**Social Stress Resilience**

Advanced Seminars in Behavioral Neuroendocrinology

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 [[Cliff](http://usdbiology.com/cliff/Courses/Advanced%20Seminars%20in%20Neuroendocrinology/Social%20Stress%20Resilience%2017/Cliff17Social%20Stress%20Resilience.pptx) – 19 January 2018](http://sunburst.usd.edu/~cliff/Courses/Advanced%20Seminars%20in%20Neuroendocrinology/Glial%20Signaling/Cliff13GlialSignaling.ppt)[The human BNST: Functional role in anxiety and addiction](http://usdbiology.com/cliff/Courses/Advanced%20Seminars%20in%20Neuroendocrinology/Anxiety%20and%20BNST%20Peptides%2018/Avery%2016%20NPP%20BNST%20Anxiety%20addiction.pdf)  Authors 2018 [*Journal*](http://www.cell.com/cell/home) *1:* 1–4

1. ***B*ed *N*ucleus of the *S*tria *T*erminalis** (**BNST**) is part of the extended amygdala
	1. a **nucleus** em**bed**ed at the end (**terminal**) of the white matter pathway (**stria**)
		1. stria terminalis is an axonal pathway from amygdala to **BNST**
			1. parallel to the fornix
	2. **** ive
		1. y
		2. s
			1. s

[[Jazmine Yaeger](http://usdbiology.com/cliff/Courses/Advanced%20Seminars%20in%20Neuroendocrinology/Anxiety%20and%20BNST%20Peptides%2018/Jaz18Anxiety%20and%20BNST%20Peptides.pptx) – 26 January 2018](http://sunburst.usd.edu/~cliff/Courses/Advanced%20Seminars%20in%20Neuroendocrinology/Glial%20Signaling/Cliff13GlialSignaling.ppt)[Cannabinoid CB1 receptors in distinct circuits of the extended amygdala determine fear responsiveness to unpredictable threat](http://usdbiology.com/cliff/Courses/Advanced%20Seminars%20in%20Neuroendocrinology/Anxiety%20and%20BNST%20Peptides%2018/Lange%2017%20MolPsych%20Cb1%20%20extended%20amgdala%20fear%20unpredictable%20threat.pdf)  MD Lange, T Daldrup, F Remmers, HJ Szkudlarek, J Lesting, S Guggenhuber, S Ruehle, K Jüngling, T Seidenbecher, B Lutz, HC Pape 2017 [*Molecular Psychiatry*](https://www.nature.com/mp/)22: 1422–1430

1. Unpredictability of **Anxious** conditions heightens the affective response
	1. the amygdala is involved
		1. basolateral amygdala (BLA) and central amygdala (CeA) especially
			1. CeA is subdivided into medial, lateral and lateral-capsular CeA
				1. mCeA, lCeA, lcCeA in medial to lateral order
		2. connected to the periaqueductal gray (PAG)
			1. Fear "on cells" in lCeA allow vlPAG signals to result in anxious behavior
				1. stimulated by BLA Glu neurons
				2. "on" cells GABAergic
				3. disinhibition of vlPAG Glu cells

blocking vlPAG GABA interneurons

* + - * 1. allowing vlPAG Glu signal to medullary then spinal cord ACh neurons
				2. and expression of anxious behavior, such as freezing
			1. Fear "off cells" lcCeA Neurons are also GABAergic
				1. inhibit lCeA "on" cells
				2. disinhibit vlPAG GABA cells
				3. disinhibit dlPAG Glu neuons

