CNS 2011

Twentieth Annual Computational Neuroscience Meeting

July 23-28
Stockholm
Sweden
www.cnsorg.org/2011

KTH VETENSKAP OCH KONST

Stockholms Universitet
Stockholm University

NORDITA

incf International Neuroinformatics Coordinating Facility

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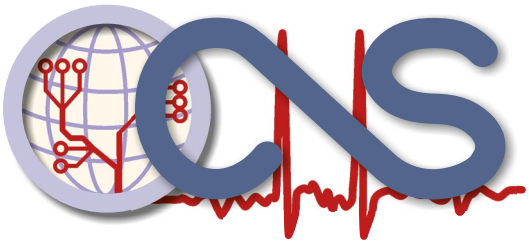


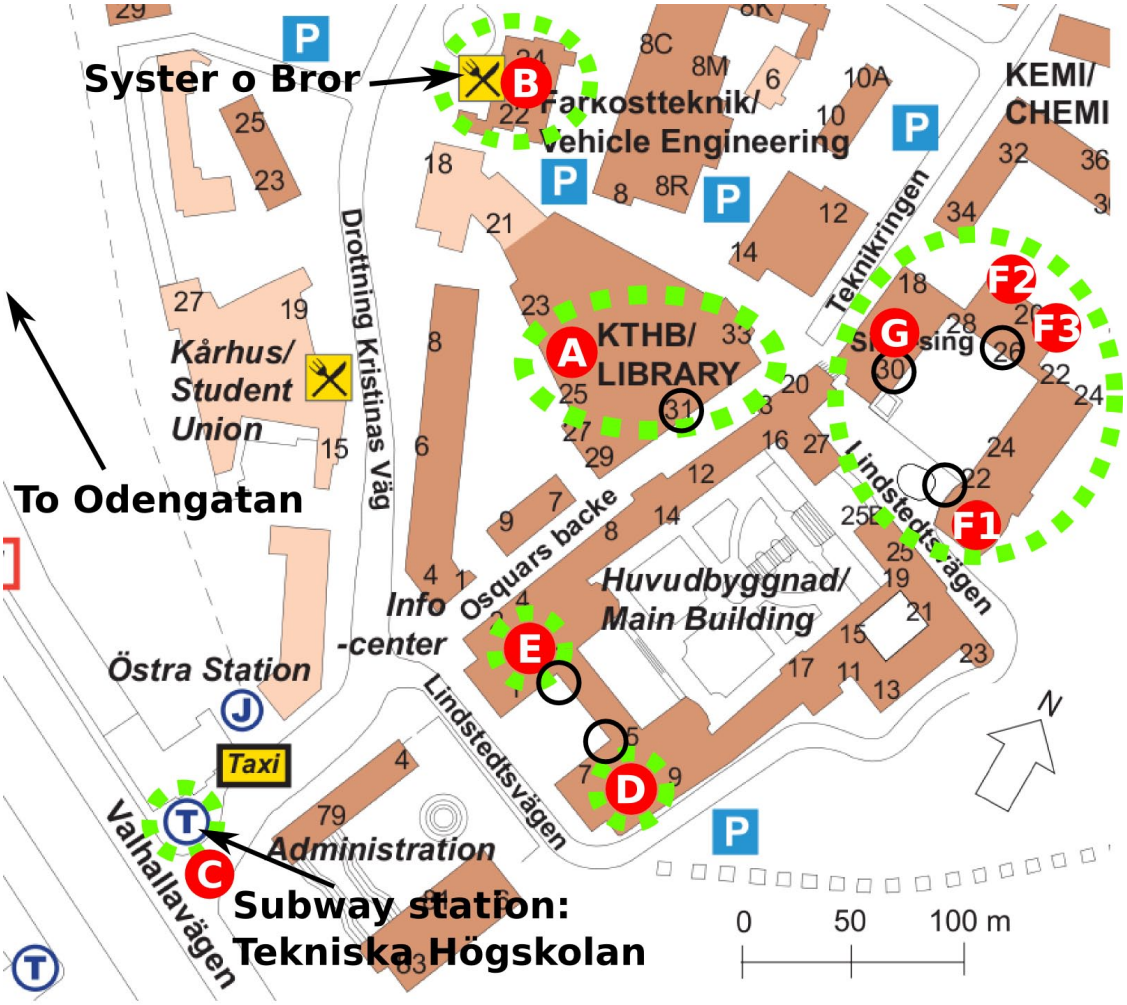
Organization for Computational Neurosciences

National Science Foundation

KTH Royal Institute of Technology

The local organizers gratefully acknowledge funding from the Swedish Research Council and the Stockholm City Council.

»Local info



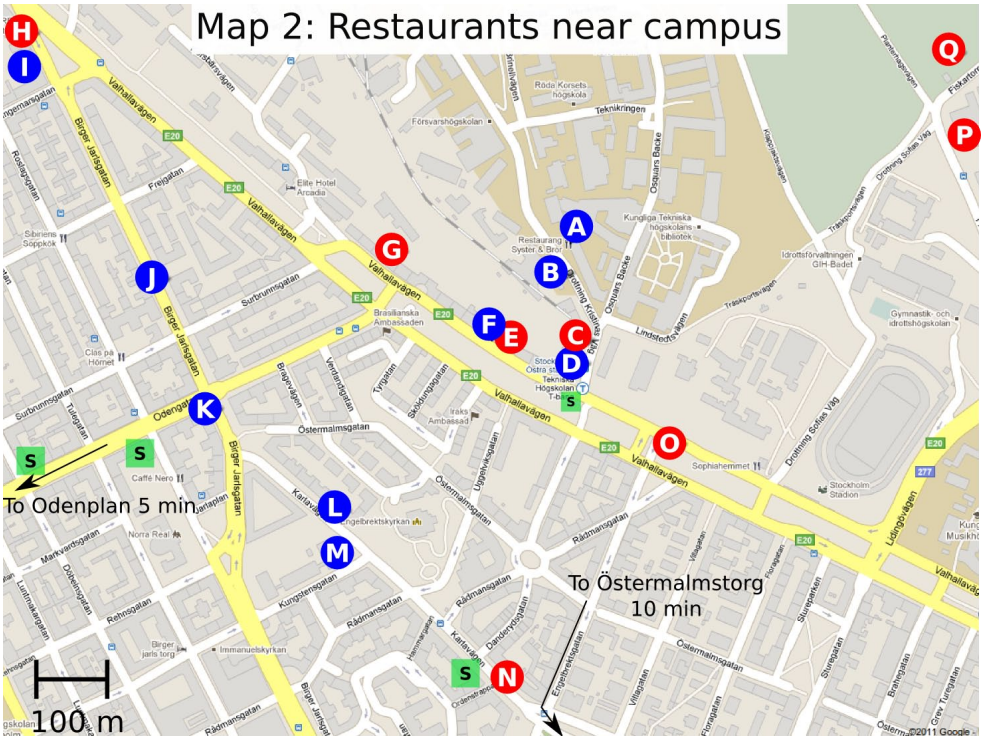
Map 1: Meeting locations

A location of interest

building entrance

T subway entrance

What?	Where?
Opening reception	(A) KTH library
Oral sessions	(F1)
Registration	(E) July 23 09.00-16.00, (A) July 23 17.00-21.00, (F1) July 24-26, (E) July 27-28
Coffee break	(G)+(F2) main meeting oral sessions, (E)+(D) tutorials + poster sessions + workshops
Exhibits	(F2)+(F3)
Tutorials	(E)+(D); rooms E1-E3, E51-E53, D1-3
Poster sessions	(E)+(D); rooms E31-E35, D31-D35
Workshops	(E)+(D); rooms E1-E3, E36, E51-E53, D1-3; seminar rooms 1439, 1537, 1625, 1635, 4523
OCNS Party	(B) Restaurant Syster&Bror
OCNS Business meetings	(E); seminar rooms (1439, 1537, 1635)
Subway exit	(C) or (T)



Map 2: Restaurants nearby

A restaurant open all days

A restaurant open weekdays

S supermarket (open all days)

All the restaurants seen on Map 2 can be reached within a 15 min walk. Restaurants marked in red are open every day, restaurants marked in blue are not open on Saturday (except for (B)) and not on Sunday. Supermarkets are open every day in Sweden. If you just want to grab a sandwich or salad and then relax in Humlegården (near (N)), you can do that in the cafe at *Östra station* or one of the supermarkets, the closest supermarket is in the subway station. More restaurants (Burger King, Hard Rock Cafe, ...) can be found near *Odenplan* which is approximately a 20 min walk from the conference venue, or around *Östermalmstorg* and *Stureplan*, which can be reached with the subway after a 5 min ride (or ca. 20-25 min walk).

NAME	FOOD	PRICE	OPEN ON DAYS	COMMENT
A Syster & bror	Swedish food	Average	Mon-Thu	
B La Raginella	Pizza	Average	Sat, Mon-Thu	
C Seven-eleven	Fast food	Average	Every day	
D Östra station	Swedish food	Average	Mon-Thu	
D Café at Östra station	Salads, sandwiches	Quite cheap	Every day	Small discount for CNS*2011
E Bullens	Salads, sandwiches	Quite cheap	Every day	
F Kebab kitchen	Kebab	Quite cheap	Mon-Thu	
G Dino	Pizza	Quite cheap	Every day	
H Hanoi	Vietnamese	Average	Every day	Small discount for CNS*2011
I Phi-Phi	Thai food	Average	Mon-Fri	
J Ramen kimama	Asian food	High	Mon-Thu	
K Kina dumplings	Dumplings	Average	Mon-Thu	
L Ciao-ciao Due	Pizza	Average	Mon-Thu	
M Råkultur	Sushi	High	Mon-Thu	
N Di Vino	Salads, sandwiches	High	Every day	
O Valhallagrillen	Fast food	Quite cheap	Every day	
P Jakttornet	Swedish food	Average	Every day	
Q Cafe Harpaviljongen	Swedish food	Average	Every day	
S Supermarkets	Salads, sandwiches	Average	Every day	

List of international restaurants for the evening:

African:

Abyssinia Södermalm - Södermalm, Ringvägen 105,
Tel. 08 643 99 91;
Abyssinia Vasastan - Vasastan, Vanadisvägen 20,
Tel. 08 33 08 40;
Gojo - Södermalm, Ringvägen 161 / Restiernasg.48

Central Asian:

Central asia - Kungsholmen, Fleminggatan 62B
Tel. 08 651 99 01 (Indian)
Little Pakistan - Vasastan, St. Eriksgatan 66
Tel. 08 30 56 46, Pakistanian
Restaurang Lilla Karachi - Gamla Stan, Little Nygatan 12,
Tel. 08 20 54 54 Pakistanian
Zartosht - Åkervägen 2, 19140 Sollentuna
Tel. 08 35 27 27 Persian
Vanak - Mariehamnsgatan 24-26, Akalla
Tel. 08 632 00 99 Persian
Alborz - Sjöängsvägen 19, 192 72, Sollentuna
Tel. 08 754 14 80 Persian
Sholeh - Albydalsvägen 4, 137 95, Österhaninge
Tel. 08 777 77 27 Persian/Arabian
Saffran - Kista Galleria
Tel. 08-75180 60 Persian

Italian:

Ciao Ciao Grande - Storgatan 11
Tel. 08 667 64 20
Dolce Vita - Kungsholmsgatan 16
Tel. 08 650 60 80
Due Fratelli Italian - Vasastan, Birger Jarlsgatan 95
Tel. 08 612 99 33
Trattoria Romana - Gamla Stan, Mälartorget 15
Tel. 08 796 90 09
Lo Scudetto - Östermalm, Kommendörsgatan 46
Tel. 08 640 42 15
Michelangelo - Gamla Stan, Västerlånggatan 62,
Tel. 08 21 50 99
Romana Bakfickan - Gamla Stan, Lilla Nygatan 16
Tel. 08 411 70 74
Runt Hörnet Pasta Bar - Norrmalm, Sveavägen 29
Tel. 08 21 55 92
Sahara Dining and Bar - Norrmalm, Olofsgatan 7
Tel. 08-20 85 65
Agaton, Gamla Stan, Västerlånggatan 72
Tel. 08 20 72 99

North and South East Asian:

Luntmakargatan - Vasastan, Luntmakargatan 65
Tel. 08 673 32 25 Korean
King Tan - Vasastan, Sveavägen 47
Tel. 08 30 47 85 Thai
Pong Asian - Norrmalm, Complete Cross Alley 3
Tel. 08 440 02 08 Thai
Libra - Liljeholmen, Södermalm, Årstaängsvägen 21
Tel. 08 20 30 13 Thai
Narknoi - Vasastan, Odengatan 94
Tel. 08 30 70 70 Thai
Orchid - Södermalm, Medborgarplatsen 3
Tel. 08 641 88 65 Thai
Siam - Gamla Stan Stora Nygatan 25
Tel. 08 20 02 33 Thai
Ho's - Hornsgatan 151
Tel. 08 84 44 20 Chinese
Rice Asian Restaurant & Bar - Östermalm, Nybrogatan 16,Tel. 08 661 32 35 Chinese
Restaurang lucky garden - Klara Norra Kyrkogata 19
Tel. 08 141850 Chinese
Kinarestaurang fair view house - Timmermansgatan 22,
08 6697695 Chinese
Restaurang Hongkong - Kungsbro strand 23
Tel. 08 653 77 20 Chinese
Ming Garden - Götgatan 41
Tel. 08 644 42 26 Chinese
Lao Wai - Luntmakargatan 74
Tel. 08 673 78 00 Chinese
Arigato Sushi Wok - Vasagatan 7
Tel. 08 20 98 15 Chinese
Malaysia, Vasastan, Luntmakargatan 98
Tel. 08 673 56 69 Malaysian
Hanoi Bar - Vasastan, Birger Jarlsgstan 121
Tel. 08 545 933 10 Vietnamese;
Shogun - Gamla Stan, German Brinken 36
Tel. 08 20 82 05 Japanese

Vegetarian:

Chutney - Catherine Bangata 19
Tel. 08 640 30 10;
Hatam - Kammakaregatan 9
Tel. 08 24 49 90;
Hermitage - Stora Nygatan 11, Gamla Stan
Tel. 08 411 95 00
Lao Wai - Luntmakargatan 74
Tel. 08 673 78 00
Martins Gröna - Regeringsgatan 91,
Tel. 08 411 58 50

Govindas - Fridhemsgatan 22
Tel. 08 654 90 04
Hermans - Fjallgatan 23
Tel. 08 643 94 80
Örtagården – Örtagården Nybrogatan 31
Tel. 08 662 17 28
Krogen tre kockar - Stormbyvägen 2
Tel. 08-795 43 44
Copacabana - Hornstulls strand 3
Tel. 08 669 29 39

Swedish:

Östgötakällaren - Södermalm, Östgötagatan 41
Tel. 08 643 22 40
Bistro 99 - Vasastan, Luntmakargatan 99
Tel. 08 15 44 70
Drottninggatan 6 - Norrmalm, Drottninggatan 6
Tel. 08 20 97 24
Eken Bar & Dining - Södermalm, Guldgränd 8
Tel. 08 517 353 36
Gaudi - Norrmalm, Sergelgatan 1
Tel. 08 216911
Washington Monument - Djurgarden, Ostermalm, Dark hook 28-30
Tel. 08 667 21 80
Citadel Canteens - Vaxholm Citadel
Tel. 08 541 333 61
JT - Gamla Stan, Järntorget 78
Tel. 08 20 44 20
Archipelago, Saltsjöbaden, Viking Road 17, Neglinge
Tel. 08 717 15 60/70;
Classroom on the Corner - Vasastan, Surbrunnsgatan 20,
Tel. 08 16 51 36
The Golden Peace - Gamla Stan, Österlånggatan 51
Tel. 08 249760
Queen Hof - Norrmalm, Drottninggatan 67
Tel. 08 22 75 22
Haga Forum - Haga Parken, Annerovägen 4, Northgate 08
Tel. 08 33 48 44
Marten Trotzig - Gamla Stan, Västerlånggatan 79
Tel. 08 442 25 30
Mälardrottningen Yacht - Gamla Stan, Riddarholmen
Tel. 08 545 187 80
Naglo - Norrmalm, Gustav Adolf Square
Tel. 08 10 27 57
Stockholms Matvarufabrik - Vasastan, Idungatan 12
Tel. 08 32 07 04
Stortorgskällaren - Gamla Stan, Stortorget 7
Tel. 08 10 55 33
Verandan - Norrmalm, Södra Blasieholmshamnen 8
Tel. 08 679 35 86

West Asian:

Tabbouli, Kungsholmen, Norra Agnegatan 39
Tel. 08 650 25 00 lebanese
Tabbouli, Norrmalm, Regeringsgatan 70
Tel. 08 20 03 04 lebanese

Stockholm unites the atmosphere of a modern metropolis, a long history and a beautiful natural scenery. Stockholm's history begins around 1250 when it was found by Birger Jarl on the Baltic sea at the mouth of Lake Mälaren as fortification to protect Sweden against sea invasions. Since then it has grown over 14 islands and to a population of 850.000 in the municipality and around 2.1 million in the metropolitan area. The conference venue is situated near the centre (marked with (A) on Map 3).

Map 3: Attractions and walks

A attraction of interest

W1 suggested walk

(A): Conference venue at KTH, subway stop: Tekniska Högskolan, to the centre take the subway line 14 to Fruängen or Liljeholmen and get off at T-Centralen.
(B): Gamla Stan (Old Town), to be reached with the subway line 14 to Fruängen or Liljeholmen.
(C): Skansen is an open-air museum and a zoo located on the island Djurgården where you can also find the Vasamuseum, the Nordic Museum or the fun park Gröna Lund. Take the subway to T-Centralen, walk to Sergels torg and take the tram, get off at Nordiska museet/Vasa or at Waldemarsudde to take a walk around the island.
(D): The area around Slussen and Medborgarplatsen on the island Södermalm is best suited to find places to drink, dine or dance (see Map 4: Going out). Slussen is reachable by the subway line 14 from Tekniska Högskolan in direction Fruängen or Liljeholmen. For Medborgarplatsen you need to either walk from Slussen (10 min) or change to the green line in direction of Hagsätra, Farsta Strand or Skarpnäck.
(E): On a walk on the island Kungsholmen you can visit the City Hall (Stadshuset) and enjoy the sun on a promenade beside the lake Mälaren. On July 25 the will be a reception at Stockholm City Hall.
(F): Perhaps the most beautiful park in Stockholm is the Haga Parken where the Swedish princess Victoria resides, 20 min walk from KTH or take the blue bus 4 from Östra station (KTH) in direction of Gullmarsplan. Get off at Stadsbiblioteket, from there take bus 515 to Sundbybergs station, get off at Haga södra or Haga norra.
(G): The botanical garden (Botaniska Trädgården) is located beside the lake Brunnsviken, reachable by walk (ca. 30 min) or by subway line 14 in direction of Mörby Centrum, stop Universitetet

General information:

Money: Visa and Mastercard accepted in most stores. Banks are tyically open 10-15 on weekdays. Prices in Sweden include VAT but tipping of about 10-15% is customary at restaurants, as is a tip of 10-20 SEK to the Taxi.
Telephone: Emergency - Dial 112. Telephone operator - Dial 118118.

Things to see & Getting around (Map 3):

Gamla Stan (the Old Town) and the Royal Palace (B), Skansen, Gröna Lund and the island Djurgården (C), Skeppsholmen (between B and C), the City Hall (E), Haga Parken (F), Botanical Garden (G)

Stockholm is a compact city, you can reach almost everything by subway or even walk. There are several types of tickets: Single fare costs 30 SEK (~3.3 EUR), slip with 16 coupons cost 180 kr (~20 EUR, a trip costs 2-4 coupons), or travel cards for 1, 3 or 7 days (costs: 100, 200, 260 SEK, respectively). You can buy from special ticket machines in the subway hall or at the bus stop, at the subway counter, by sending a text message from your cell phone or in the mini-markets "Pressbyrån". Note that *tickets cannot be bought on the bus itself*!

From the conference venue (A) on map 3 to the city center:

Public transport: Subway (red line 14) in direction of Fruängen (or Liljeholmen), get off at T-Centralen, Gamla Stan (Old Town) or Slussen, subway goes every 10 min and the ride takes 6 - 10 min. Journey planer: http://sl.se/en

A walk from the conference venue to the city center / downtown takes 30 - 35 min (e.g. W1 on Map 3 via Engelbrektsgatan, Regeringsgatan or W2 via Odengatan, Sveavägen or Drottninggatan alternatively), where you can find a lot of places to shop, dine or drink.

From T-Centralen to the OCNS Banquet (see also Map 5): Take the subway (red line 14) in direction of Fruängen (or Liljeholmen), get off at T-Centralen and when leaving the train go to left exit "Vasagatan, Centralstation, Pendeltåg, Cityterminalen" (NOT Sergel's torg). After passing the gates take the first exit on the left to Vasagatan, this exit is right after the "Pressbyrån". On Vasagatan facing the entry of the central station on the opposite site, turn left and go down the street heading south (to the water). After ca. 200 m at the crossing at the Sheraton Hotel turn right (heading west) and go under the bridge. After passing a dock where ferries leave e.g. to Drottningholm, you'll arrive at the big red building which is the Stadshuset (city hall). If you get lost don't hesitate to ask people around for the Stadshuset, almost everybody can speak English in Sweden.

Bike rental:

You can buy a 3 days card (165 SEK, ca. 18 EUR) for renting the city bikes. Cards are available at the SL centers (e.g. at the subway stops Tekniska Högskolan, T-Centralen, Slussen) or at the Stockholm Tourist Centre (Vasagatan 14). www.stockholmcitybikes.se

Museums:

In Stockholm there are around 70 museums, from which the most visited are: Skansen (open air museum and zoo on Djugården (C) on Map 3), Vasamuseum (maritime museum on Djugården, a bit west of (C)), Nationalmuseum (National Museum of Fine Arts, city centre, close to Skeppsholmen near (B)), Fotografiska (Museum of photography, Södermalm) (D), Naturhistoriska Riksmuseet (Swedish Museum of Natural History, Norra Djugården), Moderna museet (Museum of Modern Art, Skeppsholmen). The museums might close early, so please check the web before you go there!

