Modelling the Neuroanatomical Progression of Alzheimer's Disease and Posterior Cortical Atrophy

Răzvan Valentin Marinescu

Supervisors: Prof. Daniel Alexander, Dr. Sebastian Crutch, Dr. Neil Oxtoby

Centre for Medical Image Computing, University College London, UK
About me

▶ Grew up in Pitesti, Romania
▶ 2010-2014: Studied a 4-year MEng in Computer Science at Imperial College London

▶ 2014: Masters and PhD in Medical Imaging at UCL
▶ Working with Prof. Daniel Alexander on disease progression modelling
1. Study the progression of pathology in two diseases (using existing models):
   ▶ typical Alzheimer’s Disease (tAD)
   ▶ Posterior Cortical Atrophy (PCA)

2. Develop novel disease progression models (DPMs)

\[
p(X|S) = \prod_{j=1}^{J} \left[ \sum_{k=0}^{N} p(k) \left( \prod_{i=1}^{k} p(x_{s(i),j}|E_{s(i)}) \prod_{i=k+1}^{N} p(x_{s(i),j}|\neg E_{s(i)}) \right) \right]
\] (1)
My PhD Contributions

1. Modelling the Progression of PCA

2. DIVE Spatiotemporal Model

3. Disease Knowledge Transfer (DKT)

4. Novel Extensions of EBM and DEM

5. TADPOLE Competition

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Staging Consistency

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<tr>
<th>Model</th>
<th>Staging Consistency</th>
<th>Time-lapse</th>
</tr>
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<tr>
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1. Modelling the Progression of PCA

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4. Novel Extensions of EBM and DEM

5. TADPOLE Competition
Aim: Estimate the Progression of Atrophy in PCA

**Why?** No comprehensive studies modelled disease progression in PCA so far

**Demographics:**
- MRI Data from the Dementia Research Centre with uniquely large PCA population (70)

<table>
<thead>
<tr>
<th></th>
<th># Subjects</th>
<th>Gender M/F</th>
<th>Age at baseline (years)</th>
<th>Years from onset (years)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Controls</td>
<td>89</td>
<td>33/56</td>
<td>60.5 ± 11</td>
<td>-</td>
</tr>
<tr>
<td>PCA</td>
<td>70</td>
<td>27/43</td>
<td>63.0 ± 7</td>
<td>4.4 ± 2.8</td>
</tr>
<tr>
<td>AD</td>
<td>65</td>
<td>34/31</td>
<td>66.3 ± 8</td>
<td>4.8 ± 2.6</td>
</tr>
</tbody>
</table>

**Impact:** the first major investigation of PCA disease progression
Key Idea: The Event-Based Model Estimates an Atrophy Sequence from Informative Patient Snapshots

- Aim: Region 1 → Region 2 vs Region 2 → Region 1

<table>
<thead>
<tr>
<th>Region 1</th>
<th>Patient 1</th>
<th>Patient 2</th>
<th>Patient 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.1</td>
<td>0.9</td>
<td>0.1</td>
<td></td>
</tr>
<tr>
<td>0.95</td>
<td>0.0</td>
<td>0.05</td>
<td></td>
</tr>
</tbody>
</table>

Region 1

<table>
<thead>
<tr>
<th>Region 2</th>
<th>Patient 1</th>
<th>Patient 2</th>
<th>Patient 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>normal</td>
<td>normal</td>
<td>abnormal</td>
<td></td>
</tr>
<tr>
<td>normal</td>
<td>abnormal</td>
<td>abnormal</td>
<td></td>
</tr>
</tbody>
</table>

Estimated Sequence: Region 2 → Region 1
The EBM finds a Distinct Atrophy Sequence in PCA compared to tAD

- PCA → early occipital and superior parietal atrophy
- tAD → early hippocampal and inferior temporal atrophy

Firth, Marinescu and Primativo, in first revision (Brain)
Atrophy Patterns Resemble Previous Studies from the Literature

- **PCA** → early occipital and superior parietal atrophy
- **tAD** → early hippocampal and inferior temporal atrophy

Firth, Marinescu and Primativo, in first revision (Brain)
PCA Subtypes show Different Atrophy Progressions, providing Evidence for Heterogeneity within PCA

**Initial hypotheses**

1. Basic visual impairment → early atrophy in occipital lobe
2. Space perception impairment → early atrophy in superior parietal lobe
3. Visuoperceptual impairment → early atrophy in inferior temporal lobe

<table>
<thead>
<tr>
<th>Stage 1</th>
<th>Stage 2</th>
<th>Stage 3</th>
<th>Stage 4</th>
</tr>
</thead>
<tbody>
<tr>
<td><img src="image1" alt="Brain Image" /></td>
<td><img src="image2" alt="Brain Image" /></td>
<td><img src="image3" alt="Brain Image" /></td>
<td><img src="image4" alt="Brain Image" /></td>
</tr>
</tbody>
</table>

