Sleep Analytics

Emmanuel Mignot, MD, PhD

Sleep Medicine, Stanford University

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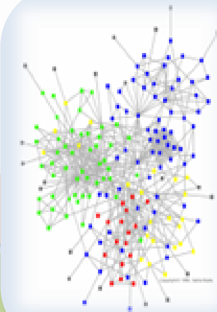
Why Study Sleep?

- Opportunity for major basic research discovery:
  - Circadian clock mechanisms are well known
  - Biological basis of sleep debt is unknown
- ~ ¼ of world population has sleep problems:
  - Sleep apnea (~10-20%)->Cardiovacular disease
  - Insomnia (~10%)->depression
  - Restless Legs Syndrome/Periodic Leg Movements (~3%)
  - Hypersomnia/Fatigue/Narcolepsy (~4%)
  - Circadian Dysregulation/shiftworker (~4%)
- Large health and economic impact of dysregulated sleep and sleep disorders on health and diseases
- Sleep affects the brain and the body

Sleep is at the confluence of several revolutions

Neurosciences
Animal Models



Analytics
Statistics
Machine Learning

Biological Understanding of Sleep

Sleep Medicine

Clinical and Consumer Applications

Scientific
understanding

Clinical
applications

Genetic
Proteomic
Revolution

Hardware
Wearable sleep
Monitoring

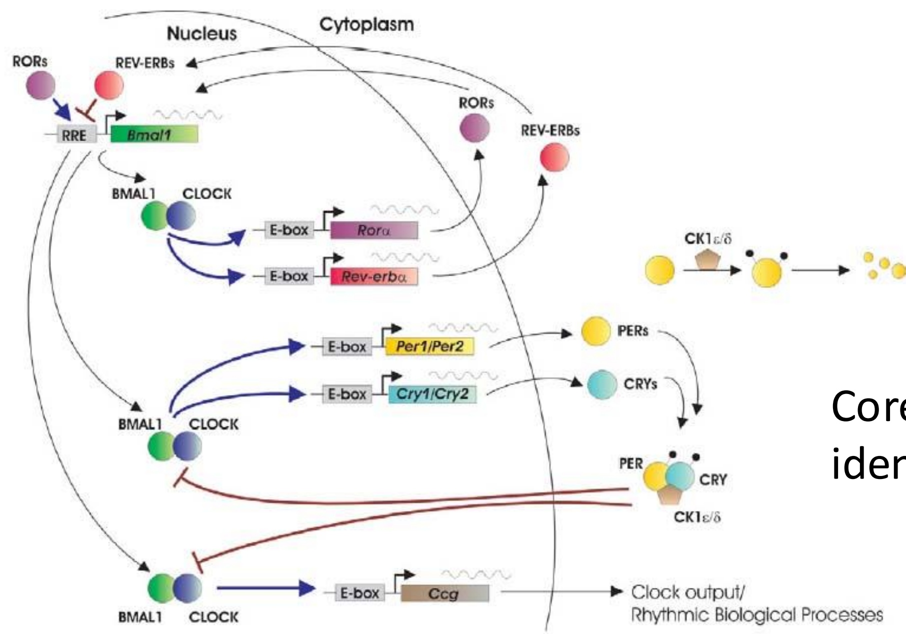
Sleep Clinic of the future

Stanford is a world leader in all these areas

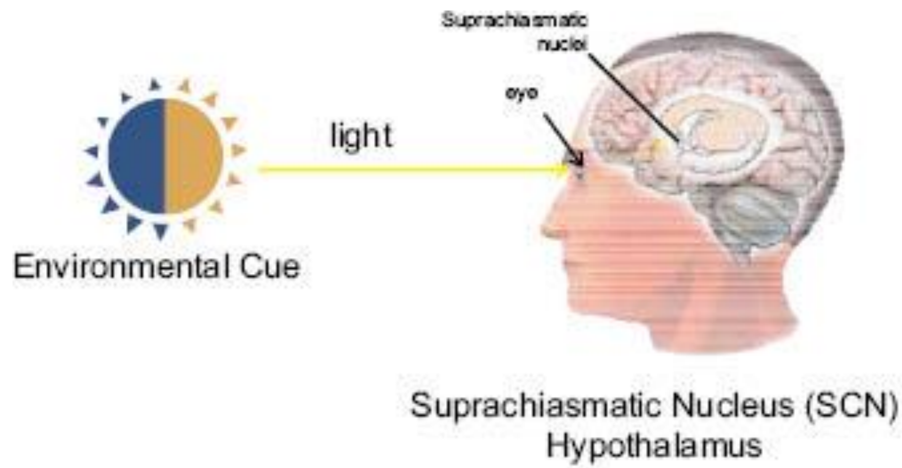
Stanford MEDICINE | Center For Sleep Sciences and Medicine

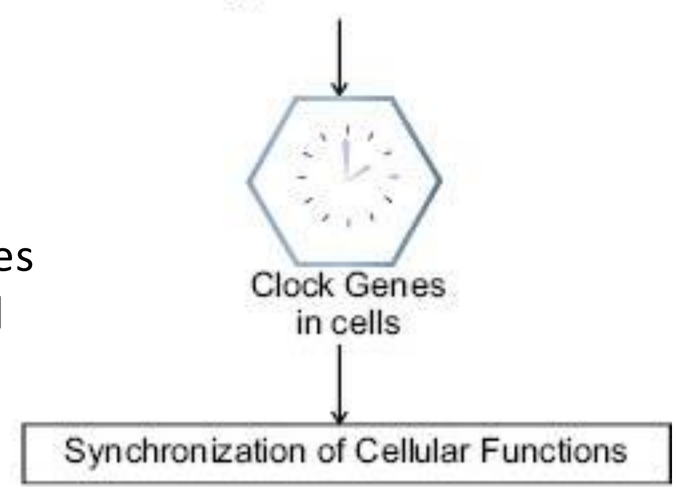
Circadian Clock well understood

4,256/7497=57% of genes are cycling
Zhang R, et al. Proc Natl Acad Sci U S A. 2014.



Core genes identified





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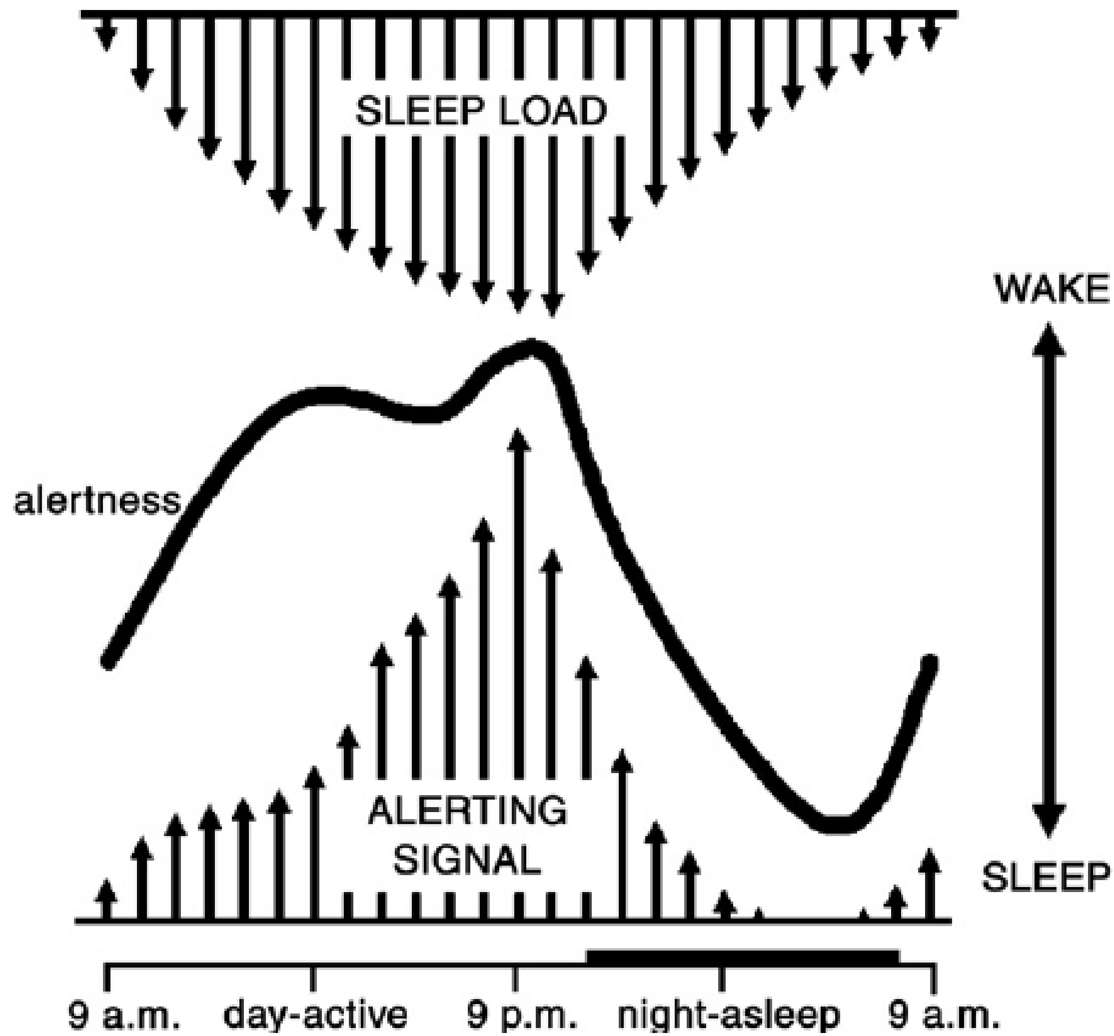
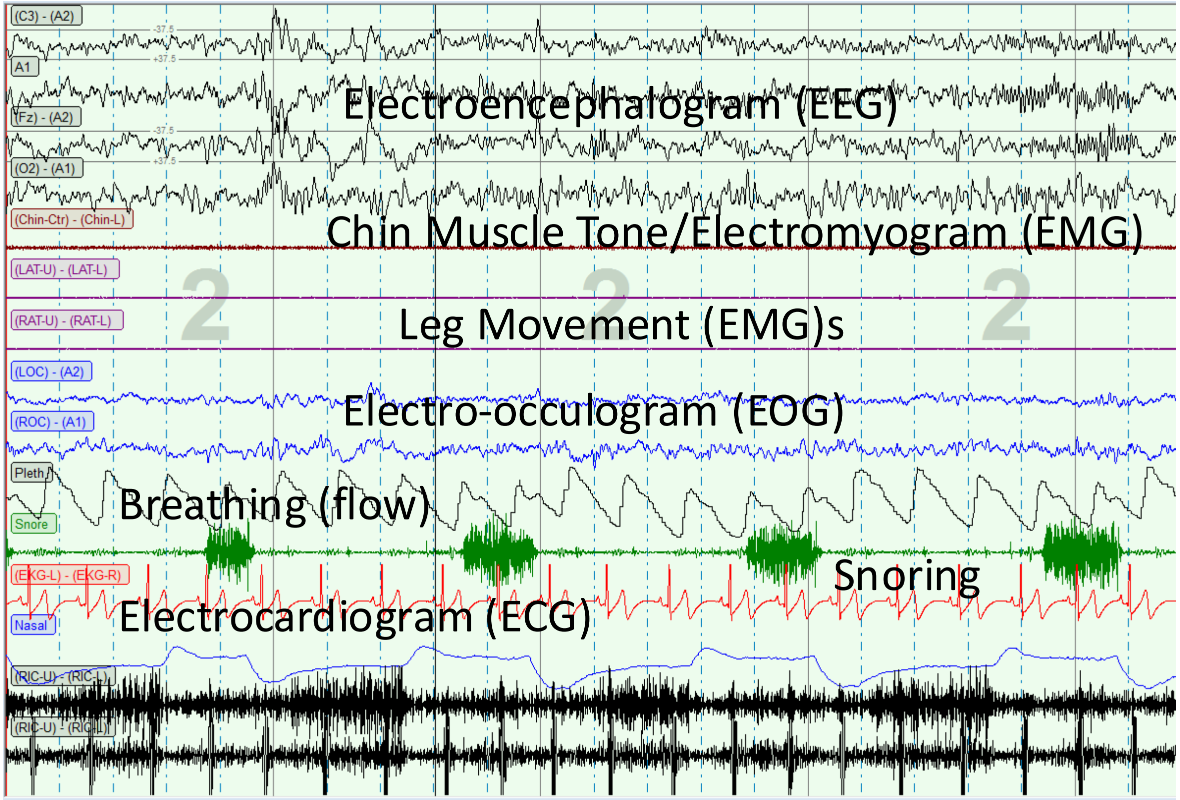
Sleep Homeostasis not understood

- Sleep and circadian physiology interact to maintain wakefulness during the day and control sleep during the night
- In the cortex and hypothamus combined, 4811/11078=43% of genes are sleep debt dependent, with about 1,520 (13%) sleep debt specific (Mackiewicz et al, Physiological Genomics, 31(3): 441-457, 2007)
- No Core gene identified

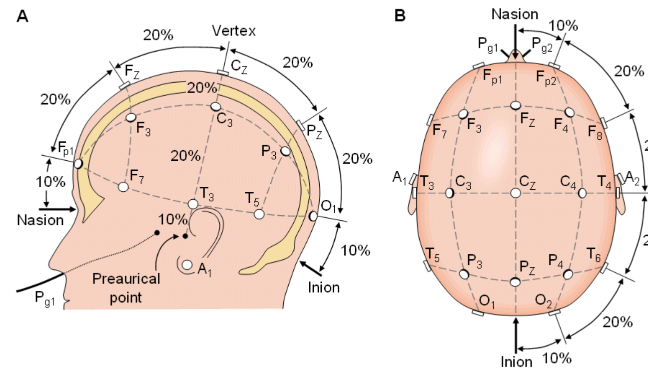



Gold Standard:
Nocturnal
Polysomnography
(PSG)

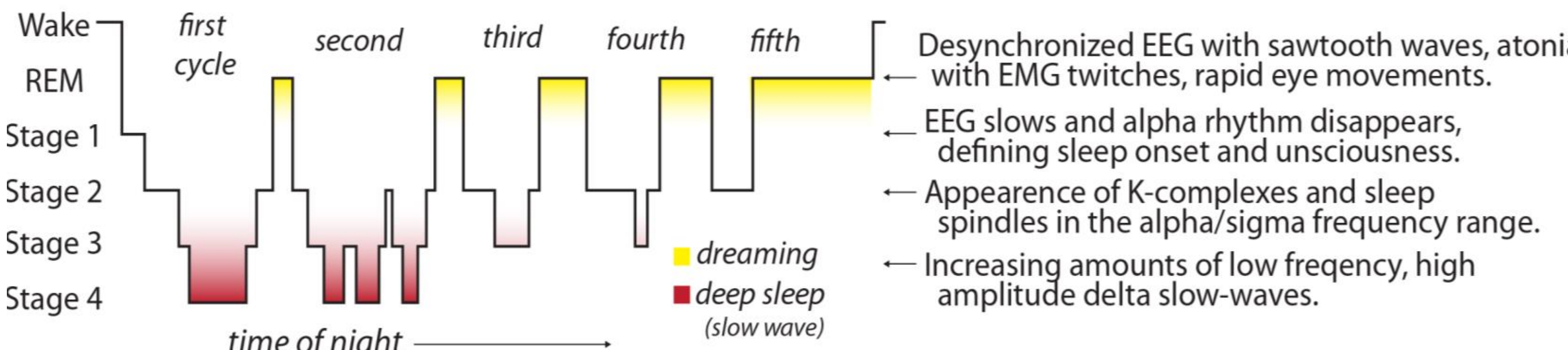
Gold Standard: Nocturnal Polysomnography (PSG)



Breathing (effort)

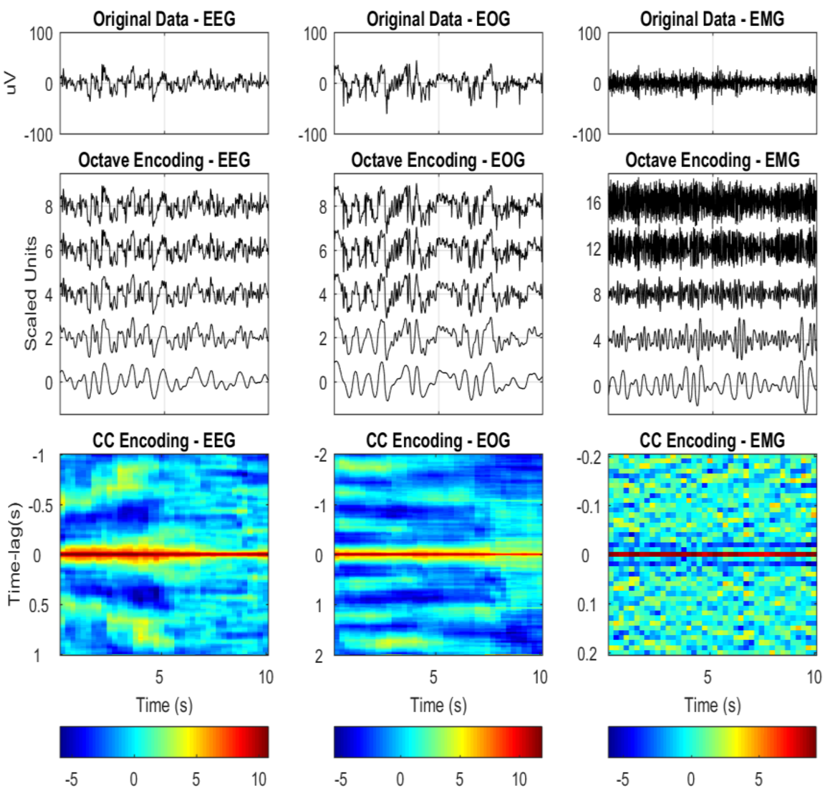


Sleep hypnogram for one night of sleep



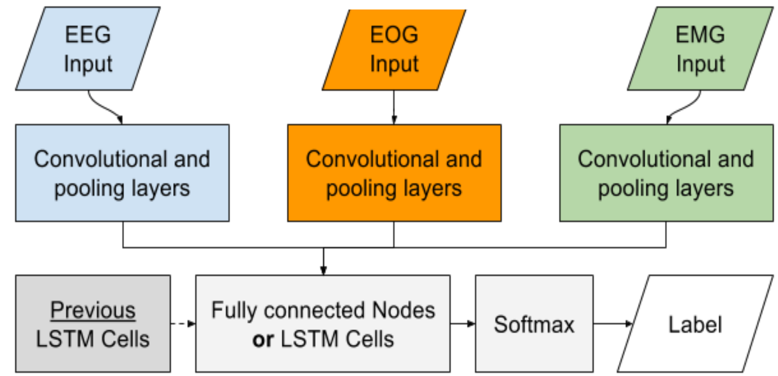
Machine learning does better than multiple human scorers in sleep staging