Other things to do:

There are many boat cruises through the archipelago available, e.g. to Drottningholm castle where the Swedish Royals reside (2h (both ways) boat trip costs 165 SEK, castle entrance extra 80 SEK, park entrance is for free, departure from Stadshusbron, next to the city hall), Birka, the Viking village (7.5h, 310 SEK, departure from Stadshusbron, next to the city hall). Be at the dock near the city hall min 45 min in advance to get your tickets even if you bought them online. Vaxholm is a nice village with a castle (1h, 80 SEK one way with Waxholmsbolaget, ferries depart every 30 min from Strömkajen, close to the Nationalmuseum, subway stop Kungsträdgården).

Internet: www.stromma.se/en/Home/ and http://www.waxholmsbolaget.se/

Viewpoints:

Gondolen (subwaystop Slussen, (D) on Map 3), an outlook on the edge of the Sodermalm neighborhood, offers a panoramic view of the city, overlooking the Old Town and harbor. Och Himlen Därtill (subwaystop Medborgarplatsen on the green line) is a bar and restaurant on the top floors of the Skrapan skyscraper located in Götgatan 78 in Södermalm offering a magnificent view. Globen (subwaystop Globen, green line): Take a ride on top of the biggest spherical building of the world. Stadshuset (subway stop T-Centralen, (E) on Map 3): You need tickets to enter the tower, so buy them early in the day before they are sold out.

Walks (Map 3):

W1, W2 on Map 3 are walks (ca. 35 min) from the conference venue to the city centre.

W3 is a nice walk around the island Djurgården (ca. 1.5 h, get off at T-Centralen, take exit Sergel's torg, close to glass obelisk leaves the tram, get off at Nordiska museet/Vasa or at Waldemarsudde).

W4 (ca. 1 h) starts at T-Centralen (exit Vasagatan) and leads you to the city hall (E), to Rålombshovparken, from there to Södermalm where you can find plenty of places to dine and drink.

W5 (ca. 40 min) is around Hagaparken ((F) free entrance), which is perhaps the most beautiful park in Stockholm. In a small castle resides the princess Victoria. The park is 20 min walk from KTH or you can take the blue bus 4 from Östra station (KTH) in direction of Gullmarsplan, get off at Stadsbiblioteket, from there take bus 515 to Sundbybergs station, get off at Haga södra or Haga norra.

W6 (ca. 1 h) starts from the conference venue and leads you (after 15 min beside a noisy road) to the Brunnsviken lake where you can have picnic, swim and enjoy the sun. Ends at the botanical garden (G), you can take the subway from there (stop Universitetet) to the centre (direction Fruängen or Liljeholmen). A nice area to walk is Björkhagen, an area south of Stockholm (15 min by subway, stop Björkhagen on the green line in direction of Skarpnäck).

Map 4: Going out

(A) KTH: The conference venue at subway stop Tekniska Högskolan
(B) Odengatan: Bars and restaurants on the way to the city
(C) Stureplan: Clubs and bars nearby, subway stop Östermalms torg
(D) Sergel's torg: City centre, subway stop T-Centralen
(E) Gamla Stan: The old town, a lot of nice restaurants and pubs to discover
(F) Slussen: Popular meeting point for starting the evening, a lot of bars and clubs around
(G) Medborgarplatsen: The centre to go clubbing, most bars can be found around this square
(H) Fridhelmsplan: Another place with a lot of possibilities to drink
Streets marked in blue also offer a lot of places to eat and drink something

Going out (Map 4):

Bars and restaurants can be found near all points marked with (B) - (G) on Map 4 (check also the CNS2011 local information web pages). (B) marks Odengatan to Odenplan which is 10 min from the conference venue. From there you can walk e.g. via Sveavägen to the centre, on the way are many restaurants

and pubs. (C) marks the Stureplan (subway stop Östermalmstorg, or 15 min walk) where you can find some poshy bars and clubs. (D) marks the city centre, at Hötorget you can find Kungshallen with a lot of comparable cheap restaurants (either walk the shopping street Sergelgatan north, or take the blue subway line to Hötorget). (E) marks Gamla Stan, the Old Town which has plenty of pubs and restaurants. (F) marks the subway stop called Slussen from which you can walk either Hornsgatan to the west or Götgatan heading south to Medborgarplatsen (G), where you find most bars and clubs in town. (H) marks Fridhelmsplan which also offerst plenty of opportunities to find something to drink (take the blue bus number 4 to Gullmarsplan, get of at Fridhemsplan (17 min), or take the subway and change at T-Centralen to the blue line in direction of Akalla).

Other useful information:

Shopping areas: Main shopping places are Drottninggatan, Sergelsgatan and Hötorget. NK in Hamngatan is a venerable Swedish department store that opened in 1902. Near Sergelstorg is also a shopping mall named Gallerian. All areas are reached by subway stop T-Centralen, exit Sergelstorg.

Souvenirs: Most souvenir shops are in Gamla Stan, but also the Touris Information between T-Centralen and Kungsträdgården has a big shop. You might also check NK and Åhléns department stores near Sergel's torg which have quite a bit.

Stockholm card: This card enables you to use public transport, sightseeing boats or ferries and free admission to 80 museums and attractions. Card costs 425 SEK for 1 day, 550 for 2 days, 650 for 3 days and 895 for 5 days. Cards can be purchased at Stockholm Tourist Center on Vasagatan 14 near the central station as well as at about 130 other locations in and around Stockholm. More information can be found online at www.visitstockholm.com.

Supermarkets: Closest supermarkets to the conference venue are one in the subway station (T-Snabben), a Coop Konsum near the crossing of Odengatan and Birger Jarlsgatan, an ICA store a bit further on Odengatan and another ICA on crossing Danderydsgatan/Karlavägen (check also Map 2: Restaurants near campus). Supermarkets in Sweden are usually opened every day of the week.

Taxis: We recomend you to use one of the following companies, and for longer trips (like to and from the airport) we recommend the fixed price. Taxi Stockholm Tel.08-150000, Taxikurir Tel.0771-860000, Taxi020 Tel.020-202020

For children: Gröna Lund is a fun park (not only for children) and Junibacken is a sort of indoor amusement park, both on Djurgården (subway T-Centralen, from Sergels torg tram, stop at Gröna Lund/Tivoli).

Map 5: To the reception at Stockholm City Hall

Map 5: Directions to the Stockholm City Hall

From KTH to the reception at Stockholm City Hall, July 25. Leave at 18:30: Take the subway (red line 14) in direction of Fruängen (or Liljeholmen), get off at T-Centralen and when leaving the train go to the left exit "Vasagatan, Centralstation, Pendeltåg, Cityterminalen" (NOT Sergel's torg). After passing the gates take the first exit on the left to Vasagatan, this exit is right after the "Pressbyrån". On Vasagatan facing the entry of the central station on the opposite site, turn left and go down the street heading south (to the water). After ca. 200 m at the crossing at the Sheraton Hotel turn right (heading west) and go under the bridge. After passing a dock where ferries leave e.g. to Drottningholm, you'll arrive at the big red building which is the Stadshuset (city hall). If you get lost don't hesitate to ask people around for the Stadshuset or City Hall, almost everybody can speak English in Sweden. The walk is around 500 m, but when you take a different exit from the subway it gets longer and might easily take you 10 min extra to get there. Please try to be in time, as you are in Sweden :)

This year's exclusive CNS*2011 party will be held on Tuesday evening at Syster & Bror, a pub located a two-minute walk from the main lecture hall, F1 - see (B) on Map 1.

They have a nice selection of beer and wine to enjoy indoors and outdoors (if the weather allows :-).

You will be served a welcome drink and snacks at arrival.

Live Stand-Up Comedy at the CNS*2011 Party

Thanks to the New Zealand-born stand-up comedian Al Pitcher, Sweden is now a much happier country.
Before he met his Swedish girlfriend and decided to move to Stockholm, Al performed mainly in english-speaking countries, including New Zealand, Australia and UK, where he received numerous awards. Here are a few examples of what people say about his shows:

"6th Best Comedian in the UK" - *Zoo Magazine*
"He has the energy of a kid who's drunk too much lemonade" - *Metro*
"Free-wheeling genious" - *Sydney Morning Herald*
"Pitcher is one of the best-kept secrets on the comedy scene" - *Time Out 2008*

»Meeting info

SATURDAY JULY 23

09:00 - 16:00	Registration ((E) on Map 1)
09:00 - 12:00	Tutorials ((E)+(D) on Map 1)
12:00 - 13:30	*Break*
13:30 - 16:30	Tutorials ((E)+(D) on Map 1)
17:00 - 21:00	Opening reception (KTH library (A) on Map 1)
17:00 - 21:00	Registration ((A) on Map 1)

SUNDAY JULY 24

09:00 - 09:20	Welcome & announcements ((F1) on Map 1)
09:20 - 10:20	Keynote 1 - INCF Distinguished lecture: Ivan Soltesz
10:20 - 10:50	*Break*
10:50 - 12:10	Oral session I: Plasticity, learning, and memory (O1-O4)
12:10 - 14:00	*Break for lunch*
14:00 - 15:40	Oral session II: Cortical networks (F1, O5-O7)
15:40 - 16:10	*Break*
16:10 - 16:30	Oral session II: Cortical networks (O8)
16:30 - 17:30	Oral session III: Development (O9-O11)
17:30 - 19:00	*Break for dinner*
19:00 - 22:00	Poster session I (Map 1: (E)+(D), ground floor) *Posters 1-128*

MONDAY JULY 25

09:00 - 09:10	Announcements ((F1) on Map 1)
09:10 - 10:10	Keynote 2: Anders Lansner
10:10 - 10:40	*Break*
10:40 - 11:20	Oral session IV: Coding (O12-O13)
11:20 - 12:00	Oral session V: Oscillators (O14-O15)
12:00 - 13:45	*Break for lunch*
13:45 - 14:45	Oral session V: Oscillators (F2, O16)
14:45 - 15:00	*Break*
15:00 - 18:00	Poster session II (Map 1: (E)+(D), groundfloor) *Posters 129-256*
18:00 - 19:00	*Transport to City Hall from conference venue and hotels (see Map 5)*
19:00 - 21:00	Reception at Stockholm City Hall ((E) on Map 3)

TUESDAY JULY 26

09:00 - 09:10	Announcements ((F1) on Map 1)
09:10 - 10:10	Keynote 3 - Frontiers lecture: Peter Tass
10:10 - 10:40	*Break*
10:40 - 12:40	Oral session VI: Cellular mechanisms (O17-O19)
11:40 - 12:40	OCNS member meeting
12:40 - 14:00	*Break for lunch*
14:00 - 15:00	Oral session VII: (De)correlation (F3, O20)
15:00 - 15:30	*Break*
15:30 - 18:30	Poster session III (Map 1: (E)+(D), groundfloor) *Posters 257-383*
18:30 - 20:00	*Break for dinner*
20:00 - 00:00	OCNS Party (Syster o Bror, (B) on Map 1)

JULY 27 - JULY 28

Workshops, see pages 27-34 for dates and descriptions

Graphic schedule

	Saturday	Sunday	Monday	Tuesday	Wednesday	Thursday
	Tutorials 23/7	Main meeting 24-26/7			Workshops 27-28/7 *	
09:00	Tutorials morning session	Welcome and announcements	Announcements	Announcements	Workshops AM	Workshops AM
09:10		Keynote 1: Ivan Soltesz	Keynote 2: Anders Lansner	Keynote 3: Peter Tass		
09:20						
09:30						
09:40						
09:50						
10:00						
10:10			Break	Break	Break	Break
10:20		Break				
10:30			Oral O-12	Oral O-17	Workshops AM	Workshops AM
10:40		Oral O-01	Oral O-13	Oral O-18		
10:50						
11:00		Oral O-02	Oral O-14	Oral O-19		
11:10						
11:20		Oral O-03	Oral O-15	OCNS Member meeting		
11:30						
11:40		Oral O-04				
11:50						
12:00	Lunch break	Lunch break	Lunch break	Lunch break	Lunch break	Lunch break
12:10						
12:30						
12:40						
12:50						
13:00						
13:10						
13:20						
13:30	Tutorials afternoon session				Workshops PM	Workshops PM
13:40						
13:50			Featured oral F-02			
14:00		Featured oral F-01		Featured oral F-03		
14:10			Oral O-16			
14:20						
14:30			Break			
14:40		Oral O-05		O-20 Oral	Break	Break
14:50						
15:00		Oral O-06	Poster session II	Break		
15:10					Workshops PM	Workshops PM
15:20		Oral O-07	*Posters 129-256*	Poster session III		
15:30						
15:40		Break		*Posters 257-383*		
15:50						
16:00						
16:10		Oral O-08				
16:20	Break					
16:30		Oral O-09				
16:40						
16:50		Oral O-10				
17:00	Welcome reception					
17:10		Oral O-11				
17:20						
17:30						
17:40		Dinner break				
17:50						
18:00			Break			
18:10						
18:20						
18:30				Dinner break		
18:40						
18:50						
19:00		Poster session I	Reception at Stockholm City Hall			
19:10						
19:20		*Posters 1-128*				
19:30						
19:40						
19:50						
20:00				CNS Party		
20:10						
20:20						
20:30						
20:40						
20:50						
21:00						
21:10						
21:20						
21:30						
21:40						
21:50						
22:00 -->						

* Individual workshop schedules may differ; please check with respective organizer

» Session 1, July 24 19:00 - 22:00

posters **1-18**	Room E31
posters **19-33**	Room E33
posters **34-48**	Room E34
posters **49-66**	Room E35
posters **67-84**	Room D31
posters **85-99**	Room D32
posters **100-117**	Room D34
posters **118-128**	Room D35

» Session 2, July 25 15:00 - 18:00

posters **129-146**	Room E31
posters **147-161**	Room E33
posters **162-176**	Room E34
posters **177-194**	Room E35
posters **195-212**	Room D31
posters **213-227**	Room D32
posters **228-245**	Room D34
posters **246-256**	Room D35

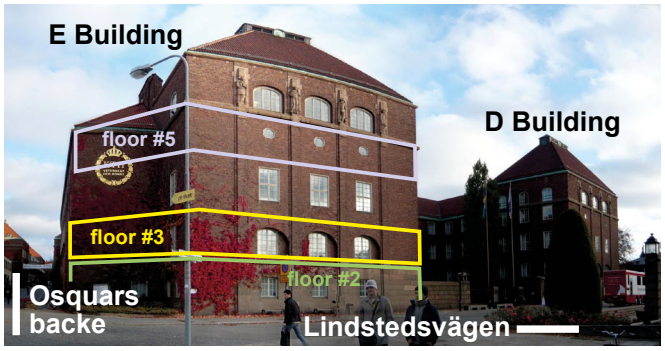
» Session 3, July 26 15:30 - 18:30

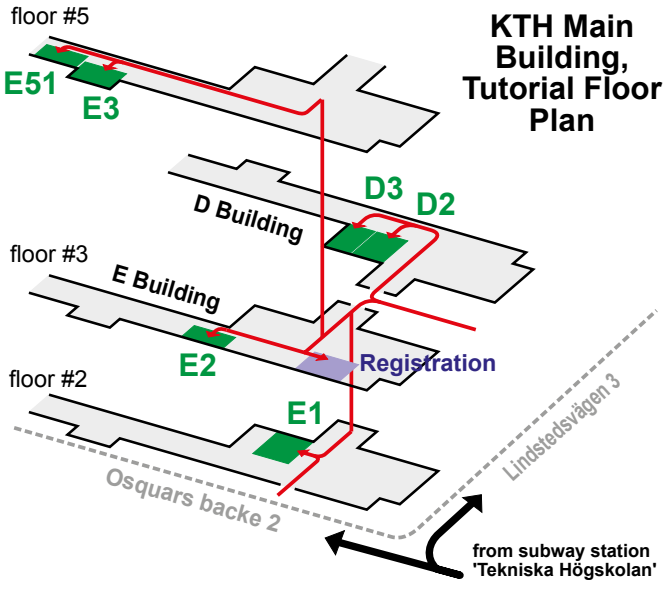
posters **257-274**	Room E31
posters **275-289**	Room E33
posters **290-304**	Room E34
posters **305-322**	Room E35
posters **323-340**	Room D31
posters **341-355**	Room D32
posters **356-373**	Room D34
posters **374-383**	Room D35

Tutorials will take place prior to the main meeting in the morning and in the afternoon of July 23rd, 2011 at the KTH main campus, houses E & D. Light snacks and beverages will be provided for all attendees. Tutorials will target the following main topics: Data analysis, Software tools, Generalized Linear Models, Neural Coding and Noisy Neural Dynamics. More detailed descriptions of the tutorials follow on the next pages.

- **A.** Introduction to Machine Learning and Kernel Methods (Matthew Blaschko)
- **B.** Analysis of Parallel Spike Trains (Sonja Grün and Stefan Rotter, FULL DAY)
- **C.** A Tutorial in Connectome Analysis: Topological and Spatial Features of Neural Networks (Marcus Kaiser)
- **D.** Multivariate techniques for fMRI analysis (Lars Kai Hansen)
- **E.** An introduction to Python for Computational Neuroscientists (Eilif Mueller and Andrew Davison, FULL DAY)
- **F.** Modelling noisy neurons: analytical approaches to characterize spontaneous activity and information transmission (Benjamin Lindner)
- **G.** Generalized Linear Models and their application to neural data - Part I (Timm Lochmann)
- **H.** Generalized Linear Models and their application to neural data - Part II (Sara Solla)
- **I.** Neural Population Coding (Matthias Bethge)
- **J.** The 'Bayesian' approach to the Brain: experimental evidence and probabilistic models of neural coding, perception and cognition (Peggy Series)

Room	Morning Tutorials 9:00-12:00	Afternoon Tutorials 13:30-16:00
E1	**C.** Connectome Analysis *Kaiser*	**F.** Noisy Neurons *Lindner*
E2	**G.** GLMs I *Lochmann*	**H.** GLMs II *Solla*
E3	**E.** Introduction to Python *Muller/Davison et al.*	
E51	**A.** Machine Learning *Blaschko*	**D.** Multi-var techniques for fMRI *Hansen*
D2	**B.** Spike Train Analysis *Gruen/Rotter et al.*	
D3	**J.** Bayesian Brain *Series*	**I.** Population Coding *Bethge*





A. Introduction to Machine Learning and Kernel Methods

Matthew Blaschko (University of Oxford, Oxford, UK)

Room E51, morning: 9:00-12:00

Kernel methods are most commonly known through the popular support vector machine (SVM) classification algorithm, but are applicable to a wide array of other learning algorithms. This short course covers the application of kernel methods to regression, dimensionality reduction, clustering, and measuring independence. These methods are unified by the common threads of the representer theorem, data centering, and second order statistics. In particular, we will talk about Kernel Ridge Regression; Kernel PCA; Spectral Clustering; Kernel Canonical Correlation Analysis; Kernel methods for measuring independence; Examples will be presented using consistent notation, and connections between various methods will be emphasized.

B. Analysis of Parallel Spike Trains

Sonja Grün (Insitute of Neuroscience and Medicine (INM-6), Research Center Juelich, Germany)
Stefan Rotter (Bernstein Center Freiburg, Germany)

Room D2, full day: 9:00 - 12:00, 13:30 - 16:00

Action potentials, or spikes, are the most salient expression of neuronal processing in the active brain, and they are very likely an important key to understanding the neuronal mechanisms of behavior. However, neurons do not act in isolation, but organize in groups or ensembles of unknown size to achieve any particular function. In experiments, this can only be observed if the action potentials of multiple individual nerve cells are recorded simultaneously. Techniques that employ multi-electrodes for parallel spike train recordings have been available for quite some time, and their use has now gained wide popularity among neuroscientists. To reliably interpret the results of such electrophysiological recordings, solid and comprehensible data analysis is crucial. It is fair to say that the development of data analysis methods has not kept pace with the advances in recording technology. Neither general concepts, nor statistical methodology are ready for the new experimental possibilities. In this tutorial we will give an overview of promising new approaches for the analysis of parallel spike trains, discuss their assumptions and applicability, and also broach some practical issues.

Topics covered in the tutorial: Point process models of neuronal spike trains. Stefan Rotter, Bernstein Center Freiburg, Germany; Firing rate estimation and spike train variability. Shigeru Shinomoto, Kyoto Univ., Japan; Population coding. Stefano Panzeri & Alberto Mazzoni, Italian Institute of Technology, Genova, Italy; Unitary events analysis. Sonja Grün, Research Center Jülich, Germany and RIKEN BSI, Japan; Higher-order correlations and cumulants for point processes. Benjamin Staude & Stefan Rotter, Bernstein Center Freiburg, Germany; Higher-order interactions for binary variables. Yasser Roudi, Kavli Institute, Trondheim, Norway; Surrogate spike trains. Michael Denker & Sonja Grün, Research Center Jülich, Germany and RIKEN BSI, Japan; Parallel and distributed computing. Michael Denker, Research Center Jülich, Germany.

C. A Tutorial in Connectome Analysis: Topological and Spatial Features of Neural Networks

Marcus Kaiser (Newcastle University, Newcastle, UK)

Room E1, morning: 9:00-12:00

High-throughput methods for yielding the set of connections in a neural system, the connectome, are now being developed. This tutorial describes ways to analyze the topological and spatial organization of the connectome at the macroscopic level of connectivity between brain regions as well as the microscopic level of connectivity between neurons. I will describe topological features at three different levels: the local scale of individual nodes, the regional scale of sets of nodes, and the global scale of the complete set of nodes in a network. Such features can be used to characterize components of a network and to compare different networks, e.g. the connectome of patients and control subjects for clinical studies. At the global scale, different types of networks can be distinguished and we will describe Erdös-Rényi random, scale-free, small-world, modular, and hierarchical archetypes of networks. Finally, the connectome also has a spatial organization and we describe methods for analyzing wiring lengths of neural systems. As an introduction for new researchers in the field of connectome analysis, I will discuss the benefits and limitations of each network analysis approach.

