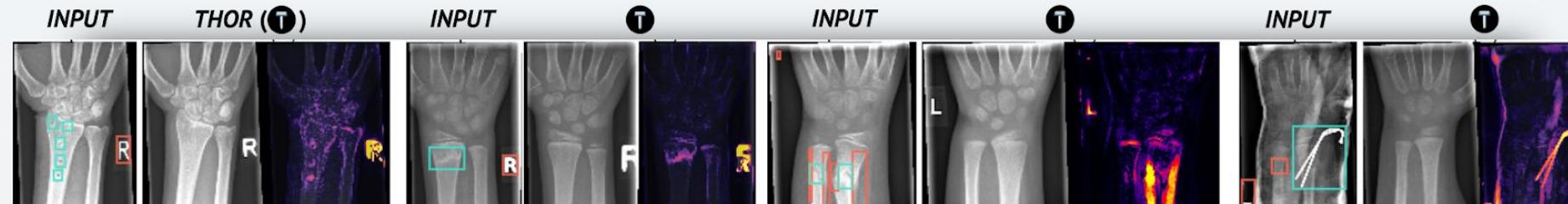
HQW?

DICE
Small
INPUT THOR (T)
[1] 9.14 ▲ 103%
[2] 1.37 ▼ 85%
[3] 4.55 ▼ 50%
Medium
INPUT THOR (T)
[1] 11.54 ▲ 44%
[2] 4.82 ▼ 58%
[3] 8.02 ▼ 31%
Large
INPUT THOR (T)
[1] 26.34 ▲ 19%
[2] 9.53 ▼ 64%
[3] 22.07 ▼ 16%
Simplex
INPUT THOR (T)
[1] 39.20 ▲ 30%
[2] 23.45 ▼ 40%
[3] 30.16 ▼ 23%
Reverse
INPUT THOR (T)

Guided harmonization by implicit anomaly maps computed directly during the reverse steps



Gradually re-introduce normal original tissues for better pseudo-healthy restoration!



Affiliations

1 Technical University of Munich, Germany
2 Helmholtz Institute for Nanobiology and Nanomedicine, Germany
3 Klinikum Rechts der Isar, Munich, Germany
4 Imperial College London, UK
5 Kings College London, UK

References

[1] Ho, Jonathan, Ajay Jain, and Pieter Abbeel. "Denoising diffusion probabilistic models." NeurIPS, 2020.
[2] Bercea, Cosmin I., et al. "Anomaly detection via denoising diffusion probabilistic models with implicit noise." CVPR, 2022.
[3] Behrendt, Finn, et al. "Patched diffusion models for unsupervised anomaly detection in brain MRI." MICCAI, 2024.
[4] Bercea, Cosmin I., et al. "Mask, stitch, and re-sample: Enhancing robustness and generalizability in anomaly detection through automatic diffusion models." ICML Workshops, 2023.