1. **Basic visual impairment** (n=21)

2. **Space perception impairment** (n=21)

3. **Visuoperceptual impairment** (n=22)

Firth, Marinescu and Primativo, in first revision (Brain)
The Differential Equation Model reconstructs Biomarker Trajectories from Short-term Longitudinal Measurements

What we want

What we have

\[
\lim_{\Delta t \to 0} \frac{\Delta x}{\Delta t} = \frac{\delta x}{\delta t} = f(x)
\]

Solve for \( x \) using the Euler method:

\[
t_1 = t_0 + \delta t \\
x_1 = x_0 + f(x_0) \delta t
\]

Reconstructed trajectory

Rate of change \( \frac{dx}{dt} \)

Time (relative)

Rate of change model

Slope of subject-wise lines

Time since baseline
Model Recapitulates Differences in PCA vs tAD Atrophy Progression

- PCA: rapid and extensive atrophy in occipital and parietal regions
- tAD: global atrophy pattern, with early hippocampal involvement

(a) PCA

(b) tAD

Firth, Marinescu and Primativo, in first revision (Brain)
My PhD Contributions

1. Modelling the Progression of PCA
   - Disease 1 (e.g. tAD)
   - Disease 2 (e.g. PCA)
   - Staging Consistency
     - EBM - Standard: 0.87 ± 0.10
     - EBM - Optimised: 0.87 ± 0.10

2. DIVE Spatiotemporal Model
   - Disease Specific
   - Disease Agnostic

3. Disease Knowledge Transfer (DKT)
   - Biomarker value
   - Staging Consistency
     - EBM - Standard: 0.71 ± 0.07
     - EBM - Sampling: 0.76 ± 0.10

4. Novel Extensions of EBM and DEM
   - Time-lapse
     - EBM - EM: 0.72 ± 0.07
     - DEM - Standard: 0.74 ± 0.91

5. TADPOLE Competition
Aim: Build a Disease Progression Model of Pathology over the Brain that Avoids Limitations of Previous Models

Avoids pre-defined ROI parcellation
Avoids simplistic spatial correlation structure
Avoids simplistic biomarker trajectories

This leads to a technique that simultaneously:

- parcellates the brain into disconnected components that undergo similar progression
- estimates biomarker trajectories
Motivation

1. Atrophy correlates with functional networks, which are not spatially connected (Seeley et al., Neuron, 2009)

2. Better biomarker prediction and disease staging

(a) Seeley et al., Neuron, 2009
Method Idea - Combine Unsupervised Learning and Disease Progression Modelling

Only Unsupervised Learning (i.e. Clustering)

- Can identify disconnected atrophy patterns ✓
- No biomarker trajectories ❌
- No disease staging of subjects ❌
- Estimate trajectories for each vertex on the cortical surface
- Vertex measures cortical thickness at that location

Only Disease Progression Modelling

- Cannot identify disconnected atrophy patterns ❌
- Can estimate biomarker trajectories ✓
- Can estimate subjects disease stages ✓
DIVE Finds Plausible Atrophy Patterns on Four Datasets

- Similar patterns of tAD atrophy in independent datasets: ADNI and UCL DRC
- Distinct patterns of atrophy in different diseases (tAD and PCA) and modalities (MRI vs PET)

Marinescu et al., NeuroImage, under second review
DIVE Estimates the Temporal Evolution of Pathology, Enabling Understanding of Disease Mechanisms

Marinescu et al., Neuroimage, under second review

▶ Open-source brain colouring/animation software to be published
**Method:** Tested the consistency of the spatial clustering in ADNI using 10-fold CV

**Results:** Good agreement in terms of spatial distribution (dice score 0.89)

Marinescu et al., Neuroimage, under second review

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University College London
DIVE
Estimated Subject Progression Scores are Clinically Relevant

**Hypothesis:**
▶ Clinical relevance → DPS correlates with other markers of disease progression

**Method:** Ran our model on ADNI using 10-fold cross-validation

**Results:** Progression scores correlate well with cognitive tests:

<table>
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<tr>
<th>Test</th>
<th>Correlation Coefficient</th>
<th>p-value</th>
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<tr>
<td>CDRSOB</td>
<td>$\rho = 0.41$</td>
<td>$p &lt; 1e^{-66}$</td>
</tr>
<tr>
<td>ADAS-COG</td>
<td>$\rho = -0.40$</td>
<td>$p &lt; 1e^{-62}$</td>
</tr>
<tr>
<td>MMSE</td>
<td>$\rho = -0.39$</td>
<td>$p &lt; 1e^{-58}$</td>
</tr>
<tr>
<td>RAVLT</td>
<td>$\rho = 0.39$</td>
<td>$p &lt; 1e^{-58}$</td>
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My PhD Contributions