D. Multivariate techniques for fMRI analysis

Lars Kai Hansen, PhD (TU Denmark, Lyngby, Denmark)

Room E51, afternoon: 13:30 - 16:00

Resampling schemes for optimization, visualization, and validation of statistical models; Linear and non-linear models for detection of networks and denoising: PCA, ICA, kPCA; Brain state decoding, visualization of SVMs; Modeling plurality and consensus

E. An introduction to Python for Computational Neuroscientists

Eilif Muller (EPFL, Lausanne, Switzerland)
Andrew Davison (CNRS, Gif-sur-Yvette, France)

Room E3, full day: 9:00 - 12:00, 13:30 - 16:00

Why Python? A Python crash-course; The scientific Python toolbox; Taming your favourite Computational Neuroscience simulator with Python; Python Guru Meditation; With special guests...

F. Modelling noisy neurons: analytical approaches to characterize spontaneous activity and information transmission

Benjamin Lindner, PhD (MPI for the Physics of Complex Systems Dresden, Germany)

Room E1, afternoon: 13:30-16:30

Neural noise, statistics of point processes, renewal theory, nonrenewal point processes, integrate-and-fire neurons, Fokker-Planck equation, Langevin equation, spike-frequency adaptation, signal transmission.

G. Generalized Linear Models and their application to neural data - Part I

Timm Lochmann, PhD (University of Maryland, College Park, USA)

Room E2, morning: 9:00-12:00

Maximum likelihood estimation, linear, logistic, and Poisson regression. Model fitting, assessment, and selection. Regularization, dimensionality reduction, smoothing, generalized additive models. Application to single cell spike-train data: estimation of receptive fields and modeling of nonlinearities. Interpretation of model parameters.

H. Generalized Linear Models and their application to neural data - Part II

Sara A. Solla, PhD (Northwestern University, Chicago, USA)

Room E2, afternoon: 13:30-16:00

Generalized Linear Models for Poisson statistics: from Point Processes to Poisson Processes. Likelihood maximization: unique maximum, hill climbing. Application to spike-train data from multielectrode arrays. Characterization of network connectivity through sparse pairwise directed filters. Applications to plasticity and motor control.

I. Neural Population Coding

Matthias Bethge, PhD (U Tuebingen & MPI for Biological Cybernetics Tübingen, Germany)

Room D3, afternoon: 13:30-16:30

This tutorial provides an introduction into the mathematical concepts, tools, and current research directions in neural population coding. In particular, it will cover topics related to stimulus reconstruction (Georgopoulos' population vector, linear decoding, pseudo-inverse decoding of integrate and fire neurons, Bayesian point estimation, approximate Bayesian point estimation, filtering, examples from current neural prosthesis research, performance measures, loss functions, cross validation), stimulus discrimination (binary decision tasks, 2AFC, linear discriminant analysis, quadratic discriminant analysis, Bayesian discrimination), probabilistic inference (approximate Bayesian inference, sampling), information theory (log-loss, entropy, KL-divergence, information gain, mutual information, rate-distortion curves, source and channel coding), Fisher information (Cramer-Rao bound, asymptotic normality, Jensen-Shannon divergence, discrimination at perceptual threshold), optimal tuning width, noise correlations, heterogeneous populations, multi-dimensional encoding, redundancy reduction, error correction, uncertainty representation (probabilistic population coding vs. sampling), and task-dependent input noise during perceptual inference.

J. The 'Bayesian' approach to the Brain: experimental evidence and probabilistic models of neural coding, perception and cognition

Peggy Series, PhD (U Edinburg, UK)

Room D3, morning: 9:00 - 12:00

In what sense is the brain 'Bayesian optimal'?, Psychophysical evidence, How could Bayesian inference be implemented in neural activity and circuits?, Bayesian models of perception and cognition.

» Sunday, July 24

09:00 - 09:20 **Welcome & announcements** ((F1) on Map 1)

09:20 - 10:20 **Keynote 1: Ivan Soltesz**

INCF Distinguished Lecture: *Ivan Soltesz*
"Functional network connectivity of the control and epileptic hippocampus"

10:20 - 10:50 *Break*

10:50 - 12:10 **Oral session I: Plasticity, learning, and memory** (O1-O4)

10:50 – 11:10 *Alex Loebel, Jean-Vincent Le Bé, Magnus JE Richardson, Andreas Herz, Henry Markram*
"The modular cross-synaptic nature of LTP/LTD following ongoing neural activity"

11:10 – 11:30 *Greg Stephens*
"The emergence of long timescales and stereotyped behaviors in C. elegans"

11:30 – 11:50 *Botond Szatmáry, Eugene M. Izhikevich*
"Spike-timing theory of working memory"

11:50 – 12:10 *Petra E Vertes, Danielle S. Bassett, Thomas Duke*
"Scale-free statistics of neuronal assemblies predict learning performance"

12:10 - 14:00 *Break for lunch*

14:00 - 15:40 **Oral session II: Cortical networks** (F1, O5-O7)

14:00 – 14:40 ***Featured oral:*** *Markus Butz, Arjen van Ooyen*
"Need for homeostasis in electrical activity may account for cortical network rewiring"

14:40 – 15:00 *Ali Mohebi, Jessica A. Cardin, Karim G. Oweiss*
"A cell-type-specific dynamic Bayesian network model for spontaneous and optogenetically evoked activity in the primary visual cortex"

15:00 – 15:20 *Oliver Weihberger, Ayal Lavi, Samora Okujeni, Uri Ashery, Ulrich Egert*
"State-dependent modulation of stimulus-response relations in cortical networks in vitro"

15:20 – 15:40 *Daphne Krioneriti, Athanasia Papoutsi, Panayiota Poirazi*
"Mechanisms underlying the emergence of Up and Down states in a model PFC microcircuit"

15:40 - 16:10 *Break*

» Sunday, July 24 (continued)

16:10 - 16:30 — **Oral session II: Cortical networks** (O8)

16:10 – 16:30 — *Henrik Lindén, Tom Tetzlaff, Tobias C Potjans, Klas H Pettersen, Sonja Grün, Markus Diesmann, Gaute T Einevoll*
"How local is the local field potential?"

16:30 - 17:30 — **Oral session III: Development** (O9-O11)

16:30 – 16:50 — *Abul Kalam al Azad, Roman Borisyuk, Alan Roberts, Steve Soffe*
"Gradient based spinal cord axogenesis and locomotor connectome of the hatchling Xenopus tadpole"

16:50 – 17:10 — *Bertrand Fontaine, Romain Brette*
"A developmental explanation of the dependence of binaural best delays on characteristic frequency"

17:10 – 17:30 — *A. Aldo Faisal, A. Victor Luria*
"Serial decision-making and noise in the assembly of neural circuits"

17:30 - 19:00 — *Break for dinner*

19:00 - 22:00 — **Poster session I** (Map 1: (E)+(D), ground floor)

» Monday, July 25

09:00 - 09:10 — **Announcements** ((F1) on Map 1)

09:10 - 10:10 — **Keynote 2: Anders Lansner**

Anders Lansner
"Perceptual and memory functions in a cortex-inspired attractor network model"

10:10 - 10:40 — *Break*

10:40 - 11:20 — **Oral session IV: Coding** (O12-O13)

10:40 – 11:00 — *Jan Clemens, Susanne Schreiber, Olaf Kutzki, Bernhard Ronacher, Sandra Wohlgemuth*
"Optimal sparse coding of song in a size-constrained auditory system?"

11:00 – 11:20 — *Christopher K. Kovach, Rick L. Jenison*
"A window to the amygdala: concurrent encoding of choice preference in multi-unit activity in the amygdala and in eye movements"

11:20 - 12:00 — **Oral session V: Oscillators** (O14-O15)

11:20 – 11:40 — *Timothy J Lewis, Jiawei Zhang, Carmen Smarandache, Brian Mulloney*
"Understanding the mechanisms underlying phase-locking behavior in the crayfish swimmeret system"

» Monday, July 25 (continued)

11:40 – 12:00 — *Christoph Kirst, Marc Timme, Demian Battaglia*
"Local control of non-local information flow in oscillatory neuronal networks"

12:00 - 13:45 — *Break for lunch*

13:45 - 14:45 — **Oral session V: Oscillators** (F2, O16)

13:45 – 14:25 — ***Featured oral:*** *Svetlana Postnova, Peter A Robinson*
"Neuronal mechanisms of shift workers' sleepiness"

14:25 – 14:45 — *Angela C.E. Onslow, Matthew W. Jones, Rafal Bogacz*
"Oscillatory mechanisms of selective integration during decision making"

14:45 - 15:00 — *Break*

15:00 - 18:00 — **Poster session II** (Map 1: (E)+(D), groundfloor)

18:00 - 19:00 — *Transport from conference venue to city hall: approximately 30 min. Transport from hotels to city hall: 10 min – 45 min depending on hotel location, see Map 5 for directions.*

19:00 - 21:00 — **Reception at Stockholm City Hall** ((E) on Map 3)

» Tuesday, July 26

09:00 - 09:10 — **Announcements** ((F1) on Map 1)

09:10 - 10:10 — **Keynote 3: Peter Tass**

Frontiers lecture: *Peter Tass*
"Long-lasting neuronal desynchronization caused by coordinated reset stimulation"

10:10 - 10:40 — *Break*

10:40 - 12:40 — **Oral session VI: Cellular mechanisms** (O17-O19)

10:40 – 11:00 — *Mark E. J. Sheffield, Tyler K. Best, Brett D. Mensh, William L. Kath, Nelson Spruston*
"Slow integration leads to persistent action potential firing in distal axons of coupled interneurons"

11:00 – 11:20 — *Magnus J. E. Richardson, Rupert Swarbrick*
"Exact firing-rate response of the integrate-and-fire neuron receiving finite amplitude excitatory and inhibitory postsynaptic potentials"

11:20 – 11:40	*Alexey Kuznetsov, Joon Ha* **"An interlocked oscillator model for high-frequency firing of the midbrain dopaminergic neuron"**
11:40 - 12:40	**OCNS member meeting**
12:40 - 14:00	*Break for lunch*
14:00 - 15:00	**Oral session VII: (De)correlation** (F3, O20)
14:00 – 14:40	***Featured oral:*** *Charles J. Wilson, Bryce Beverlin II, Theoden Netoff* **"Chaotic decorrelation of Globus Pallidus by periodic forcing: A possible mechanism for the therapeutic effects of deep brain stimulation"**
14:40 – 15:00	*Jiannis Taxidis, Stephen Coombes, Robert Mason, Markus Owen* **"Modeling sharp wave - ripple complexes and their interactions with cortical slow oscillations through a cortico-CA3-CA1 model"**
15:00 - 15:30	*Break*
15:30 - 18:30	**Poster session III** (Map 1: (E)+(D), groundfloor)
18:30 - 20:00	*Break for dinner*
20:00 - 00:00	**OCNS Party** (Syster o Bror, (B) on Map 1)

27 July

Emerging standards for network modeling in neuroscience

Sean Hill, Ivan Raikov, Anatoly Gorchetchnikov, Eilif Muller, Andrew Davison, Mikael Djurfeldt, Yann Le Franc, Malin Sandström

The growing number of large-scale neuronal network models has created a need for simulator-independent description languages to ease model sharing and facilitate the replication of results across different simulators. To coordinate and promote community efforts towards such standards, the International Neuroinformatics Coordinating Facility (INCF) has formed its Multiscale Modeling program, and has assembled a task force to propose a declarative computer language for descriptions of large-scale neuronal networks: NineML. The INCF Task Force responsible for NineML development contains delegates from several relevant projects like the Blue Brain Project, GENESIS, KInNeSS, MOOSE, NEST, NeuroML, NEURON and PyNN.

This workshop aims to present an overview of the tools available to the community that support the development and exchange of network models. One key new community development is NineML, which provides a simulator independent description of model dynamics. At the same time, the next generation of NeuroML, (NeuroML V2.0) is under active development and will introduce important new advances in interoperable model description.

The focus of the morning session will be to present the current development in model description languages, with the presentation of two complementary initiatives, NineML and NeuroML. This will be followed by a presentation on the Connection Set Algebra, a recent development for describing network connectivity, which is currently being integrated with NEST and PyNN.

The afternoon session will then be focused on demonstrating interoperability using model description, APIs and runtime data exchange, with use cases that demonstrate the integration of these elements. We will present the current status of the interactions between NeuroML/LEMS and NineML, the integration of NineML within PyNN and the interrelation between PyNN and the multi-simulation coordination software MUSIC, developed as an INCF initiative. We will conclude this session by a presentation of the potential use of NineML as a native language for multiscale modeling. As all the presented initiatives were developed for and rely on the comments of the computational neuroscience community, we would like to invite the workshop participants to share their experiences, their questions and comments to help us to further develop these tools and their interactions for the community of modelers.

Finally, deeply aware of the dynamism of the community, we have invited interested scientists to present in a lightning talk (4 + 1 minutes) their related work, developed outside of the major initiatives.

The target audience is computational neuroscientists, developers of modeling tools, and anyone interested in replication and reuse of modeling results across platforms and research groups.

Speakers: Sean Hill, Sharon Crook, Mikael Djurfeldt, Padraig Gleeson, Robert Cannon, Andrew Davison, Ivan Raikov

Modeling Central Pattern Generators: Neuronal Network Design Principles and Problems

Erik Sherwood (Boston University), Silvia Daun-Gruhn (Universitaet Koeln)

CPGs represent a great success story for neuroscience in general and computational neuroscience in particular, being an area in which there has been great progress over the past 30 years in understanding circuit function and design. It might even be said that the success is too great for it to remain a cutting-edge area of research: in a recent review article, it is argued that for invertebrate CPG circuit mechanisms, at least, “it is no longer necessary to ask more questions.”

Is this truly the case? Multiple mechanisms of rhythmogenesis, phasing control, etc. have been elucidated, but have we really worked most everything out, aside from some details?

The aim of this workshop is to bring together active modelers of (vertebrate and invertebrate) CPGs to present recent work and to discuss, inter alia,

- CPG design principles: what is firmly established and where are open questions?

- Modeling approaches: what works and what doesn't work, and why? What are (in)effective strategies for inferring the architectures and intrinsic dynamics appropriate for particular modeling biological CPGs, given limited experimental data?

- Multistability and transitions: what architectures and intrinsic mechanisms support multiple rhythms? What kinds of transitions do they admit and how?

- Complications: functional rewiring by neuromodulation, modulation by sensory feedback, overlapping CPGs (sharing one or more neurons), large scale (100+ neuron) CPGs

The workshop is intended to foster an open exchange of ideas and constructive criticism, and to include discussion of problems or failures, in addition to successes, in the CPG modeling process.

Speakers: John H. Byrne, Ron Calabrese, Gennady Cymbalyuk, Nalin Harischandra, Auke Ijspeert, Brian Mulloney, Farzan Nadim, Astrid Prinz, Jonathan Rubin, Ilya Rybak

Enabling Super-Computational Neuroscience: Low-Cost GPU-Parallel Analyses And Simulations

Dan Gardner (Weill Cornell Medical College)
Progress in many significant areas of neuroscience is limited by computational barriers. For example, our current capacity to acquire data from a hundred or more simultaneously recorded neurons or networks far outstrips our ability to analyze them, especially in real time.

To address this imbalance, this workshop will review the demonstrated ability, and explore the enormous potential for computational neuroscience, of inexpensive and newly-available computational architectures. These are derived from graphics processing units (GPUs) and yield supercomputer-level 500 GFLOPS to 1 TFLOPS performance from a single inexpensive ($2,500, £2,200, or 2.400 Euros) card. Such cards have the potential to speed analyses or simulations by up to two orders of magnitude, newly enabling real-time decoding or analyses during experiments or toward enhanced brain-machine interfaces. Similarly, previously-impractical algorithms or models may be explored in reasonable compute times.

As innovative programming techniques and extensive testing are required to achieve these transformative results, the workshop will also review successful case studies and derive criteria for which analyses are best suited for such development.

Three groups of invited speakers will:

- present very recent successful examples of GPU-enabled computations for neural modeling and analysis,

- offer open source routines for neurophysiology data analysis, with bottlenecks particularly amenable to GPU parallelization, and

- explore optimization strategies, and avoidable pitfalls, for GPU parallelization.

Additional short talks or poster presentations on any of these areas or related topics are being solicited. In all cases, the twin emphases will be on identifying areas where these advances can have the greatest impact on multielectrode neurophysiology and other areas of computational neuroscience, and on community-enabling open source software to most appropriately leverage GPU architecture and capabilities.

Participants: Esther Gardner, Steven Hsiao, Conor Houghton, Thomas Nowotny, Rodrigo Quian-Quiroga, Charles Schroeder, Ruggero Scorcioni, Gabriel Silva

New approaches to spike train analysis and neuronal coding

Conor Houghton (Trinity College, Dublin), Thomas Kreuz (Institute of Complex Systems, Italy)
A myriad of putative properties have been proposed for the spiking responses of neuronal populations. However, spike train properties like sparseness, temporal coding, synchrony and population coding are easier to describe intuitively than to match to quantitative definitions that can be applied to experimental data. As a consequence, it may be that some of the important questions being posed about neuronal coding are difficult to answer precisely because they are difficult to phrase in an answerable way. The purpose of this workshop is to discuss how different approaches, such as measures of spike train (dis)similarity and methods from information theory, can be used to define quantitative properties of neuronal signalling, properties which could be used to analysis the large quantities of experimental data now available in a way that would help explain neuronal coding. Contributions will include both experimental and theoretical studies, data analysis as well as modelling.

Speakers: Romain Brasselet, Daniel Chicharro, Daniel Gardner, Sonja Gruen, Richard Naud, Jose Principe, Ralph G. Andrzejak, Matthias H. Hennig, Mikail Rubinov, Juan Carlos Vasquez

Method of Information Theory in Computational Neuroscience

Simon Schultz (UCL), Michael Gastpar (UC Berkeley), Aurel A. Lazar (Columbia), Todd Coleman (UIUC)
Methods originally developed in Information Theory have found wide applicability in computational neuroscience. Beyond these original methods there is a need to develop novel tools and approaches that are driven by problems arising in neuroscience.

A number of researchers in computational/systems neuroscience and in information/communication theory are investigating problems of information representation and processing. While the goals are often the same, these researchers bring different perspectives and points of view to a common set of neuroscience problems. Often they participate in different fora and their interaction is limited.

The goal of the workshop is to bring some of these researchers together to discuss challenges posed by neuroscience and to exchange ideas and present their latest work.

The workshop is targeted towards computational and systems neuroscientists with interest in methods of information theory as well as information/communication theorists with interest in neuroscience.

Speakers: Ryan T. Canolty, Simona Cocco, Todd P. Coleman, Susanne Ditlevsen, A. Aldo Faisal, Olivier Faugeras, Michael C. Gastpar, Erol Gelenbe, John A. Hertz, Kilian Koepsell, Christian Machens, Eftychios A. Pnevmatikakis, Sridevi V. Sarma, Simon R. Schultz, Tatyana Sharpee

Advances in deep brain stimulation - computation approaches

Peter Tass, Christian Hauptmann (Institute of Neuroscience and Medicine, Juelich, Germany)
Medically refractory Parkinson patients are treated with deep brain stimulation (DBS). For this, chronic electrodes are implanted into target structures and high--frequency (HF) pulse trains are applied to counteract the over--activation of the output structures of the basal ganglia. The development of HF DBS was basically an empirical process. HF DBS basically mimics the effect of tissue lesioning and strongly alters, e.g. suppresses, the neuronal firing in a way that is not yet sufficiently understood. In the past ten years computational neuroscience had a strong impact on the understanding of the mechanisms of DBS and numerical simulations triggered concepts for the optimization of the therapeutic outcome of DBS. The workshop intents to present these achievements and to identify promising strategies to utilize computational approaches for further advancing the usability of DBS for various diseases like Parkinson's disease, Dystonia and mental disorders.

Participants: Warren Grill, Jean-Pascal Pfister, Cameron McIntyre, Bard Ermentrout, Wassilios Meissner, C.R. Butson, Jonathan Rubin, David Terman, Roman Borisyuk, Jeff Wickens

Multistability in Neurodynamics

Gennady Cymbalyuk, Georgia State University
This workshop is focused on the co-existence of regimes of activity of neurons. Such multistability enhances potential flexibility to the nervous system and has many implications for motor control, dynamical memory, information processing, and decision making.

The goal of this workshop is to identify the scenarios leading to multistability in the neuronal dynamics and discuss its potential roles in the operation of the central nervous system under normal and pathological conditions.

It is intensively studied on different levels. On the cellular level, multistability is co-existence of basic regimes like bursting, spiking, sub-threshold oscillations and silence. On the network level, examples of multistability include co-existence of different synchronization modes, ''on'' and ''off'' states, polyrhythmic bursting patterns, and co-existence of pathological and functional regimes.

Speakers: Maxim Bazhenov, Gennady Cymbalyuk, Fernando R. Fernandez, Erik Fransén, Boris Gutkin, Bóris Marin, Farzan Nadim, Thomas Nowotny, Frances Skinner, Peter Tass

Basal Ganglia: Dynamics, Function and Learning

Arvind Kumar (University of Freiburg, Germany), Jeanette Helgren Kotaleski (Royal Institute of Technology, Stockholm), Dieter Jaeger (Emory University)
The basal ganglia (BG) are involved in a wide range of motor and cognitive processes, and accordingly, their dysfunction can lead to several neurological diseases. Extensive experimental characterization at multiple scales of the BG in normal and pathological conditions have provided important insights about the BG. However, a coherent computational theory linking these observations to function has eluded the neuroscientists. Bottom-up computational approaches have addressed the dynamical properties and interaction of the neural activity in the BG nuclei, while top-down approaches rather have described BG function inspired by machine learning algorithms. In this workshop, we will discuss progress made in our understanding of the BG at multiple scales with the aim to bridge between bottom-up and top-down approaches. The main emphasis of the workshop will be to understand how the dynamics relate to function and dysfunction of BG. In additional the workshop will foster an interaction between experimentalists and theoreticians.