by inhibiting mCeA GABA neurons

* 1. **BNST** modifies the circuit
		1. BLA Glu stimulates **BNST** Glu neurons
			1. stimulates Orx neurons in the KH-DMH/PeF hypothalamus
		2. lcCeA "on cells" inhibit **BNST** Glu neurons
			1. inhibits **BNST** GABA interneurons
1. unpredictably presented CS (tone) prior to footshock (US) during conditioning
	1. produces longer term freezing conditioned response (CR)
		1. longer duration CR than predictable CS time prior to US
		2. unpredictability related to variable time between tone and shock
	2. longer-term freezing due to unpredictable presaging of aversive stimulation can be reversed
		1. shorter duration freezing produced by blocking cannabinoid Cb1 receptors in **BNST**
			1. Cb1 antagonist = AM251
2. ****endocannabinoid (eCb) Cb1 activation promotes prolonged fear responses
3. stimulation of BLA Glu neurons stimulates eCb release at terminals in **BNST**
	1. optogenetically stimulated via AAV6-Syn-hChR(H134R)-eYFP transfection
	2. eCb binding to Cb1 inhibits release of presynaptic (BLA) Glu = DSE
		1. eCb = 2-arachidonylglycerol (2-AG) or anandamide (AEA)
		2. DSE = Depolarization-induced Suppression of Excitation
			1. reduced Glu-Na+ stimulated postsynaptic current
	3. reduced postsynaptic current reversed by Cb1 antagonist AM251
		1. demonstrates that Cb1 actions produce DSE
4. Optogenetic stimulation of CeA GABA neurons also stimulates eCb at **BNST** terminals
	1. Cb1 activations in **BNST** inhibits release of CeA presynaptic GABA = DSI
		1. DSI = Depolarization-induced Suppression of Inhibition
			1. reduced GABA-Cl- stimulated postsynaptic current
	2. reduced Cl- current reversed by Cb1 antagonist AM251
		1. demonstrates that Cb1 actions also produce DSI
5. stimulation of MeA does ***not*** produce DSE or DSI in **BNST**
6. BLA and CeA signals act via DSE or DSI only on Glu (non-GABA) neurons in **BNST**
	1. both GABA and Glu neurons in **BNST** are capable of DSE or DSI
	2. BLA and CeA synapse with **BNST** GABA and Glu neurons
	3. BLA and CeA terminals only contain Cb1 on Glu connections
7. Mice transfected with flox-STOP codon upstream of Cb1 gene reduces duration of **anxiety**
	1. Stop-Cb1 mice do not express Cb1 receptors
	2. Removal of Cb1 activity produces shortened duration of freezing
		1. To mice with unpredictable CS
	3. Mice with Cre removal of stop codon (AAV-Cre) return to long duration freezing
		1. Inhibition of Cb1 (AM251) shifts long-to-short-term freezing
8. Floxed-Cb1 + AAV-Cre to remove Cb1 shifts long-to-short-term freezing
9. ****Cb1 receptors are essential for the transition from short to prolonged fear responses
	1. freezing
10. Local rescue of Cb1 activity (floxed stop + AVV-Cre) in BLA Glu neurons partially reinstates prolonged freezing
	1. Local rescue of Cb1 activity in CeA GABA neurons partially reinstates prolonged freezing
	2. ****Both BLA Glu and CeA GABA inputs to **alBNST** Glu neurons are necessary for Cb1 stimulation of transition from short to prolonged fear responses
11. Orexin A (OrxA) deprolarizes **BNST** neurons
12. OrxA in **BNST** reduces social interaction (SI) time = ↑ **anxiety**
	1. OrxA in **BNST** increases **anxiety** on EPM
		1. Not in septum
		2. Glu antagonists in **BNST** reverses OrxA-induced SI **anxiety**
			1. AMPA antagonist = CNQX, NMDA antagonist = AP5
13. OrxA-induced **anxiety** is NMDA-dependent
14. OrxA in vlPAG produces analgesia
	1. Similar to morphine
15. restraint stress also produces analgesia and stimulates LH Orx neuron activity
	1. OrxA + cFos double staining
	2. Restraint stress → ↑ plasma corticosterone (B)
		1. Not influenced by Orx1 receptor antagonist
	3. Restraint stress → ↑ vlPAG OrxA levels
16. Orx1 antagonist in vlPAG blocks restraint stress-induced analgesia
	1. Orx1 receptor antagonist = SB334867
17. Cb1 antagonist in vlPAG blocks restraint stress-induced analgesia
	1. Cb1 antagonist in vlPAG inhibits Orx1-induced analgesia