Our latest experiment

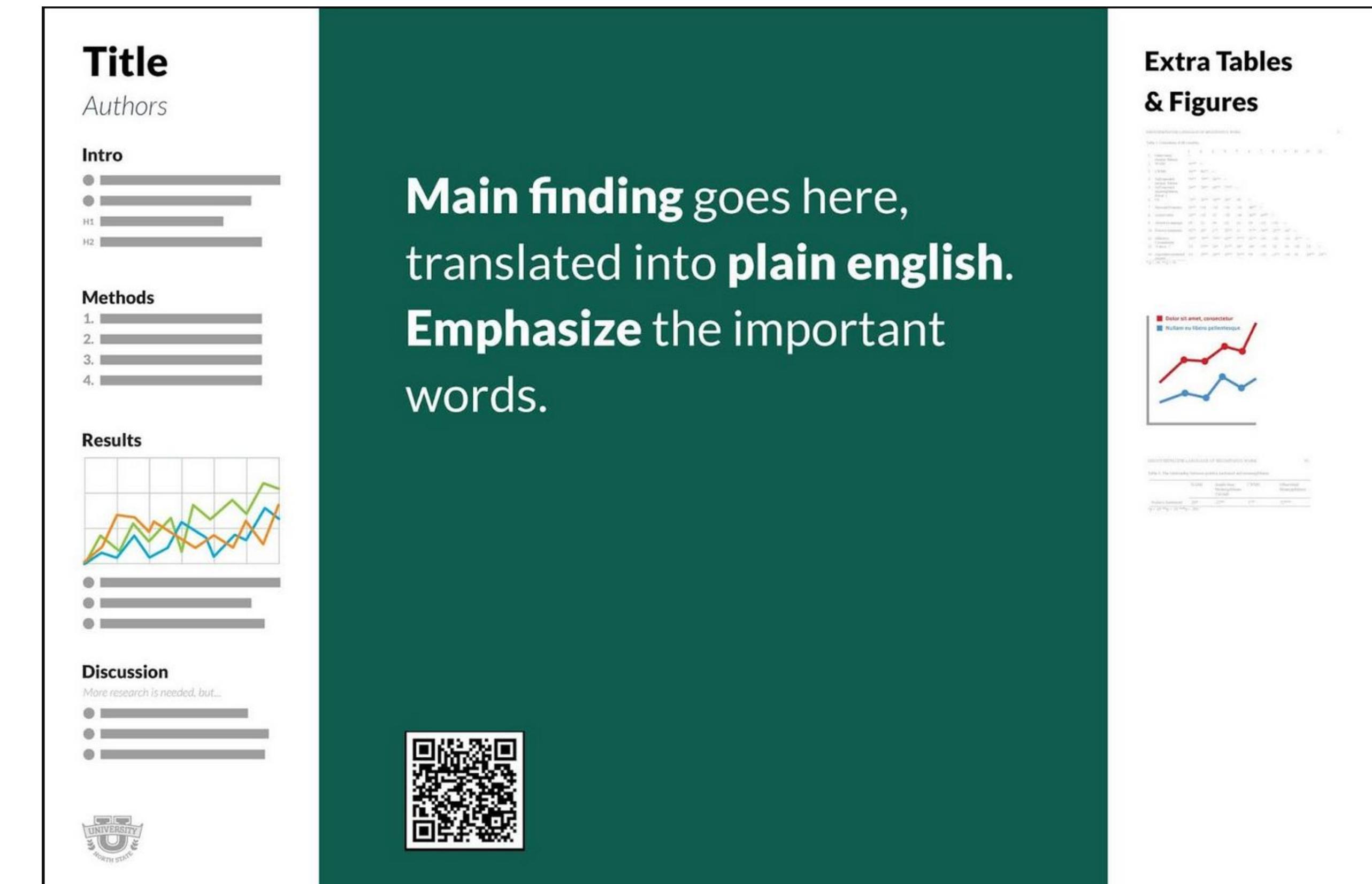
Good

- Story is easy to follow (Why, How, Results)
- Separation is clear with nicer boxes
- Clear teaser + main finding on the top —> It's enough for “lazy” audience
- Interested people would look also at the bottom part
- Quite visual, not too many words

Could be better?

- Augment with some videos on a screen?
- Should it contain more detailed information?

The ‚better poster‘ format



Title
Authors

Intro

-
-
- H1
- H2

Methods

- 1.
- 2.
- 3.
- 4.

Results

Discussion

Extra Tables & Figures

Title
Authors

Intro

-
-
- H1
- H2

Methods

- 1.
- 2.
- 3.
- 4.

Results

Discussion

More research is needed, but...

Extra Tables & Figures

<https://www.youtube.com/watch?v=SYk29tnxAzs>

The ,better poster format

Pro

- Looks professional
- Forces you to use little text
- Nice structure
- Easy to use template

Con

- Many papers might look alike

To reconcile observations and predictions of the RGB bump, one may include **convective boundary mixing** with an **efficiency that increases with decreasing metallicity**.

THE RED-GIANT BRANCH BUMP REVISITED: CONSTRAINTS ON CONVECTIVE BOUNDARY MIXING IN A WIDE RANGE OF MASSES AND METALLICITIES

Saniya Khan, Andrea Miglio, Josefina Montalbán, Oliver J. Hall, Guy R. Davies, Benoît Mosser, Leo Girardi

★ The RGB bump allows investigation of the internal structure of low-mass stars. One can use its luminosity to predict the occurrence and efficiency of mixing processes beyond convective boundaries.
★ By combining asteroseismic and spectroscopic constraints, we expand the analysis of the bump to masses and metallicities beyond those previously accessible using globular clusters.

Using statistical mixture models, we are able to detect the bump in the average seismic parameters v_{max} and $\langle \Delta v \rangle$, in a sample of nearly 3000 RGB stars observed by Kepler and APOGEE.