Speakers: Izhar Bar-Gad, Gilberto Fisone, Anne Graybiel, Sten Grillner, Kevin Gurney, Dieter Jaeger, Jeanette Hellgren Kotaleski, Arvind Kumar, Abigail Morrison, Gilad Silberberg, Jeff Wickens, Charles Wilson

Noise and nonlinear dynamics in neural information processing (in honor of the late Frank Moss)

Hans Liljenström (SLU, Uppsala and Agora for Biosystems, Sigtuna, Sweden), Christian Finke (University of Oldenburg, Germany), Hans A. Braun (University of Marburg, Germany)
The objective of this workshop is to bring together different theoretical and experimental approaches for a better understanding of how noise and nonlinear neurodynamics is related to cognitive and autonomous brain functions. This implies elucidating the functionally relevant interdependences between different organisational levels and time scales of brain dynamics - from ion channels over neurons and networks to system functions and behaviour.

Different modelling concepts with different realizations of neuronal elements and couplings shall be presented. Neuron-based models shall be compared with higher level approaches and physical/technical systems to elucidate general rules of stochastic systems control as well as dynamically relevant differences. Special emphasis will be put on the analysis of phase transitions between different types of oscillatory and chaotic activity and the accompanying alterations of the system's responses to external stimuli and noise.

Speakers: Hans A. Braun, Adi R. Bulsara, Dante Chialvo, Christian Finke, Jan Freund, Hans André Longtin, Jose Mateos, Svetlana Postnova, Luigi M. Ricciardi, Epaminondas Rosa Jr., Kenneth Showalter, C. Eugene Bennett, Daisuke Takeshita

Dendrite function and wiring: experiments and theory

Hermann Cuntz, Michiel Remme, Ben Torben-Nielsen and Jaap van Pelt
Neuronal dendritic trees are complex structures that endow the cell with powerful computing capabilities and allow for high neural interconnectivity. Studying the function of dendritic structures has a long tradition in theoretical neuroscience, starting with the pioneering work by Wilfrid Rall in the 1950's. Recent advances in experimental techniques allow us to study dendrites with a new perspective and in greater detail. For example, dendritic function can now be studied in awake, behaving animals. Also, owing to the precise characterization of neural circuits, the role of the single dendrite can be studied in the context of its connectivity. The goal of the workshop is to provide a resume of the state-of-the-art in experimental, computational and mathematical investigations into the functions of dendrites in a variety of neural systems.

28 July

Computational Approach to the etiology of Alzheimer's disease

Jaeseung Jeong, Ph.D (KAIST, South Korea) Justin Dauwels, Ph.D (Nanyang Technological University, Singapore)
Alzheimer's disease is a complex, dynamic disorder, thus computational approach to its etiology based on integrative network models is so significant particularly for diagnosis and prediction of the disease. Recently, computational modeling of Alzheimer's disease has been extensively performed from cellular to systems levels arising from different hypotheses (amyloid beta, tau, and other proteins; hub attack and lethality; synaptic compensation mechanism for disease progression etc). In this workshop, we introduce recent computational studies on the etiology and disease progression of Alzheimer's disease using single cell model and damage model of complex network for the Alzheimer's brain. Firstly, neurobiology of Alzheimer's disease is briefly reviewed and computational approaches (dynamic, statistical, complex network, connection models) are presented, respectively. Computational models for EEG generation are also demonstrated. Finally, the implication of computational models is actively discussed for early diagnosis, and disease progression prediction of Alzheimer's disease. We believe that this workshop is helpful for integrative understanding of Alzheimer's disease.

Speakers: Michael E Hasselmo, Carmen Canavier, Lydia S. Glaw or Thomas C. Skalak, Fernando Buarque de L. Neto, Glosser G., Samanwoy Ghosh-Dastidar, Włodzisław Duch, Justin Dauwels, Jaeseung Jeong

Multi-Scale Modeling in Computational Neuroscience: Challenges and Opportunities

James M. Bower, UTSA; Ilya Rybak, Drexel University
The U.S. National Institutes of Health, the U.S. National Science Foundation, and several other US federal funding agencies have recently organized a cross agency consortium on Multi-Scale Modeling with working groups covering a wide range of biological systems and research approaches. The IMAG (Interagency Modeling and Analysis Group) has organized a wiki to centralize the discussion and description of multi-scale modeling in biological research (http://www.imagwiki.nibib.nih.gov/mediawiki/index.php?title=Main_Page).

One of working groups that is part of this larger effort is focused on the development and use of multi-scale modeling in computational neuroscience. Drs. Bower and Rybak are the organizing leaders for this working group. In this CNS 2011 workshop, Drs. Bower and Rybak will lead a discussion around the challenges and opportunities represented by multi-scale modeling in computational neuroscience. The workshop will start with several short presentations of case studies in the current application of multi-scale modeling in computational neuroscience. The majority of the workshop will then be devoted to discussing the technical issues, scientific opportunities, and developments and directions necessary to support multi-scale modeling. The results of this discussion will be added to the IMAG wiki and will be presented to the Multi-scale Modeling Consortium in August.

This workshop represents an opportunity for the CNS community to influence the direction of future funding for modeling in general and multi-scale modeling efforts in particular. The organizers would be happy to receive proposals for short talks (no more than 15 minutes long) demonstrating the actual biological applications of multi-scale modeling.

Reciprocal interactions of dynamical changes in network structure and function

Markus Butz and Arjen van Ooyen (Neuroscience Campus, VU University Amsterdam)
Network structure determines the flow of electrical activity in every neural network and determines its functional and computational properties. Electrical activation of the neuron goes along with an intracellular increase in calcium which induces morphological alterations of the neuron on a slower time scale. Morphological changes, such as changes in dendritic spine and axonal bouton numbers as well as elongation, retraction and branching of axons and dendrites have direct impact on network connectivity (structural plasticity) even in the adult brain: As a consequence of morphological changes, synapses may break, new synapses can form and axonal branches can be re-routed. Rewired network connectivity, in turn, gives rise to an altered activity dynamic and may hold as a source for long term memory formation. Experimental data further support the notion that structural plasticity is not necessarily Hebbian-like but may serve as a neuronal mechanism to maintain electrical activity at a certain setpoint (neuronal homeostasis). Local structural changes at the single neuron compensating lasting disturbances in electrical activity may entail alterations in global network topology. Conversely, global topology can have impact on local compensation since certain network topologies may better support local structural plasticity mechanisms for maintaining neuronal homeostasis than others. These complex reciprocal interactions between structural changes and activity dynamics as well as local and global effects of structural plasticity necessitate theoretical modeling approaches to elucidate rapidly growing experimental data showing structural plasticity during memory formation and following brain lesions and degeneration. Therefore, the goal of this workshop is to bring together experimentalists and theoreticians in each session. Every session addresses one particular aspect of this topic and closes with a plenary discussion with all speakers of this session to integrate the different perspectives and approaches.

Supercomputational Neuroscience – tools and applications

Abigail Morrison, Markus Diesmann, Anders Lansner
The increasing availability of supercomputers presents the neuroscientific community with unprecedented opportunities to perform novel and groundbreaking brain research, but also with formidable challenges. These challenges include integrating data from multiple brain areas to define large-scale models and developing appropriately scalable tools to specify, simulate, visualize and analyze such complex models. This workshop will feature front-line research on the use of supercomputers and other parallel computers (e.g. GPUs) for neuroscience. The intention is to highlight different aspects of data analysis and tool development for large-scale modeling as well as new biological insights resulting from the application of such tools.

Potential speakers: Eugene Izhikevich, Dharmendra Modha, James Kozloski, Nikil Dutt, Klaus Hilgetag, Markus Kaiser, Ingo Bojak, Tobias Potjans, Mikael Djurfeldt, Susanne Kunkel

Relevance of coherent neural activity for brain functionality

Alessandro Torcini (Institute of Complex Systems, Italy) Michael Rosenblum (Universitaet Potsdam, Germany)
Collective neural oscillations have been observed in many various contexts of brain activity, ranging from ubiquitous gamma-oscillations to theta-rhythm in the hyppocampus, and their role is considered to be crucial for computation and information processing in neural systems. Furthermore, coherent population activities are believed to provide the key mechanism linking sensory stimulation, internal memory, and actual behavior of humans and higher animals. The topic of collective dynamics has been also extensively addressed in mathematical and numerical studies of networks and networks of networks, with a recent emphasis on non-trivial collective dynamics like collective chaos and chimera

states. Following the pioneering studies by Abbot and van Vreeswijk on asynchronous states and partial synchronization in neural networks, new non-trivial collective phenomena have been revealed in neural systems with linear and nonlinear synaptic coupling within the last few years.

This workshop aims to provide a forum to discuss state-of-the-art on coherent behaviours in neuronal populations from the point of view of computational neuroscience. The main focus will be to understand the relevance of the observed population dynamics for the brain activity, therefore a frank and open discussion with experimental neuroscientists is planned at the end of the workshop.

The workshop will be organized in 10 presentations of 20 minutes each plus 5 minutes for questions/discussion. A final session of 30 minutes is planned for an ample discussion.

Speakers: Mario Di Poppa, Denis Goldobin, Moritz Grosse-Wentrup, David Hansel, Kari Hoffmann, Simona Olmi, Carl van Vreeswijk, Kyle Wedgwood, John A. White, Michael Zaks

Method of Information Theory in Computational Neuroscience (day 2)

Simon Schultz (UCL), Michael Gastpar (UC Berkeley), Aurel A. Lazar (Columbia), Todd Coleman (UIUC)
Methods originally developed in Information Theory have found wide applicability in computational neuroscience. Beyond these original methods there is a need to develop novel tools and approaches that are driven by problems arising in neuroscience.

A number of researchers in computational/systems neuroscience and in information/communication theory are investigating problems of information representation and processing. While the goals are often the same, these researchers bring different perspectives and points of view to a common set of neuroscience problems. Often they participate in different fora and their interaction is limited.

The goal of the workshop is to bring some of these researchers together to discuss challenges posed by neuroscience and to exchange ideas and present their latest work.

The workshop is targeted towards computational and systems neuroscientists with interest in methods of information theory as well as information/communication theorists with interest in neuroscience.

Speakers: Ryan T. Canolty, Simona Cocco, Todd P. Coleman, Susanne Ditlevsen, A. Aldo Faisal, Olivier Faugeras, Michael C. Gastpar, Erol Gelenbe, John A. Hertz, Kilian Koepsell, Christian Machens, Eftychios A. Pnevmatikakis, Sridevi V. Sarma, Simon R. Schultz, Tatyana Sharpee

Methods of Systems Identification for Studying Information Processing in Sensory Systems

Aurel A. Lazar (Columbia), Mikko I. Juusola (Sheffield University)
A functional characterization of an unknown system typically begins by making observations about the response of that system to input signals. The knowledge obtained from such observations can then be used to derive a quantitative model of the system in a process called system identification. The goal of system identification is to use a given input/output data set to derive a function that maps an arbitrary system input into an appropriate output.

In neurobiology, system identification has been applied to a variety of sensory systems, ranging from insects to vertebrates. Depending on the level of abstraction, the identified neural models vary from detailed mechanistic models to purely phenomenological models.

The workshop will provide a state of the art forum for discussing methods of system identification applied to the visual, auditory, olfactory and somatosensory systems in insects and vertebrates.

The lack of a deeper understanding of how sensory systems encode stimulus information has hindered the progress in understanding sensory signal processing in higher brain centers. Evaluations of various systems identification methods and a comparative analysis across insects and vertebrates may reveal common neural encoding principles and future

research directions.

The workshop is targeted towards systems, computational and theoretical neuroscientists with interest in the representation and processing of stimuli in sensory systems in insects and vertebrates.

Speakers: Mikko I. Juusola, Aurel A. Lazar, Stefano Panzeri, Gonzalo G. de Polavieja, Barani Raman, Hugh P.C. Robinson, Shihab A. Shamma, Garrett B. Stanley

Towards multimodal modeling: Linking cortical network models to various types of neural measurements

Gaute T. Einevoll (Aas, Norway), Alain Destexhe (CNRS, Gif-y-Survette), Jeanette Hellgren Kotaleski (Stockholm, Sweden), Marja-Leena Linne (Tampere, Finland), Daniel Wojcik (Warsaw, Poland)
A host of experimental techniques are now available for studies of activity in cortical columns or cortical areas. In addition to intracellular and extracellular recordings with various types of single-contact or multi-contact electrodes, several imaging techniques (e.g., intrinsic optical, two-photon calcium, voltage-sensitive dye) have been developed and refined in the last decade. Until now cortical network models have typically been compared with measured spike trains only, and comparison of candidate models against other measurements as well offers new possibilities for distinguishing between good and bad models. This requires, however, that the links between neural activity and what is recorded in the experiments are well understood and accurately modeled. These links, i.e., "the physics of neural activity measurements", are the topic of the workshop. In the first part the underlying physics of the most prominent experimental techniques measuring population activity is presented. In the second part the focus is on the development of neuroinformatics tools aiming to facilitate such multimodal modeling.

Non-invasive imaging of non-linear interactions

Alex Ossadtchi (St. Petersburg State University)
Interactions between neuronal assemblies form a basis for the mechanism of functional integration that underlies most of our actions. Studying these interactions is important for understanding the principles of brain operation. However, such neuronal communications are hallmark of a living brain and therefore it is of paramount importance to develop tools for imaging of these interactions on the basis of non-invasively collected data.

The interactions manifest themselves in synchrony of activity of neuronal assemblies. Recent years demonstrated a dramatic increase in the number of studies dealing with synchrony detection and analysis on the basis of EEG and MEG data. Several reliable algorithms have been developed, however very few studies dealt with the task of source-space analysis of non-linear (cross-frequency) interactions on the basis of non-invasive data.

Validation of real data analysis results are complicated due to the difficulty of establishing the gold standard as the interaction picture may differ from subject to subject. One way to approach this problem is to do validation of the new techniques for imaging of synchrony in application to simultaneously recorded ECoG and EEG/MEG datasets.

Synchrony detection methods should either be built based on statistical considerations or should be followed by a statistical testing step. It is important that such tests include physiologically plausible models balancing the accuracy and the relatively low level of spatial details yielded by EEG and MEG measurements.

Speakers: Richard Greenblatt, Michael Rosenblum, Felix Darvas, Matias Pavla, Alex Ossadtchi, Vadim Nikulin, Vladimir Litvak / Will Penny

»Abstracts

K-01 Functional network connectivity of the control and epileptic hippocampus

Ivan Soltesz

Department of Anatomy & Neurobiology, University of California, Irvine, USA
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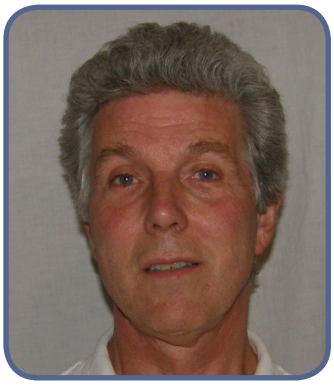
With the rapid rise in our knowledge about the structural and functional properties of hippocampal microcircuits, it has become possible to closely integrate experimental findings with large-scale, anatomically and biophysically realistic computational simulations of control and epileptic neuronal networks with unprecedented precision and predictive power. We are developing full-scale realistic network models of the control and injured temporal lobe in order to investigate fundamental questions related to normal hippocampal microcircuit function and the mechanistic bases of epilepsy. I review the conceptual framework and biological basis of model development and show specific applications, including new computational and experimental results concerning the phase-related firing of various interneuronal subtypes during learning and memory-related hippocampal network oscillations and the roles of aberrant hyper-connected hub-like neurons in seizures. The talk will highlight the unprecedented predictive and analytic power of increasingly user-friendly, freely shared, highly realistic, large-scale computational models in understanding normal circuit function and temporal lobe epilepsy.

K-02 Perceptual and memory functions in a cortex-inspired attractor network model

Anders Lansner

Dept of Computational Biology, School of Computer Science and Communication, Stockholm University and Royal Institute of Technology, Stockholm Brain Institute partner.
E-mail: ala@csc.kth.se



I will initially present the foundations of this work in Hebbs hypotheses of synaptic plasticity and cell assemblies and how perception and memory can be understood in those terms. Then I will turn to the question about biological plausibility, which we have studied by means of modeling and computer simulation, typically using very-large scale Hodgkin-Huxley based network models running on supercomputers. We developed and studied a computational model of cortical layers 2 and 3 where the horizontal connectivity critical in this context is most prominent. Sparse, distributed memory items were embedded in such a network by means of Hebbian synaptic plasticity. I will show how this model can perform basic perceptual and memory functions like perceptual completion and rivalry as well as reconstructive associative recall from fragments of a stored memory. It also reproduces key features of the attentional blink phenomenon. More recently we analyzed the dynamics of these processes in terms of spike discharge patterns, oscillatory dynamics in theta to gamma frequencies, as well as spontaneous ongoing activity, phase locking and coherence. It was shown that some of the dynamic properties depend prominently on the modular structure of the network. It is concluded that a network model set up along the lines proposed by Hebb with cortex-like composition, microcircuit structure, and connectivity can to a significant extent reproduce basic cognitive functions as well as dynamics of real cortex. Finally, directions for further development of this type of models will be discussed, e.g. to use them as components in network-of-network architectures representing complex interacting cortical feedforward, lateral and processing streams.

Acknowledgments: This work is supported by Swedish Science Council, European Commission (FACETS, NeuroChem, and BrainScaleS projects), VINNOVA, and Swedish Foundation for Strategic Research.

K-03 Long-lasting neuronal desynchronization caused by coordinated reset stimulation

Peter A. Tass

Institute of Neuroscience and Medicine - Neuromodulation (INM-7), Research Center Juelich, 52425 Juelich, Germany & Department of Stereotactic and Functional Neurosurgery, University Hospital, 50924 Cologne, Germany

A number of brain diseases, e.g. movement disorders such as Parkinsons disease, are characterized by abnormal neuronal synchronization. Within the last years permanent high-frequency (HF) deep brain stimulation became the standard therapy for medically refractory movement disorders. To overcome limitations of standard HF deep brain stimulation, we use a model based approach. To this end, we make mathematical models of affected neuronal target populations and use methods from statistical physics and nonlinear dynamics to develop mild and efficient control techniques. Along the lines of a top-down approach we test our control techniques in oscillator networks as well as neural networks. In particular, we specifically utilize dynamical self-organization principles and plasticity rules. In this way, we have developed coordinated reset (CR) stimulation, an effectively desynchronizing brain stimulation technique. The goal of CR stimulation is not only to counteract pathological synchronization on a fast time scale, but also to unlearn pathological synchrony by therapeutically reshaping neural networks. The CR theory, results from animal experiments as well as clinical applications will be presented: Animal and human data will be shown on electrical CR stimulation for the treatment of Parkinsons disease via chronically implanted depth electrodes.

Furthermore, acoustic CR stimulation for the treatment of subjective tinnitus will be explained. Subjective tinnitus is an acoustic phantom phenomenon characterized by abnormal synchronization in the central auditory system. In a multicenter proof of concept study it has been shown that acoustic CR stimulation significantly and effectively counteracts tinnitus symptoms as well as the underlying pathological neuronal synchronization processes.

F-01 Need for homeostasis in electrical activity may account for cortical network rewiring

Markus Butz[1], Arjen van Ooyen[1]

1 Department of Integrative Neurophysiology, Computational Neuroscience Group, VU University Amsterdam
Email: mbutz@falw.vu.nl

Current thinking about how the brain learns from experience and encodes memories is focused on changes in the strengths of connections between neurons, whereby the pattern of connections is considered fixed. However, the adult brain is not as hard-wired as traditionally thought. Neurons establish new connections and break existing ones (structural plasticity). Although structural plasticity associated with brain lesions (stroke) and degeneration is known since the late 1960s, its relevance for cognitive functions such as learning and memory [4,8] and its contribution to network repair after lesions [5,7,9] still remain unclear. Current computational models are insufficient to address this issue since network structure is considered as fixed with plasticity merely arising from changes in connection strengths of existing synapses. The present work aims to elucidate the underlying organizing principles that drive structural plasticity and brain repair induced by lesions. We hypothesize that the need of neurons to maintain their average electrical activity at a particular level (homeostatic regulation) drives lesion-induced restructuring of cortical circuits, and can predict changes in connectivity and spine/bouton/synapse numbers experimentally observed following cortical deafferentations such as a focal retinal lesion [5,9]. In this view, loss of input due to lesions disturbs the homeostasis of neuronal activity, which, through activity-dependent neurite outgrowth and synapse formation, triggers compensatory structural changes that attempt to regain homeostasis [2].

To explore whether the experimentally observed structural changes can be accounted for by the neuronsâ€™ need for homeostasis in electrical activity, we constructed a neuronal network model in which each neuron receives a vertical input stream (from the eye via the thalamus) and a horizontal input stream (from other neurons within the cortical network). The focal retinal lesion is modeled as a circumscribed removal of the vertical input stream. To model structural changes of the neuron, we created a novel model formalism in which each neuron has a number of axonal elements (representing boutons) and dendritic elements (representing spines) [1,3]. Synapses are formed by merging axonal and dendritic elements. In line with the experimental data, the number of these elements can change depending on the neuronâ€™s own level of electrical activity, which may cause existing synaptic connections to break or new ones to form. A neuron will change the number of its elements so as to try to maintain its average activity at a particular set-point. So, when its activity is above the set-point, it will eliminate (dendritic) elements, and when activity is below the set-point it will generate new elements. However, when activity is too low, neurons will also lose axonal and dendritic elements. Electrical activity is generated by a conductance-based spiking neuron model with intracellular calcium as a measure of the average electrical activity of the single neuron [6].