[[Shaydie Davies](http://usdbiology.com/cliff/Courses/Advanced%20Seminars%20in%20Neuroendocrinology/Anxiety%20and%20BNST%20Peptides%2018/Shaydie18Anxiety%20and%20BNST%20Peptides.pptx) – 23 February 2018](http://sunburst.usd.edu/~cliff/Courses/Advanced%20Seminars%20in%20Neuroendocrinology/Glial%20Signaling/Cliff13GlialSignaling.ppt)[Corticotropin releasing factor in the bed nucleus of the stria terminalis in socially defeated and non-stressed mice with a history of chronic alcohol intake](http://usdbiology.com/cliff/Courses/Advanced%20Seminars%20in%20Neuroendocrinology/Anxiety%20and%20BNST%20Peptides%2018/Albrect-Souza%2017%20FrontPharm%20CRF%20BNST%20Social%20Defeat%20ETOH.pdf)  L Albrechet-Souza, TW Biola, R Grassi-Oliveira, KA Miczek, RMM de Almeida 2017 [*Frontiers in Pharmacology Neuropharmacology*](https://www.frontiersin.org/articles/10.3389/fphar.2017.00762/full) 8: 762: 1–15

1. Chronic Alcohol (ETOH) consumption stimulates corticotropin releasing factor (CRF)
	1. leading to corticosterone release
	2. CRF from the paraventricular nucleus (PVN) activates HPA axis
		1. PVN CRF projects to Amygdala, VTA, and **BNST**
	3. overactivity of CRF leads to alcohol abuse
		1. **anxiety**
			1. **anxious** phenotypes exhibit upregulation of CRF
		2. depression
2. High ETOH preference rats (msP) have modified CRF function
	1. upregulation of CRF1 receptor gene expression
		1. in cingulate gyrus (CG), motor cortex (M1), sensory cortex (S1)
		2. hippocampal CA1, CA3 and CA4
		3. nucleus accumbens (NAc), medial mygdala, (MeA), basolateral amygdala (BLA) and central amygdala (CeA)
			1. but not in **BNST**
	2. msP mice have increased freezing acquisition and recall in fear conditioning
	3. decreased threshold of stress-induced reinstatement of ETOH seeking
	4. systemic CRF1 antagonist reduces ETOH self-administration
		1. systemic CRF1 antagonist blocks ETOH reinstatement
3. ****CRF1 receptors influence ETOH self-administration and reinstatement
4. CRF1/2 receptor antagonist into BNST had NO effect on ETOH self-administration
	1. CRF1/2 receptor antagonist = d-Phe-CRF12-41
	2. CRF1/2 receptor antagonist in CeA reduces ETOH self-administration
	3. ETOH vapor had no effect on BNST CRF cell density
		1. decreases CeA CRF cell density
5. Social defeat in mice increases voluntary ETOH drinking
	1. social defeat transiently → ↑ hedonic response
		1. increased preference for saccharin solution vs water
	2. social defeat → ↑ **anxiety**
		1. defeat → ↓ open arm entries and time on elevated plus maze (EPM)
6. Social defeat induces increases CRF mRNA in **BNST**
	1. but *no* ∆ in CRF1 receptor mRNA in **BNST**
	2. slight/non-significant increase in CRF2 receptor mRNA in **BNST**
7. CRF1 antagonism in **BNST** → ↓ acute ETOH self-administration
	1. no ∆ in stressed mice
		1. CRF1 antagonist = CP376,395
	2. no ∆ in due to CRF2 antagonism
		1. CRF2 antagonist = Astressin 2B
	3. CRF1 antagonism in **BNST** → ↓ ETOH self-administration at 24h
		1. but not in stressed mice
	4. CRF2 antagonism in **BNST** → ↑ ETOH self-administration at 24h
		1. in both defeated and non-stressed mice
8. ****CRF-CRF1 activity in **BNST** promotes ETOH consumption
9. CRF-CRF2 activity in **BNST** limits ETOH consumption
10. **BNST** CRF- CRF1 & CRF2 activity influence Alcohol addiction
	1. but the CRF1 & CRF2 actions are opposite

[Brock Baade](http://usdbiology.com/cliff/Courses/Advanced%20Seminars%20in%20Neuroendocrinology/Anxiety%20and%20BNST%20Peptides%2018/Brock18Anxiety%20and%20BNST%20Peptides.pptx) – 23 March 2018

[Serotonin inputs to the dorsal BNST modulate anxiety in a 5-HT1A receptor-dependent manner](http://usdbiology.com/cliff/Courses/Advanced%20Seminars%20in%20Neuroendocrinology/Anxiety%20and%20BNST%20Peptides%2018/Garcia-Garcia%2017%20MolecPsych%205-HT1A%20dBNST%20modulate%20anxiety.pdf) AL Garcia-Garcia, S Canetta, JM Stujenske, NS Burghardt, MS Ansorge, A Dranovsky, ED Leonardo 2018 [Molecular Psychiatry](https://www.nature.com/mp/)