INTRODUCTION

METHODS



UNIVERSITY OF
BIRMINGHAM

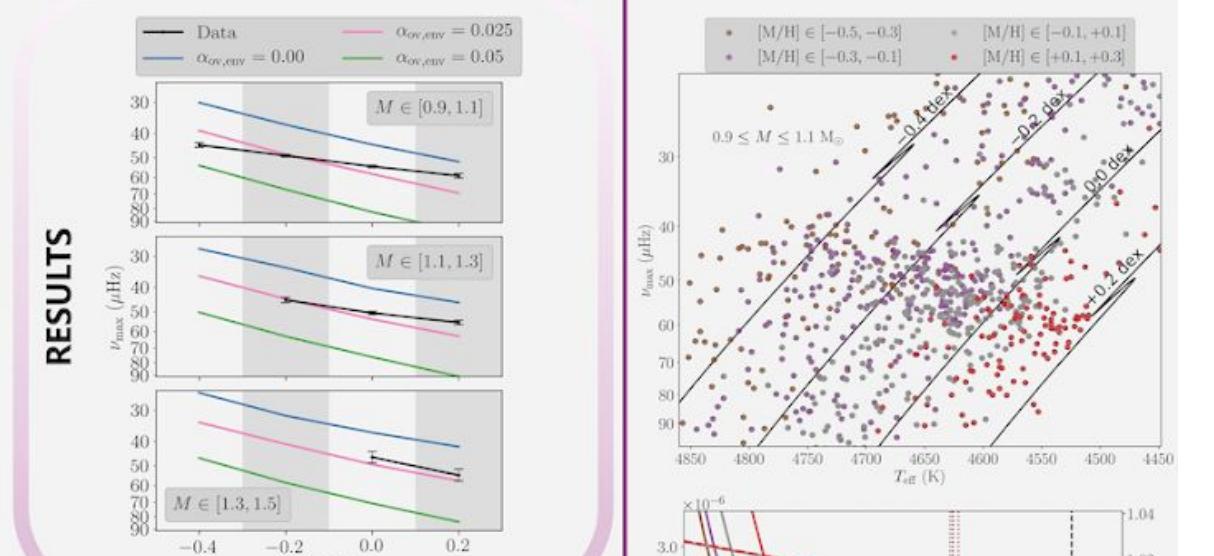
SCHOOL OF
PHYSICS AND
ASTRONOMY

Source:
<https://astrobites.org/2020/02/28/fixing-academic-posters-the-betterposter-approach/>

Exponential CBM prescription
(Freytag et al. 1996; Herwig 2000)

$$D = D_0 \exp(-2z/\alpha_{\text{ov,env}} H_{\text{P0}})$$

• $[\text{M}/\text{H}] \in [-0.5, -0.3]$ • $[\text{M}/\text{H}] \in [-0.1, +0.1]$
• $[\text{M}/\text{H}] \in [-0.3, -0.1]$ • $[\text{M}/\text{H}] \in [+0.1, +0.3]$



★ The dependence of CBM on metallicity could possibly be related to the stiffness of convective boundaries.
★ Drawing the link with CBM prescriptions from 2D/3D simulations is the way forward to physically explain this dependence.

ASTER
CHRONOMETRY
erc
European Research Council

doi.org/10.3847/1538-4357/aabf90



Scan me
for full text

How to present a research poster



- **Be welcoming!** Stand next to your poster, always face the audience, actively approach people
- **Be enthusiastic!** If you don't sound enthusiastic about your work, why should others be excited?
- **Be early and stay late!** Don't miss the great opportunity to have interesting, long conversations with people genuinely interested in your work
- **Be concise!** Prepare an elevator pitch for ca. 1-2 minutes. Hook the audience and then respond to any questions
- **No “Do you know X”.** Give a 10 sec intro to X and then focus on your work
- **Build your network!** Thank each person for stopping by and/or asking good questions. Say their name (and ask how to pronounce it). Greet them the next time you meet them
- **Have backup material for questions.** Have a laptop around to show more results when required

Where to begin?



Answer these three questions

1. What is the most important / interesting finding?

2. How can I visually share the research?
Should I use charts, graphs, photos, images?
(Which figures of the paper are most important and illustrative?)

3. What kind of information can I convey during my talk that will complement the poster?

Put your sections into a logical flow so the audience knows where to read.

Your task

- **Submit your poster as PDF to Our GitHub Repo**
 - Ahead of your presentation slot
 - Include your own name as the presenter
 - We will print the poster
- **Present your poster briefly at your presentation slot**
 - Use only the poster, no slides!
 - 2 minutes/person
 - Use it as a pitch to emphasize the main message of the paper and main results of the coding project



That's all folks!



Coffee-loving Cosmin

Postdoctoral Researcher

cosmin.bercea@tum.de



Jasmine tea-loving Jun

PhD Student

june.li@tum.de