By this approach we could show that the neuronal need for homeostasis in electrical activity can account for the structural changes observed in the visual cortex after focal retinal lesions [5,9]. We hypothesize that the precise interplay between axons and dendrites as well as the topology of the network matter for the experimental results. In this study, we investigated different scenarios depending on the calcium-dependency of axonal growth, which may give insight into potential approaches for promoting network repair in experimental deafferentation studies. In addition to making testable predictions, our network model is the first to describe the reciprocal dynamics of activity-dependent network rewiring. Our findings on topological changes are in line with recent data from stroke patients [7] and may imply a more general principle of the mechanisms involved in brain repair. A better understanding of neuronal network repair following lesions is urgently needed for a better treatment of neurological diseases such as stroke.

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F-02 Neuronal mechanisms of shift workers' sleepiness

Svetlana Postnova[1,2], Peter A Robinson[1,2,3]

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2 Centre for Interdisciplinary Research and Understanding of Sleep (CIRUS), Woolcock Institute of Medical Research, University of Sydney, New South Wales 2037, Australia
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E-mail: postnova@physics.usyd.edu.au

In an attempt to use all 24 hours of the day, shift work is becoming increasingly popular. Long term shift work leads to multiple health problems, including higher risk of cardiovascular diseases, mood disorders, and diabetes. Other consequences include loss of concentration and increase of sleepiness resulting in accidents [1]. Given the prevalence of shift work and the severity of the associated hazards, understanding of the mechanisms underlying sleepiness and predictions of shift workers' fatigue would prove highly valuable. A number of mathematical models addressing these questions exist in a current literature. However, most of them are phenomenological and are limited to studying short-term effects of shift work; i.e., sleepiness only during the first couple of days on the shift [2].

The model that is used here follows a different, physiologically-based approach, combining a quantitative model of sleep-wake switch [3] with a model of the human circadian pacemaker entrained by light and non-photic inputs [4]. This model accounts for the state-related transitions in the firing of wake-active monoaminergic (Fig.1A) and sleep-active ventrolateral preoptic nuclei in the brain (Fig.1B) under the influence of homeostatic and circadian drives shown in Fig.1C and D. Homeostatic drive is responsible for accumulation of sleep pressure during wakefulness, while the circadian drive, which is controlled by the suprachiasmatic nucleus of the hypothalamus, provides a 24 hour periodicity of sleep-wake cycles, and is entrained by external light/dark cycle (Fig.1F) and non-photic stimuli. During shift work (pink areas in Fig.1) external cues, as well as sleeping times (blue areas in Fig.1) are changed, affecting the total sleep drive and sleepiness of shift workers (Fig.1E).

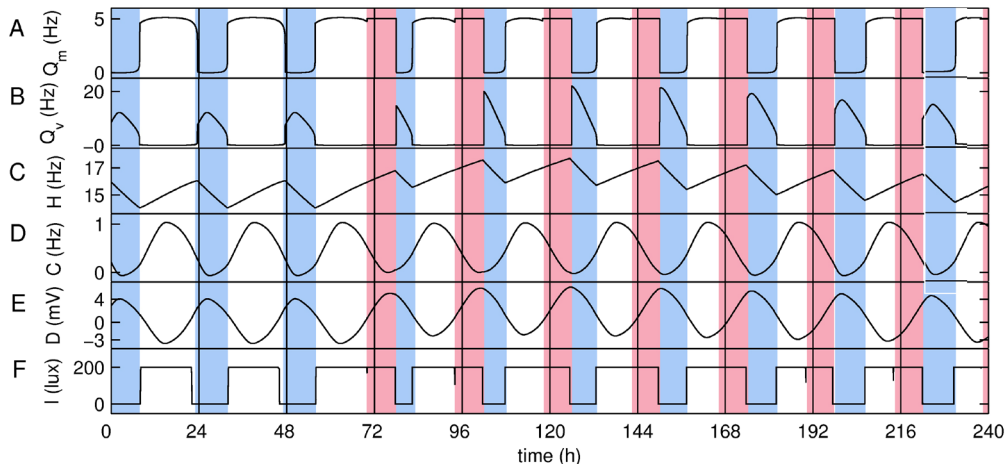

Figure 1: Sleep-wake activity with and without shift work. Permanent shifts are introduced on the 3rd day of the simulation between 22.00 and 06.00. Variables shown on the plot are: mean firing rate across the neurons in (A) monoaminergic and (B) ventrolateral preoptic nuclei, (C) homeostatic sleep drive, (D) circadian process, (E) total sleep drive, and (F) intensity of light to which the worker is exposed.

Using this model the physiological mechanisms responsible for shift-related changes in sleepiness are examined in the simplest case of permanent shift work. In good agreement with experimental data sleepiness was shown to increase

during the first days on the evening, night and early morning shifts. This is explained by the inability to sleep enough during the active circadian phase and the thereby increased homeostatic pressure. After this initial increase, sleepiness decreases, and stabilizes due to circadian entrainment to the new external cues provided by the shifts. The entrainment time and the degree of sleepiness are higher for the shifts leading to a stronger change of the circadian phase comparing to the no-shift situation. The performance of shift workers was shown to be improved by increasing lighting intensity at work place and by decreasing light during breaks. Altogether, this model has shown to be a powerful tool for the research of mechanisms of sleepiness, and for design of optimal shift schedules.

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F-03 Chaotic decorrelation of Globus Pallidus by periodic forcing: A possible mechanism for the therapeutic effects of deep brain stimulation

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High frequency (~100 Hz) stimulation of the subthalamic nucleus is an effective therapy for the symptoms of Parkinson's disease. Presumably, stimulation partly normalizes a pathological change in rate or pattern of neuronal activity in the output neurons of the globus pallidus. The rate effects of subthalamic stimulation are paradoxical, because firing rate in the globus pallidus is increased in Parkinson's disease, and subthalamic input is excitatory. Parkinson's disease is also accompanied by changes in globus pallidus cell firing pattern. Pallidal neurons are autonomous single-spiking oscillators that fire continuously and usually show no periodic bursting. They fire independently of each other, having flat cross-correlations. In Parkinson's disease the cells burst at 5-10/s and are highly correlated [1]. Rubin and Terman [2] have proposed that subthalamic stimulation entrains globus pallidus cells interfering with the low frequency bursting. In their model, the synchrony of globus pallidus cells should be high, because all cells are entrained by the same stimulus. The periodic nature of deep brain stimulation is inconsequential in their model.

A recent clinical study has shown that the periodicity of deep brain stimulation is critical to its effectiveness [3]. Poisson stimulation at the same rate was much less beneficial. This observation, coupled with the frequency dependence of DBS effectiveness, suggests that disruption of low frequency bursting may not be the only mechanism of action. Globus pallidus cells are oscillators, so it is possible that periodic stimulation may not only suppress low frequency bursting, but also disrupt synchrony by driving the cells into chaotic firing patterns.

We examined the range of stimulus frequencies and amplitudes required to chaotically desynchronize pallidal neurons. Oscillating pallidal cells were represented by their phase resetting curves. We used a type 1 phase resetting curve similar to that measured from model pallidal neurons [4]. The variance of the phase resetting curve was estimated as in Ermentrout et al [5] and used to form a stochastic phase map. The stationary distribution of latencies and the phase resetting curve were used to calculate Lyapunov exponents for both hyperpolarizing and depolarizing stimulation, and compared to the synchronizing or desynchronizing effect of stimulation on a population of simulated pallidal neurons that were weakly synchronized by common noisy input. Powerful synchronization and desynchronization were evoked at different frequencies as predicted by the sign of the Lyapunov exponents. Chaotic decorrelation in response to de-

polarizing stimulation occurred at frequencies similar to those known to be most effective for deep brain stimulation clinically.

We suggest that in addition to its effects on low frequency oscillations in the pallido-thalamic projection, deep brain stimulation decorrelates the activity of pallidal neurons by driving them into a chaotic firing pattern. When the stimulus is randomized the chaotic response is destroyed, explaining why it is less effective. This chaotic effect may be essential to the clinical benefit of stimulation, being responsible for its frequency sensitivity and the requirement for periodicity in the stimulus pattern.

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O-01 The modular cross-synaptic nature of LTP/LTD following on-going neural activity

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While synaptic efficacies are modified continuously by on-going spiking activity, it is yet unclear whether the underlying pre- and post-synaptic processes occur independently, or in accordance. To elucidate the effects of sustained spiking communication on synaptic properties, we patch-clamped paired pyramidal neurons in-vitro at both ends of 12h intervals of spontaneous or glutamate-induced spiking activity. We found that the synaptic efficacies either increased, or decreased, with the ratio between the second and first measurement ranging between 0.08-14. Using quantal and failure analyses we show that this slow form of long-term potentiation and depression is explained by changes in the estimated number of release sites, alongside overall post-synaptic changes that maintain the quantal size per release site. Our findings suggest that sustained neural activity results in matched pre- and post-synaptic modifications, in which elementary modules that span the synaptic cleft are added or subtracted as a function of experience.

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O-02 The emergence of long timescales and stereotyped behaviors in C. elegans

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Animal behaviors are often decomposable into discrete, stereotyped elements, well-separated in time. In one model, such behaviors are triggered by specific commands; in the extreme case, the discreteness of behavior is traced to the discreteness of action potentials in the individual command neurons. Here, we use the crawling behavior of the nematode C. elegans to demonstrate the opposite view, in which discreteness, stereotypy and long timescales emerge from the collective dynamics of the behavior itself. In previous work [1], we found that as C. elegans crawls, its body moves through a ``shape space'' in which four dimensions capture ~95% of the variance in body shape. Here we show that stochastic dynamics within this shape space predicts transitions between attractors corresponding to abrupt reversals in crawling direction. With no free parameters, our inferred stochastic dynamical system generates reversal time scales and stereotyped trajectories in close agreement with experimental observations. We use the stochastic dynamics to show that the noise amplitude decreases systematically with increasing time away from food, resulting in longer bouts of forward crawling and suggesting that worms can use noise to adaptive benefit.

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O-03 Spike-timing theory of working memory

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It is commonly assumed that long-term memory is represented by patterns of synaptic connections within groups of

neurons and that memory recall corresponds to an activation of a group triggered, e.g., by a sensory stimulus. Sustained spiking activity of one or a few selected long-term memory representations is believed to be the neural correlate of working memory.

The capacity of working memory is referred to as being the number of neuronal groups that could be maintained active at the same time. This short-term memory capacity is, for example, thought to be seven plus or minus two items for ordered lists. We distinguish the short-term capacity from long-term memory capacity or repertoire, which is the large number of neuronal groups required to store many distinct memories formed by past sensory experience.

We propose [1] a mechanism for working memory that allows for a greatly expanded long-term memory capacity, and we demonstrate how this mechanism can simultaneously maintain in working memory a few items out of this huge repertoire (which is far greater than the number of neurons).

We assume that (i) the groups of neurons representing long-term memories are largely overlapping and (ii) neurons (within a group) are capable of exhibiting precise firing patterns, so different representations are distinguished not only by which neurons fired, but also by their exact spiking patterns. This is in contrast with previously suggested mechanisms of working memory, where the spike-timing nature of neuronal activity is ignored and the models' explanatory power is limited to systems having small repertoires of long-term memories represented by, e.g., carefully selected non-overlapping populations of neurons.

Using associative short-term synaptic plasticity in the form of short-term STDP, we demonstrate that a few neuronal groups can be simultaneously selected to transiently be part of working memory, i.e., show elevated and precise firing activity for seconds after initial activation.

Our theory explains the relationship between precise spikes and slowly changing firing rates of neurons engaged in active maintenance of working memory, and it points to the connection between working memory and perception of elapsed time on the order of seconds.

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O-04 Scale-free statistics of neuronal assemblies predict learning performance

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Recent years have seen a profusion of research and controversy surrounding the hypothesis that the brain may be operating at criticality. While experimental evidence supporting the presence of scale-free avalanches in neuronal activity characteristic of a critical state has been steadily accumulating, the interpretation of these data as evidence for critical brain dynamics has simultaneously been questioned (see [1, 2] and references therein). From a neuroscientific point of view, the principal appeal of the criticality hypothesis is that critical dynamics have been repeatedly linked to improved information processing, including increased memory capacity, dynamic range, and computational power (see [2] and references therein). Thus far, however, the role that neuronal avalanches - whether hallmarks of criticality or not - might play in complex mental processes such as learning and memory has remained largely unexplored.

In this paper, we show that scale-free cascades of neuronal activity also characterize artificial neural networks performing Hebbian learning tasks [3, 4]. Our experimental framework enables us to simultaneously measure the network's performance on the task while observing the corresponding size-distribution of neuronal assemblies. These phenomena are assessed as a function of both network topology and the degree of excitation versus inhibition.

Smoothly tuning the degree of order in the network topology, we find that task-performance is optimized in the dy-

namical state associated with scale-free cascades of exponent $2 < \alpha < 3$ which occur in so-called 'small-world' networks characterized by high local clustering and short path-length. A transition to ordered, lattice-like, networks is accompanied by both the sudden onset of severely impaired performance and the loss of scale-invariance in neuronal responses. Furthermore, we find that this correlation between scale-free neuronal cascades and performance also holds true when variations in performance are achieved by altering the network's excitatory-inhibitory balance. Performance is optimized in balanced networks, whilst excitation-dominated states are characterized by a simultaneous drop in performance and the loss of scale-invariance. This is in keeping with experimental studies in cortical slices where the application of picrotoxin, a GABA-receptor antagonist that reduces inhibitory activity, has been shown to result in the loss of power laws in local field potential recordings [5].

Finally, we note that the performance of the system is more robust to deviations from excitatory-inhibitory balance in small-world networks than in random ones. These results may provide insight into psychiatric diseases such as schizophrenia where decreased inhibitory activity [6] and a disruption of topological organization [7] have both been observed alongside the well-known cognitive impairments associated with the disease.

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O-05 A cell-type-specific dynamic Bayesian network model for spontaneous and optogenetically evoked activity in the primary visual cortex

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Reciprocal interaction between excitatory and inhibitory neurons within and between layers of the cerebral cortex is a major element of brain function. Ensemble extracellular recording techniques using mircroelectrode arrays have permitted recording spiking activity of many neurons simultaneously to characterize network function [1]. Identifying the type of neurons in these recordings is not straightforward due to the variability in extracellular spike shapes, and the irregularities often observed in their interspike interval characteristics. In this study, we used optogenetic tools to genetically target fast spiking interneurons in the primary visual cortex of mice [2]. We modulated their spiking activity by illuminating the region with very short pulses (<1 ms) of light (~470 nm wavelength)in mice primary visual cortex. Using Dynamic Bayesian Network analysis [3], we assessed the effective connectivity between the recorded neurons in the presence and absence of light stimuli under distinct cortical states observed under light anesthesia. DBNs could identify the effective connectivity between putative excitatory pyramidal cells and inhibitory interneurons. These findings suggest a novel and unprecedented way to identify cortical neuronal circuits and characterize the dynamics of their computations in vivo.

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O-06 State-dependent modulation of stimulus-response relations in cortical networks in vitro

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Variable responses of neuronal networks to repeated identical electrical or sensory stimuli reflect the interaction of the stimulus' response with ongoing activity and its modulation by adaptive mechanisms such as cognitive context, network state, cellular excitability or synaptic transmission capability. To identify the rules that underlie the modulation of stimulus-response relations we set up a state-dependent stimulation paradigm in generic neuronal networks in vitro. Extracellular neuronal activity was recorded and stimulated from rat cortical cell cultures on microelectrode arrays at 60 sites. Spontaneous and evoked network activity was examined under control conditions, under blockage of GABAA-receptors as well as under overexpression of the synaptic protein DOC2B [1]. We were interested in the interactions that arise between spontaneous and stimulus-evoked activity dynamics and how these shape and modulate stimulus-response relations.

Spontaneous network activity consisted of recurring periods of globally synchronized burst firing, so-called network bursts. The duration of intervals that preceded network bursts best predicted the length of the following network burst. This supported a process of network depression to a low threshold during bursts followed by subsequent recovery [2]. Facilitation of synaptic transmission by overexpressing DOC2B yielded ~30 % more spikes per network burst. The intervals between bursts increased by ~ 75 %, suggesting interdependence between resource activation and the time it needs for them to be recovered.

Response length and delay depended on the timing of stimulation relative to preceding bursting. Response length increased exponentially and saturated with increasing duration of pre-stimulus inactivity t, $y(t) = A(1-e^{-\alpha t})$. Response delay, in turn, decreased exponentially and saturated at a low level, $y(t) = Be^{-\beta t} + C$. The rate constant β describes the coupling between recovery from depression and response delay. We found activity-dependent recovery dynamics with longer spontaneous bursts yielding smaller β and vice versa.

Stimulus-response modulation persisted under the blockage of inhibition, that introduced overall longer responses and shorter delays. Longer network bursts with more spikes occurred less frequently and recovery rates concomitantly decreased. The average firing rate was, however, unchanged, supporting a pool of available resources that is repeatedly used and replenished during and between network bursts, respectively.

Conclusion The timing of stimulation relative to spontaneous bursting modulates stimulus-response relations in cortical networks in vitro following distinct rules. The interrelation between resource depletion and replenishment determines the temporal evolution of the network's excitability state. Our findings can be explained by short-term synaptic depression and activity-dependent adaptation of excitability as underlying mechanisms.

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O-07 Mechanisms underlying the emergence of Up and Down states in a model PFC microcircuit

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Up and Down states are oscillations between periods of prolonged activity (Up state) and quiescence (Down state) and are recorded both in vivo and in vitro in layer V prefrontal cortex (PFC) pyramidal neurons. Biophysical mechanisms that have been proposed to underlie this phenomenon include the balance of excitation and inhibition within local PFC networks [1] along with certain intrinsic membrane mechanisms such as the afterdepolarization [2]. Using a biophysical compartmental network model of PFC layer V pyramidal neurons that incorporates anatomical data (as described in [3]), we investigated the role of synaptic input, intrinsic currents and local interconnectivity in the following features of Up and Down states: (a) the emergence of Up and Down states, (b) the duration of Up states, (c) the frequency of Up states and (d) the firing frequency during the Up state.

We found that Up and Down states could emerge in our model microcircuit (see Figure 1), provided the existence of background synaptic activity. Among the various conditions we examined, statistically significant results were obtained when:

- Increasing the firing frequency of the background synaptic input or the number of activated background synapses (Up frequency increased by ~150% and 60%, respectively, firing frequency increased by ~30% and 50%, respectively).

- Blocking the NMDA current, while compensating for the reduced excitability by enhancing the AMPA current (no emergence of Up and Down states).

- Increasing the iNMDA-to-iAMPA ratio from 1 to 1.5 (Up frequency increased ~190%, firing frequency increased by 25%, Up duration doubled).

- Activating the dADP mechanism at a physiological value (4mV) (Up frequency increased by ~ 200%, firing frequency increased by 60%, Up duration doubled).

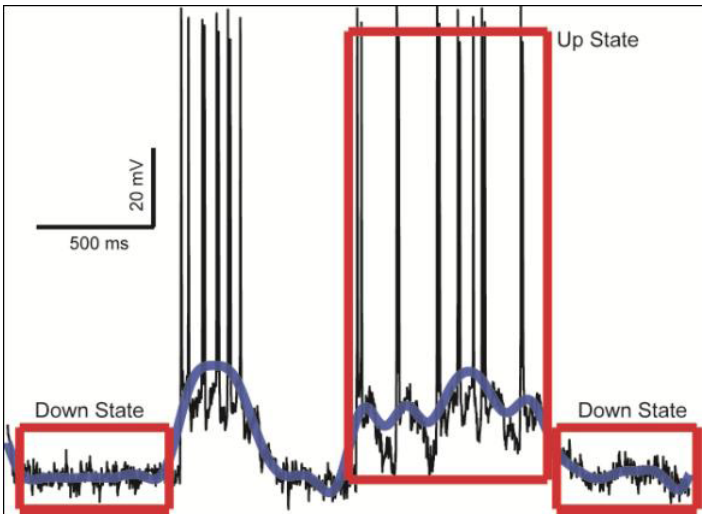

Figure 1: Representative trace (black) of Up and Down states. Blue trace is the signal after it has been filtered with the Butterworth filter. Red boxes are indicative of Down States and an Up state that meets the criteria (500 ms duration and above -60 mV depolarization plateau).

Conclusions: Our results indicate that the generation of Up states in PFC is likely to involve not only a balance of excitation/ inhibition provided within a microcircuit but also single-neuron dynamics shaped by intrinsic mechanisms. Interestingly, the duration of the Up state was significantly altered in only two of the conditions tested, namely, the enhancement of the NMDA current and the activation of the dADP mechanism. These findings suggest that the transition to more prolonged depolarizations is carefully controlled by the same mechanisms that have been associated with persistent firing during working memory tasks.

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O-08 How local is the local field potential?

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The local field potential (LFP), usually referring to the low-frequency part of an extracellularly recorded potential (< 500 Hz), is nowadays routinely measured together with the spiking activity. The LFP is commonly believed to mainly reflect synaptic activity in a local population surrounding the electrode [1] but how large this population is, i.e. how many neurons contribute to the signal, is still debated. In this modeling study we investigate which factors influence the spatial summation of contributions that generate the LFP signal. A better understanding of this is crucial for a correct interpretation of the LFP, especially when analyzing multiple LFP signals recorded simultaneously at different cortical sites.

We use a simplified two-dimensional model of a cortical population of neurons where the LFP is constructed as a weighted sum of signal contributions from all cells within a certain radial distance to the recording electrode. First we consider a general formulation of the model: if the single-cell LFP contributions can be viewed as current dipole sources [2], the single-cell amplitude will decay as 1/r2 with distance r to the electrode. On the other hand, for the two-dimensional geometry considered here, the number of neurons at a given distance increases linearly with r. In addition to these two opposed scaling factors the amplitude of the summed LFP signal also depends on how correlated the single-cell LFP sources are. We calculate the LFP amplitude as a function of the population radius and relate it to the above factors. We show that if the single-cell contributions decay as dipole sources or more steeply with distance, and if the sources are uncorrelated, the LFP is originating from a small local population. Cells outside of this population do not contribute to the LFP. If, however, the different LFP sources are uniformly correlated, cells at any distance contribute substantially to the LFP amplitude. In this case the LFP reach is only limited by the size of the region of correlated sources. This result highlights that the spatial region of the LFP is not fixed; rather it changes with the dynamics of the underlying synaptic activity.