1. Acute selective serotonin reuptake inhibitor (SSRI) → ↑ **anxiety**
	1. SSRI → ↑ synaptic 5-HT after release
	2. SSRIs include fluoxetine (Prozac), sertraline (Zoloft), citalopram (Celexa), ecitalopram (Lexapro), fluvoxemine (Faverin), Paroxetine (Paxil)
2. **Anxiety** activates serotonergic raphé neuron release of serotonin (5-HT) in **BNST**
	1. 5-HT receptors differentially expressed throughout **BNST**
		1. 18 types of 5-HT receptors
	2. 5-HT1A receptors are heavily expressed in **BNST**
		1. 5-HT1A are Gi →x ~~cAMP~~ inhibitory receptors
			1. high affinity receptors
	3. 5-HT2C receptors are also found in **BNST**
		1. 5-HT2C are GP/q/11 → PLC → PIP2 → IP3 → ↑Ca++ excitatory receptors

 → DG → PKA

* + - 1. low affinity receptors
1. 5-HT → **BNST** → hyperpolarized 37% of **BNST** neurons
	1. likely via 5-HT1A receptors
		1. 5-HT1A are Gi→ αsubunit→x ~~AC~~ →→ βγ → GIRK1-GIRK4 → K+ efflux
			1. GIRK → K+ efflux → K+ hyperpolarization
				1. 25% of hyperpolarizations
			2. GABA reversal potential is 75% of hyperpolarizations
	2. depolarized 25% of **BNST** neurons
		1. likely via 5-HT2C receptors
	3. hyperpolarized and depolarized 19% of **BNST** neurons
		1. likely via 5-HT3 receptors
	4. 19% do not respond to 5-HT
2. 5-HT injected intraBNST blocks fear potentiated startle response
	1. 5-HT1 agonist (5-CT) intra**BNST** blocks fear potentiated startle
		1. 5-HT and 5-CT produce similar hyperpolarizations
	2. ****5-HT1 receptors disinhibit neurons that block fear potentiated startle
	3. ****2 synapse disinhibition
3. optogenetic TpH-ChR2 simulation of raphé → **BNST** projections → 5-HT inhibition of **BNST**
	1. blue light on BNST terminals → ↓ c-fos in dorsal **BNST**, not **vBNST**
	2. raphé → CeA optogenetic release of 5-HT inhibits c-fos in CeL and CeM
		1. CeA = central amygdala, CeL = lateral CeA, CeM = medial CeA
4. optogenetic -blue TpH-ChR2 → 5-HT release in **dBNST** → ↓ **anxiety**
	1. ↑5-HT in dBNST → ↑ center time in open field (OF)
	2. ↑5-HT in dBNST → ↑ open arm time and entries in elevated plus maze (EPM)
	3. ↑5-HT in dBNST → ↓ latency to feeding in a novel stressful setting (NSF)
	4. optogenetic-blue TpH-ChR2 → 5-HT release in CeA does not affect **anxiety**
	5. optogenetic-green-Pet-ARC → ↓ 5-HT release in **dBNST** → ↑ c-fos
		1. no effect on vBNST
		2. optogenetic-green-Pet-ARC → ↓ 5-HT release in **dBNST** → ↑ **anxiety**
			1. ↓ center time in OF, ↓ open arm time in EPM
5. 5-HT1A antigonist → ↑ **anxiety** paired with optogenetic → ↑ 5-HT release in **dBNST**
	1. in OF, EPM and NSF
	2. ****5-HT1A stimulation is necessary for ↓ **anxiety**
6. optogenetic stimulation of local **BNST** CRF/GABA cells inhibits outputs to the VTA
	1. BNST projects widely through the brain
	2. Local **BNST** CRF/GABA are depolarized by 5-HT via 5-HT2C receptors
	3. 5-HT → 5-HT2C → ↑ activity in CRF/GABA local neurons
		1. local **BNST** CRF/GABA neurons do not project to the VTA
	4. VTA projecting neurons are not stimulated by 5-HT
		1. hyperpolarized
7. opto blue stimulation 10mW of **d+vBNST** ↑↑5-HT → ↑ **anxiety**
	1. 5-HT receptors differentially expressed throughout **BNST**
	2. ↑↑5-HT → BNST → ↑ freezing in cued and contextual fear conditioning
	3. ↑↑5-HT → via optogenetic stimulation of BNST → ↓ open arm time in EPM
	4. ↑↑5-HT → via optogenetic stimulation of BNST → ↑ latency to feeding NSF
8. Projection CRF/GABA **BNST** neurons are inhibited indirectly by 5-HT
	1. via activation of local CRF/GABA **BNST** neurons through 5-HT2C receptors
9. Acute SSRI fluoxetine activates GABA currents from local CRF/GABA **BNST** neurons
10. Acute SSRI → ↑ **anxiety**
	1. Acute SSRI → ↓ open arm time and entries in EPM
	2. Acute SSRI → ↑ freezing in contextual + cued fear conditioning
		1. blocked by CRFCre:hM4Di + CNO inhibition of CRF/GABA interneurons
		2. also blocked by HSVCre:hM3Dq + CNO activation of BNST projection neurons to VTA and LH