Feature extraction

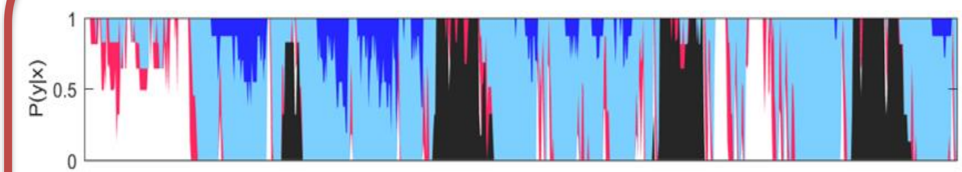


Supervised, unsupervised ML

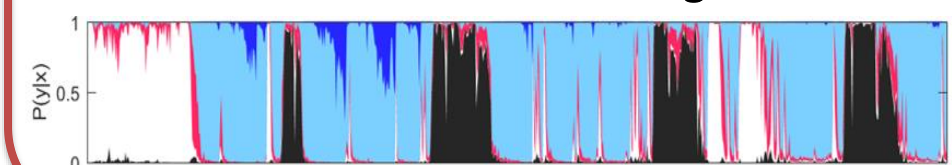
Network

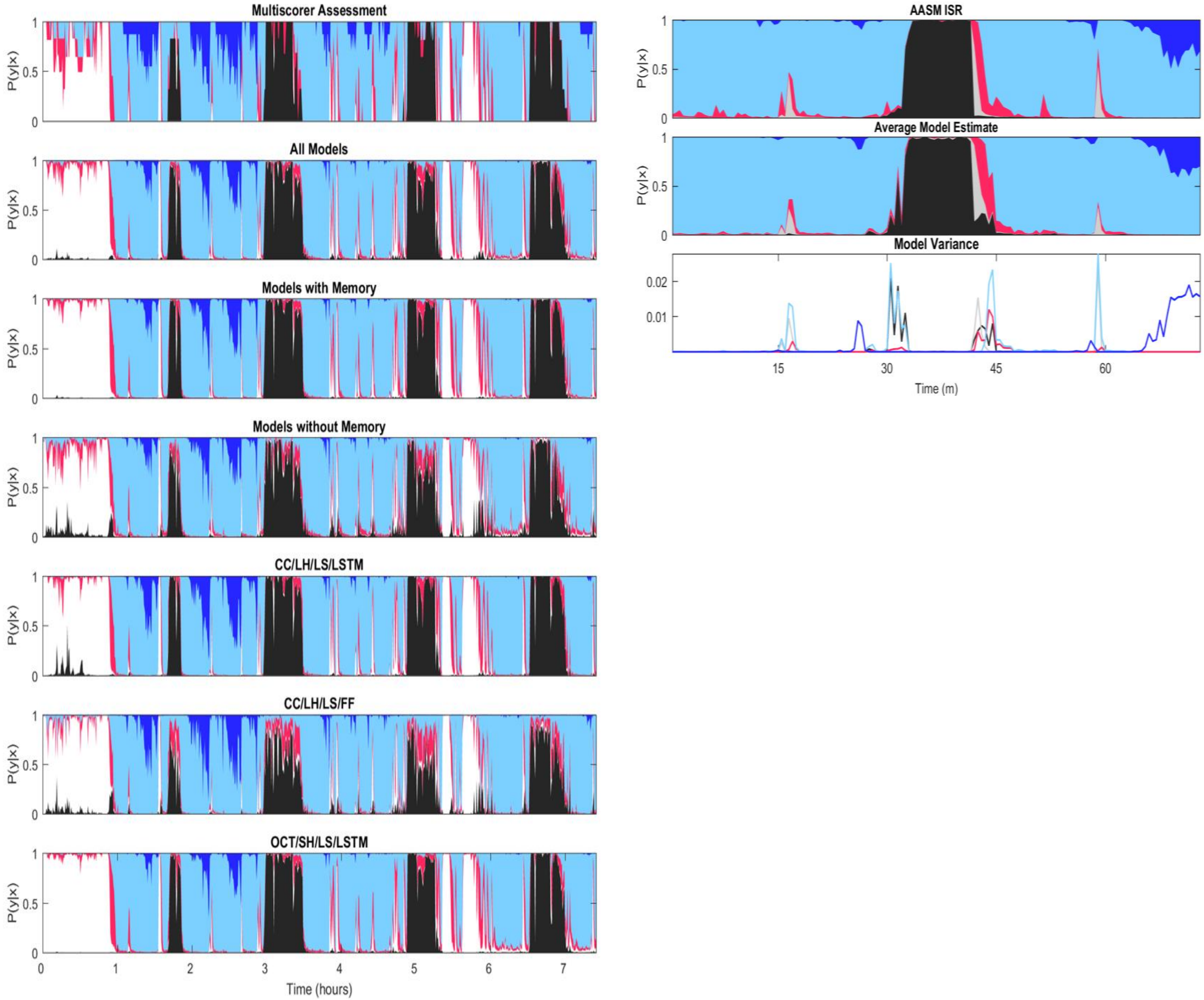


Mean of six scorers



Machine learning




Multiscorer Assessment
All Models
Models with Memory
Models without Memory
CC/LH/LS/LSTM
CC/LH/LS/FF
OCT/SH/LS/LSTM
P(y|x)
Time (hours)
AASM ISR
Average Model Estimate
Model Variance
Time (m)

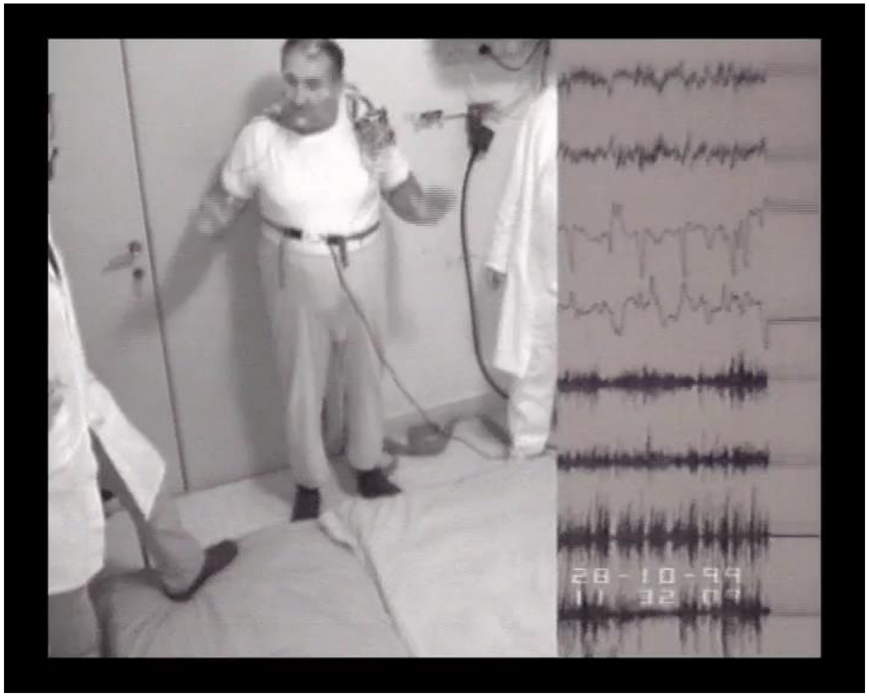
Effect of pathology on automatic scoring accuracy

- no strong effect of sex, age common sleep pathologies

Condition	% (n)	Sum Sq.	P value
Insomnia (343)	41%	0	0.76
OSA (871)	50% none 23% mild 14% moderate 12% severe	0.137	0.005
RLS (768)	20%	0.07	0.02
PLMI (472)	39% none 29% mild 18% moderate 12% severe	0	0.79
Type 1 narcolepsy (917)	21%	0.94	$5.6\ 10^{-22}$

Stephansen et al., Nature communication, 2019

Type 1 narcolepsy-cataplexy, a REM sleep and wakefulness disorder

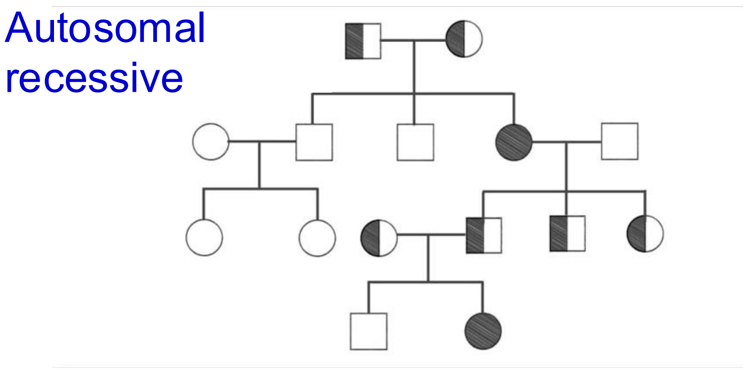


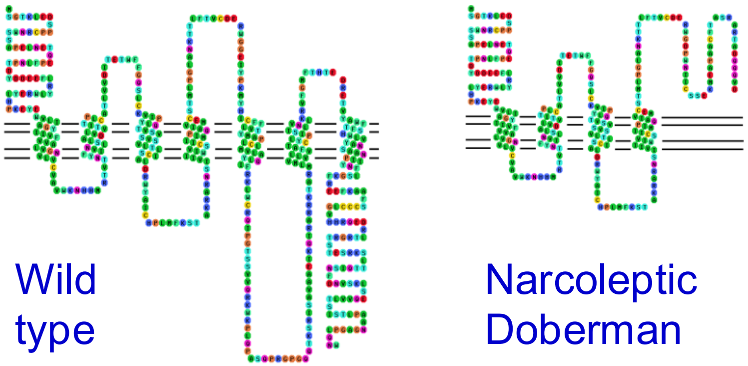
Giuseppe Plazzi

- Excessive daytime sleepiness
- Cataplexy
- Sleep paralysis
- Hypnagogic hallucinations
- Disturbed nocturnal sleep
- Sleep Onset REM periods on Multiple Sleep Latency Test (MSLT)

Sporadic disorder, prevalence: 0.05% (3-4M worldwide)
classically treated using amphetamine, antidepressants, sedatives
Model to understand sleep and REM sleep?

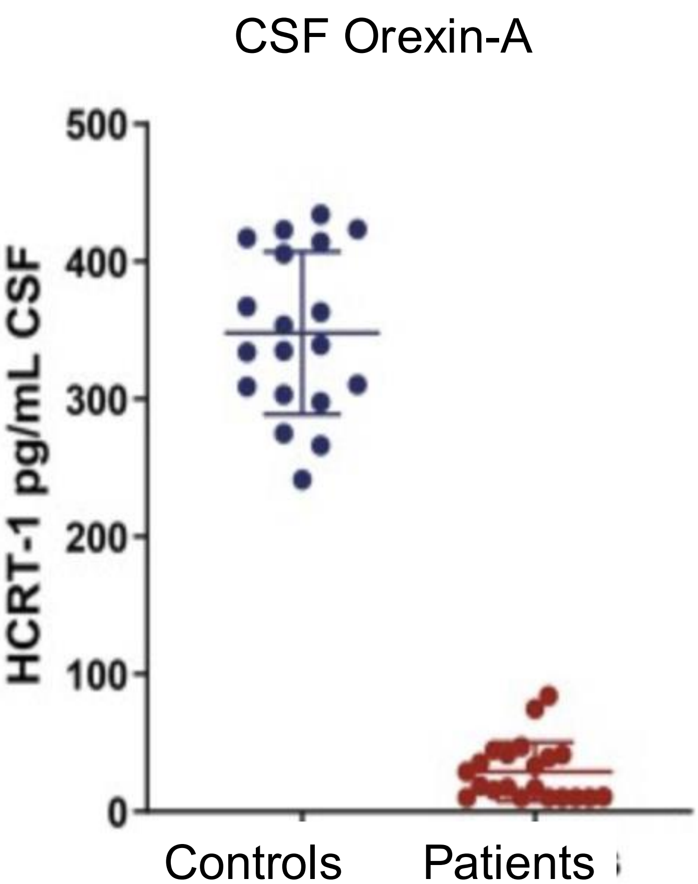
Unlike in humans, canine narcolepsy is genetically transmitted as a single gene

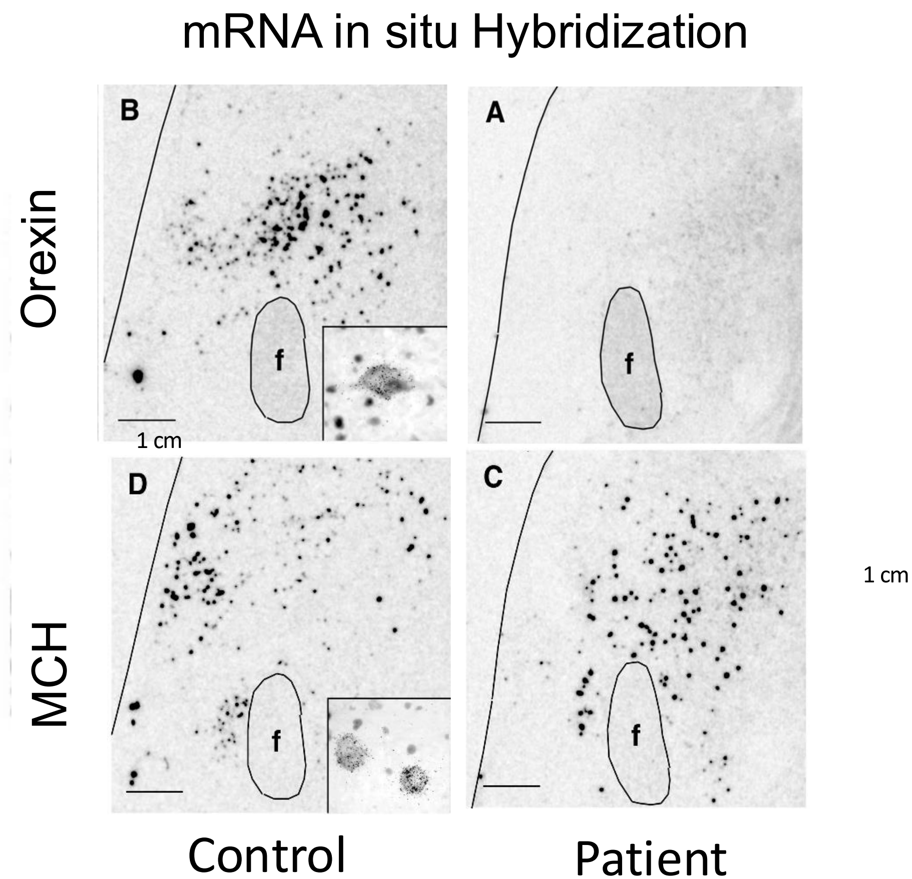




OXR2 mutations in 3 breeds
Lin *et al.*, Cell, 1999

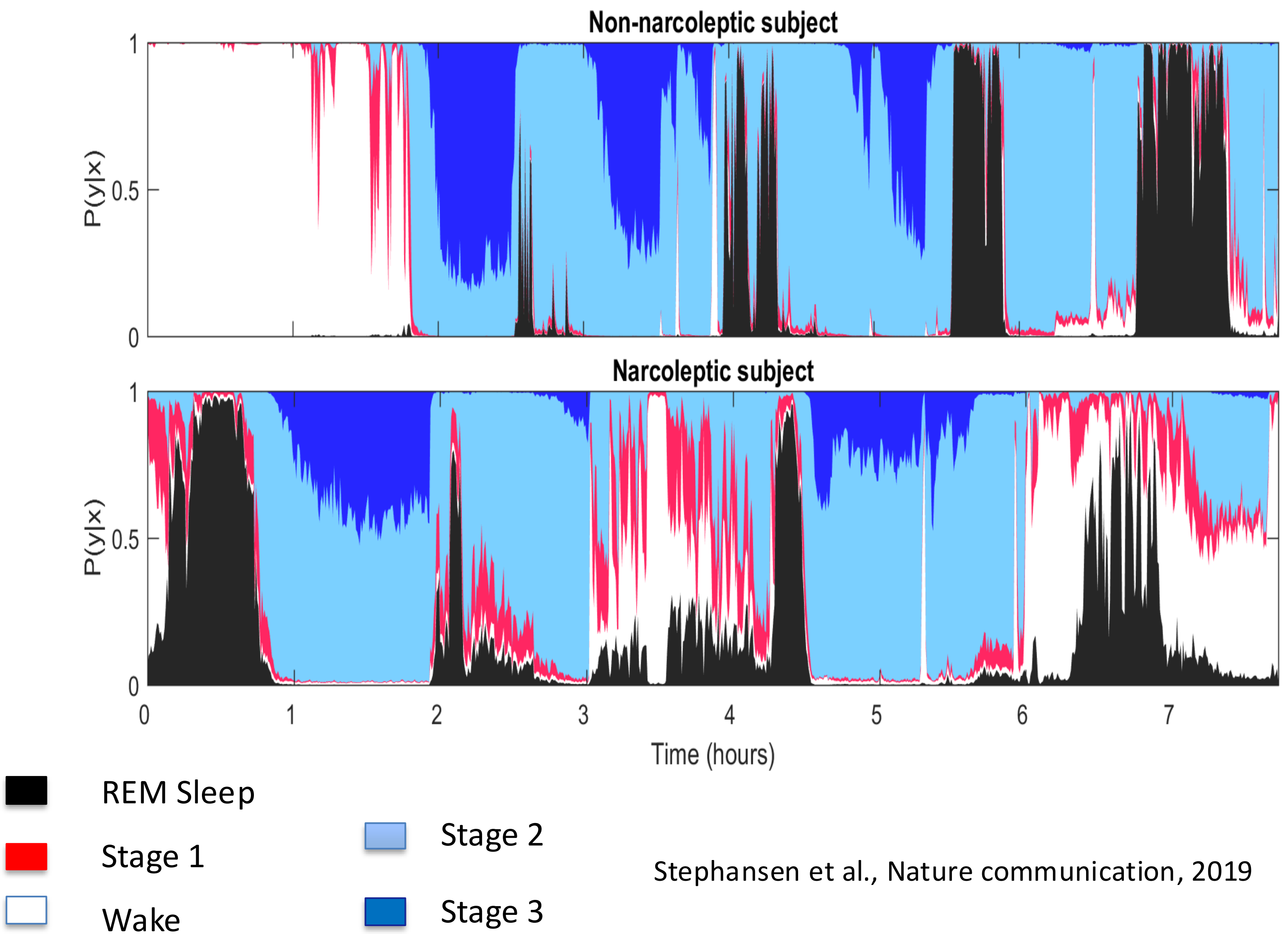
Hypocretin deficiency in human type 1 narcolepsy





Peyron *et al.,* Nature Med 2000; Mignot *et al.,* JAMA Neurol, 2002; Scammell *et al*., Neurology, 2004

Hypnodensity in narcolepsy displays sleep state dissociation



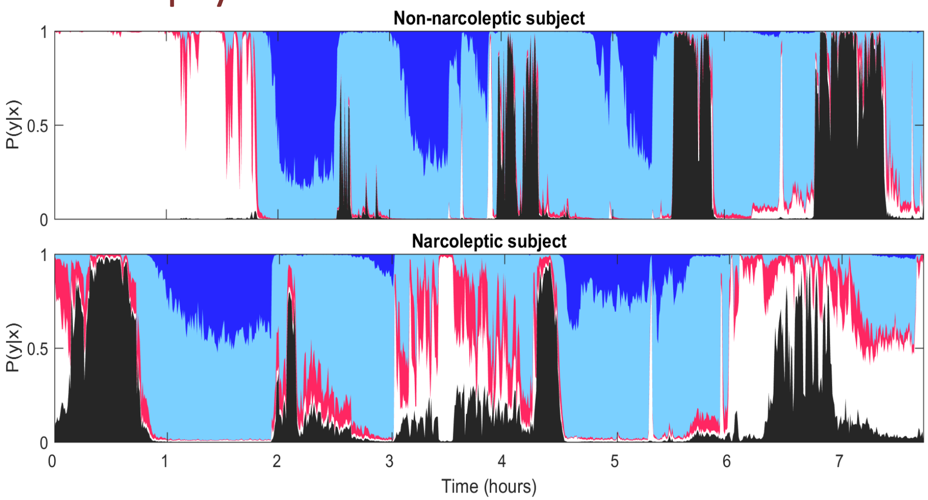
Stephansen et al., Nature communication, 2019

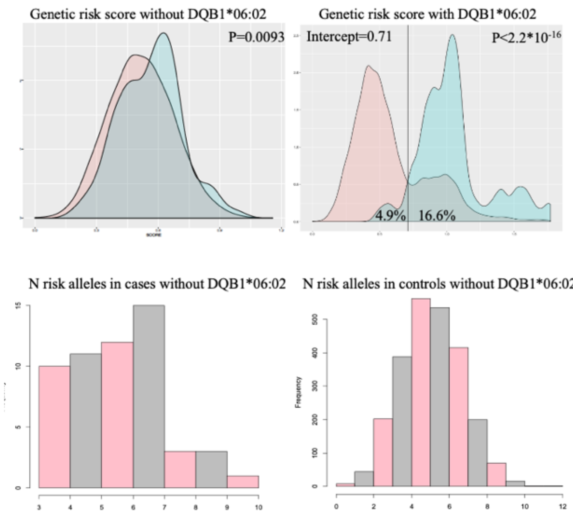
Selection of narcolepsy features using Lasso regression

#	Description	Relative importance
1	REM latency (Automatically extracted)	1
2	Mean co-occurence of REM and wake	0.80
3	Mean value of REM x the time till 10 % of the cumulative fraction of REM is reached.	0.68
4	The maximum value of co-occurrence of N2 and REM.	0.66
5	The mean value of N2 x the time till 10 % of the cumulative fraction of N2 is reached.	0.61
6	Time till 10 % of the cumulative fraction of the co-occurrence of all 5 stages is reached.	0.55
7	Mean value x the Shannon entropy of the co-occurrence of N2 and N3.	0.40
8	Time till 10 % of the cumulative fraction of the co-occurrence of N1 and N2 is reached.	0.37
9	Maximum value of co-occurrence of wake and N1	0.36
10	Maximum value of co-occurrence of wake and REM	0.35

Narcolepsy predictions

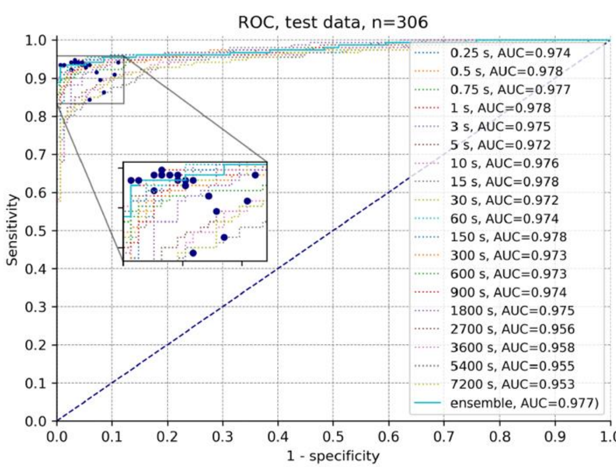
Narcolepsy





Stephansen et al., *Nat Communication,* 2018.