1. Modelling the Progression of PCA
   - Stage 8
   - Stage 16
   - Stage 24

2. DIVE Spatiotemporal Model

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5. TADPOLE Competition

Razvan V. Marinescu
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University College London
DKT
Aim: Estimate the *Longitudinal, Multimodal* Progression of Rare Neurodegenerative Diseases

- Current disease progression models require large, multimodal datasets
- Applications to rare neurodegenerative diseases are challenging due to lack of data
- Deep transfer learning techniques exist, but are not interpretable

<table>
<thead>
<tr>
<th>Typical Neurodegenerative Diseases</th>
<th>Rare Neurodegenerative Diseases</th>
</tr>
</thead>
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<tr>
<td>• Large datasets ✓</td>
<td>• Small datasets ✗</td>
</tr>
<tr>
<td>• Multimodal imaging ✓</td>
<td>• MRI only ✗</td>
</tr>
<tr>
<td>• Longitudinal ✓</td>
<td>• Cross-sectional only ✗</td>
</tr>
</tbody>
</table>
Disease Knowledge Transfer (DKT) can estimate multimodal trajectories in rare diseases by transferring information from larger datasets of typical diseases.
DKT Accurately Estimates Ground Truth Parameters on Synthetic Data

Subject shifts

\[ R^2 = 0.997 \]

True shifts

Estimated shifts

\[ R^2 = 0.988 \]

Subject shifts

Dis0 estimated trajectories

\[ \text{MAE} = 0.057 \]

Dis0 true trajectories

Dis1 estimated trajectories

\[ \text{MAE} = 0.055 \]

Dis1 true trajectories

Unit0 estimated trajectories

\[ \text{MAE} = 0.058 \]

Unit0 true trajectories

Unit1 estimated trajectories

\[ \text{MAE} = 0.015 \]

Unit1 true trajectories

Razvan V. Marinescu
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University College London
On Patient Data, DKT Estimates Plausible Multimodal PCA Trajectories

- only MRI data was available in PCA
Validation: DKT has Favourable Performance Compared to Other Approaches

<table>
<thead>
<tr>
<th>Model</th>
<th>Cingulate</th>
<th>Frontal</th>
<th>Hippocampus Prediction Error (MSE)</th>
<th>Occipital</th>
<th>Parietal</th>
<th>Temporal</th>
</tr>
</thead>
<tbody>
<tr>
<td>DKT</td>
<td>0.09±0.04</td>
<td>0.03±0.01</td>
<td>0.18±0.03</td>
<td>0.04±0.02</td>
<td>0.06±0.02</td>
<td>0.04±0.02</td>
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<tr>
<td>Latent stage model</td>
<td>0.09±0.04</td>
<td>0.03±0.01</td>
<td>0.17±0.03</td>
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<tr>
<td>Linear Model</td>
<td>0.05±0.02*</td>
<td>0.15±0.04*</td>
<td>0.09±0.03*</td>
<td>0.07±0.02*</td>
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**Rank Correlation (Spearman rho)**

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<tr>
<td>DKT</td>
<td>0.76</td>
<td>0.48</td>
<td>0.76</td>
<td>0.55</td>
<td>0.55</td>
<td>0.33</td>
</tr>
<tr>
<td>Latent stage model</td>
<td>0.76</td>
<td>0.49</td>
<td>0.80*</td>
<td>0.56</td>
<td>0.51*</td>
<td>0.33</td>
</tr>
<tr>
<td>Linear Model</td>
<td>0.48*</td>
<td>0.31*</td>
<td>0.64*</td>
<td>0.61*</td>
<td>0.57*</td>
<td>0.27*</td>
</tr>
</tbody>
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- Latent stage model: assumes PCA and tAD all follow the same progression
- Linear model: estimates DTI from MRI using ROI-wise linear model
My PhD Contributions

1. Modelling the Progression of PCA
   - Stage 8
   - Stage 16
   - Stage 24

2. DIVE Spatiotemporal Model
   - Disease Knowledge Transfer (DKT)

3. Disease Knowledge Transfer (DKT)

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5. TADPOLE Competition
Aim: Improve the Parameter Estimation in EBM and DEM Models

Why:
▶ EBM assumed parameter independence
▶ DEM trajectory alignment challenging due to measurement noise.
▶ Accurate parameters → better disease staging → better patient stratification

Secondary Aim:
▶ Develop performance criteria for evaluation of disease progression models

Why?
▶ Comparative performance of disease progression models currently unknown
Novel EBM and DEM Extensions Perform Better than Standard Implementations