We further validate these results through LFP simulations of morphologically reconstructed cortical cells [2,3,4] where we study the effects of neuronal morphology on the size of the region contributing to the LFP. Finally, we show the laminar dependence of the reach measure used here and discuss potential implications of the interpretation of experimentally recorded LFPs.

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O-09 Gradient based spinal cord axogenesis and locomotor connectome of the hatchling Xenopus tadpole

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Understanding the mechanisms underlying the self-assembly and organization of functional neuronal networks is a crucial problem confronting both experimental and theoretical neuroscience alike. Early in development, functional neuronal networks self-assemble with astonishing rapidity. It is, therefore, imperative to investigate and understand how far simple basic mechanisms can allow primary functioning neuronal circuits to develop. To address this 'structure-function' issue, we model anatomy and electrophysiology of young hatchling Xenopus tadpole's spinal cord [1, 2, 3]. Our bottom-up approach to modeling of neuronal connectivity is based on developmental process of axon growth – we develop a gradient-based mathematical model for axon growth. It is known that in the developing vertebrate spinal cord, neurons arise from progenitor cells in the neural tube and thereafter the axons grow under influence of chemical morphogenes released from the dorsal roof plate ('BMP'), ventral floor plate ('shh') and hindbrain regions ('Wnt'). Distribution of these guidance molecules along the spinal cord set up a gradient field which steer the axons in appropriate locations and thus ensure formation of proper connections. We grow axons of spinal neurons and generate synaptic connections similar to biological developmental process based on the data from Professor Alan Roberts Lab at University of Bristol [4]. Using the gradient-based model we were able to grow axons for all seven types of spinal neurons which are believed to be involved in swimming and struggling behavior of tadpole. These spinal neurons include sensory neurons which are responsible for receiving stimulus from environment; interneurons which process the sensory information and pass it onto the motor which translate these signals into appropriate locomotive activities. We successfully modeled both morphologically different types of spinal neurons– firstly commissural neurons grow axons ventrally on the same side of the spinal cord at first and turn longitudinally on the other (contralateral) side of the spinal cord, whereas non-commissural neurons grow their axons on the same (ipsilateral) side of spinal cord. The model incorporates experimental data for somata distribution in the spinal cord, the outgrowth angles, axon lengths, etc. The computer modeling of the axon growth of spinal neurons has enabled both us and our Biology partners to construct and test various hypotheses particularly the roles of the different guidance cues in the axon growth. We also implemented an optimization technique to determine the model parameters for a particular type of neuron based on minimizing a cost function which comprises of the difference between the axon trajectory distribution of the model axons and the corresponding experimental data and also difference between the tortuisities of the model and experimental axons. The complete reconstruction of the spinal cord includes about 2000 neurons. In the reconstruction, both the neurons and the dendrites are randomly distributed along the spinal cord on the 'left' and 'right' side of the body according to the experimental measurements. Thus, the reconstruction of the connectivity is biologically realistic. To model spiking activity we use Hodgkin-Huxley type conductance model. Parameters of the model are chosen according to the available neurophysiological measurements. Activity dynamics of neural network comprising both the reconstruction model and conductance based spiking neurons demonstrates swimming patterns, i.e., anti-phase oscillations on opposite sides of the body and metachronal wave in longitudinal direction in a wide range of model parameters.

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O-10 A developmental explanation of the dependence of binaural best delays on characteristic frequency

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Both mammals and birds use interaural time difference (ITD) as a cue to determine the horizontal position of a sound source in space. In the barn owl brainstem, neurons of the Nucleus Laminaris (NL) have firing curves which vary with the acoustic stimulus ITD. Each NL neuron is tuned to a certain ITD in that their response is maximal for a certain ITD, i.e., they respond preferably when the stimuli from the two ears have a certain delay, called best delay (BD) of that neuron. Recently, it has been shown that the distribution of BDs along the tonotopical axis depends on the best frequency (BF) of their corresponding neurons. In particular, BDs rarely exceed 1/(2BF), an approximate constraint called the pi-limit. The origin of this BF-dependent distribution of BDs is still a matter of debate.

In this work, we use a modeling approach to test whether the pi-limit could emerge from activity-driven plasticity, in particular Spike-Timing Dependent Plasticity (STDP). Using a standard peripheral model and physiologically plausible spiking neuron models, we first feed binaural acoustic noise with ITDs in the physiological range of the barn owl (smaller than 250 µs) to NL neurons undergoing STDP and analyze the distribution of the resulting BDs. We show that STDP selects the inputs based on their synaptic delays and that the resulting BD distribution mainly falls between plus or minus half the characteristic period of the corresponding neurons. We then use uncorrelated binaural white noises and show that the ITD selectivity and the dependence of binaural best delays on characteristic frequency also emerges after a simulated developmental period. We finally check that our conclusions also hold in more realistic settings, by feeding the model with real forest recordings.

In conclusion, our results suggest that the frequency-dependence of BDs may simply be a by-product of the way ITD tuning develops in binaural neurons. It does not impair the ability of these neurons to represent the azimuth of sound sources, because BDs that differ by an integer number of characteristic periods are mostly redundant. Perhaps more interestingly, it also provides a complete representation of ITDs which is functional in any acoustical environment, even if the head of the animal continues to grow after the critical development period. This suggests that the distribution of BDs does not simply mirror the statistics of binaural sounds during development, but instead provides a robust representation of changing environments.

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O-11 Decision-making and noise in the assembly of neural circuits

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Accurate decision-making is essential for the correct assembly of sensory-motor circuits, which are composed of neurons whose axonal growth cones execute discrete, binary decisions at sequential trajectory selection points to connect motor neurons to target muscles. Limb motor axon trajectory selection is controlled by guidance cues: ephrin ligands and Eph receptors [1], whose expression levels are variable. Some cues direct axons to opposite trajectories. Genetic inactivation of Eph and ephrin cues results in inaccurate trajectory decisions and incorrect neural circuit topology [2]. To understand this decision making process we developed a model of an axon growth cone as it grows from the spinal chord,passing binary decision regions to innervate its target muscle. Our model linearly sums noisy guidance cues at receptors on the growth cone surface, which in turn drive cytoskeletal dynamics of theor nearest-neighbour microtubule (resulting in the protraction/ of individual filopodia of the growth cone). Our simple model incorporates three basic constraints: 1. guidance cues are noisy signal due to gene expression variability and stochastic ligand-receptor interactions, 2. guidance cue combination is linear and additive and, 3. raw material constraints during growth, limit the total size of the growth cone. Combining these basic constraints suggests a decision-making model that explains in a unified way 4 experimental findings:

First, at binary decision points genetically homogenous populations of axons can partition on the two possible trajectories tissues with an unequal ratio. Unequal ratios are observed in very different developmental decisions e.g. Bacillus subtilis cell fate (80:20), Drosophila photoreceptor type(70:30), the optic chiasma of vertebrate retinae(97:3) and in frog, chicken and mouse motor systems(93:7-96:4) [2,3]. It was unclear how such unequal ratios are reliably produced, as deterministic mechanisms for decision-making could account for 100:0, and 50:50 ratios but not intermediate ones. Second, shifts in partition ratios were observed in genetic mutation studies where cues were removed or added, in our ephrin and Eph mouse mutants, these decision were inordinately variable (from 95:5 in wild-type to 80:20, 60:40, 0:100) [2]. Third, axons growing in genetically symmetrised limbs (where dorsal or ventral limb halves are duplicated) grow with equal probability towards either target or fail to enter the limb [4]. Fourth, experiments showed that growth cones faced with decisions slow their movement considerably (500-1,000%) and grow in diameter (300-1000%). Thus, the decision-making machinery grows commensurately in size and sensing capacity (receptor-covered axon surface area). Our model reproduces this as the repulsive cues for each target competitively engage cytoskeletal material towards targets and reducing availability of material devoted to forward movement. Once decisions are made, our model growth cone shrinks as cytoskeletal material is used for rapid axon growth in accordance with experiments.

In conclusion, we developed a mechanistic model of serial decision-making in axonal growth cones that explains all four experimental findings. Our findings highlight the axons and generally neurons during circuit assembly in development, face computational problems that are comparable to decision making under uncertainty in whole animals, such as sensory processing, decision-making and control of movement [5].

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O-12 Optimal sparse coding of song in a size-constrained auditory system?

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Optimal coding theory has been successfully applied to understand the principles of organization in many sensory systems. However, these systems were usually large and relatively generic stimulus encoders, like the early visual and auditory system of vertebrates or the early olfactory system in insects [1, 2, 3]. Do the principles of optimal coding also apply to small sensory systems with a specialized task and a restricted set of relevant stimuli? We studied the early auditory system of grasshoppers as an example for such a sensory system. These insects use genetically fixed songs to recognize mates with high fidelity. First steps of the processing of song take place in a 3-layer feed-forward network consisting of only a few dozen neurons.

Analyzing the transformation of the neural code for song in grasshoppers, we find that a temporally sparse representation of song is created. Additionally, responses of populations of cells in each of the three network layers get more diverse due to a higher diversity in the stimulus selectivity. That is, while neurons in the first two layers are selective for very similar temporal features of the song, each neuron in the network's output stage responds to a specific temporal feature.

This transformation has implications for the population code in the network: By asking whether a population decoder needs to incorporate information about which neuron fired which spike to discriminate stimuli optimally, we find that neuronal identity becomes more and more important for an effective read out of the population the higher one ascends in the network [4]. Thereby, an explicit, labeled-line like population code for temporal features of the song is created: This means that each neuron in the output layer signals the presence or absence of a specific temporal feature by the presence or absence of spikes in its response. In contrart, preceding layers encode temporal features implicitly by the temporal patterns of spikes.

Although the creation of a sparse, labeled-line like code resembles the transformations happening in large sensory systems, the small size and restricted task of the early auditory system of grasshoppers leads us to a different conclusion about the objective of these transformations. Early sensory areas in mammals like V1 encode the stimulus largely comprehensively – they filter the stimulus only little according to behavioral relevance as these networks serve as hubs which distribute information to more specific processing stages. In contrast, the grasshoppers' songs have a genetically fixed structure, freeing the animals from the task to learn the significance of a stimulus feature during life time. This allows grasshoppers to hard-wire and specialize their representation early in the sensory pathway, leading to a compression of the stimulus representation based on behavioral relevance. We have evidence that some of the neurons at the output of the network indeed do encode stimulus features directly related to behavior. Despite being at the very beginning in the grasshopper's auditory pathway, this representation might thus be similar to higher order areas in vertebrates, as it produces specific representations of behaviorally relevant features.

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O-13 A window to the amygdala: information on choice preference evolves concurrently in eye movements and neural responses in the limbic system

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Research on the functions of the amygdala has pointed towards multifaceted roles in learning, emotion, attention and decision making. The degree to which the various functions of the amygdala reflect different aspects of a common set of operations, such as the modulation of attention, remains unknown. A recent observation links deficits in facial expressions following bilateral amygdala damage to a specific attentional mechanism, reflected in an abnormal pattern of gaze directed to faces [1], raising the question whether other aspects of amygdala function can similarly be traced to the modulation of attention. For example, when choosing a preferred item from multiple alternatives, eye movements reveal the emergence of preference for one of the [3,4], suggesting an integral role for attention in the formation of choice preference, whose nature, however, remains uncertain. We consider whether preference-related modulation of eye movement depends on encoding of value in amygdala responses, which has recently been described during economic decision-making in humans [2]. Using concurrent eye tracking and recordings from the amygdala we aim to compare the time-course of information about choice encoded in unit activity with that encoded in patterns of gaze. To provide a framework for this comparison we combine a novel and sensitive generalized linear modeling approach to eye movement analysis with Bayesian particle filtering, and apply it towards identifying the time-evolution of information contained in eye movements, which will be compared with spike-encoding using equivalent procedures on spiking activity.

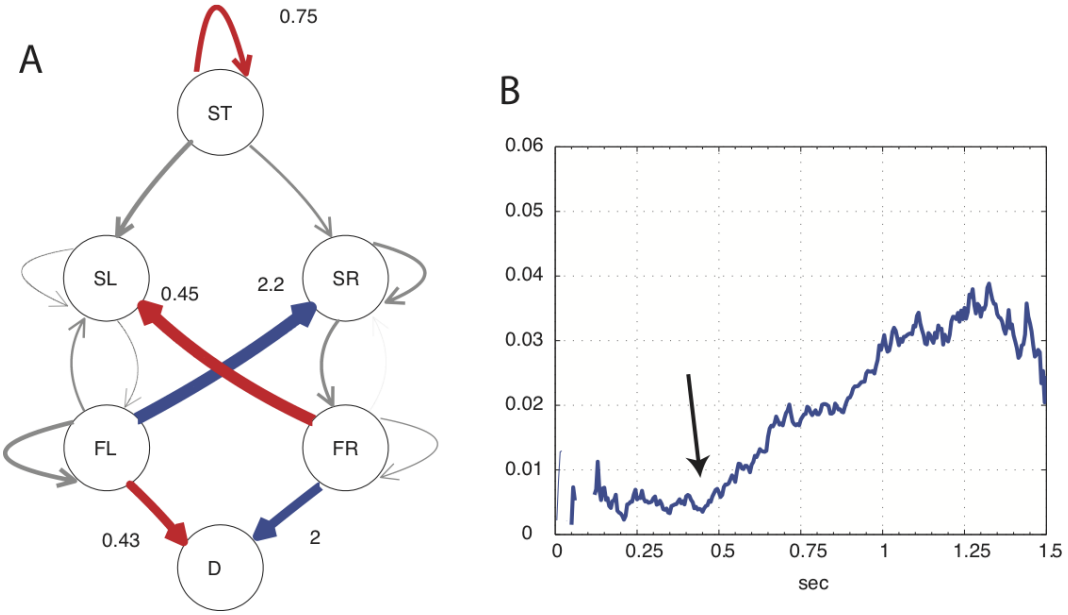

Figure 1: A Markov model of eye movement during a two-alternative choice task. A: Modeled states are start of trial (ST), saccade-to-left item (SL), saccade-to-right item (SR), fixate-on-left item (FL), fixate-on-right item (FR), decision (D). Multinomial logistic regression reveals an interaction between transition probabilities and choice side ($P < .001$). Significantly increasing (blue) and decreasing (red) changes in transition probability show an increase in saccades towards the chosen item and in choosing the item fixated last in the trial. B. The time course of average mutal information reveals to the onset of information accumulation at 500 ms after stimulus onset.

For each of 4 blocks of 138 trials, a Markov model of eye movement transitions (Fig 1A) was fitted to a subset of data which excluded that trial. Choice outcome was then predicted at each time point in the trial using a sequential Bayesian particle filter from likelihoods generated by the fitted model. The filter was considered to favor the right option over

the left when the proportion of particles favoring right exceeded some threshold. The quality of the prediction was quantified at each time point using mutual information between predicted and observed outcomes across multiple thresholds. Averaging over thresholds reduces noise variance in the estimate of MI, allowing for a clearer representation of the time course of the prediction quality (Fig 1B). This procedure reveals that Information about choice in eye-movements appears with a monotonically increasing trend having an abrupt onset around 500 ms after the stimulus onset. The emergence of information in eye movements therefore resembles the previously reported time course of value encoding in amygdala neurons [2], implying that value encoding in the amygdala and preference-related biases of gaze emerge concurrently. Further work will focus on the direct comparison of information in simultaneously recorded sets of eyetracking data and multiunit activity.

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O-14 Understanding the Mechanisms Underlying Phase-Locking Behavior in the Crayfish Swimmeret System

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One of the fundamental challenges in neuroscience is to understand how the intrinsic properties of neurons and the properties of neural networks combine to produce behavior. Networks that produce rhythmic motor behaviors, such as locomotion, provide important model systems to address this problem. A particularly good model for this purpose is the neural circuit underlying the coordinated rhythmic limb movements in the crayfish swimmeret system.

During forward swimming, rhythmic movements of swimmerets on different segments of the crayfish abdomen progress from back to front with the same period, but neighboring swimmerets are phase-lagged by 25% of the period. This coordination of limb movements is maintained over a wide range of frequency. The exact mechanisms underlying this robustly stable phase-locking are not known. Here, we use mathematical modeling and analysis in conjunction with recent experimental results to obtained insight into these mechanisms.

The rhythmic behavior of each swimmeret is driven by a local pattern generating circuit consisting of a half-center oscillator (HCO). These local pattern generating circuits are connected through well-described intersegmental connections. We model the neural circuitry of the swimmeret system as a chain of HCOs. First, we examine the phase response properties of HCOs for two fundamentally different mechanisms that produce anti-phase activity in HCOs: the “escape” and “release” mechanisms. We demonstrate that the “escape” and “release” mechanisms give rise to very different phase response properties, and we use phase plane arguments to explain the different shapes of the phase response curves. We then examine a chain of four nearest-neighbor coupled HCOs. We use the coupled oscillator theory and symmetry arguments to show that the phase-locking 25% phase-locking observed in the crayfish swimmeret system arises naturally from the network connectivity, but this phase-locking is robustly stable for only some combinations of connectivity and the escape/release mechanisms.

O-15 Local Control of Non-Local Information Flow in Oscillatory Neuronal Networks

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Control of information flow between neurons or groups of neurons is essential in a functional brain, e.g. for context and brain state dependent processing. In line with recent experimental and theoretical studies [1-5] we show that phase relations between synchronized oscillatory local circuits or brain areas may dynamically create information channels and induce changes in the effective connectivity.

Reducing neuronal oscillatory dynamics to a phase - amplitude description [6,7], we show how alternative phase shifts between different neurons or groups of neurons result in different effective connectivities. In particular, to quantify the information flow, we analytically calculate the time delayed mutual information and transfer entropy between oscillators in a phase locked state. We further present a theoretical framework to predict phase lag patterns within and between groups of oscillators in hierarchical networks. Combining both results we derive the information flow between the oscillators as a function of structural and dynamical network parameter.

We use our results to reveal how effective connectivity is controlled by the underlying physical connectivity and the intrinsic single oscillation frequencies. Interestingly, we find that local changes in the strength of a single link can remotely control the effective connectivity between two different physically unchanged oscillators. Similarly, local inputs modulating the intrinsic frequencies can dynamically and remotely change the information flow between distal nodes.

We link our results to biophysically more realistic networks of spiking neurons. In a clustered network of groups of type I neurons exhibiting gamma oscillations emanating from a PING mechanism [8], we numerically show that local changes of the connectivity or the inputs strengths within a cluster can non-locally control the phase relations and the information flow between distant clusters.

Conclusion Our findings reveal that local changes, e.g. in the physical strength of a local link or in the local frequency due to variation in the local inputs, can remotely and dynamically control the direction of non-local global information flow between distal nodes/clusters in a network. This might provide an efficient local mechanism to control global information processing in neuronal systems and to account for contextual and attentional modulation.

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O-16 Oscillatory mechanisms of selective integration during decision making

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Coordinated, rhythmic neuronal activity is proposed to allow selective routing of information by downstream targets which can filter a rhythmic input from noisy, asynchronous inputs. This mechanism was recently implemented in a model by Akam and Kullman [1]. Our work extends their concept to incorporate a neural mechanism for integrating the routed information, intended to model decision making. In our model, oscillatory input signals encode information relevant to the decision making task which must be evaluated in order to make a correct decision. The model is based on hippocampal-prefrontal interactions during a spatial working memory task. In this context the oscillatory signal represents the working memory item being retrieved and is assumed to be generated through an interactive process between the hippocampus and prefrontal cortex. This information is then integrated by prefrontal neurons which will initiate a decision once an integration threshold is reached. An oscillatory (as opposed to asynchronous) signal allows the relevant information to be filtered from other simultaneous activity and attended to by the cortex.

Two variations of the model are explored: one which combines routing and integration in a single subunit of the model (equivalent to a single processing step); the other utilizes two separate subunits, one for routing and one for integration. The model components represent excitatory pyramidal cells and inhibitory interneurons in prefrontal cortex. The two models make different experimental predictions which are evaluated through comparison with data recorded in rats during a task which requires spatial working memory (specifically, the memory of the last turn made) in order to make a correct decision [2].

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O-17 Slow integration leads to persistent action potential firing in distal axons of coupled interneurons

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The conventional view of neurons is that synaptic inputs are integrated on a timescale of milliseconds to seconds in the dendrites, with action potential initiation occurring in the axon initial segment. In a subset of rodent hippocampal and neocortical interneurons in acute slices prepared from serotonin 5b receptor (Htr5b) BAC transgenic mice [1], we found a much slower form of integration leading to action potential initiation in the distal axon. In approximately 80% of these interneurons (n=214 of 274), and in 23% of hippocampal interneurons in wild-type C57BL/6 mice (n=6 of 26), hundreds of spikes, evoked over a period of minutes, resulted in persistent firing that lasted for a similar duration.

Persistent firing was observed in response to step current injections, synaptic stimulation, sine wave current injections or in response to stimulation with natural spike trains [2]. With all of these protocols, multiple stimuli were required to induce persistent firing. While axonal action potential firing was required to trigger persistent firing, somatic depolarization was not; antidromic stimulation of the axon while hyperpolarizing the soma with current injection produced persistent firing. In addition, phase plots of persistent firing revealed that spikes had two components: an initial com-

ponent represented spiking in the axon and a second component that overlapped with the current-evoked spikes, indicative of a somato-dendritic spike following an initial, axonally initiated spike.

In some recordings (n = 11), partial spikes (spikelets) were observed during persistent firing. These spikelets overlapped the first component of the full-amplitude spikes, with the peak of the spikelets corresponding to an inflection on the rising phase seen in the full-amplitude spikes. These observations suggest that the first component of each action potential during persistent firing is an axonal spike, which sometimes fails to evoke a somato-dendritic spike. Furthermore, in some cells (n=3), spikelets were observed if the soma was hyperpolarized during persistent firing. These spikelets were smaller than those observed without hyperpolarization, suggesting that they are caused by propagation failures at a more distal axonal location.