Ollila et al., *Nat Communication, 2023*

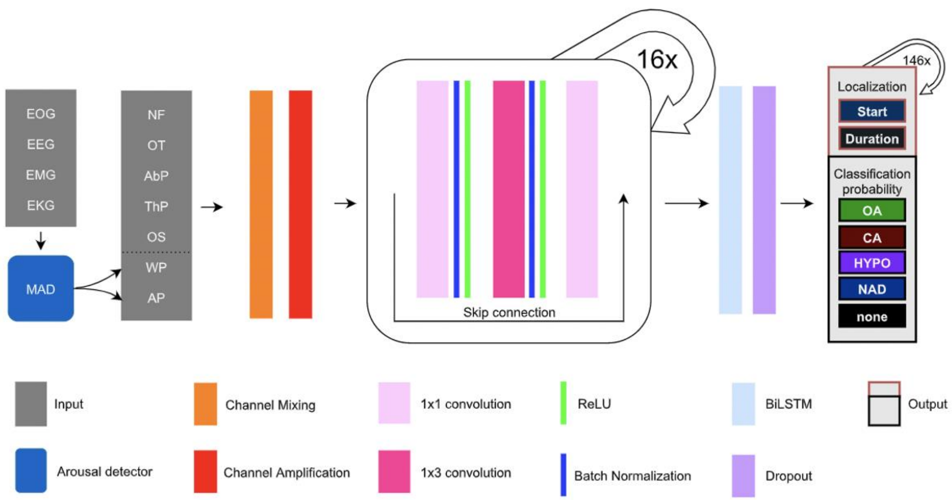


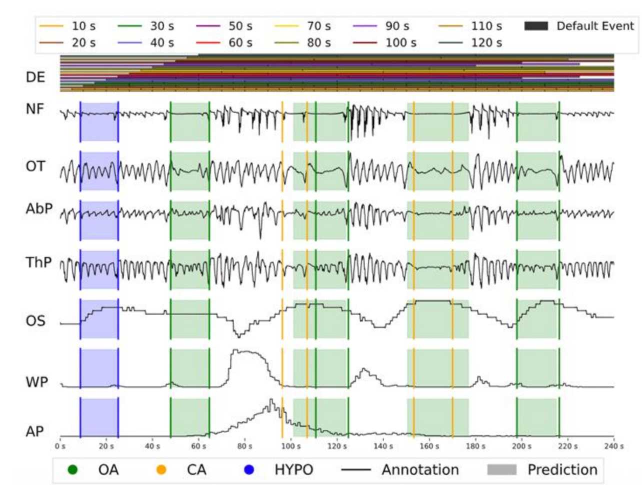
Multimodal prediction with genetics (HLA, PRS, LD prep)

Collaboration Dmitri Wolfson @ Takeda

ABED: Automatic Sleep-Disordered Breathing Event Detection

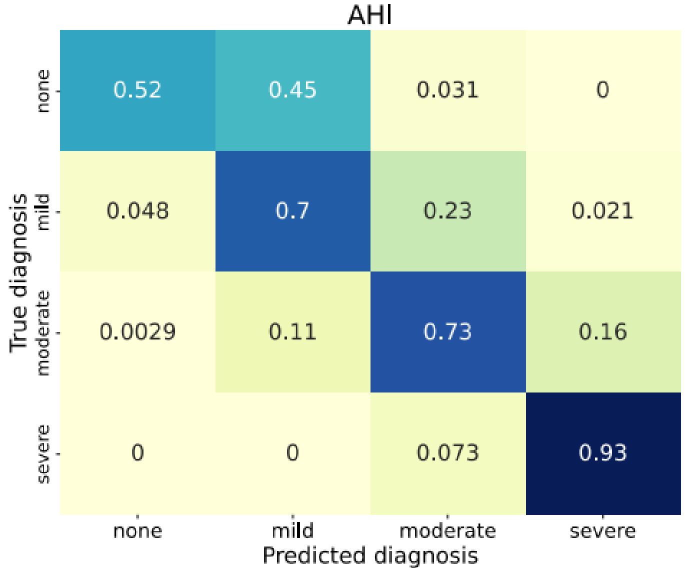
Cohort	OA	CA	HYPO	NAD	PSGs (test)	Age	BMI	(F - M)
Single scored								
MESA *	32,249	3,865	145,119	131,600	1,424 (249)	69.2 ± 9.0	28.8 ± 5.3	54 - 46
MrOS *	97,544	26,919	214,605	198,641	2,837 (500)	76.4 ± 5.5	27.2 ± 3.8	0 - 100
WSC *	28,964	4,753	155,517	0	1,567 (250)	57.3 ± 8.0	31.8 ± 7.3	46 - 54
CFS *	16,245	2,384	74,091	0	727 (100)	41.5 ± 19.4	32.4 ± 9.5	55 - 45
Total	205,063	42,718	803,438	196,044	6,555 (1,099)	∼	∼	∼
Consensus scored								
Alliance *	1,521	228	6,050	0	58 (58)	51.1 ± 4.2	32.9 ± 9.2	100-0
DREEM *	2,166	501	4,026	0	55 (55)	45.6 ± 16.5	29.6 ± 6.4	36 - 64
Total	3,687	729	10,076	0	103 (103)	∼	∼	∼

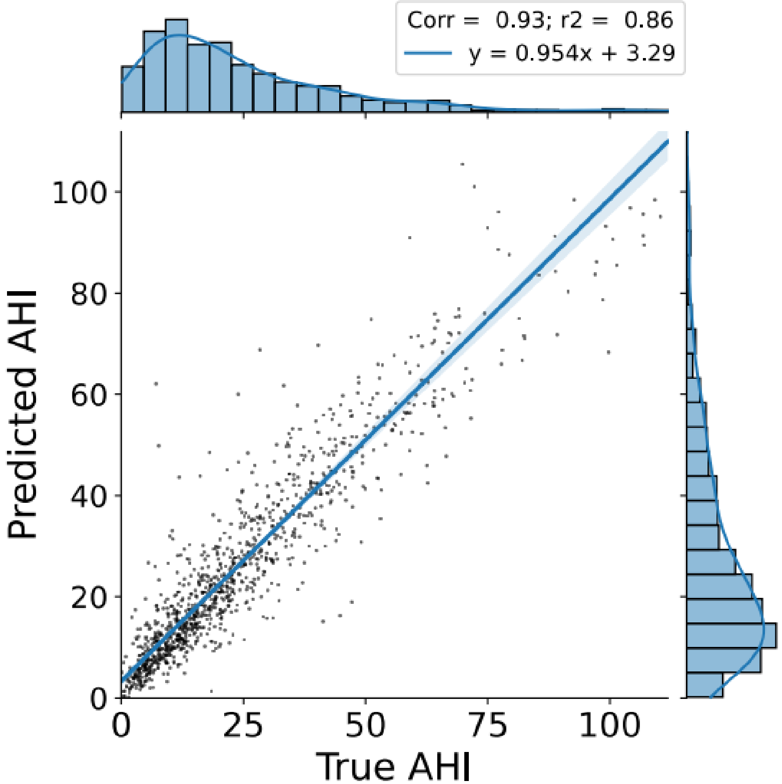




Magnus Ruud Kjær

Sleep apnea Diagnosis Using Detected Events to Estimate AHI





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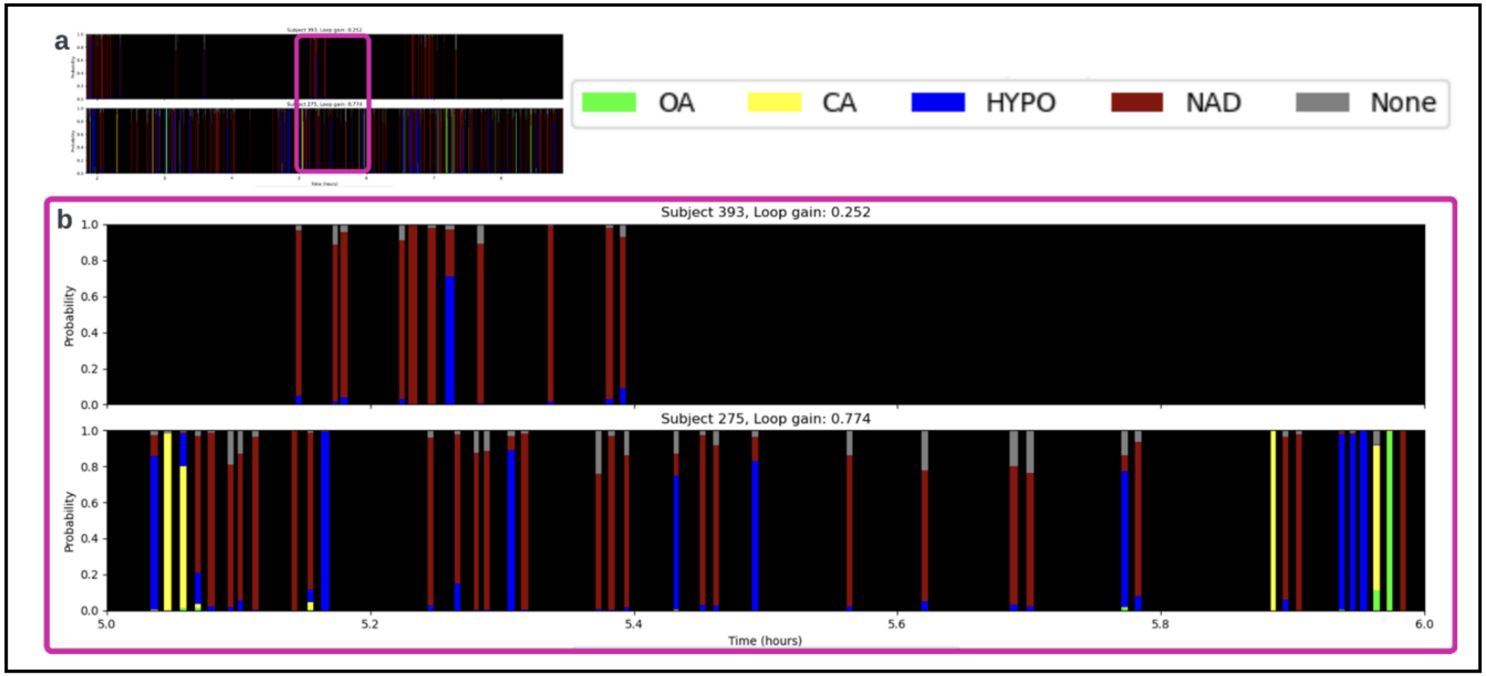
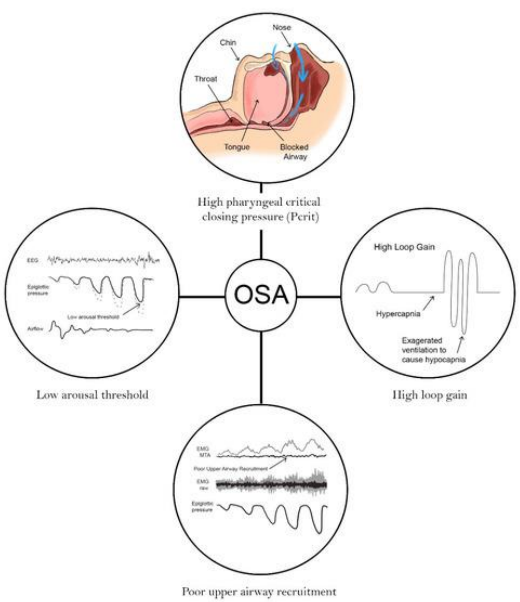
Sleep "apneatype" predicts cause of sleep apnea





MODEL (LGN)	VARIABLE	EFFECT UNIVARIATE (+ BASE)	EFFECT MULTIVARIATE	R2(ADJ)
BASE	BMI AGE Sex			0.088
BASE + TRUE	OAI_{true}			0.129
BASE + PREDICTED	HI_{pred} CAI_{pred} NI_{pred}			0.209
BASE + PROBABILITY X PREDICTED	$OAI \cdot \overline{P(CA \mid OA)}$ $OAI \cdot \overline{P(HYPO \mid OA)}$ $NI \cdot \overline{P(NAD \mid NAD)}$ $CAI \cdot \overline{P(CA \mid CA)}$			0.226
BASE + PROBABILITY + PREDICTED	$\overline{P(CA \mid NAD)}$ CAI_{pred} $\overline{P(NAD \mid HYPO)}$ NI_{pred}			**0.266**
		-0.05 0 0.05	-0.05 0.00 0.05	

Magnus Ruud Kjær

Toward a single, multimodal PSG analyzer

- ✓Doing classic and novel sleep stage identification
- ✓Detecting macro and microarousals (autonomic and EEG)
- ✓Detecting periodic leg movements during sleep, with and without arousal
- ✓Diagnosing narcolepsy and REM behavior disorder
- ✓Detecting breathing abnormalities during sleep, including subtypes of sleep apneas (with and without arousals or hypoxia)
- ✓To apply in the context of large population studies

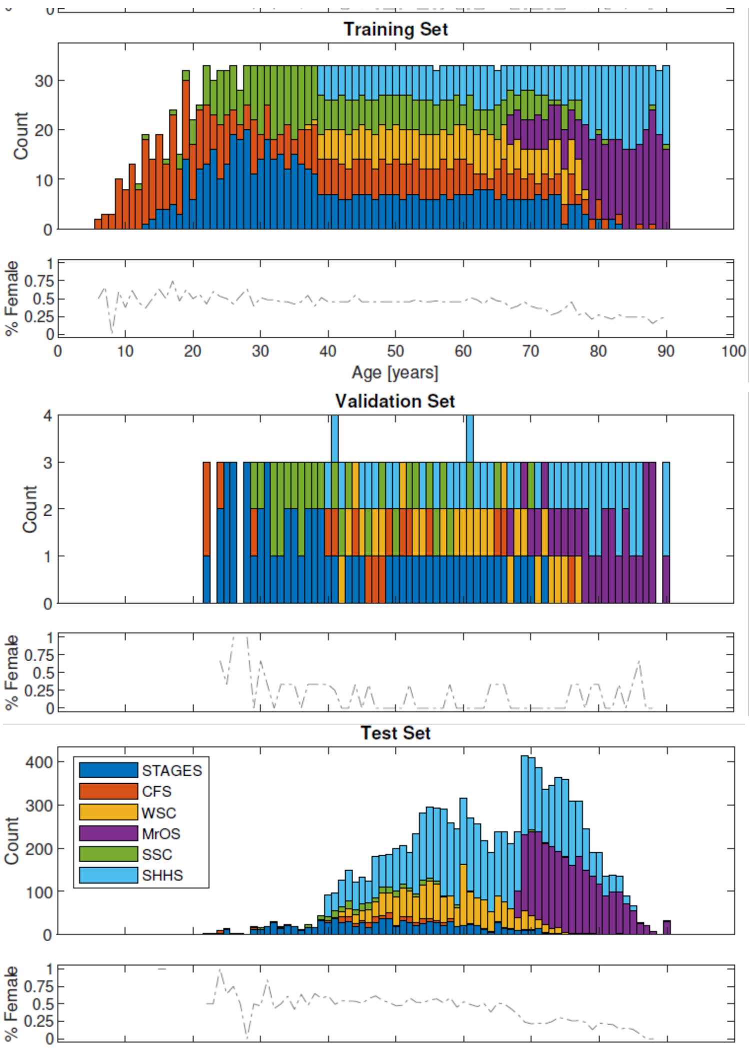
Polysomnography “ Sleep” Age

Andreas Brink Kjaer MS

- New deep learning **framework** for end-to-end processing of polysomnograms.
- Train a deep neural network to predict age based on 5-minute epochs of PSG data across six cohorts (2,500 subjects)
- Use a learned latent representation of each 5-minute epoch to model age based on a whole-night’s PSG data.

1	CFS	STAGES	WSC	MrOS	SSC	SHHS	All
Train (n)	521	555	267	287	419	447	2500
Age ($\mu \pm \sigma$)	37.902 ± 20.869	42.0 ± 17.6	58.7 ± 11.3	81.3 ± 6.4	44.8 ± 16.9	68.8 ± 15.5	52.7 ± 22.3
Val (n)	17	58	26	30	28	41	200
Age ($\mu \pm \sigma$)	46.2 ± 15.5	42.8 ± 14.8	60.7 ± 10.9	80.1 ± 6.5	44.8 ± 11.5	68.5 ± 15.2	56.6 ± 19.1
Test (n)	192	969	1627	2587	251	5218	10844
Age ($\mu \pm \sigma$)	50.4 ± 10.6	48.8 ± 11.8	58.3 ± 7.5	75.8 ± 5.0	47. ± 9.0	62.7 ± 10.6	63.4± 12.4

*Preliminary work published in EMBC 2020: Brink-Kjaer, E. Mignot, H. B. D. Sorensen and P. Jennum, "Predicting Age with Deep Neural Networks from Polysomnograms," 2020 42nd Annual International Conference of the IEEE Engineering in Medicine & Biology Society (EMBC), Montreal, QC, Canada, 2020, pp. 146-149, doi: 10.1109/EMBC44109.2020.9176254.



Input PSG signals

Input size [C, H, W]	Operations	
[1, 12, L ~ 4500000]	Epoch split	
N x [1, 12, 38400]	Channel mixer	conv2d (12x1, 32, s = 1)
N x [32, 1, 38400]	Small MobileNetV2	conv2d (1x3, 32, s = 2)
N x [32, 1, 19200]		bottleneck (1x3, 16, s = 2, t = 1)
N x [16, 1, 9600]		bottleneck (1x3, 32, s = 2, t = 6)
N x [32, 1, 4800]		bottleneck (1x3, 32, s = 1, t = 6)
N x [32, 1, 4800]		bottleneck (1x3, 64, s = 2, t = 6)
N x [64, 1, 2400]		bottleneck (1x3, 64, s = 1, t = 6)
N x [64, 1, 2400]		bottleneck (1x3, 128, s = 1, t = 6)
N x [128, 1, 2400]	Average pooling	avg pool (1x20, s = 20)
Input size [seq_len, C]	Operations	
N x [120, 128]	Recurrent layer	GRU (128, bidirectional)
N x [120, 256]	Additive attention	att (512)
N x [1, 256]	Dense layer	linear (256)

Latent representation of each 5-minute epoch

N x [1, 256]	Dense layer	linear (1)
N x [1, 1]	Epoch age prediction	

[N, 256]	Recurrent layer	GRU (128, bidirectional)
[N, 256]	Additive attention	att (512)
[1, 256]	Dense layer	linear (256)
[1, 256]	Dense layer	linear (1)
[1, 1]	PSG age prediction	

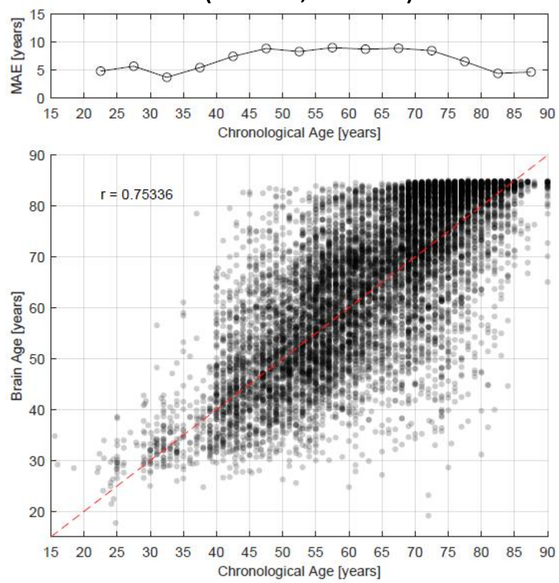
Age prediction

Andreas Brink Kjaer MS

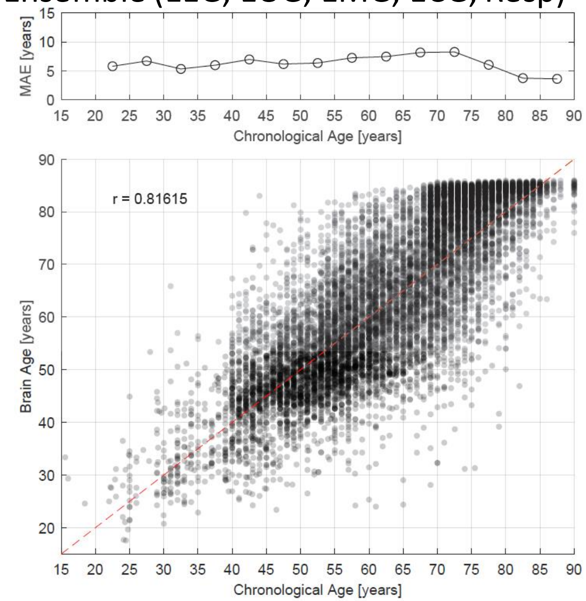
Results: Performance

- Scatter plot with MAE in 5-year age intervals

EEG (C3-A2, C4-A1)