► Novel extensions vs standard implementations

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<td>EBM - Standard</td>
<td>0.88 ± 0.12 0.66 ± 0.09</td>
<td>-</td>
</tr>
<tr>
<td>EBM - Sampling</td>
<td>0.96 ± 0.06 0.70 ± 0.06</td>
<td>-</td>
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<td>EBM - EM</td>
<td>0.95 ± 0.10 0.68 ± 0.11</td>
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<tr>
<td>DEM - Standard</td>
<td>0.94 ± 0.06 0.95 ± 0.05</td>
<td>0.54 ± 0.31 0.52 ± 0.29</td>
</tr>
<tr>
<td>DEM - Optimised</td>
<td>0.95 ± 0.05 0.95 ± 0.04</td>
<td>0.56 ± 0.28 0.52 ± 0.27</td>
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Table 1: PCA - DRC cohort

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<tr>
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<td>0.74 ± 0.92 0.69 ± 0.92</td>
</tr>
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</table>

Table 2: tAD - DRC cohort
Novel Performance Criteria More Sensitive than Accuracy of Diagnostic Predictions

Table 3: PCA - DRC cohort

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Table 4: Accuracy of diagnosis prediction - DRC data

<table>
<thead>
<tr>
<th>Model</th>
<th>PCA vs AD</th>
<th>Controls vs PCA</th>
<th>Controls vs AD</th>
</tr>
</thead>
<tbody>
<tr>
<td>EBM - Standard</td>
<td>0.72 ± 0.13</td>
<td>0.95 ± 0.05</td>
<td>0.90 ± 0.06</td>
</tr>
<tr>
<td>EBM - Simultaneous Sampling</td>
<td>0.79 ± 0.09</td>
<td>0.94 ± 0.06</td>
<td>0.90 ± 0.05</td>
</tr>
<tr>
<td>EBM - EM</td>
<td>0.80 ± 0.07</td>
<td>0.95 ± 0.05</td>
<td>0.87 ± 0.05</td>
</tr>
<tr>
<td>DEM - Standard</td>
<td>0.81 ± 0.07</td>
<td>0.95 ± 0.05</td>
<td>0.90 ± 0.11</td>
</tr>
<tr>
<td>DEM - Trajectory Alignment</td>
<td>0.82 ± 0.09</td>
<td>0.93 ± 0.06</td>
<td>0.88 ± 0.14</td>
</tr>
<tr>
<td>Support Vector Machine</td>
<td>0.79 ± 0.14</td>
<td>0.91 ± 0.06</td>
<td>0.88 ± 0.07</td>
</tr>
</tbody>
</table>

▶ Work still in progress
My PhD Contributions

1. Modelling the Progression of PCA
   - Stage 8
   - Stage 16
   - Stage 24

2. DIVE Spatiotemporal Model

3. Disease Knowledge Transfer (DKT)

4. Novel Extensions of EBM and DEM
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5. TADPOLE Competition

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TADPOLE
TADPOLE is a Challenge to Predict the Progression of Individuals at Risk of AD

- Train on existing data from ADNI subjects, then predict future values over the next 5 years

**Dates:**
- **Open webinar 1 on challenge:** 14:00 GMT+1 Wed 12th July 2017
- **Open webinar 2 on challenge:** 14:00 GMT+1 Thu 10th Aug. 2017
- **Open webinar 3 on challenge:** 14:00 GMT+1 Thu 14th Sept. 2017
- **Submission deadline:** 15th Nov. 2017
- **Test set complete:** Nov. 2018
- **Evaluation results on website:** Jan. 2019
- **Publication submitted:** March 2019
- **Review first phase:** March 2019
My TADPOLE Contributions

- Assembled the training datasets from several ADNI spreadsheets
- Helped create the website
- Built an automated evaluation system and leaderboard
- Wrote the challenge design paper
Join the TADPOLE Challenge!

- URL: https://tadpole.grand-challenge.org/
- Deadline: 15 November 2017
- Prize fund: £30,000
Team Locations

- USA 9
- UK 8
- France 4
- Denmark 2
- Netherlands 2
- Mexico 2
- Australia 1
- Romania 1
- Canada 1
- Israel 1
- Finland 1
Prediction Methods

Breakdown by number of teams

- Regression: 10
- Machine learning: 3
- Disease Progression Model: 7
- Other: 3

Breakdown by number of entries

- Regression: 20
- Machine learning: 17
- Disease Progression Model: 3
- Other: 23
Next steps

- Run final evaluation with ADNI data so far
- Submit publication with results
Acknowledgements

Collaborators

1. Leon Aksman
2. Maura Bellio
3. Arman Eshaghi
4. Nicholas Firth
5. Sara Garbarino
6. Kyriaki Mengoudi
7. Marco Lorenzi
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Funders

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