Using a stylized computational model constructed with the NEURON simulation environment [3] of a branching axon attached to a soma, we simulated both small- and large amplitude spikelets, as well as full-amplitude spikes, by depolarizing a branch of the axon during somatic hyperpolarization. Large-amplitude spikelets corresponded to failure of the action potential to invade the soma, whereas small-amplitude spikelets corresponded to failures at the axon branches, 40 µm from the soma. Similar results were obtained with a full morphological model of a branching axonal arborization.

Additionally, in paired recordings, persistent firing was not restricted to the stimulated neuron; it could also be produced in the unstimulated cell (n=3). None of these pairs exhibited direct electrical coupling, and both glutamate and GABA receptors were blocked.

Consolidating these results suggests the existence of a previously unknown operational mode for some mammalian neurons. These interneurons can slowly integrate spiking, share the output across a coupled network of axons and respond with persistent firing even in the absence of input to the soma or dendrites.

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O-18 Exact firing-rate response of the integrate-and-fire neuron receiving finite amplitude excitatory and inhibitory post-synaptic potentials

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Neurons in active networks are subject to a fluctuating synaptic drive comprising excitatory and inhibitory post-synaptic potentials arriving at high rates relative to the integration time of the cell. The standard approach for treating stochastic synaptic currents and conductances has been to approximate them by a Gaussian white-noise or coloured-noise process in which the amplitudes of the underlying post-synaptic potentials are considered small. Importantly, this diffusion approximation has allowed for various firing-rate properties of populations of fluctuation-driven neurons to be calculated using Johanessma's [1] and Ricciardi's [2] Fokker-Planck framework. This framework in turn has proven to be a powerful theoretical tool for deriving the emergent states of coupled neuronal networks as a function of the properties of constituent neurons. The Gaussian/diffusion model of fluctuating synaptic conductance is in widespread use theoretically, computationally and experimentally and, in many cases, is an excellent approximation to the synaptic drive experienced by neurons in active networks in vitro or in vivo.

The typical synaptic coupling between neurons in many brain regions, however, is often sufficiently strong such that relatively few synchronous synaptic events are required to bring a neuron from rest to the spiking threshold. For ex-

ample, pairs of neocortical layer-5 pyramidal cells form synaptic connections with amplitudes in the range 0.5mV-6mV with a mean of 1.3mV [2] and, when correlations are taken into account, near-synchronous pre-synaptic activity will lead to even stronger aggregate EPSPs. Given that the difference between rest and threshold is around 10mV for this class of cell [3] it is likely that finite-amplitude effects missed by the standard Gaussian/diffusion approximation will play an important role in shaping the steady-state and response properties of neurons in vivo.

Here we present a significant generalization of the standard Gaussian/diffusion approach to treating synaptic fluctuations by exactly solving the firing-rate response for a neuron receiving finite-amplitude Poissonian excitatory and inhibitory post-synaptic potentials [5]. The mathematical framework involves the solution of a master equation and can be applied to synaptic-amplitude distributions decomposable into combinations of exponential functions -similar to those seen in experiment. It will be shown that this more general mathematical description of the neuronal response to stochastic synaptic dynamics is significantly richer and qualitatively distinct from that predicted by the Gaussian/diffusion approximation. As well as providing analytical solutions for the firing-rate response of the leaky integrate-and-fire model, it will be demonstrated how an efficient numerical scheme can be used to calculate, numerically but exactly, the response properties of non-linear integrate-and-fire neurons and also to better capture the effects of synaptic reversal potentials.

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O-19 An interlocked oscillator model for high-frequency firing of the midbrain dopaminergic neuron

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Dopamine neurotransmission has been found to play a role in addictive behavior and is impaired in psychiatric disorders. Dopaminergic (DA) neurons display two functionally distinct modes of electrophysiological activity: low- and high-frequency firing. The puzzling feature of the DA neuron is the combination of its high-frequency response to N-methyl-D-aspartate (NMDA) receptor activation coupled with the inability of other treatments to elevate its frequency effectively. We suggest a new computational model that reproduces this combination of responses and accounts for recent experimental data. The model is presented in two morphologies: (1) a reconstruction of a DA neuron and (2) a single compartment that ignores the spatial structure of the neuron. We show that these two model morphologies display very similar patterns. Therefore, an equipotential representation of the DA neuron is sufficient for combining its high- and low-frequency firing. Our comparison of the reconstructed morphology and the one-compartment model suggests that different regions of the neuron contribute differently to the high- and low frequencies. The model suggests how NMDA current restricted to the soma evokes high-frequency oscillations (Fig. 1 A) - a recent experimental result. Alternatively, distal NMDA stimulation must span an extensive part of the dendritic tree to evoke the burst. The two distinct patterns of stimulation suggest that the burst may report different cue types, such as saliency and reward. In both cases, the voltage dependence of the NMDA current is central for this capability. Additionally, we introduced a putative potassium current that allows for sustained oscillations under blockade of the calcium-dependent (SK-type) potassium current. Given multiple de- and repolarizing currents that sustain pacemaking, the neuron has two interlocked mechanisms (calcium-dependent and independent; Fig. 1 B) for producing oscillatory activity.

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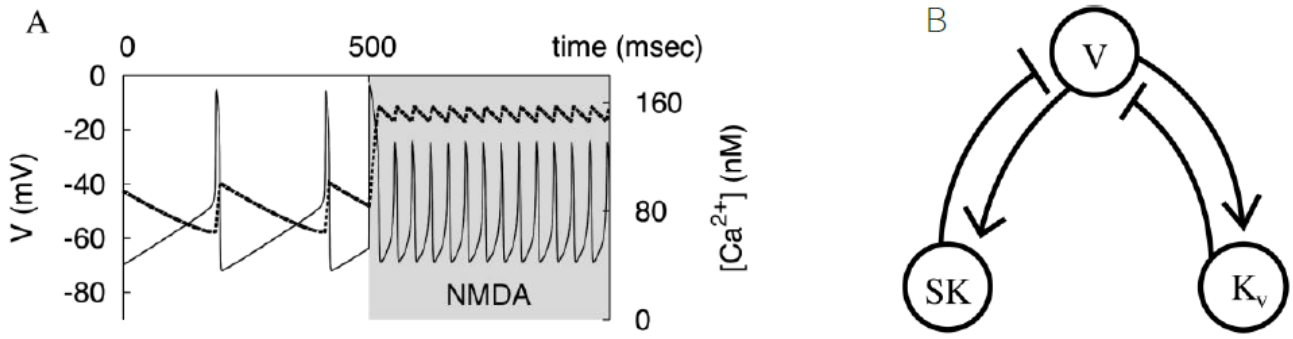

Figure 1: (A) The model switches to high-frequency oscillations at the onset of NMDA receptor stimulation at 500 msec. Dashed is the Ca2+ concentration; Solid is the voltage. The increase in the frequency is based on the reduction in the amplitude of Ca2+ oscillations. (B) The structure of the model. The SK-type Ca2+-dependent potassium current and the putative voltage-dependent potassium current create two negative feedback loops. The loops are interlocked by the voltage variable.

O-20 Modeling sharp wave - ripple complexes and their interactions with cortical slow oscillations through a cortico-CA3-CA1 model

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The hippocampus, and particularly the CA3 and CA1 areas, exhibit a variety of oscillatory rhythms that span frequencies from the slow theta range (4-10 Hz) up to fast ripples (~200 Hz). Various computational models of different complexities have been developed in an effort to simulate such population oscillations. Nevertheless the mechanism that underlies the so called sharp wave-ripple complex, observed in extracellular recordings in CA1, still remains elusive.

We present a combination of two computational models of the rat CA3 and CA1 hippocampal areas respectively. Both models are simple one-dimensional arrays of pyramidal and interneuronal populations, interacting only via fast AMPA and GABAA synapses. Connectivity schemes and postsynaptic potentials are based on biological data, rendering the network topology as realistic as possible.

Both models are first validated individually by showing that they reproduce a number of established anatomical and/or functional characteristic properties of the corresponding areas, including population bursts in CA3 and gamma oscillations in CA1.

The two networks are coupled in a full CA3-CA1 model through a feedforward scheme mimicking Schaffer collaterals. Quasi-synchronous population bursts in CA3 result in transient responses in CA1, consisting of deep depolarizations in the dendritic layer accompanied by transient ~150-200 Hz field oscillations in the somatic layer. These responses are shown to accurately reproduce a number of basic characteristic features of sharp wave-ripple complexes, reported previously in a variety of neurophysiological studies. By examining these features through our model, we are led to the formulation of a novel mechanism for the generation of ripples, based purely on chemical interactions and avoiding any use of gap junctions. The depolarizing input from CA3, produces intense interneuronal firing in CA1 which is regulated and synchronized by strong, fast-decaying, recurrent inhibition, resulting in a field oscillation within the ripple frequency range. Pyramidal cells, trapped within this inhibitory barrage, have a more passive role, with a small subset of them producing most of the excitatory spikes in a ripple. Through our proposed mechanism, we are able to interpret neurophysiological observations, such as the ripple disruption by halothane and the selective firing of pyramidal cells during ripples, which has various implications on memory consolidation.

Finally, the CA3-CA1 model is coupled with a cortical network exhibiting slow oscillations, in a feedback loop involving the mossy fiber and temporoammonic pathway inputs to the hippocampus and monosynaptic projections from CA1 to prefrontal cortex. We study the resulting correlations between hippocampal and cortical activity in an effort to uncover important parameters and mechanisms on which they depend. We show how the spiking activities of CA3 and CA1 depend on the inhibition-excitation ratio, induced by the two hippocampal inputs, and how this ratio can affect established correlations between cortical UP states and ripples. Such correlations have been suggested to be important for the transferring of memories to the cortex for long-term storage.

P-1 Reverse engineering of metabotropic glutamate receptor-dependent long-term depression in the hippocampus.

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P-2 Phase precession through acceleration of local theta rhythm: a biophysical model for the interaction between complex spike cells and theta cells.

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P-3 Measuring real-time synchronization in both spike trains and continuous time series.

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P-4 Evaluation and comparison of different machine learning approaches to auditory spectro-temporal receptive field estimation.

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P-5 Complex behavior in a modified Jansen and Rit neural mass model.

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P-6 Dynamical Behavior of Neural Ensembles: Bifurcation Analysis Approach.

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P-7 Binding features by relaying modulator group of neurons.

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P-8 Effects of Stochastic Inputs on Calcium-Dependent Synaptic Plasticity.

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P-9 Evaluation of functional arterial spin labeling data using a perfusion template.

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P-10 Stable and robust development of orientation maps and receptive fields.

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P-11 Disruption of tonic-clonic seizures using periodic stimulation of model neurons.

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P-12 Inferring ion channel densities from spike data.

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P-14 Reconstructing neuronal inputs from voltage recordings: application in the auditory system.

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P-15 GENESIS 3.0 functionality enhanced by interface to multiple types of database.

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P-16 Establishing community models as the underpinning of neuroscience: Linking computational and experimental data.

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P-17 Backwards-compatibility in GENESIS 3.0 and beyond: Bridging between procedural and declarative modeling.

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P-18 Maximized directed information transfer in critical neuronal networks.

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P-19 Evoking transitions from states of motor readiness to motor action in a phase-coupled oscillator model of motor cortex.

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P-20 A functional spiking model of the ITD processing pathway of the barn owl.

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P-21 Encoding The Pitch Of Sounds Using Synchrony Receptive Fields.

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P-22 A generative model of the crab cardiac neuromuscular system with modulation.

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P-23 Analytical and computational results regarding scalar property in a striatal-beat frequency model of interval timing.

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P-24 A bifurcation analysis of a modified neural field model: Conductance-based synapses act as an anti-epileptic regulatory mechanism.

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P-25 Mathematical model for frequency modulation in the respiratory network.

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P-26 Analyzing how neuronal parameters influence network activity.

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P-27 The role of ERG current in pacemaking and bursting in dopamine neurons.

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P-28 Phase locking and resetting in human subthalamic neurons.

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P-29 Development of NeuroML version 2.0: greater extensibility, support for abstract neuronal models and interaction with Systems Biology languages.

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P-30 The influence of structure on the response properties of biologically plausible neural network models.

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P-31 Architectural constraints on learning and memory function.

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P-32 Functional networks and dynamics in human seizure activity.

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P-33 Coding Motion Direction by Action Potential Patterns.

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P-34 Burst dynamics enable contrast coding via synchrony.

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P-35 A convolutional neural network model of the neural responses of inferotemporal cortex to complex visual objects.

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P-36 Spike trains in posterior parietal and premotor cortex encode trained and natural grasping behaviors.

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P-37 A bi-directional neuro-robotic system to study computational properties of cell assemblies.

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P-38 Theory of neural communication based on spatio-temporal coding.

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P-39 Switching of cortical Up and Down states: Reproduction of the Shu-Hasenstaub-McCormick experiment from a conductance-based model.

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P-40 Inferring Interactions in Assemblies of Stochastic Integrate-and-Fire Neurons from Spike Recordings: Method, Applications and Software.

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P-41 Emergence of Synfire Chains with Triphasic Spike-Time-Dependent Plasticity.

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P-42 A minimal model of C. elegans forward locomotion: the larval L1 circuit.

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P-43 Assessing time-varying causality network of ensemble neural spiking activity.

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P-44 The influence of network topology on synchrony and oscillations in networks of spiking neurons.

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P-45 A computational and behavioral study of the precision of visuo-spatial working-memory for several items.

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P-46 The effect of network structure on epileptic dynamics: Analysis of the synchronisation properties of an inter-network of cortical columns.

Andre D.H. Peterson[1], Iven M.Y. Mareels[2], Anthony N. Burkitt[2,3,4], David B. Grayden[2,3,4], Hamish Meffin[2,4], Mark J. Cook[1,3]

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P-47 Computational modeling of the electric potential in biological membrane. A comparison between healthy and cancerous neurons.

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P-48 A Biological Plausible Recurrent Model of V1 Hypercolumns.

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P-49 A model study for the progressive disruption of CA1 firing properties during Alzheimer's Disease.

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P-50 Dejittering of neural responses by use of their metric properties.

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P-51 Network properties of control and epileptic human slice recordings.

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P-52 Bringing rest into consideration: analyzing a database of computational models for multistability of oscillatory and stationary regimes.

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P-53 How and where does the brain predict the when: a Bayesian approach to modeling temporal expectation.

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P-54 Decoding Choice and Mutual Information on a Moment-by-Moment Basis from Single Neurons in Rat Prefrontal Cortex.

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P-55 A dynamical neural simulation mapping feature-based attention to location with non-linear cortical circuits.

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P-56 Does Calcium diffusional global feedback leads to slow light adaptation in Drosophila photoreceptors? - A 3D biophysical modelling approach.

Zhuoyi Song[1,2], Marten Postma[3], Weiliang Chen[5], Daniel Coca[2], S.A. Billings[2], Roger C. Hardie[3], Mikko Juusola[1,4], Erik De Schutter[5,6]

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P-57 SBML and MathML support for the stochastic reaction-diffusion simulator STEPS.

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P-58 Improving Performance of the STochastic Engine for Pathway Simulation (STEPS).

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P-59 Boundary representation of neural architecture and connectivity.

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P-60 Compensating the effect of dendritic diameters on calcium transients: A modeling study.

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P-61 Efficient Estimation of Phase Response Curves via Compressive Sensing.

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P-62 Neural mechanisms of audio tactile integration in the flutter range.

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P-63 Simulated functional networks in health and schizophrenia: a graph theoretical approach.

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P-64 A computational study of visual working memory capacity in the presence of saliency effects.

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P-65 Neurodynamical model of confidence decision-making in LIP.

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P-66 A biophysically detailed model of the primary auditory cortex explains physiological forward masking, co-tuning of excitation and inhibition and cortical signal amplification.

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P-67 A Model of Propagating Waves in Cerebral Cortex Across Network States.

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P-68 Emergence of direction- and orientation-selectivity and other complex structures from stochastic neuronal networks evolving under STDP.

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P-69 Forming cooperative representations via solipsistic synaptic plasticity rules.

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P-70 Hippocampal Population Dynamics Underlying Memory Trace Activation in a Tactile Classification Task.

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P-71 Structural analysis of functional connectivity related to memory reveals network changes in memory function over the life span.

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P-72 Macaque structural connectivity revisited: CoCoMac 2.0.

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P-73 Towards a unified theory of correlations in recurrent neural networks.

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P-74 Robustness vs. flexibility: How do external inputs shape the activity in a data-based layered cortical network model?

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P-75 High-resolution mapping of single neurons provides insight into neuron structure and LFP generation.

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P-76 Phase shift in hippocampal circadian rhythm during the latent period of epileptic rats.

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P-77 Connecting MOOSE and NeuroRD through MUSIC: Towards a communication framework for multi-scale modeling.

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P-78 Interfacing a parallel simulation of a neuronal network to robotic hardware using MUSIC, with application to real-time figure-ground segregation.

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P-79 Efficient spike communication in the MUSIC multi-simulation framework.

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P-80 The Connection-set Algebra: A formalism for the representation of connectivity structure in neuronal network models, implementations in Python and C++, and their use in simulators.

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P-81 An analytical approximation to the AdEx neuron model allows fast fitting to physiological data.

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P-82 Attracting states in frontal cortex networks associated with working memory and decision making.

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P-83 A network model of a multi-item working memory task based on competitive reverberating neural activity.

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P-84 Network inhomogeneity supports burst initiation in vitro.

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P-85 Burst initiation and propagation in cortical cultures requires an inhomogeneous connectivity distribution and synaptic rescaling.

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P-86 Modeling the LFP footprint of unitary thalamic inputs to sensory cortex.

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P-87 Laminar population analysis of multielectrode recordings from rat primary auditory cortex.

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P-88 Spatial reach of the local field potential is frequency dependent.

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P-89 Dynamics of self-sustained activity in random networks with strong synapses.

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P-90 Rate dynamics of the retina-LGN connection.

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P-91 The ball and stick neuron model accounts both for microscopic and macroscopic power laws.

Klas H Pettersen[1], Henrik Lindén[1], Tom Tetzlaff[1], Gaute T Einevoll[1]

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P-92 Flexible and efficient recording of neuronal properties in large network simulations: The NEST Multimeter.

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P-93 Impairments in the prefronto-hippocampal interactions explain associative learning deficit in schizophrenia.

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P-94 High-performance classification of contour percepts from EEG recordings.

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P-95 A model using mutual influence of firing rates of corticomotoneurons for learning a precision grip task.

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P-96 A stochastic model describing transport of PSD-95 molecules in spiny dendrites provides the basis for synaptic plasticity.

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P-97 Genes for adaptation and learning spanning evolution: computational comparison between synaptic transmission and chemo-tactic signaling protein networks.

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P-98 The implied exploration-exploitation trade-off in human motor learning.

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P-99 Does the cost function of human motor control depend on the internal metabolic state?

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P-100 The metabolic efficiency of myelinated vs unmyelinated axons.

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P-101 Sensory limits in the rodent whisker system predict an internal forward model for sensorimotor estimation of object touch.

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P-102 The structured variability of finger coordination in daily tasks.

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P-103 Ultra-low cost eyetracking as an high-information throughput alternative to BMIs.

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P-104 Detecting the presence of long-range temporal correlations in a time-varying measure of phase synchrony.

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P-105 Adaptive time-varying detrended fluctuations analysis: A new method for characterizing time-varying scaling parameters in physiological time series.

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P-106 Modeling Effects of GABAA Receptors in Basal Ganglia Computational Models.

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P-107 Experience-dependent Reactivations of Ventral Tegmental Area Neurons in the Rat.

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P-108 Inferring functional brain connectivity from field-potential oscillations in health and disease.

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P-109 The role of intrinsic dynamics and noise for neural encoding and synchronization.

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P-110 Role of TRP channels in dendritic integration and subthreshold membrane potential plateaus.

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P-111 Policy gradient rules for populations of spiking neurons.

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P-112 Conjoint Computational and Morphological Optimization by Cortical Neurons.

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P-113 Dependence of spatial filtering by spike timing dependent synaptic plasticity on learning window.

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P-114 Layer dependent neural modulation of a realistic layered-microcircuit model in visual cortex based on bottom-up and top-down signals.

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P-116 A neural field model of decision making in the posterior parietal cortex.

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P-117 State-dependent network reconstruction from calcium imaging signals.

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P-118 Neural dynamics and network topology interact to form critical avalanches.

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P-119 Automatic Characterization of Three Cortical Neuron Types Reveals Two Distinct Adaptation Mechanisms.

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P-120 Plasticity and stability in recurrent neural networks.

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P-121 Fast and richly structured activity in cortical networks with local inhibition.

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P-122 Synaptic Tagging and Capture: a bridge from molecular to behaviour.

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P-123 Efficient modeling of neural activity using coupled renewal processes.

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P-124 General conditions for spiking neurons and plasticity rules to perform independent component analysis.

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P-125 Self-organizing neural maps for multi-modal associations.

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P-126 Interaction of inhibition and synaptic plasticity in a model of the hippocampal CA1 microcircuit.

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P-127 The Role and Function of Noise and Neural Heterogeneity in the Integrated Population Response of the Vestibulo-Ocular Reflex.

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P-128 Interaction between synaptic dynamics and synaptic configuration determines the phase of the response to rhythmic inputs.

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P-128X Representation of dynamical stimuli in threshold neuron models.

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P-129 Relating thalamic neuronal activity and EMG for validating predictive control of deep-brain stimulation in Essential Tremor patients.

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P-130 A Computational Study of Action Planning in Syntax Evolution.

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P-131 Towards guiding principles in workflow design to facilitate collaborative projects involving massively parallel electrophysiological data.

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P-132 Cross-frequency coupling of eye-movement related LFP activities of freely viewing monkeys.