Ensemble (EEG, EOG, EMG, ECG, Resp)



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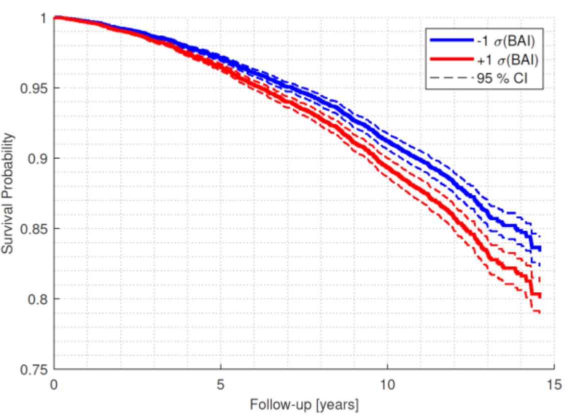
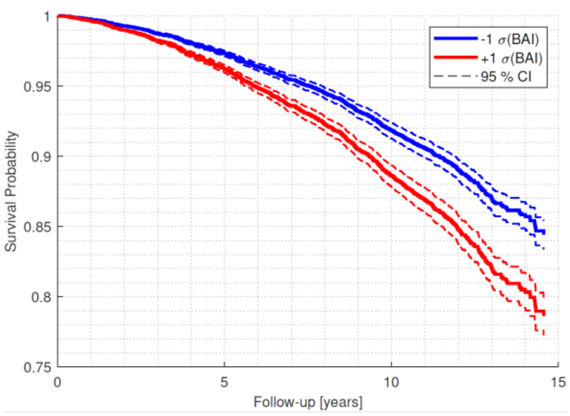
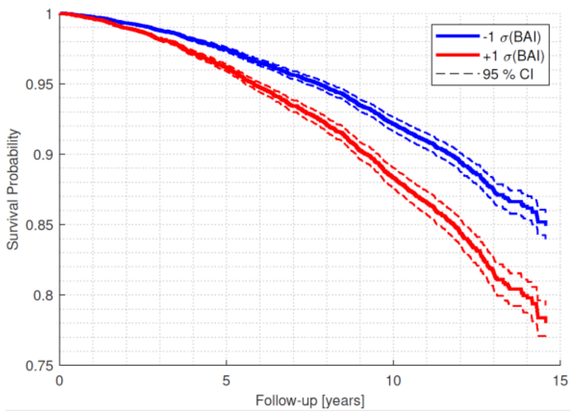
![Stanford Medicine]

Results: Mortality Analysis (SHHS test set n = 5218)

Variable	(a) AEE_{Full}	(b) AEE_{EEG}	(c) $AEE_{EEG+EOG+EMG}$	(d) AEE_{ECG}	(e) AEE_{Resp}	(f) $AEE_{Ensemble}$
# Channels	12	2	5	1	5	11
HR (95% CI)	1.24, (1.15, 1.33)	1.12, (1.04, 1.20)	1.19, (1.10, 1.28)	1.11, (1.03, 1.20)	1.16, (1.07, 1.25)	1.24, (1.16, 1.32)



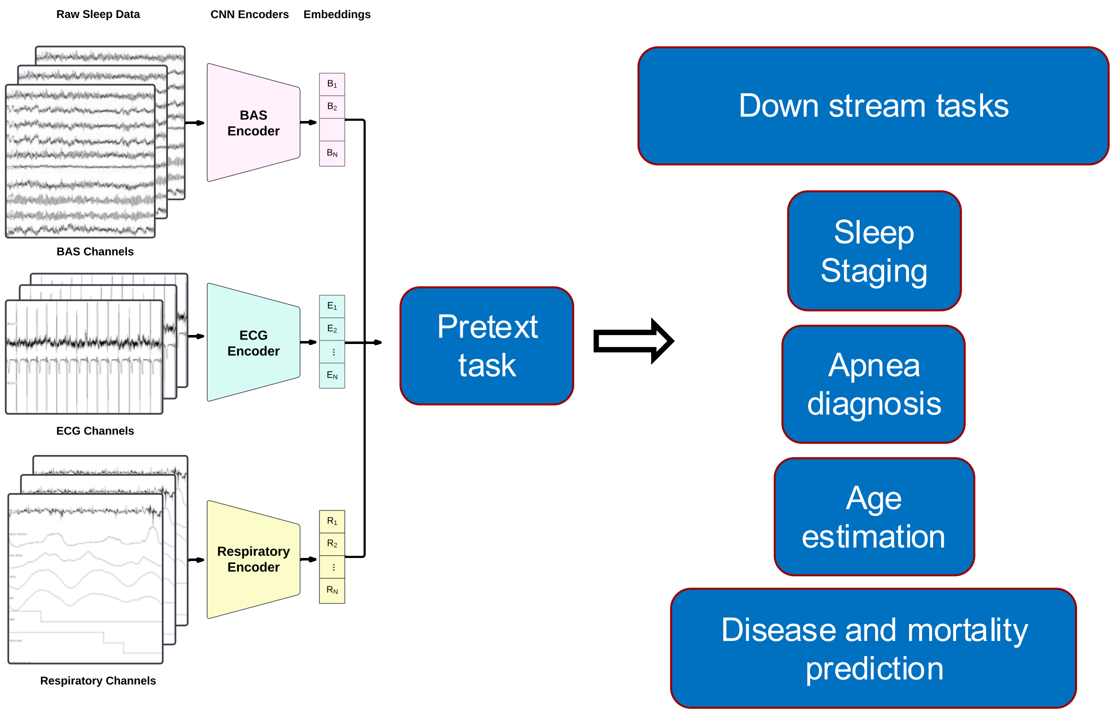




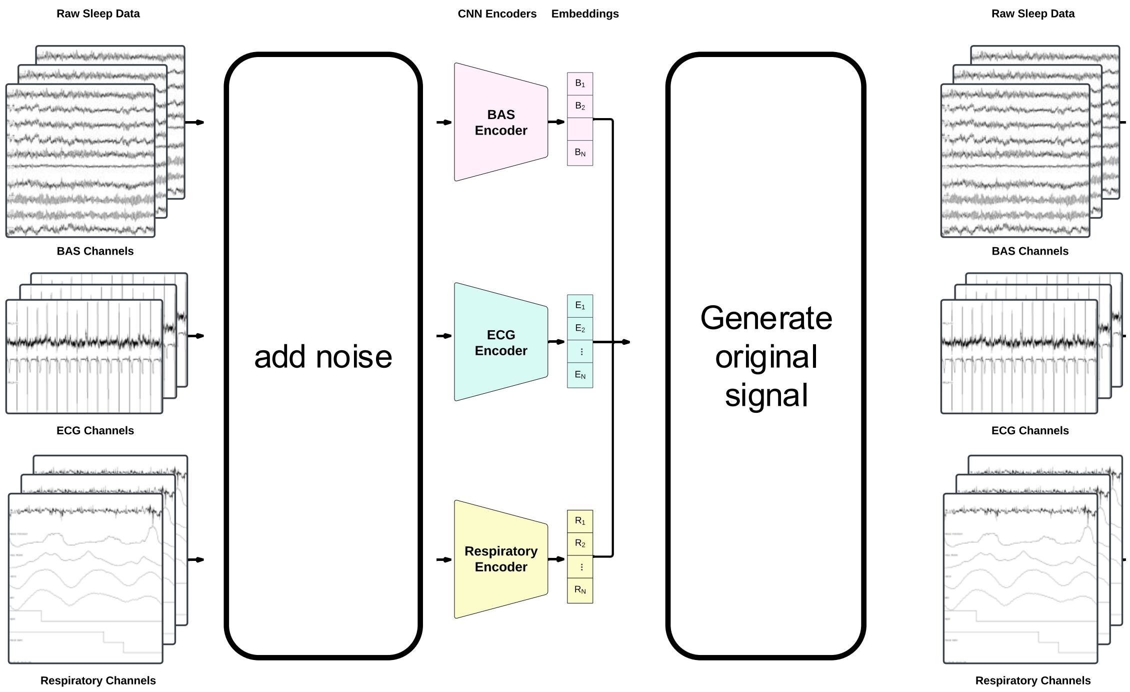
Next: predict mortality directly, attention network

Andreas Brink Kjaer MS

Next step: Foundation models

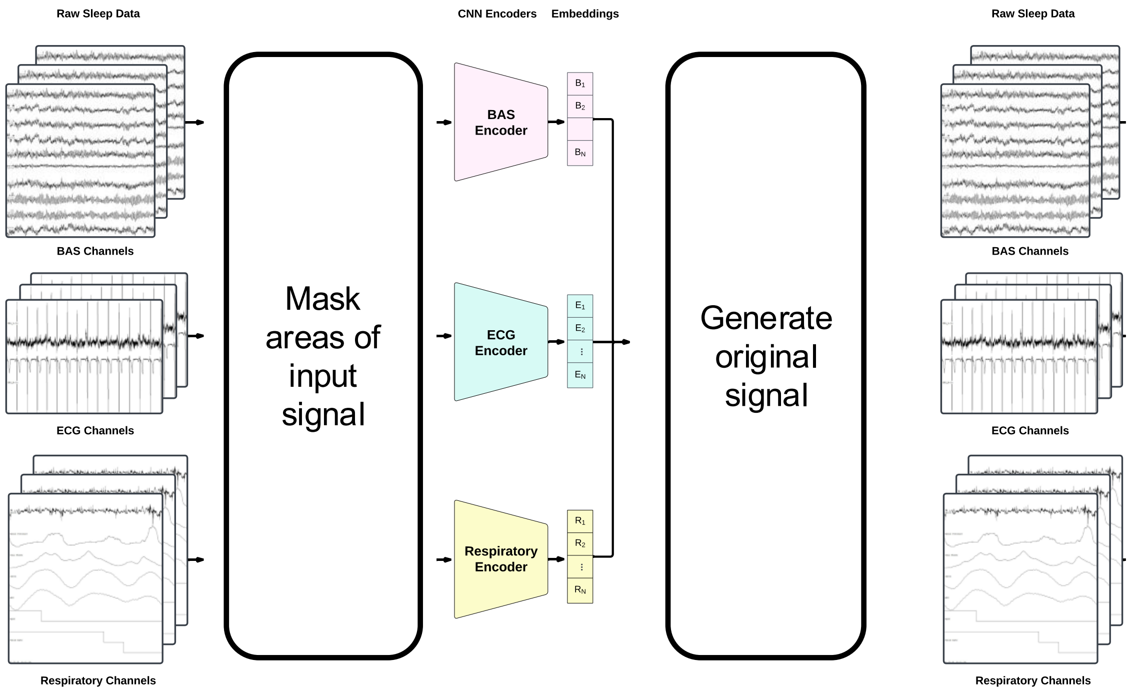


What is a foundation model (denosing auto-encoder)



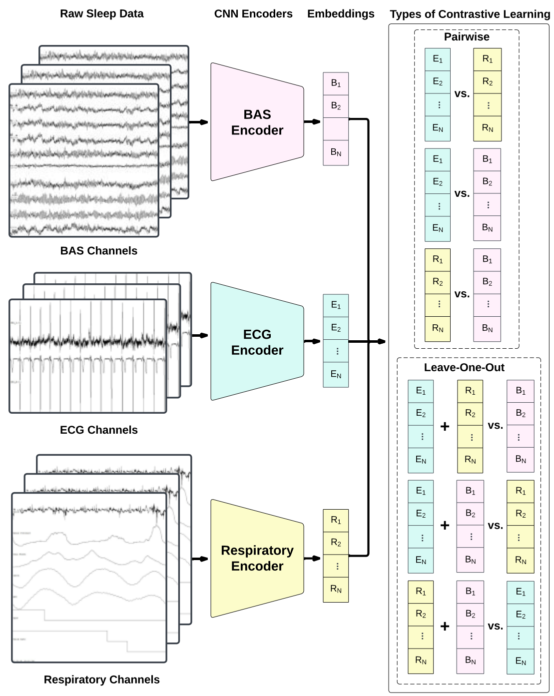
DAE

What is a foundation model (Masked auto-encoder)



MAE

What is a foundation model (contrastive learning)

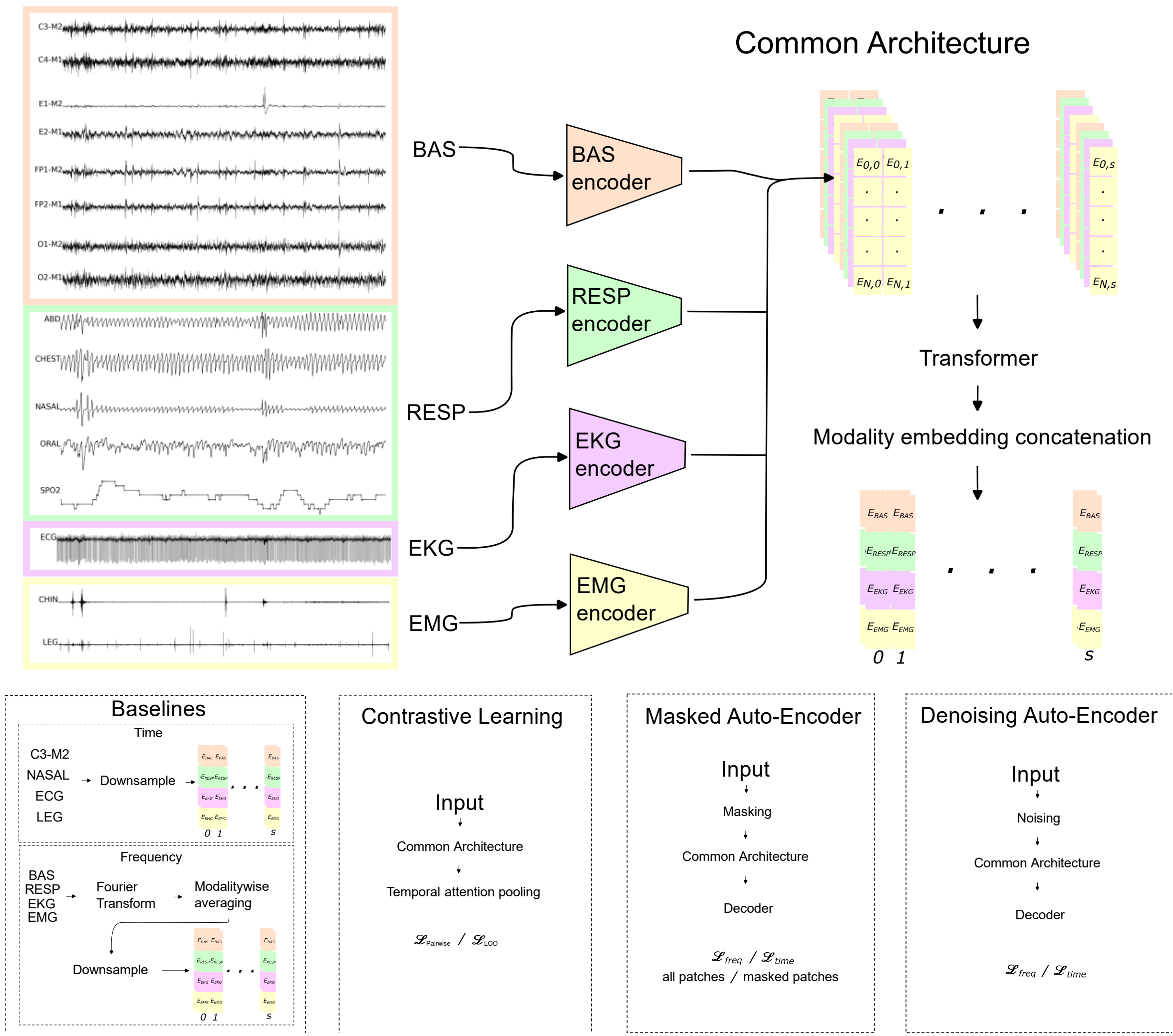


PW

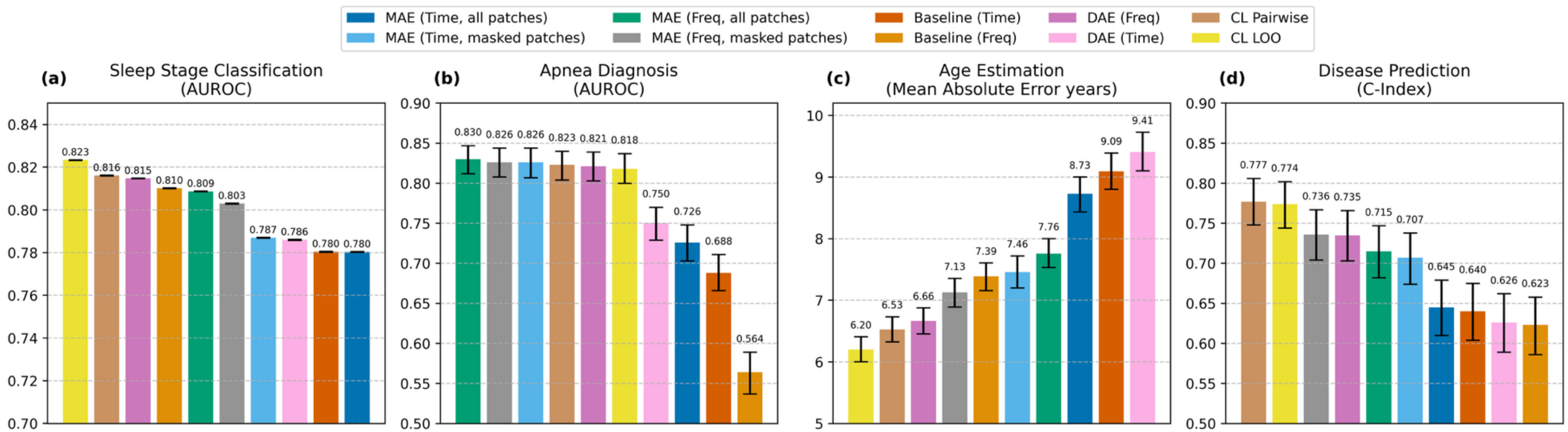
LOO

CL

How do we make a Foundation model for Multi modal PSG data



What is the optimal SSRL task for multimodal PSG data (Best performing model)



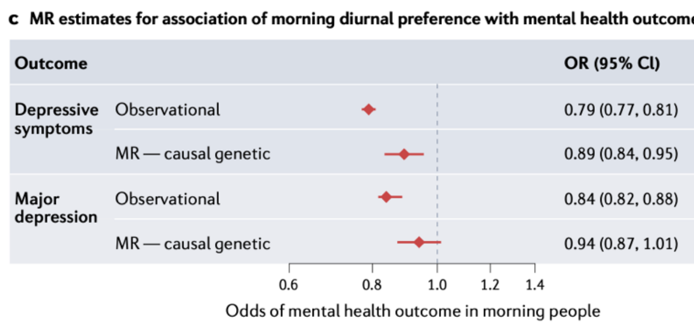
Genetics of circadian rhythms and sleep in human health and disease

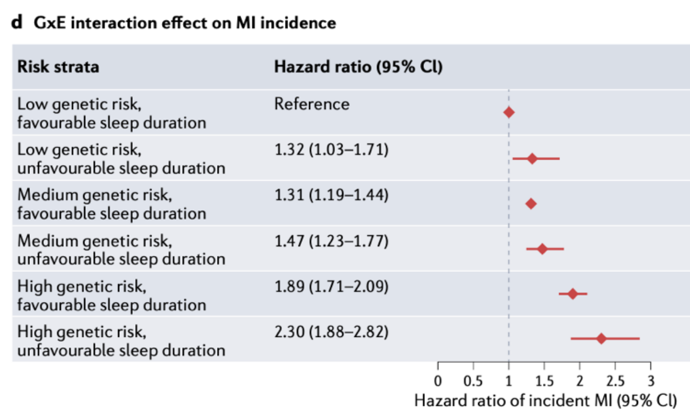
Jacqueline M. Lane[1,2,3], Jingyi Qian[2], Emmanuel Mignot[4], Susan Redline[2], Frank A. J. L. Scheer[2]✉ and Richa Saxena[1,2,3]✉

Abstract | Circadian rhythms and sleep are fundamental biological processes integral to human health. Their disruption is associated with detrimental physiological consequences, including cognitive, metabolic, cardiovascular and immunological dysfunctions. Yet many of the molecular underpinnings of sleep regulation in health and disease have remained elusive. Given the moderate heritability of circadian and sleep traits, genetics offers an opportunity that complements insights from model organism studies to advance our fundamental molecular understanding of human circadian and sleep physiology and linked chronic disease biology. Here, we review recent discoveries of the genetics of circadian and sleep physiology and disorders with a focus on those that reveal causal contributions to complex diseases.