[Ben Onserio](http://usdbiology.com/cliff/Courses/Advanced%20Seminars%20in%20Neuroendocrinology/Anxiety%20and%20BNST%20Peptides%2018/Ben18Anxiety%20and%20BNST%20Peptides.pptx) – 6 April 2018

[Acute engagement of Gq-mediated signaling in the bed nucleus of the stria terminalis induces anxiety-like behavior](http://usdbiology.com/cliff/Courses/Advanced%20Seminars%20in%20Neuroendocrinology/Anxiety%20and%20BNST%20Peptides%2018/Mazzone%20Kash%2018%20MolPsych%20Gq%20BNST%20anxiety.pdf) CM Mazzone, D Pati, M Michaelides, J DiBerto, JH Fox, G Tipton, C Anderson, K Duffy, JM McKlveen, JA Hardaway, ST Magness, WA Falls, SE Hammack, ZA McElligott, YL Hurd, TL Kash 2018 [Molecular Psychiatry](https://www.journals.elsevier.com/neuroscience-letters) 23: 143–153

1. Stress/**anxiety** reduces α1-adrenoreceptor long-term depression (LTD) in **vlBNST**
	1. reduced depression of excitatory postsynaptic current (EPSC) signal over time
		1. via PLC and cAMP 2nd messengers
	2. also in **dlBNST**
	3. not in mGluR5 stimulated LTD
	4. α1-adrenoreceptor stimulation → ↑ spontaneous EPSCs
	5. CRF1 antagonist NBI 27914 blocked α1-induced sEPSCs
		1. α1 activity stimulates CRF→ CRF1 receptors → ↑ sEPSCs s
2. Acute and Chronic Alcohol (EtOH) also reduces α1-induced LTD in **vlBNST**
	1. coincident with **Anxiety**
3. **Anxiety** acts in a similar way to stress, reducing LTD in **BNST**
4. Chronic variable stress → ↑ **anxiety** (**anxiogenic**)
	1. increased by bright lights
		1. dependent on **BNST** activity
	2. measured by amplitude of acoustic startle response
		1. stress or **anxiety** or fear results in fear potentiated acoustic startle response
	3. increases PACAP and its receptor PAC1 in **dlBNST**, not **vlBNST**
		1. PACAP = Pituitary Adenylate Cyclase Activating Protein
		2. BDNF and its TrKB receptor are also increased in **dlBNST** by stress
			1. also in dRN and PVN for BDNF
			2. in PVN for PAC1
5. PACAP infusion into **aBNST** is **anxiogenic**
	1. ↑ acoustic startle - is dose dependent with respect to PACAP
	2. long-lasting effect (7 days)
		1. suggests that PACAP-PAC1 activity → neural plasticity
6. Acute chemogenetic (hM3Dq-CNO) activation of **BNST** GABA neurons → ↑ **anxiety**
	1. rAAV8-hsyn-DIO-hM3Dq-mCherry transfection in both **d and vBNST**
		1. + vGATCre + *Rosa26*-floxed1-stop-L10-GFP
		2. reduced EPSC signal = LTD
			1. Cb1 receptor antagonist blocked LTD
				1. Cb1 receptor antagonist = SR14176A = rimonabant
		3. not reduced by rAAV8-hsyn-DIO-hM3Di
		4. nor by rAAV8-hsyn-DIO-hM3Ds
	2. ****Cb1 is required downstream of Gq receptor to produce LTD
	3. less time in Elevated Plus Maze (EPM) open arms
	4. less time in the center of the open field (OF)
	5. less time in the light side of the Light-Dark box
7. DREADD hM3Dq-CNO activation of **BNST** GABA neurons → ↑ metabolic activity in
	1. VTA = ventral tegmental area (makes DA)
	2. PBN = parabrachial nucleus (arousal)
	3. LC = locus ceruleus (makes NE)
8. Chemogenetic hM3Dq-CNO → ↑ **BNST** GABA neurons reveals ↑expression of Gq receptors
	1. mGluR5 - 95% of GABA cells express this Gq receptor
	2. Neurotensin 2 receptor - 88%
	3. M1 muscarinic ACh receptor - 70%
	4. 5-HT2C - 55%
	5. mGluR1 - 50%
	6. α1 adrenergic receptors - 30%
9. ↑ synaptic 5-HT stimulates similar ↑ acoustic startle as DREADD hM3Dq
	1. mCPP = SSRI → ↑ synaptic 5-HT
	2. hM3Dq may reflect the actions to 5-HT2C receptors
10. activation of **BNST** 5-HT2C receptors → Gq → ↑ **anxiety**