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P-133 Simple spatio-temporal transformation with sub-threshold integration in the saccadic system.

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P-134 Cellular and network mechanisms of cholinergically-induced transition between gamma and sharp wave – ripple network states in the hippocampus.

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P-135 Entrainment of a Thalamocortical Neuron to Periodic Sensorimotor Signals.

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P-136 A computational model of interconnected basal ganglia-thalamocortical loops for goal directed action sequences.

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P-137 Sequential tests and biologically grounded multi-alternative decision making.

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P-138 Dopamine-mediated action discovery promotes optimal behavior 'for free'.

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P-139 Decoding of spatiotemporal activity of auditory information in the cortex.

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P-140 Role of ICAN in rate, spike time, and theta phase coding by persistent spiking neurons of the medial entorhinal cortex.

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P-141 Identifying brain effective connectivity patterns from EEG: Performance of Granger Causality, DTF, PDC and PSI on simulated data.

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P-142 STDP, Hebbian cell assemblies, and temporal coding by spike synchronization.

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P-143 Use of Granger causality analysis and artificial spike trains to examine pause coding in Purkinje cell spike activity related to rhythmic licking.

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P-144 Correlation transmission of spiking neurons is boosted by synchronous input.

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P-145 Short term plasticity within the basal ganglia - a systems level computational investigation.

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P-146 What can MaxEnt reveal about high-density recordings and what can high-density recordings reveal about MaxEnt?

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P-147 Differential contribution of A-type potassium currents in shaping neuronal responses to synaptic input.

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P-148 Nonlinear Integration of Evidence in a Dynamic Motor Task.

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P-149 Development of Goal-Oriented Behavior in Self-Learning Robots.

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P-150 Network inference from non-stationary spike trains.

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P-151 Monocular eye position specificity in the oculomotor neural integrator.

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P-152 A metric space approach to the information channel capacity of spike trains.

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P-153 A dendrogram approach to the structure of spike trains.

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P-154 The effect of glutamate-gated chloride current on the excitability of a Purkinje cell: a modeling study.

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P-155 Neurodynamic regimes of phase relation and behavior in robotic models.

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P-156 External stimulation induces switches between neural oscillations: an illustrative feedback model.

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P-157 Modeling axial spinal segments of the salamander central pattern generator for locomotion.

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P-158 Effects of muscle dynamics and proprioceptive feedback on the kinematics and CPG activity of salamander stepping.

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P-159 A computational model of a strongly facilitating synapse.

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P-160 Conditional Intensity/Point Process Model of Task-Related Prefrontal Spiking: Effects of Performance.

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P-161 A unified computational model of the genetic regulatory networks underlying synaptic, intrinsic and homeostatic plasticity.

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P-162 Self-generated off-line memory reprocessing on different layers of a hierarchical recurrent neuronal network.

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P-163 Modelling human connectome development: precursors to neural circuits.

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P-164 Model of metabolic and temperature fluctuations in the brain.

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P-165 Degree of locking to network activity of neurons with similar movement tuning in the motor cortex of awake, behaving rats differs by layer.

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P-166 The Study and Date analysis of Interictal Electroencephalogram in Epileptic Patients.

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P-167 Inferring computational function of neuronal networks from multi-electrode array recordings: An evolutionary approach.

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P-168 Burrow-Centered Neural Model for Burrow Surveillance in Fiddler Crabs.

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P-169 Neural Representation for Distance and Direction of Resource for Honeybees.

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P-170 Mathematical Model for Metabolic Neuro-Hemodynamic Coupling.

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P-171 Decaying diffusion-based similarity measure of nodes and multi-scale modular structure finding in brain networks.

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P-172 Signal propagation and neuronal avalanches analysis in networks of formal neurons.

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P-174 A neural field model using advanced anatomical connectivity information.

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P-175 Spontaneous State Switching in Realistic Mean-Field Model of Visual Cortex with Heteroclinic Channel.

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P-176 Optimization of weak signal propagation in a feedforward network.

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P-177 On the Relation between Encoding and Decoding of Neuronal Spikes.

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P-178 Comparison of a Bayesian and a Regression Model for Stimulus Classification.

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P-179 Stimulus reconstruction based on postsynaptic potentials of leech interneurons.

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P-180 Encoding of tactile stimulus parameters by mechanosensory P cells of the medicinal leech Hirudo medicinalis.

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P-181 Shaping corticostriatal connectivity with STDP.

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P-182 Uncorrelated inputs enhance signal representation in the inhibitory striatum network.

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P-183 The impact of structural embeddedness of neurons on network dynamics.

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P-184 A hybrid model of the primary visual cortex.

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P-185 A large-scale model of the three first stages of the mammalian olfactory system implemented with spiking neurons.

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P-186 Activity-Dependent Memory Organization in the Early Mammalian Olfactory Pathway for Decorrelation, Noise Reduction, and Sparseness-Enhancement.

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P-187 A cortical attractor network with dynamic synapses.

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P-188 Odor segmentation and identification in an abstract large-scale model of the mammalian olfactory system.

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P-189 An Abstract Model of the Basal Ganglia, Reward Learning and Action Selection.

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P-191 Scaling of a biophysical neocortical attractor model using Parallel NEURON on the Blue Gene/P.

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P-192 Inferring and quantifying causality in neuronal networks.

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P-193 Spike timing jitter is beneficial in neural spike coding - A case of the mammalian MSO sound localization circuit.

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P-194 A Physiologically Inspired Model for Global Remapping in the Hippocampus.

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P-195 Inhibition enhances Capacity of Sequence Replay: A Mean Field Model.

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P-196 Capacity Measurement of a Recurrent Inhibitory Neural Network.

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P-197 Metabifurcation analysis unveils hidden dynamical structure of a neural population model.

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P-198 Complex dynamics for a reduced model of human EEG: implications for the physiological basis of brain activity.

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P-199 How stochastic adaptation of neurons shapes interspike interval statistics – theory and experiment.

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P-200 TRENTOOL: An open source toolbox to estimate neural directed interactions with transfer entropy.

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P-201 Contribution of SERCA and IP3 sensitivity to calcium signaling in astrocytes: a computational study.

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P-202 Effects of local structure of neuronal networks on spiking activity in silico.

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P-203 Computational study of structural changes in neuronal networks during growth: A model of dissociated neocortical cultures.

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P-204 Different LFP frequency bands convey complementary information about the BOLD signal.

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P-205 Signal detection in neural populations: the importance of heterogeneity.

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P-206 Spontaneous onset of irregular collective oscillations in heterogeneous neural networks.

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P-207 Improved parameter fitting for models of young and aged neurons.

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P-208 Interlaminar Granger causality and alpha oscillations in a model of macaque cortex.

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P-209 Simulating the spread of activation in neocortical circuits.

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P-210 Local unsupervised learning rules for a spiking neural network with dendrite.

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P-211 Spike timing jitter is beneficial in neural spike coding - A case of the mammalian MSO sound localization circuit.

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P-212 A computational model of the moth macroglomerular complex.

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P-213 Computational Mechanism of Postponed Decisions.

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P-214 Interdependence between network dynamics and connectivity in dissociated cortical cultures: a theoretical and experimental approach.

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P-215 System identification of spiking neuron networks: a model-driven approach.

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P-216 ITD sensitivity to naturalistic sounds in the superior olivary complex.

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P-217 The role of the large-conductance calcium-dependent potassium channel, BK/Slowpoke, in shaping motor neuron firing during rhythmic activity.

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P-218 Biophysical modeling of excitability and membrane integration at the single cell and network levels.

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P-219 Synchronization and spontaneous dynamics in the Locus Coeruleus network.

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P-220 Is the circadian clock a limit cycle oscillator?

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P-221 Self-organized criticality in a model for developing neural networks.

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P-222 A multi-compartment model for interneurons in the dorsal lateral geniculate nucleus.

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P-223 Controlling Spike Timing and Synchrony in Oscillatory Neurons.

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P-224 Adaptive Cluster Algorithm to infer Boltzmann Machines from Multi-Electrode Recording Data.

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P-225 Single cell dynamics determine strength of chaos in collective network dynamics.

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P-226 Quantifying the visual information sourced from melanopsin photoreceptors in mouse LGN field responses.

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P-227 Does the information in the phase of low frequency LFP reflect the low frequency envelope of local spike rates?

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P-228 Identification of striatal cell assemblies suitable for reinforcement learning.

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P-229 Fail-safe detection of threshold crossings of linear integrate-and-fire neuron models in time-driven simulations.

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P-230 Emergence of beta/gamma oscillations: ING, PING, and what about RING?

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P-231 Modulation of frequency-tuning property of subcortical neurons elicited by corticofugal signals in bat's auditory cortex.

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P-232 Role of neuromodulators in regulating Hippocampal encoding and retrieval in anxiety disorders.

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P-233 Benchmarking the impact of information processing in the insect olfactory system with a spiking neuromorphic classifier.

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P-234 Modeling phonotaxis in female Gryllus bimaculatus with artificial neural networks.

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P-235 Coarse-grained statistics for attributing criticality to heterogeneous neural networks.

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P-236 The effect of intrinsic subthreshold oscillations on the spontaneous dynamics of a ring network with distance-dependent delays.

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P-237 Transient Dynamics between Displaced Fixed Points: An Alternate Nonlinear Dynamical Framework for Olfaction.

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P-238 Dynamic Observer: Ion Channel Measurement beyond Voltage Clamp.

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P-239 Flexible neuronal network simulation framework using code generation for NVidia® CUDA™.

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P-240 Effects of adaptation and synaptic plasticity on synchronization of coupled oscillating neurons.

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P-241 A neural model of human fear pathways based on anatomical and neuroimaging data.

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P-242 Coherent periodic activity in excitatory neural networks : The role of network connectivity.

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P-243 Non-equilibrium diffusive fluxes of ions and their impact on the refractory period of tightly packed neurons.

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P-244 Spiking Neural Network Model of Free-Energy-based Reinforcement Learning.

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P-245 Prediction of Spatiotemporal Patterns of Neural Activity Using a Higher-Order Markov Representation of Instantaneous Pairwise Maximum Entropy Model.

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P-246 Network dynamics associated with experience-dependent plasticity in the rat somatosensory cortex.

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P-247 Different neural codes result in bidirectional connectivity formed by the same model of spike-timing-dependent plasticity.

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P-248 Local field potential phase and spike timing convey information about different visual features in primary visual cortex.

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P-249 Scaling of temporal correlations in densely connected networks of LIF neurons.

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P-250 Stochastic gradient ascent learning with spike timing dependent plasticity.

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P-251 Compressed Sensing with Stochastic Spikes.

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P-252 An activity based model of grating and plaid adaptation in the human visual system.

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P-253 Neural coding tools, based on Information Theory, applied to discrete time series: from electrophysiology to neuroethology.

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P-255 Measures of statistical dispersion based on Entropy and Fisher information.

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P-256 A Limbic System Model for the Control of Release of Tonic Dopamine and it's Effect on Response Vigor.

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P-257 Each to their own beat: periodicity in temporal inference.

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P-258 A novel model of an identified Drosophila crawl motoneuron for investigating functional effects of ion channel type across larval developmental stages.

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P-259 An offline correction method for uncompensated series resistance and capacitance artifacts from whole-cell patch clamp recordings of small cells.

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P-260 INEX – A binary neuronal model with inhibitory and excitatory synapses.

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P-261 Spatiotemporal information transfer pattern differences in motor selection.

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P-262 The dynamics of cost and benefit representations by noradrenaline and dopamine neuronal activity, and their relation to goal-directed behavior.

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P-263 Spatial stereoresolution for depth corrugations may be set in primary visual cortex.

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P-264 The influence of stationary synaptic activity on the PRC.

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P-265 A dynamical model of perceptual categorization.

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P-266 Hippocampal theta oscillations synchronize with rhythmical head movements during locomotion.

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P-267 How attention and reinforcers jointly optimize the associations between sensory representations, working memory and motor programs.

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P-268 Detection and localization of multiple rate changes in Poisson spike trains.

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P-269 Interplay of periglomerular and granule cell inhibitory synapses on mitral cell spiking.

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P-270 Cross-modular processing in a spiking neural network model.

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P-271 Firing frequency response to current and conductance periodic inputs in a Ih/INap biophysical neuron model.

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P-272 Effect of Network Structure on Spike Train Correlations in Networks of Integrate-and-Fire Neurons.

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P-273 Effective neuronal refractoriness dominates the statistics of superimposed spike trains.

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P-274 Neural network reconstruction using Kinetic Ising Models with Memory.

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P-275 Modulation of thalamocortical relay by basal ganglia in Parkinson's disease and dystonia.

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P-276 Possible mechanisms underlying intermittent synchronous activity in the networks of excitatory and inhibitory bursting neurons.

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P-277 Synchronization hubs may arise from strong rhythmic inhibition during gamma oscillations in primary visual cortex.

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P-278 The emergence of synchronized bursting in a heterogeneous, highly clustered respiratory network.

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P-279 Motion-based predictive coding is sufficient to solve the aperture problem.

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P-280 Identification of a molecular system that regulates growth cone membrane potential during growth cone guidance.

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P-281 Inscrutable Games? Facial Expressions Predict Economic Behavior.

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P-282 Optimization of battery strengths in the Hodgkin-Huxley model.

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P-283 Studying functional networks in human brain through intracerebral spontaneous EEG.

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P-284 Dynamical switching between different hippocampal rhythms.

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P-285 Spike distributions of a population based hierarchical network of the rat amygdaloid complex.

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P-286 Self-organization of virtual odorant receptors inspired by insect olfaction.

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P-287 Temperature differentially affects subsequent layers of auditory neurons in the locust.

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P-288 Enthorinal phase precession revisited – single-run analysis of in-vivo grid cell data.

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P-289 Dendritic versus somatic resonance.

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P-290 Robustness of STDP-induced memory to perturbations of presynaptic activity: a simulation study.

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P-291 Inhibitory coverage of dendritic excitation.

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P-292 Complex Symptoms of Demyelination and Nerve Damage Explained by Nonlinear Dynamical Analysis of Conductance-based Models.

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P-293 Reinforcement learning in dendritic structures.

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P-294 Modeling maladaptive decision-making in a rat version of the Iowa Gambling Task.

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P-295 The influence of behavioral context on sensory encoding.

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P-296 Analysis of simultaneous multielectrode recordings with 4,096 channels: changing dynamics of spontaneous activity in the developing retina.

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P-297 Single neuron transient activity detection by means of tomography.

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P-298 Effect of spatial arrangement of presynaptic calcium channels on the calcium current cooperativity of neurotransmitter release.

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P-299 Assessing predictive capability of neuronal network models by computing Lyapunov exponents.

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P-300 The emergence of functional connectivity patterns bound by an underlying structural connectivity substrate.

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P-301 Synaptic integration and NMDA spikes in a layer 5 pyramidal neuron model.

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P-302 Object Localization with Electrosensory Mechanism in Weakly Electric Fish.

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P-303 Analyzing possible pitfalls of cross-frequency analysis.

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P-304 A Cruise Control for Parkinson's Disease.

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P-305 Basket cell contributions to the generation of theta rhythms in model hippocampal CA1 networks.

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P-306 Identifying dendritic processing in a [Filter]-[Hodgkin Huxley] circuit.

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P-307 High dimensional phase resetting curves and their use in predicting network dynamics.

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P-308 State-dependent control of the respiratory pattern and coupled oscillators.

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P-309 Improving visualization and analysis of relationships between neuronal model parameters in discrete parameter spaces.

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P-310 MoNETA: massive parallel application of biological models navigating through virtual Morris water maze and beyond.

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P-311 What decision-making models can tell us about tactile remapping.

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P-312 Dependence of Rapid Spike-Based Neural Learning upon Neural Parameters.

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P-313 Extension of the Kuramoto model to encompass time variability in neuronal synchronization and brain dynamics.

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P-314 Non-specific LTD at parallel fibre - Purkinje cell synapses in cerebellar cortex provides robustness against local spatial noise during pattern recognition.

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P-315 The effect of dendritic morphology on pattern recognition in the presence of active conductances.

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P-316 The beneficial effects of non-specific synaptic plasticity for pattern recognition in auto-associative memory.

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P-317 The effect of synaptic noise on dendritic morphology.

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P-319 Homeostasis causes hallucinations in a hierarchical generative model of the visual cortex: the Charles Bonnet Syndrome.

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P-320 Unifying low-level mechanistic and high-level Bayesian explanations of bistable perceptions: neuronal adaptation for cortical inference.

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P-321 Matching synaptic type with postsynaptic firing class shapes the encoding of either stimulus rate or rate change.

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P-322 Modeling UBC intrinsic excitability.

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P-323 Detailed visualization and morphometric analysis of reconstructed neurons using Blender and Python.

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P-324 The applicability of effective connectivity measures to time series of neuronal oscillators.

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P-325 Model parameter estimation for Channelrhodopsin-2 light gated ion channels.

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P-326 Quantification of emotional bias by an Emotional-Gain Model.

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P-327 Quantification of fairness bias by an Fairness-Equity Model.

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P-328 Gender difference of emotional bias in sharing love.

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P-329 Contributing factors in judgment of fairness by monetary value.

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P-330 NineML: The Network Interchange for Neuroscience Modeling Language.

Ivan Raikov[1,2], Robert Cannon, Robert Clewley, Hugo Cornelis, Andrew Davison, Erik De Schutter, Mikael Djurfeldt, Padraig Gleeson, Anatoli Gorchetchnikov, Hans Ekkehard Plesser, Sean Hill, Mike Hines, Birgit Kriener, Yann Le Franc, Chung-Chuan Lo, Abigail Morrison, Eilif Muller, Subhasis Ray, Lars Schwabe, Botond Szatmary

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P-331 A model of healthy aging and motor inhibition in the basal ganglia.

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P-332 Networks of phase-amplitude neural oscillators.

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P-333 Finding the event structure of neuronal spike trains.

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P-334 Restoring ion channel pathology by parameter optimization.

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P-335 Resolution of ITD detection: stochastic vs. exquisite system.

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P-336 Chimera states and collective chaos in pulse-coupled neural networks.

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P-337 A model of the levator-depressor neuro-mechanical system of the stick insect leg.

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P-338 Interplay between dendritic non-linearities and STDP.

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P-339 Free concepts association: a neural model.

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P-340 Reorganization of spatial maps in the hippocampal circuit.

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P-341 BLISS: an Artificial Language for Learnability Studies.

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P-342 Sensitivity analysis to explain the excitability in a pyramidal neuron with application to Alzheimer's disease.

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P-343 Active exploration is important for reinforcement learning of interval timing.

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P-344 IKCa-Cav3 complex creates a high pass filter for parallel fiber input in cerebellar Purkinje cells.

Jordan DT Engbers[1], Dustin Anderson[1], Renata Rehak[1], W Hamish Mehaffey[1], Bruce E McKay[1], Mirna Kruskic[1], Gerald W Zamponi[1], Ray W Turner[1]

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P-345 The relationship between cortical network structure and the corresponding state space dynamics.

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P-346 Simulation Platform: Cloud-computing meets computational neuroscience.

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P-347 Visualization and analysis of peripheral drift illusion.

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P-348 Generating realistic retinal image for whole visual system modeling.

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P-349 The flatness of bifurcations in 3D neuronal branching patterns.

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P-350 A multi-scale modeling approach for studying cortical lesions as a cause for epilepsy.

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P-351 The effect of spike time dependent plasticity on activity patterns in the basal ganglia.

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P-352 Building a Neuronal Infrared Lens.

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P-353 The impact of resource competition on neurite outgrowth.

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P-354 Modeling neuronal dynamics during brain ischemia.

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P-355 Hebbian Cross-Correlation Learning Emerges as Spike Timing Dependent Plasticity.

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P-356 Independently outgrowing neurons with a geometric synapse formation model develop realistic network connectivity patterns with small-world properties.

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P-357 Small-world topology is most efficient for homeostatic neuronal network repair.

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P-358 Online video tracking for activity-dependent stimulation in neuroethology.

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P-359 A model study for causal relationships between voltage and calcium dynamics.

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P-360 A computational approach for modeling the biological olfactory system during an odor discrimination task using spiking neuron.

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P-361 Towards massively-parallel analytic capabilities for multielectrode recordings.

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P-362 Stabilisation of beta and gamma oscillation frequency in the mammalian olfactory bulb.

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P-363 CLONES : A Closed-Loop Simulation Framework for Body, Muscles and Neurons.

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P-365 Effects of synaptic and intrinsic parameters in shaping dynamic responses to olfactory input: A combined computational-experimental study of two glomerular microcircuits.

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P-366 Automated generation of compartmental models via database tools for neurophysiology data management, analysis, and simulation.

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P-367 Information coding by single spikes and bursts in thalamocortical relay neurons.

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P-368 Modeling habituation of auditory evoked fields using neural mass models.

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P-369 Phase-of-firing Information Coding in Laminar Cortical Architecture.

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P-370 Phase Response Curves of Subthalamic Neurons: Experimental Measurement and Theoretical Prediction.

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P-371 Synaptic Bouton Sizes Are Tuned to Best Fit their Physiological Performances.

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P-372 Synaptic Scaling Generically Stabilizes Circuit Connectivity.

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P-373 Cortical modulation of neuronal activity in the cat's lateral geniculate and perigeniculate nuclei: a modeling study.

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P-374 Extracting activity of individual cell populations from multielectrode recordings.

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P-375 Kernel Current Source Density method.

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P-377 Persistent localized activity in a two-population neural-field model with spatio-temporal external input.

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P-378 Real-time emulation of parallel channel responses in the vertebrate retina and the primary visual cortex.

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P-379 Modeling network phenomena in the Inferior Olive: I. Keeping track of time.

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P-380 Modeling network phenomena in the Inferior Olive: II. Modulation of sub-threshold oscillations.

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P-381 The effect of movement noise on internal estimations: possible implications for neural coding.

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P-382 The effects of conduction delay on temporal ordering in leaky integrate and fire neuronal networks.

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P-383 Early Signs of Tinnitus in a Simulation of the Mammalian Primary Auditory Cortex.

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