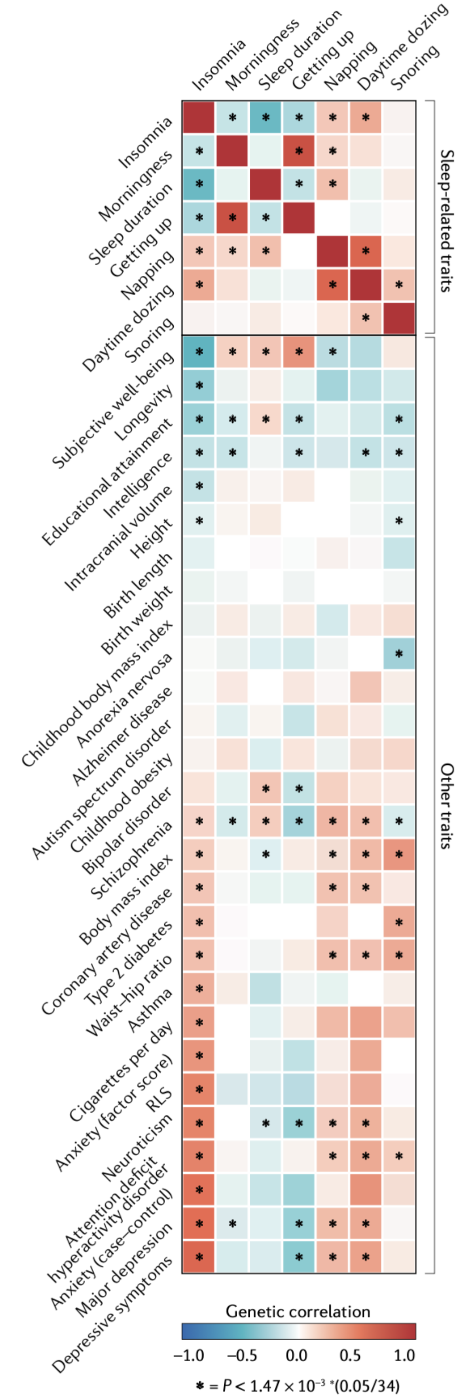
Nature Reviews Genetics volume 24, pages4–20 (2023)

Genetics is key to get to pathophysiology of NT1/IH sleepiness

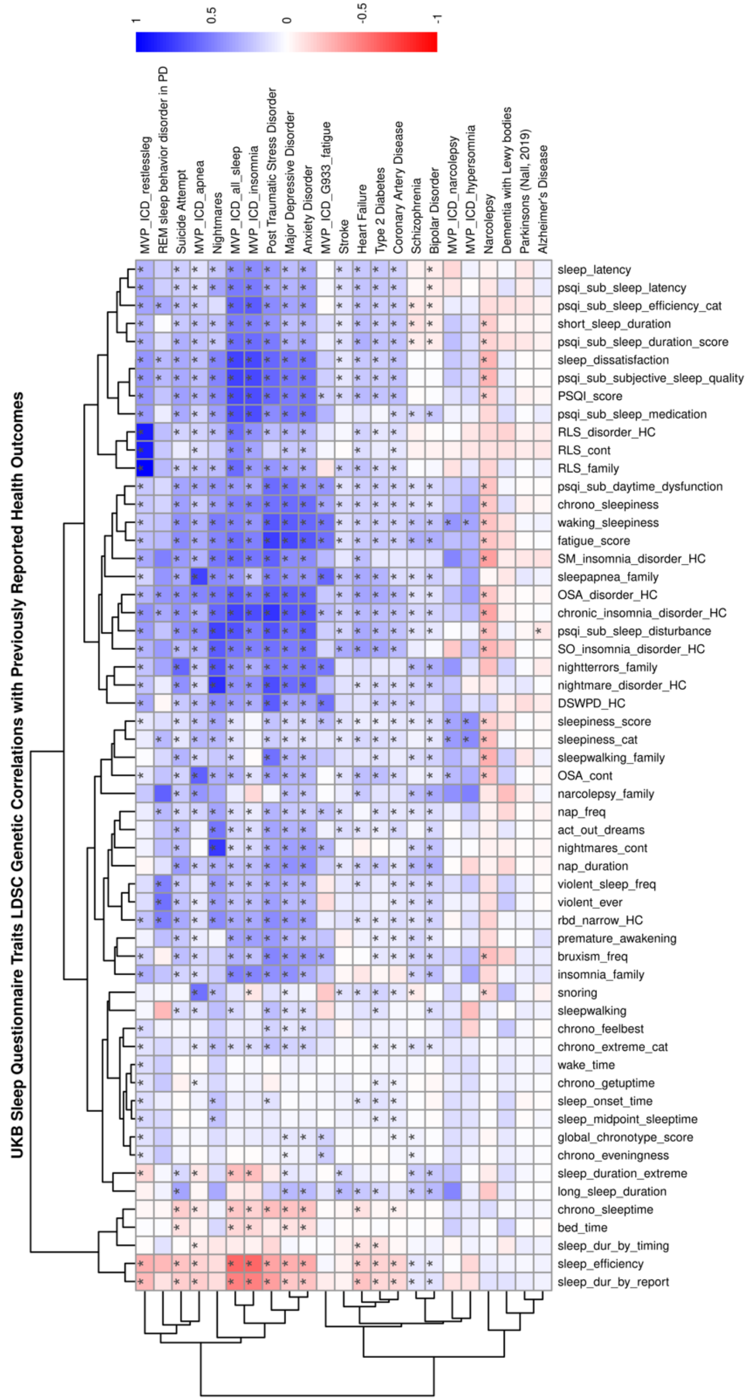


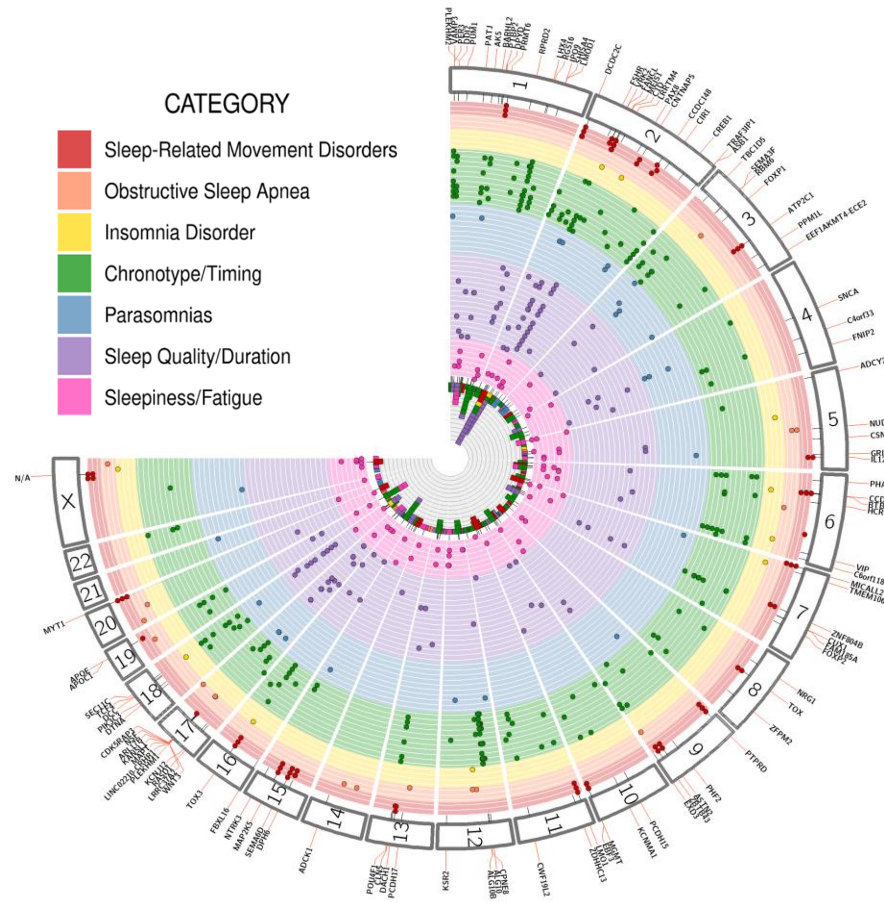


Daghlas et al., 2019



Correlations and loci involved in sleep (UKB)

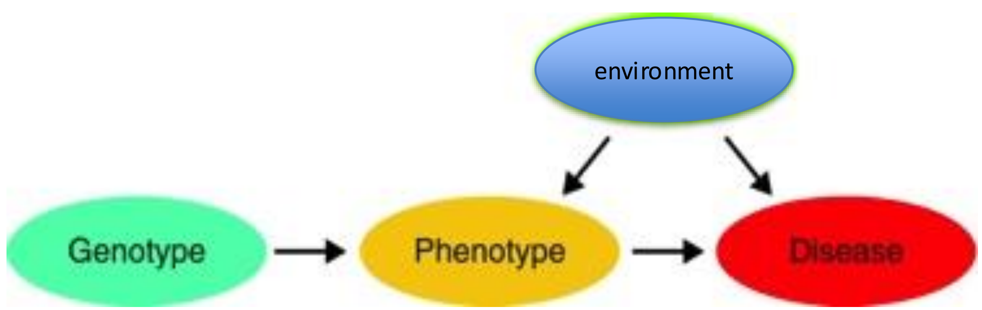




Courtesy of Angus Burns

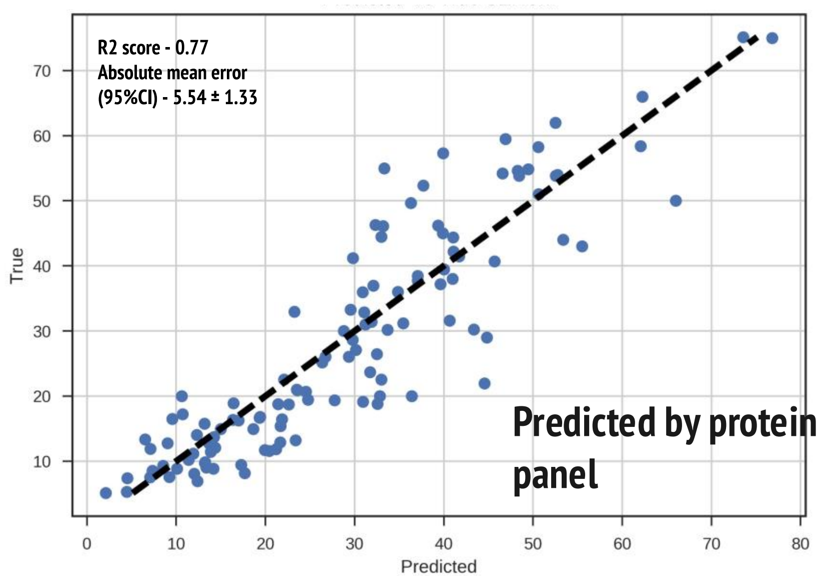
Why Use Proteomic Analysis?

- Proteins are modified by both genes and the environment
- They are closer to physiology
- They are potential drug targets; can be modulated/modified
- With genetic analysis, and a process called mendelian randomization, it is possible to create causality pathways, and get to function

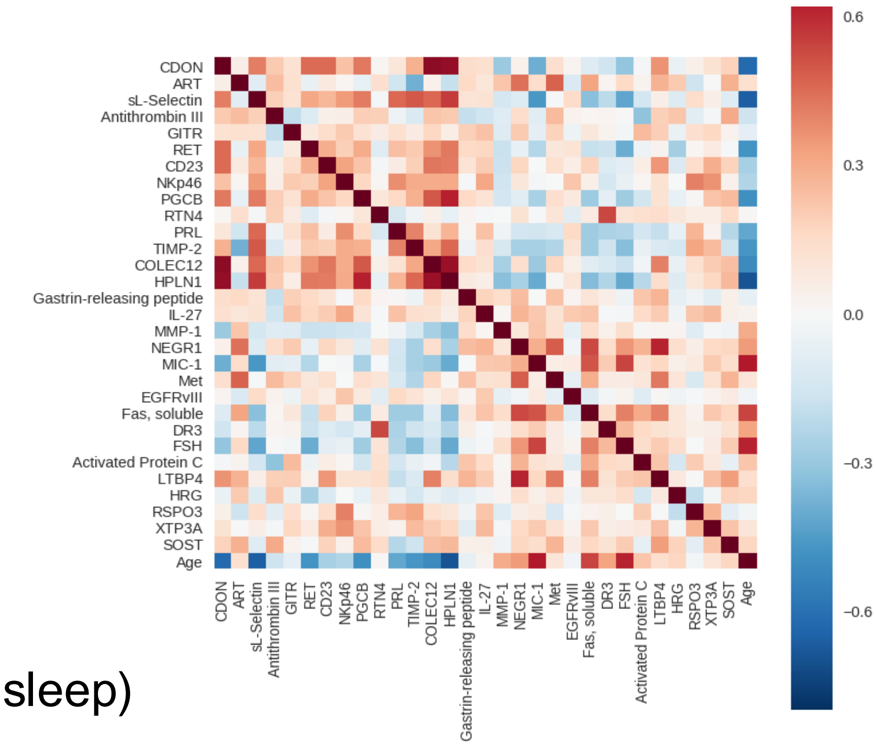


Proteomics (1.3K)

Predicted age versus actual age



Lasso model features correlation with age



Genes → Proteins → Phenotype (sleep)

Environment → Proteins

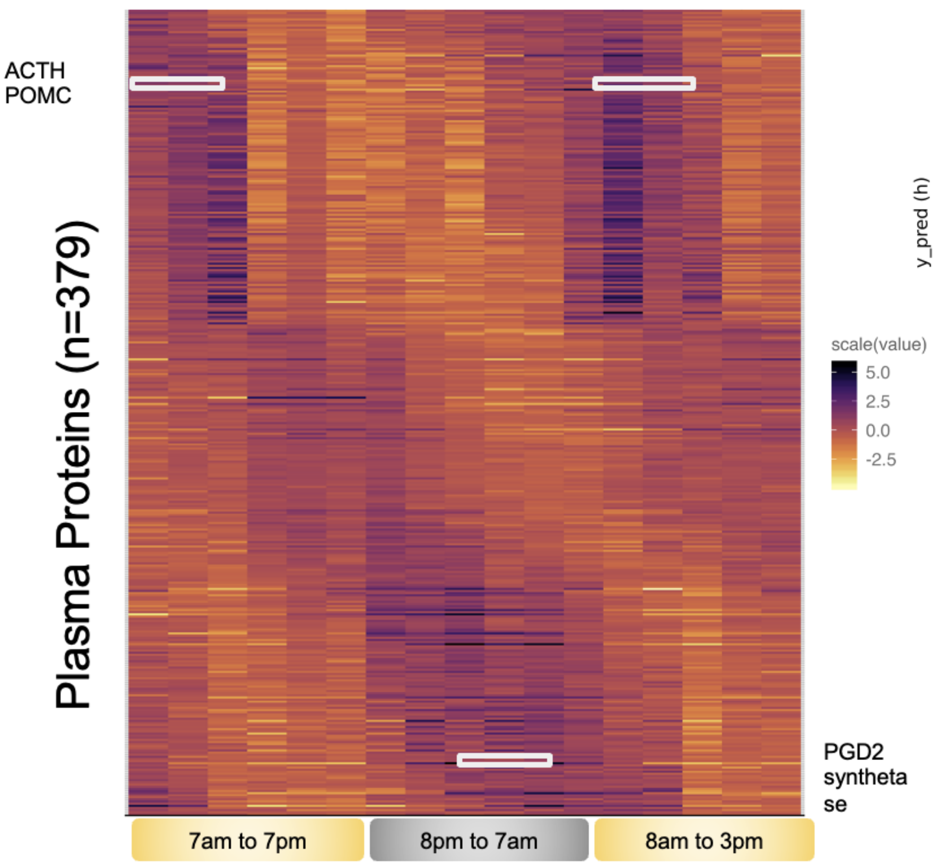
Aditya Ambati, PhD

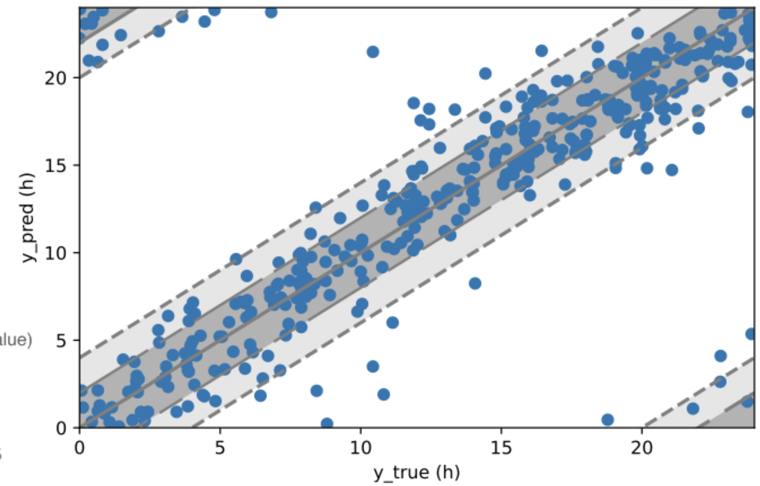
Stanford MEDICINE | Center For Sleep Sciences and Medicine

What are the drives of Wakefulness?

- Circadian phase or amplitude
  - Sleep and wake disruption (abnormal phase/amplitude)
  - REM sleep strongly circadian
  - Circadian disruption is associated with sleep deprivation
- Insufficient sleep promotion
  - Sleep deprivation profile (sleep deprivation proteins)
  - Sleepiness due to an inability to sleep enough
  - Positive effect of GHB?
- Insufficient wake promotion
  - Sleep excess profile (sleep specific proteins)
  - Low wake amounts
  - Role of motivation/depression
- Profile likely mixed in many cases

Proteomics (5.5K): Circadian phase

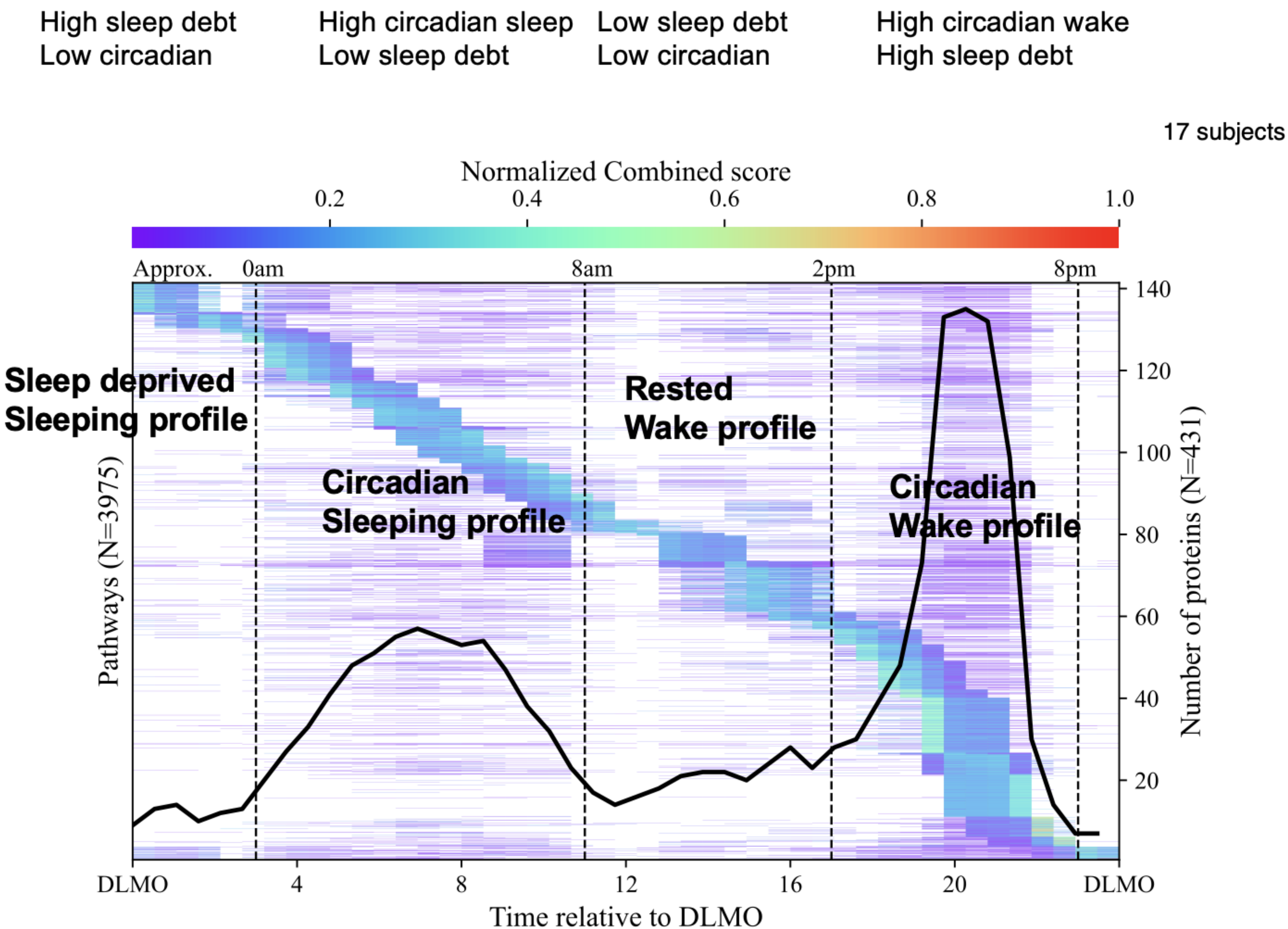




Protocols H - Heparin / E - EDTA Plasma * expected within a year	# Samples (Subjects)
28h-Forced desynchrony	179 (6) H
36h-Constant routine	338 (17) E
Chronic sleep restriction	149 (8) H
Control condition	47 (7) H
Control condition	103 (8) E
Recovery	46 (7) E
Modified constant routine	310 (103) E
Insomnia cases	35 (35) E
Various clinical patients	20 (9) E
Shift workers	30 (20) * E
Total	1236 (183)