[Kevin Krupp](http://usdbiology.com/cliff/Courses/Advanced%20Seminars%20in%20Neuroendocrinology/Anxiety%20and%20BNST%20Peptides%2018/Kevin18Anxiety%20and%20BNST%20Peptides.pptx) – 13 April 2018

[Neuropeptide Y2 receptors in anteroventral BNST control remote fear memory depending on extinction training](http://usdbiology.com/cliff/Courses/Advanced%20Seminars%20in%20Neuroendocrinology/Anxiety%20and%20BNST%20Peptides%2018/Verma%20Pape%2018%20NPY2%20avBNST%20control%20remote%20fear%20memory.pdf) D Verma, R Tasan, G Sperk, H-C Pape 2018 [Neurobiology of Learning and Memory](https://www.journals.elsevier.com/neurobiology-of-learning-and-memory) 49: 144–153

1. In the **BNST**, NYP secretion influences **anxiety**
	1. NPY is the most abundant peptide in the brain
		1. 36 amino acid peptide
		2. NPY family: NPY, Peptide YY (PYY), and pancreatic polypeptide (PP)
	2. 7 NPY receptors
		1. Y1, Y2, Y4, Y51, Y6, Y7, Y8
			1. All inhibitory: Gi coupled
			2. NPY has high affinity for all
		2. Y2 are the only presynaptic NPY receptors
			1. Auto-receptors = negative feedback
	3. NPY is often expressed in GABA neurons
		1. Colocalized
		2. Also colocalized with somatostatin, nitric oxide (NO), and parvalbumin
	4. NPY is found in Amygdala, Hippocampus, Cortex, Hypothalamus
		1. Moderate Y1 and Y2 mRNA in BLA
		2. NPY into hippocampus stimulates FGF and neurogenesis
		3. NPY intraBLA or CeA is anxiolytic
			1. Footshock → ↑ NPY gene expression
	5. Y1 receptors are **Anxiolytic**
	6. NPY polymorphisms are related to stress sensitivity in humans
2. NPY in the **BNST**
	1. High density of NPY fibers
		1. Projections from other brain regions
			1. From the arcuate nucleus (ARC) of the hypothalamus
	2. 5-HT1A expressed in **BNST**

Spring 2017 Decision Making during Depression

Fall 2017 Social Stress Resilience

Spring 2018 Anxiety and BNST Peptides

****∆ → ↑ ↓ ↔   ° é ∑ α β γ δ κ λ θ μ π τ ± ≤ ≈ ≠ ≥ ♀ ♂ ∞

Possible topics Fall 2015 1st vote 2nd Vote 3rd Vote 4th Vote

Sleep I’ll put in the votes later

Decision Making during Depression

Mirror Neurons

Endocannabinoids +

IGF1

Epilepsy

Happiness

Plasticity

Possible topics Fall 2014 1st vote 2nd Vote 3rd Vote 4th Vote

Sleep IIIII 1 -

Optogenetics in nucleus accumbens IIIII 6 7

Mirror Neurons IIII 3 -

Endocannabinoids IIIII 6 11

IGF1 IIII 6 6