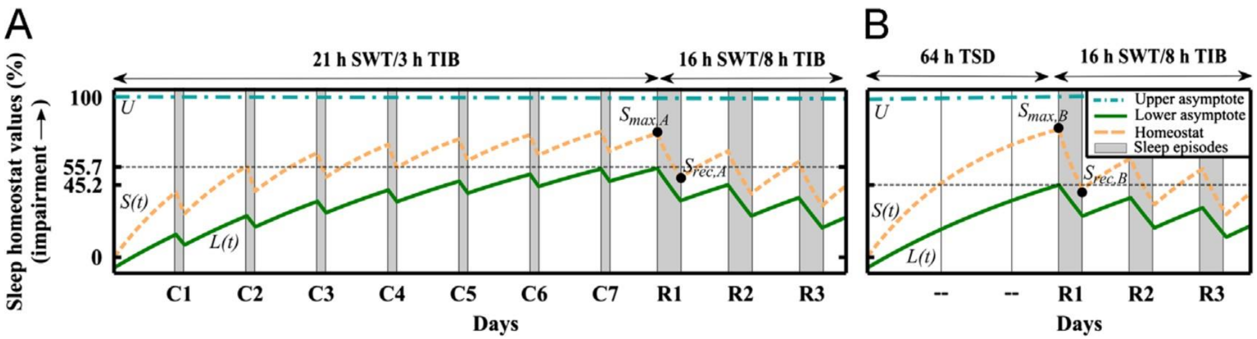
Median Absolute Error (MdAE) ~1.4h vs DLMO, with 16% of subjects having a mean error > 3h and 7% > 4h (571 samples in 75 subjects)

Other possibilities: single cell expression (body time) (Achim Kramer)



High sleep debt
Low circadian
High circadian sleep
Low sleep debt
Low sleep debt
Low circadian
High circadian wake
High sleep debt
17 subjects
Normalized Combined score
0.2
0.4
0.6
0.8
1.0
Approx.
0am
8am
2pm
8pm
Sleep deprived
Sleeping profile
Circadian
Sleeping profile
Rested
Wake profile
Circadian
Wake profile
Pathways (N=3975)
Number of proteins (N=431)
140
120
100
80
60
40
20
DLMO
4
8
12
16
20
DLMO
Time relative to DLMO

Unified model of chronic and acute sleep



02/14/38/29

Potential implications

- Incorporates sleep debt into the classical two-process model
- Adaptive capacity to recover
- Models both CSR and TSD
- Adds additional parameters

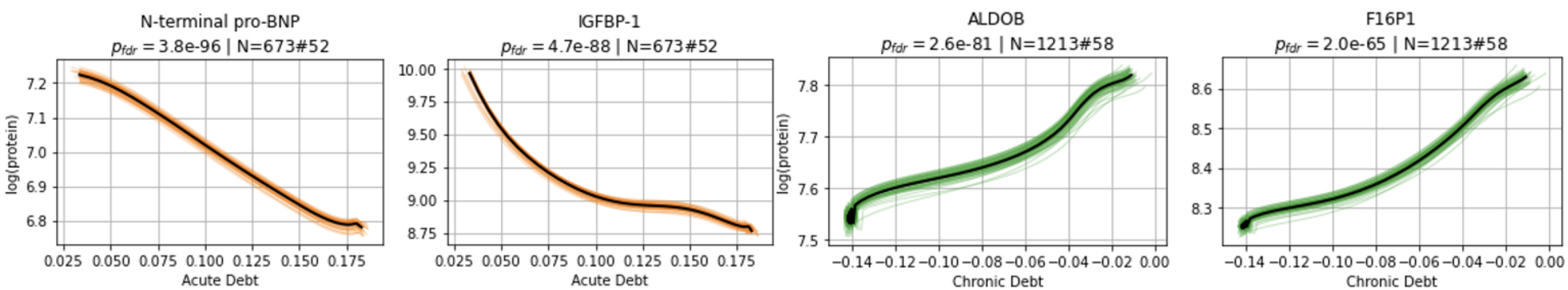
Sleep Biomarkers results

Sleep debt effect on proteins within subject

For each protein, we fit

$$log(protein) \sim debt + (1|subject)$$

where $debt$ represents either chronic or acute sleep debt from the unified model.



1348
(18%)

775
(11%)

2658
(38%)

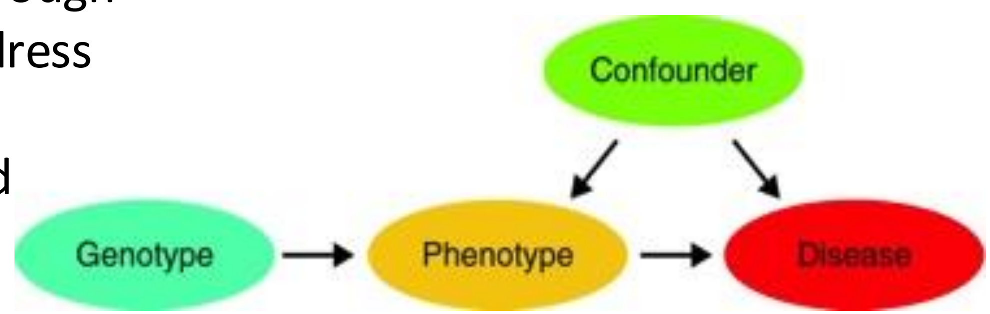
Proteomics goals

Biomarkers

- Find a panel of proteins that predicts circadian phase using a single blood sample (time stamp, see Agostinelli et al., Bioinformatics, 2016)
- Find a panel of proteins that predicts sleep debt using a single blood sample
- Find panels of proteins that predicts sleep apnea (hypoxia), and Periodic Leg Movements during sleep using the STAGES and WISC studies

Basic biology

- Link genetics to proteinomics through mendelian randomization to address causality
- Study basic function of identified gene-protein pathways



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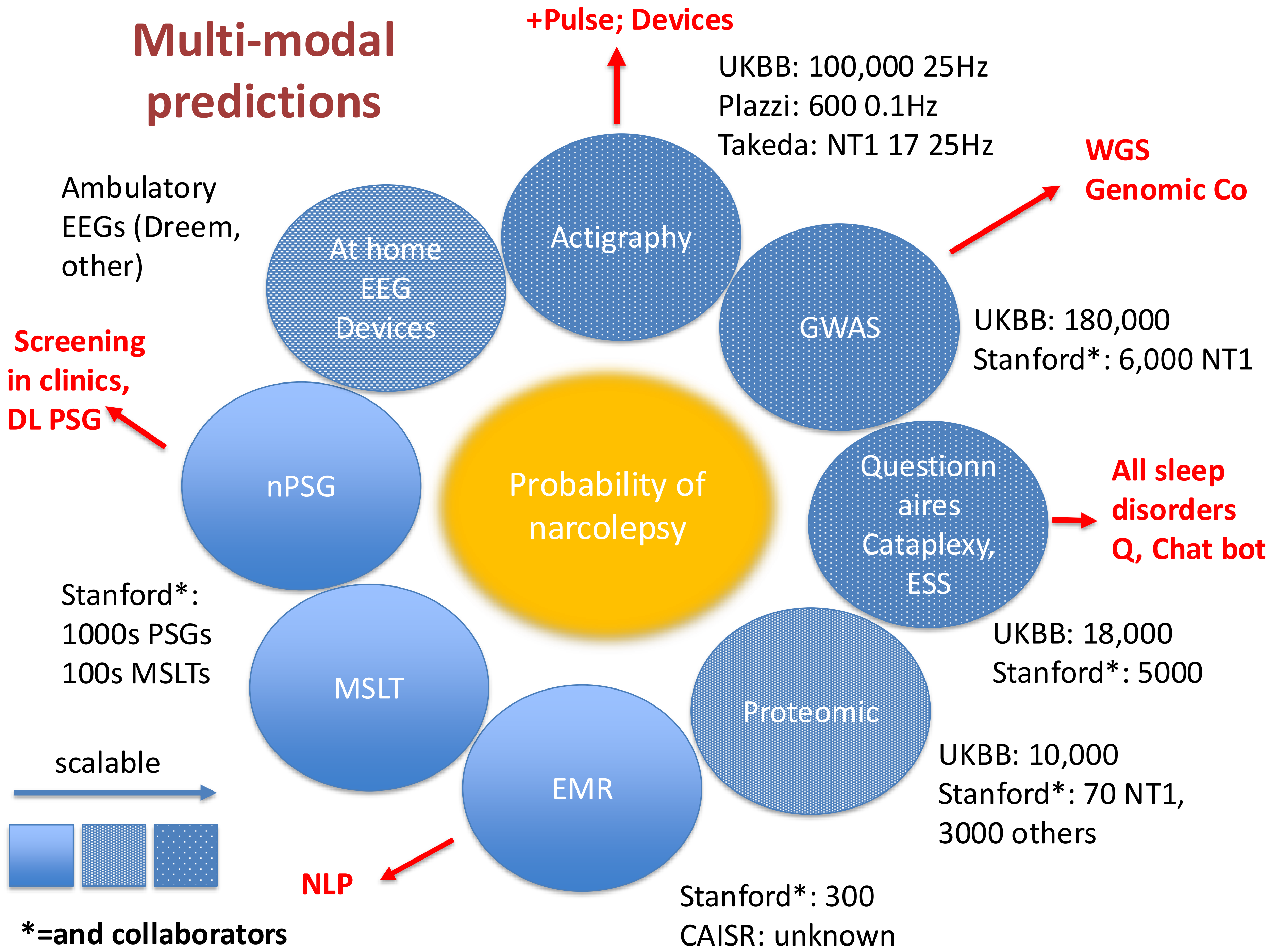
Multi-modal predictions



Simplified Sleep Devices without EEG

<u>Non- contact sensors</u>

- Smart beds, Bedits etc
- Radio-frequency sensor
- Sound/Snore Apps
- Video processing

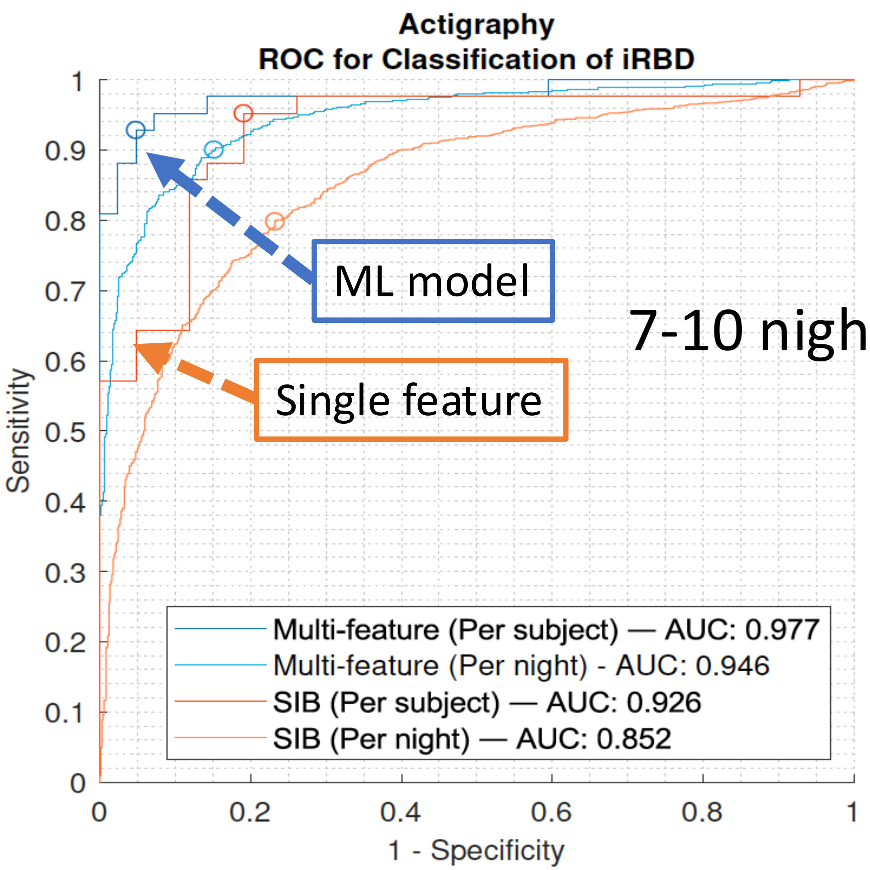
<u>Contact sensors</u>

- Accelerometers
- Pulse measurements
- ECGs, EEG
- Oxygen saturation

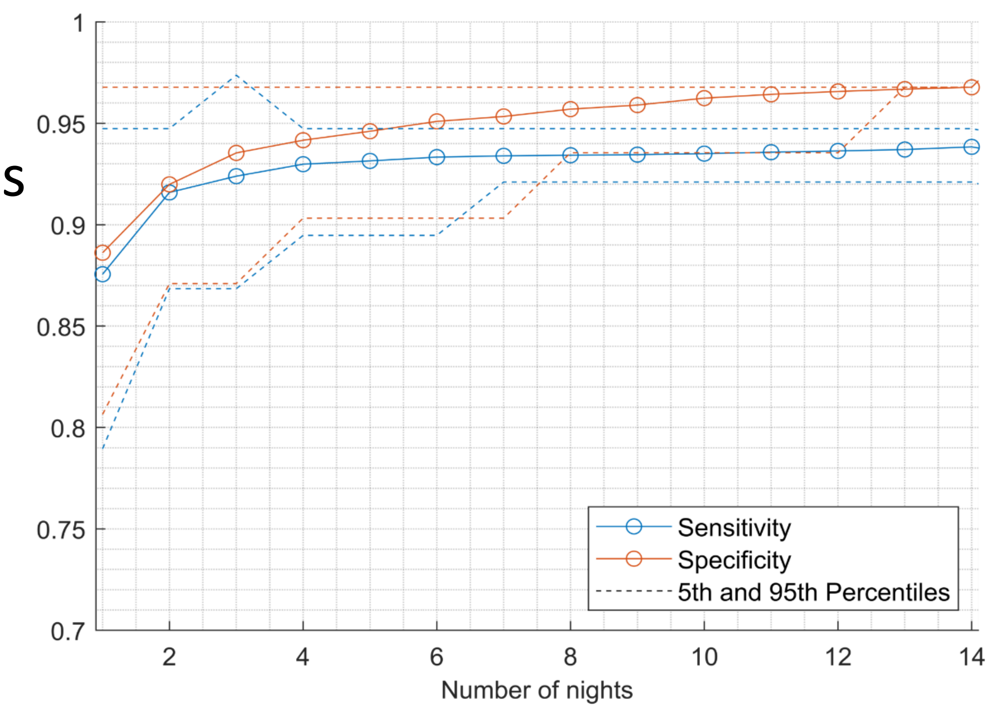
>Rings, Watches, Actigraphs

Performance Actigraphy in REM Behavior Disorder

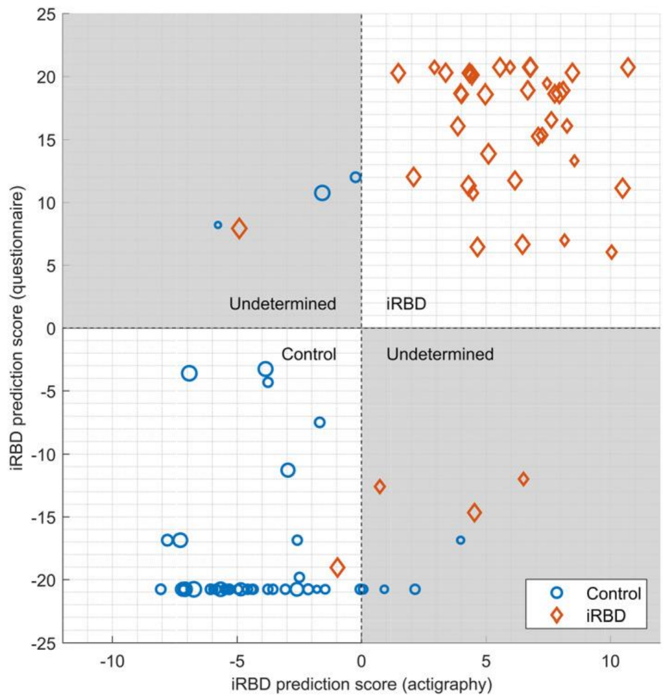
	Sensitivity	Specificity	Accuracy	Precision
ACTIGRAPHY ML	0.95 (.88 - .99)	0.90 (.82 - .96)	0.93	0.91



7-10 nights



REM sleep Behavior disorder



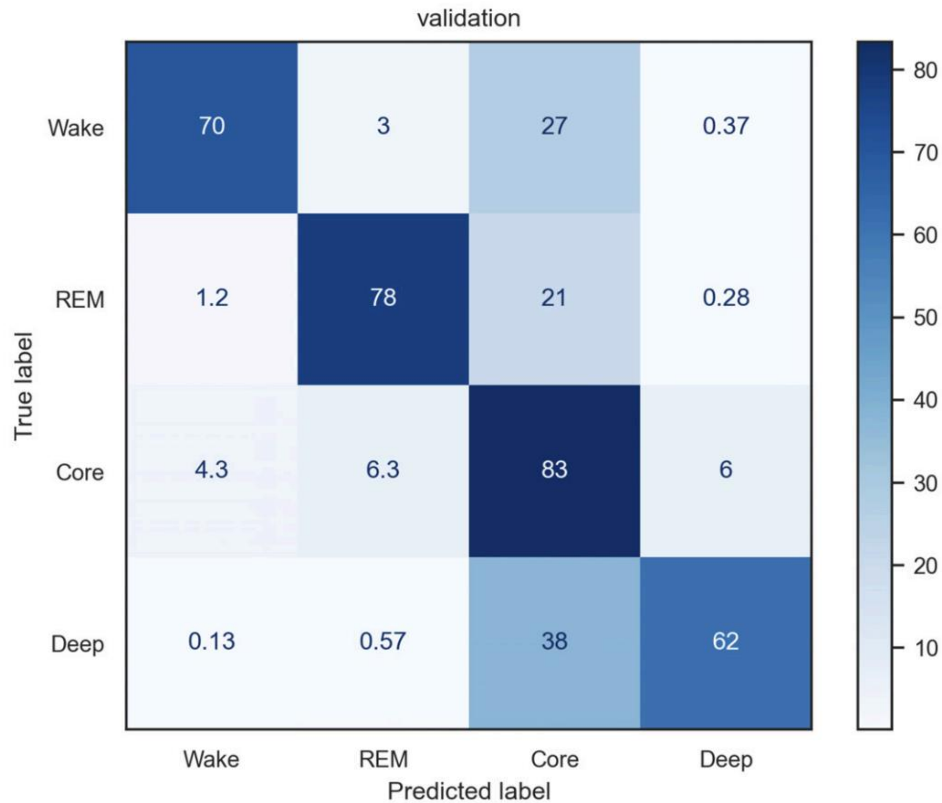
- 7-night actigraphy, RBD Innsbruck inventory, hyposmia, constipation, and orthostatic dizziness
- 42 iRBD vs 42 non-RBD:
  - ✓ Specificity/precision=100% (95.7–100.0);
  - ✓ Sensitivity= 88.1% (79.2–94.1)

Mov Disord. 2023 Jan; 38(1): 82–91.

Ambulatory Detection of Isolated Rapid-Eye-Movement Sleep Behavior Disorder Combining Actigraphy and Questionnaire

Andreas Brink-Kjaer, PhD,[1,2,3] Niraj Gupta, BSc,[3] Eric Marin, MD,[4] Jennifer Zitser, MD,[4,5] Oliver Sum-Ping, MD,[4] Anahid Hekmat, MD,[4] Flavia Bueno, MD,[3] Ana Cahuas, BSc,[3] James Langston, MD,[6,7] Poul Jennum, MD,[2] Helge B.D. Sorensen, PhD,[1] Emmanuel Mignot, MD, PhD,[3] and Emmanuel During, MD[4,7,8*]

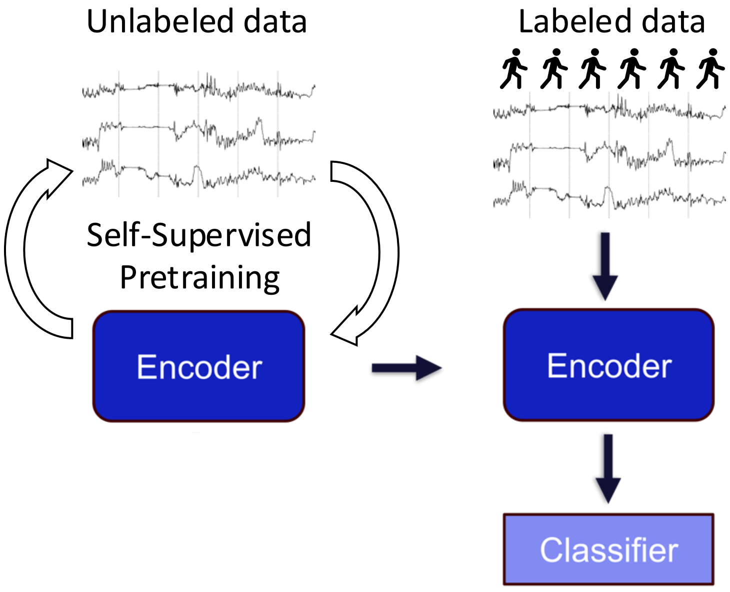
ACTIGRAPHY: Apple Watch sleep stages
Training: 858, Validation: 166
(September 23, 2023)



4 stage Kappa= 0.55 (0.65 to 0.57); 97.9% sensitivity and 72.5 % percent specificity

Optimizing Self-Supervised Pretraining for Accelerometer-based Activity Recognition

- Supervised learning is limited by labeled data.
- Self-supervised pretraining allows learning from large unlabeled datasets.
- Our focus is how to do this effectively for accelerometer data.
- Our main hypothesis is that sequence learning during pretraining will improve activity recognition performance.

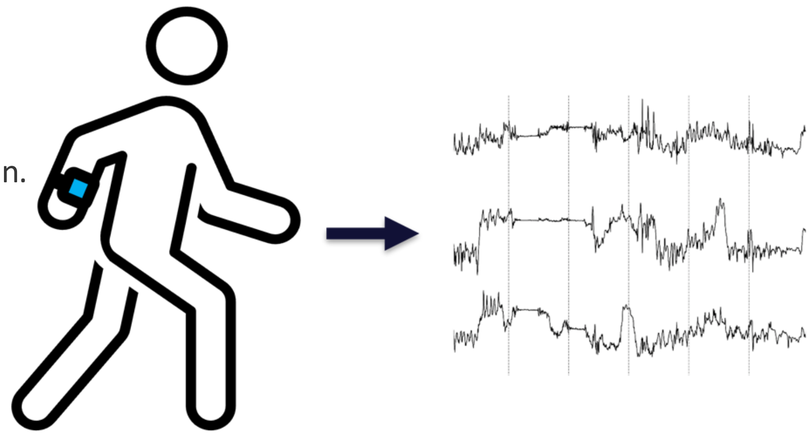


Data Description

- Tri-axial wrist-worn accelerometry (Axivity AX3).
- 100 Hz resampled to 30 Hz.

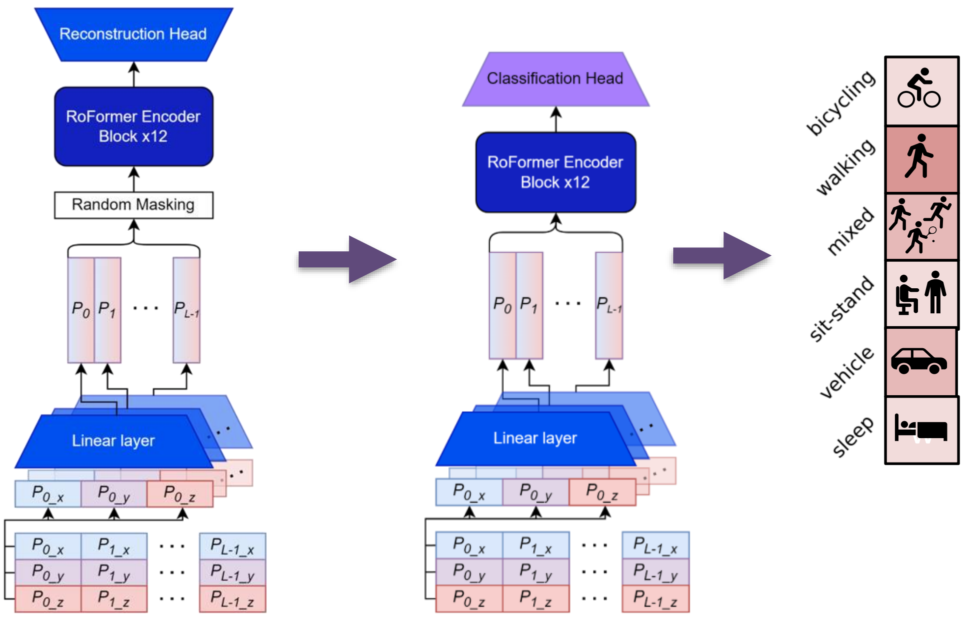
- UK Biobank
  - 103,618 participants.
  - 115,390 recordings, 108,933 after exclusion.
  - ~7 days of data pr. recording.
  - Unannotated.
  - 90-10 train-validation split.

- Capture24
  - 151 participants, 149 after exclusion.
  - ~ 24 hrs. of data pr. participant.
  - Body-cam and sleep diary-based annotations.
  - 80-20 train-test split, 5-fold CV on train set.

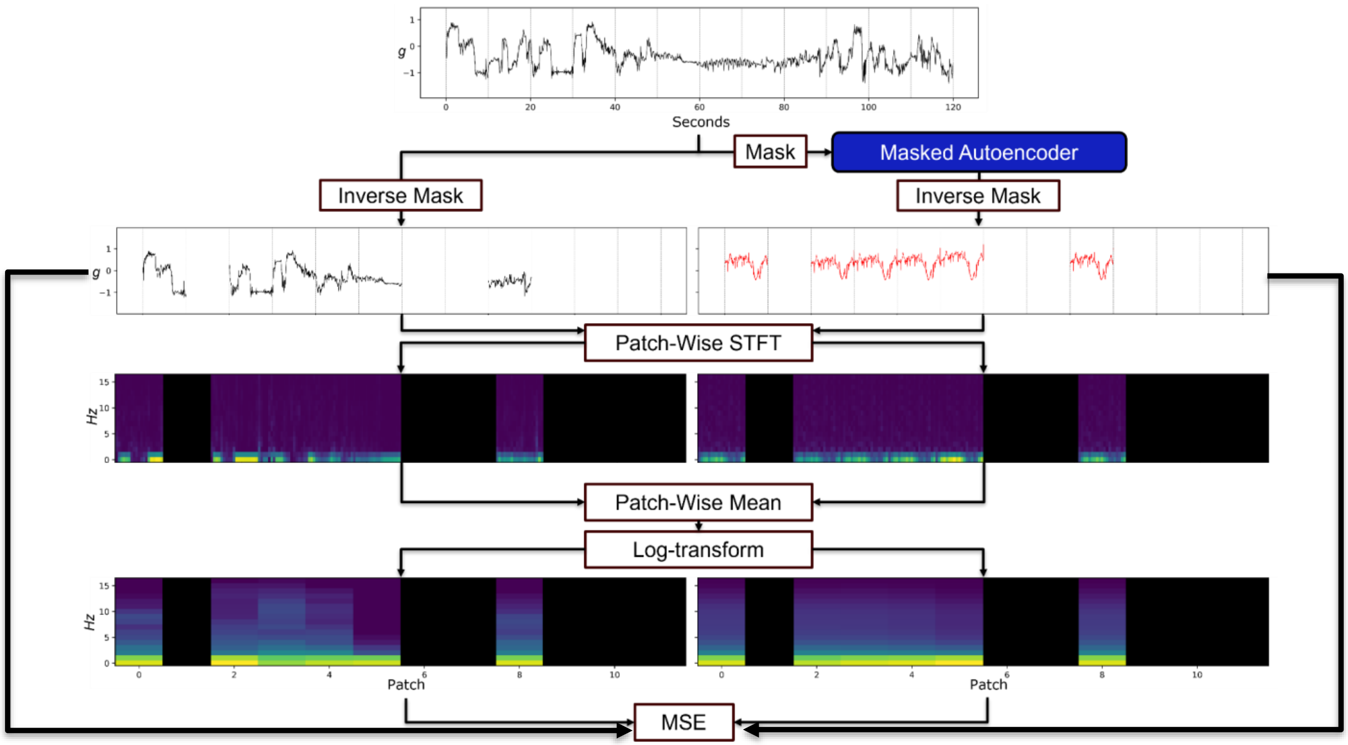


Masked Autoencoder (MAE) Pretraining and Finetuning Architectures

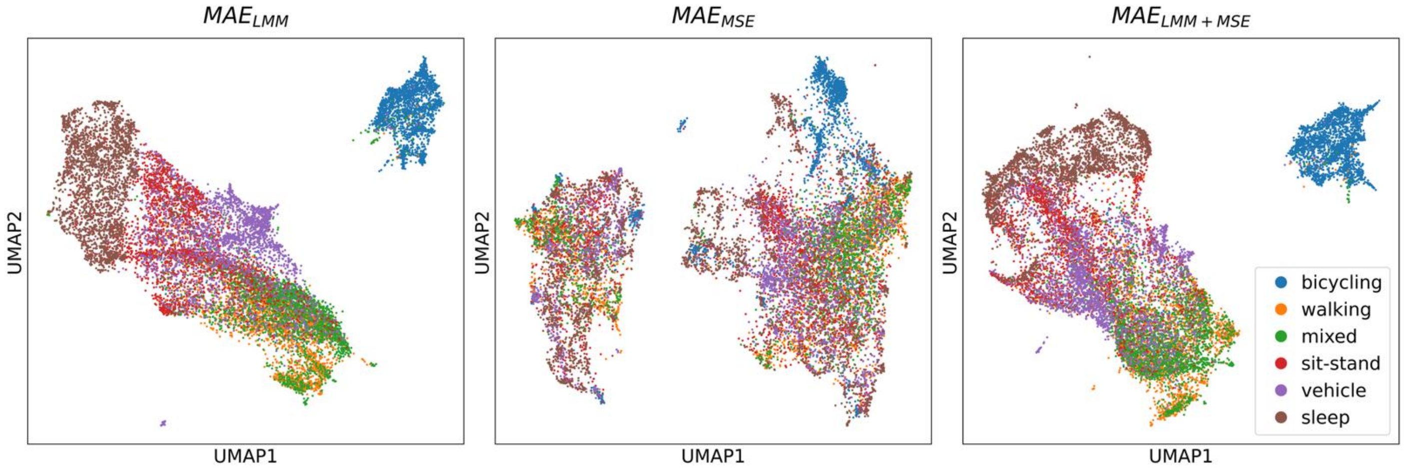
- 300 10-s patches (50 m).
- Pretraining using a Masked Autoencoder (MAE).
- Transfer encoder and linear embedding layer.
- Train a linear classifier on encoder outputs.



The Log-scale Mean Magnitude (LMM) Loss vs. the Mean Squared Error (MSE) Loss

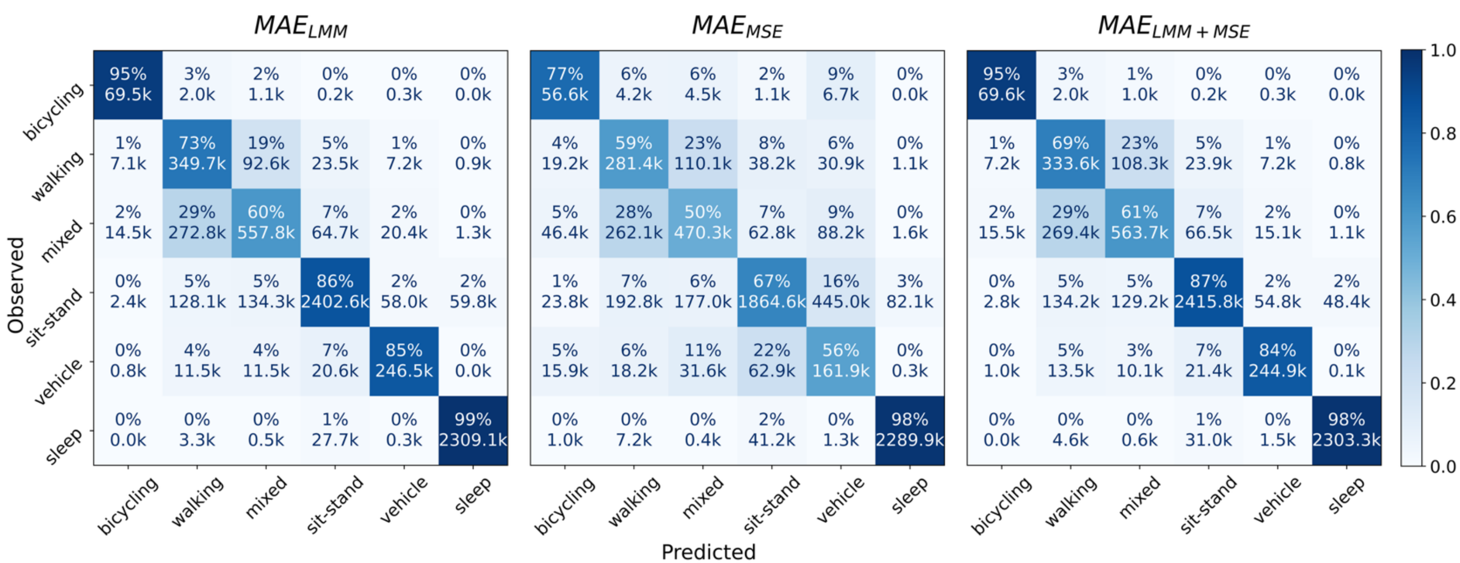


Visualizing Activities in the Latent Spaces of Encoders Pretrained with Different Losses



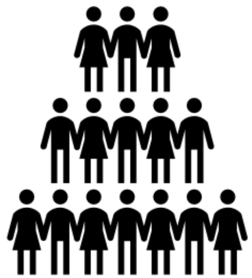
- The LMM-only model had a clearly structured latent space compared to the MSE-only model.
- No clear difference between the LMM-only model and the mixed-loss model.

Class-wise Confusions for Models Pretrained with Different Losses

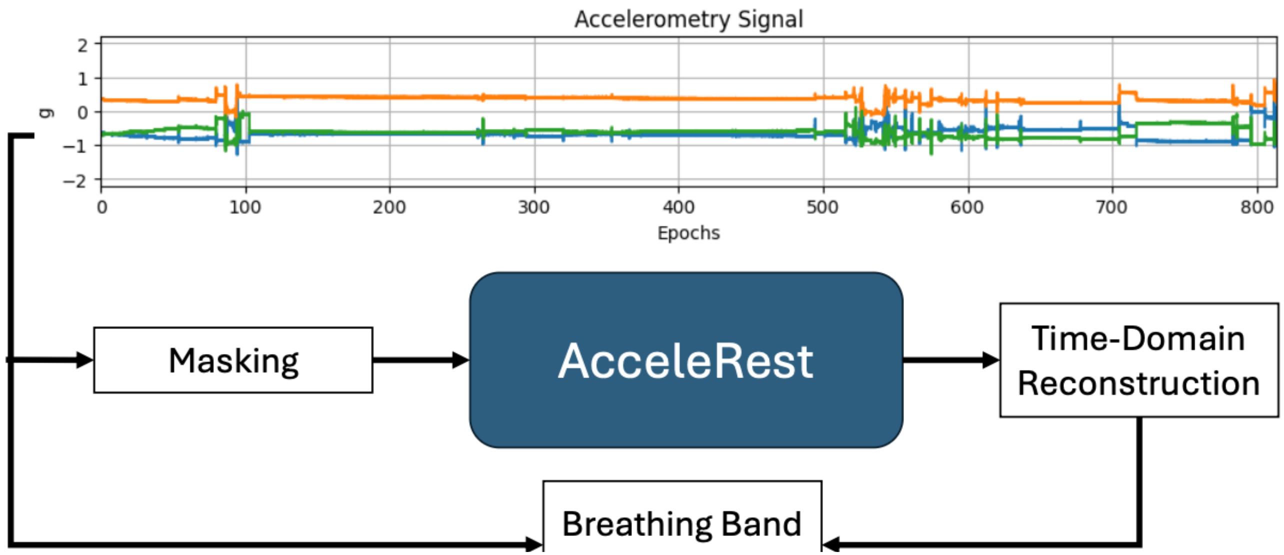


- The LMM-only model achieved better performance compared to the MSE-only model for all classes except sleep.
- Again, no clear difference between the LMM-only and the mixed-loss model.

Sleep stage detection



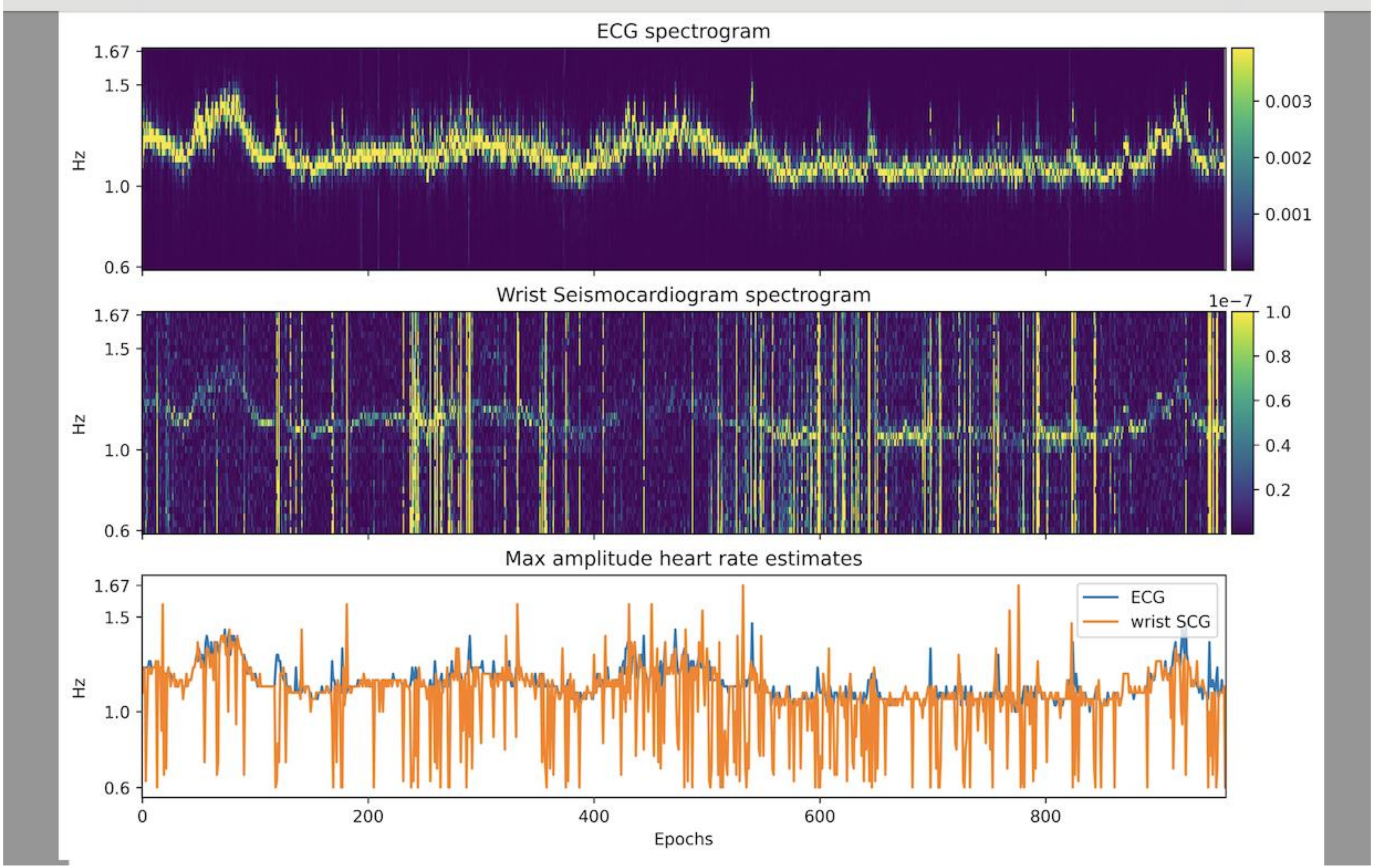
700,000 hours of free-living wrist-accelerometry recorded with **Axivity Ax3** from the UK Biobank was used for breathing-band focused masked reconstruction.



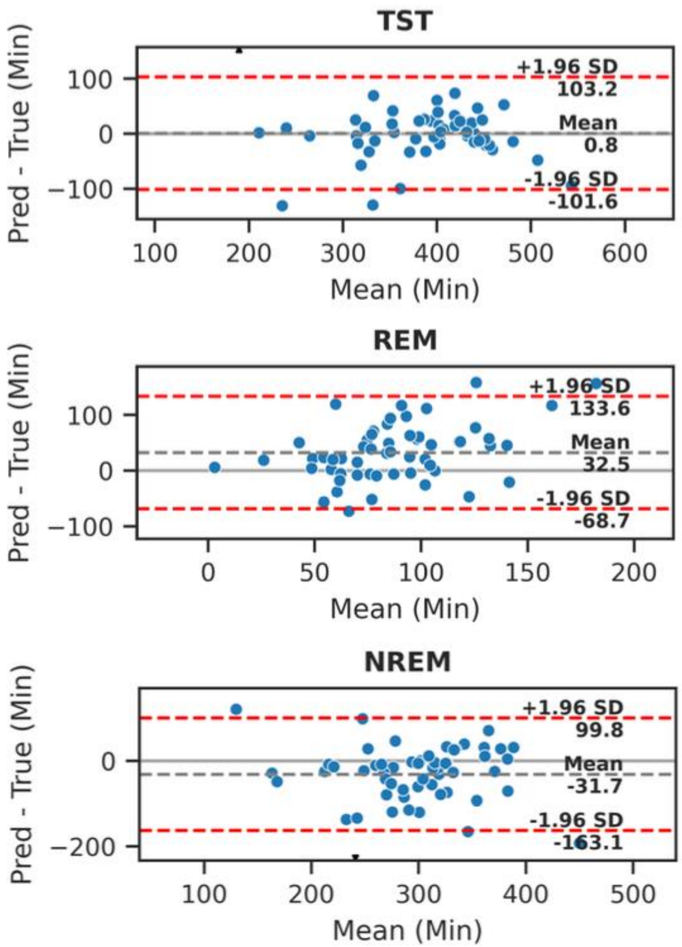
	Training Data				Test Data	
Device	GT3X	Empatica E4	Amazfit Arc	GENEActiv	Amazfit Health	Apple Watch
N	238	98	32	27 (53)*	35	21*

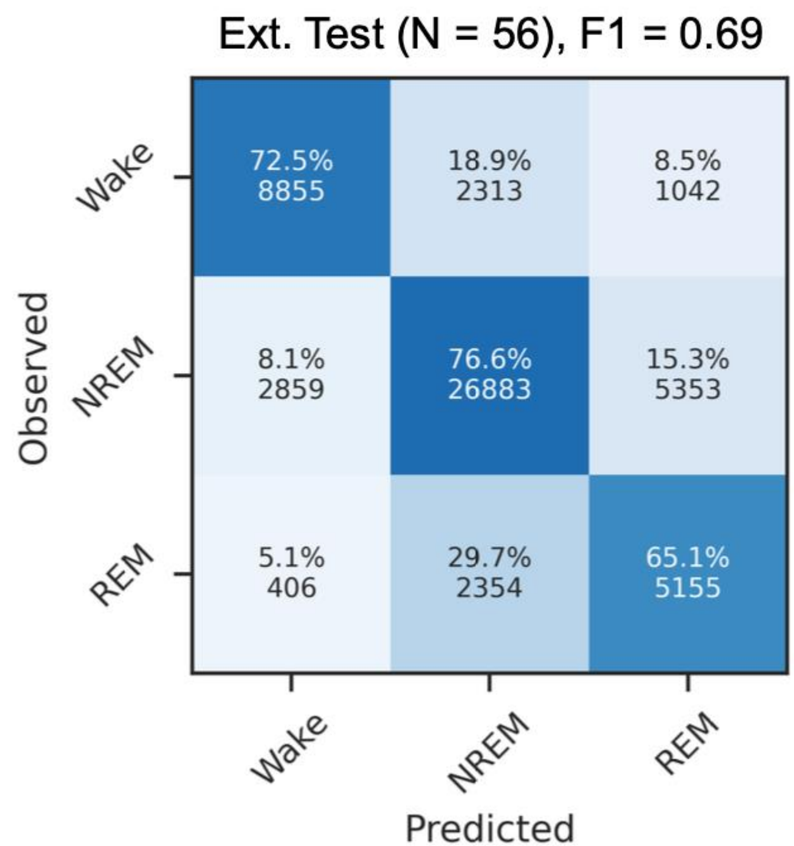
* Datasets did not have apnea annotations.

Breathing detection

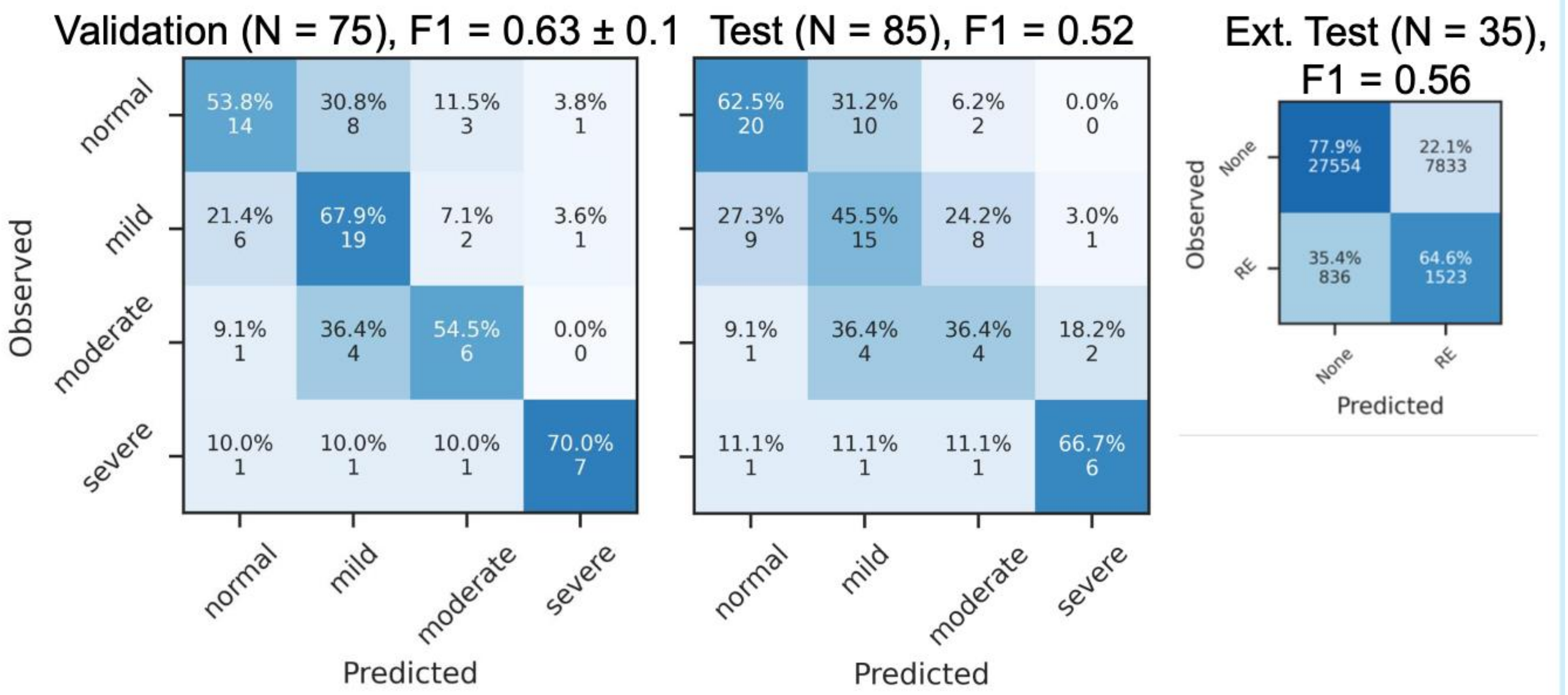


Sleep staging





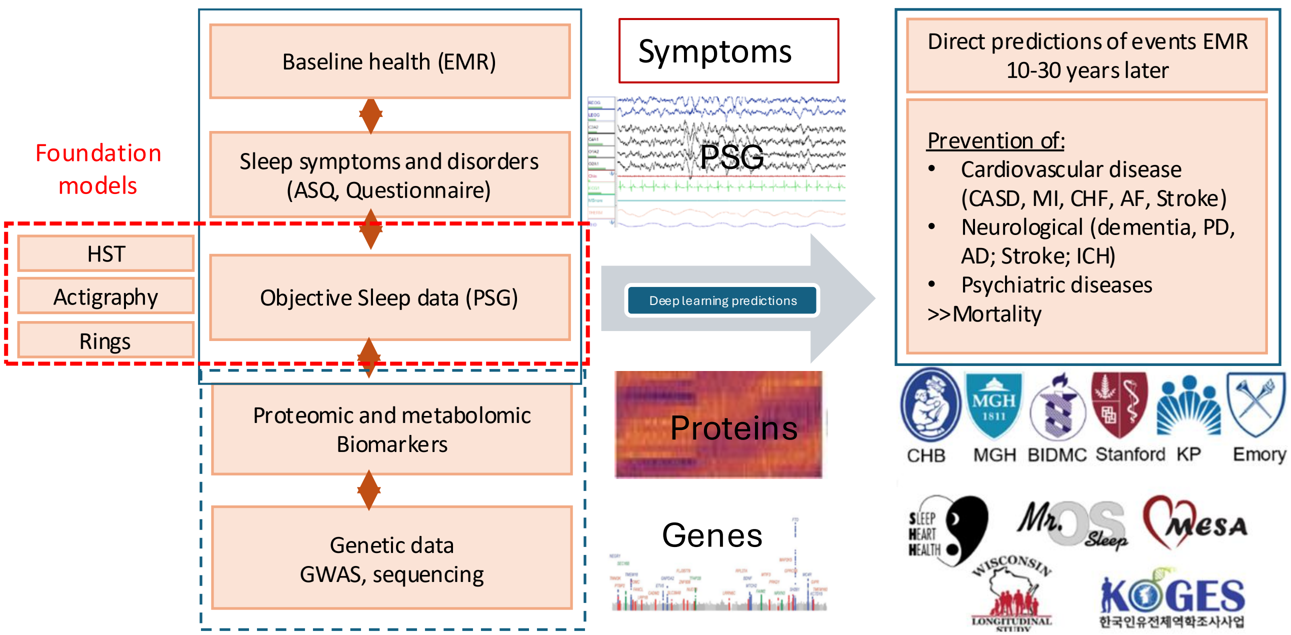
Detection of severe SDB



Foundation models

- SleepFM (Thapa et al., Nature Medicine, 2026)
  - Contrastive learning >Masked autoencoders
  - Does well to at basic tasks (scoring sleep, AHI etc)
  - Predicts future diseases
- AcceleRest (Lorenzen et al. Annu Int Conf IEEE Eng Med Biol Soc 2025; MedArch 2026)
  - Physiology-aware masked autoencoders to capture pulse- and respiration-related motion
  - Predicts basic behaviors of walking, running, cycling, sitting/standing, sleep/rest very well
  - Predicts 4-class OSA severity F1=0.67
  - Predicts 3-class wake-NREM-REM scoring F1=0.65

Omics and technology predicting health in 250-500,000 subjects



Brink-Kjaer et al., *Nature Digital medicine, 2022*
Thapa et al., *Nature Medicine, 2026*

HST=home sleep tests

NIH
HL161253

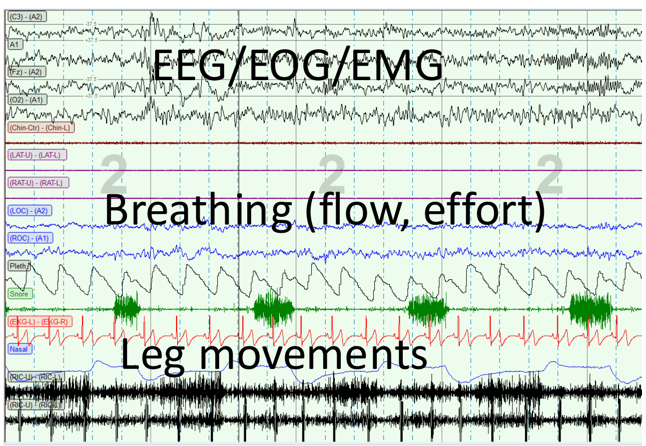
Need new sleep monitoring system

Now

Material science
Chemical engineering
System integration

Sleep testing
(daytime, nighttime,
in natural environment)

Future



Sleep stages (insomnia
Hypersomnia)

AHI (sleep apnea)

PLMI (RLS)

- EEG, sound, actigraphy
- New skin-like material
- Miniaturization
- Wireless transmission
- Automatic analysis/machine learning
- Monitoring response

At home polysomography EEG

- Medical oriented: Nox A1, Somnomedic HST etc
  - ✓ Need wireless
- Dreem (helmet), Nextsense (earbud), Xtrodes
  - ✓ Usually not a true PSG: lack leg movement etc
  - ✓ Issues with comfortability remain
  - ✓ Use during wakefulness unvalidated
- Need to merge devices for modalities
  - ✓ for example, leg actigraphy, sound and stretch sensor for breathing, O2 saturation with ring

CBT–i Coach

By US Department of Veterans Affairs (VA)

Open iTunes to buy and download apps.

View More by This Developer

The future: at home therapies if there is reimbursement: death by absurdity

Description

CBT–i Coach is for people who are engaged in Cognitive Behavioral Therapy for Insomnia with a health provider, or who have experienced symptoms of insomnia and would like to improve their sleep habits. The app will guide users through the process of learning about sleep, developing positive sleep routines, and improving their sleep

US Department of Veterans Affairs (VA) Web Site ▸ CBT–i Coach Support ▸ Application License Agreement ▸ ...More

CBT-i

View in iTunes

Free

Category: Health & Fitness
Released: Jun 05, 2013
Version: 1.0
Size: 33.0 MB
Language: English
Seller: US Department of Veterans Affairs (VA)
© US Department of Veterans Affairs
Rated 4+

Compatibility: Requires iOS 4.3 or later. Compatible with iPhone, iPad, and iPod touch. This app is optimized for iPhone 5.

Customer Ratings

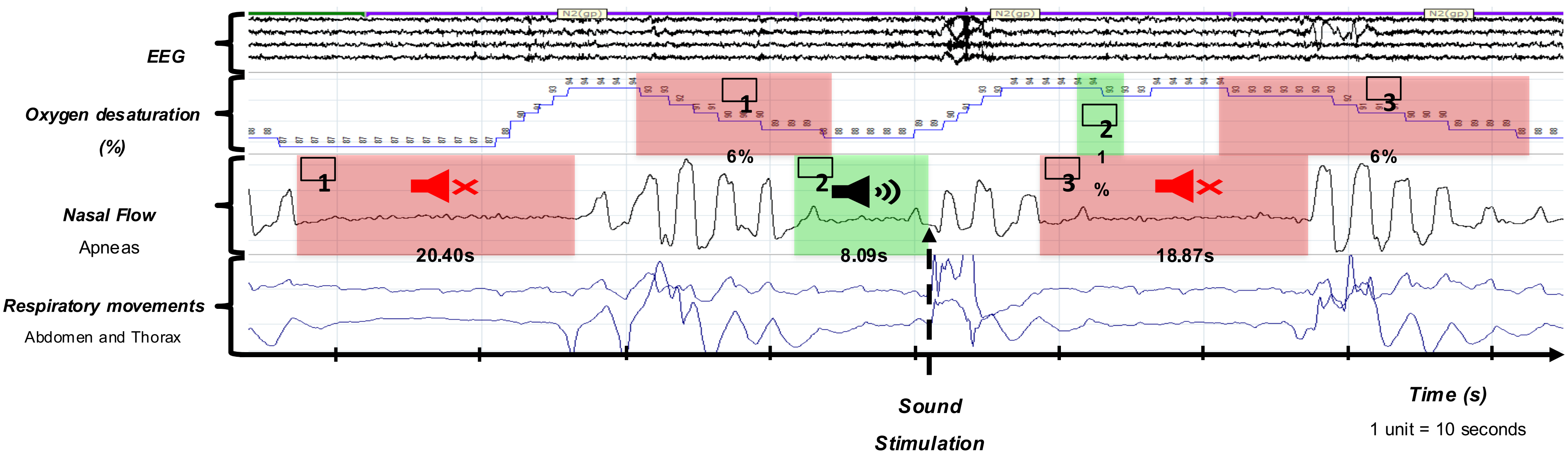
Current Version:
★★★★½ 12 Ratings

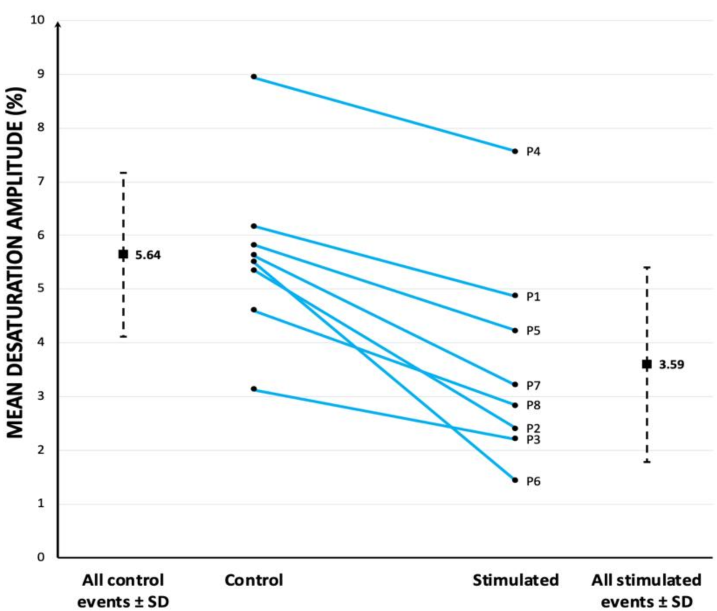
More iPhone Apps by US Department of Veterans Affairs (VA)

PTSD Coach

iPhone Screenshot

Real time interventions





Decreased desaturations
No effect on subjective daytime sleepiness

Waeber et al. Sleep Med. 2021 Feb;78:38-42.

Stanford MEDICINE
Mignot lab
http:/mignotlab.com

(when) will we become all obsolete?

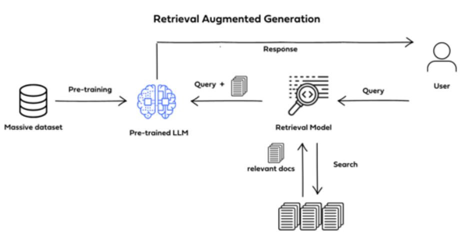
Clinical evaluation

Testing

Treatments

CPAP-Doctors
cBTI-Psychologists
Drugs--Doctors

Chat-Bot LLM
RAG augmented



Devices+ Artificial Intelligence

HST

Biochemical tests

Agentic AI

Chat-Bot LLM
Auto CPAP
Online CBTi
MA prescribing
Device for surveilling efficacy

What can we achieve?

- Extend SleepFM, integrate with AcceleRest, add high resolution ring foundation models as they are integral to sleep apnea, which cannot be ignored
- Integrate Proteomics and Genetic markers in these models; health and molecular pathways
- Design and use novel minimalist wearable devices at home to monitor sleep and brain health, allowing longitudinal data, which will be more predictive
- Finally, use automatic at home feedback to improve outcomes through intervention studies

"Sleep belongs to the home not to a laboratory"

What will we achieve?

- **Molecular components generating sleep at the core of sleep disorders**
- **Blood biomarkers of sleep disorders, sleep debt and internal circadian clock**
- **Automatic, non-human based analysis of sleep signals**
- **Novel minimalist wearable device used at home to monitor sleep and brain health**
- **Need basic research in animal models for functional testing to understand function**

“Sleep belongs to the home not to a laboratory”

Participants to work presented

Genetic/immunology:

- Eric Yu

Deep learning PSG students:

- Niels Lorensen PhD student
- Mads Olsen, Post Doc/Takeda
- Andreas Brink-Kjaer PhD Assistant Prof
- Umear Hanif, MS
- Dmtri Volson, Takeda

Proteomics:

- Adrien Specht, PhD student
- David Benacom, MD, PhD student

Main Collaborators

- Andreas Brink-Kjaer PhD (Danmarks Tekniske Universitet, Denmark)
- Poul J. Jennum MD (Rigshospitalet Glostrup, Denmark)
- Chuck Czeisler MD and Jeanne Duffy PhD (Harvard University)

mignot@stanford.edu

Website: Mignotlab.com