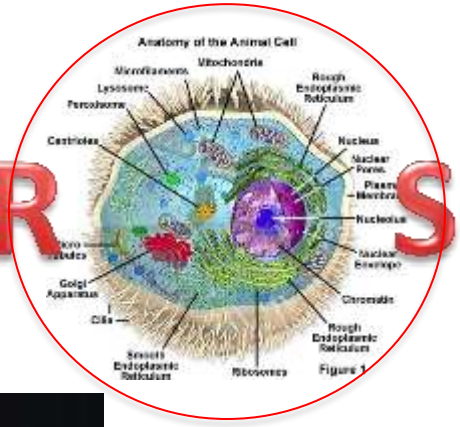
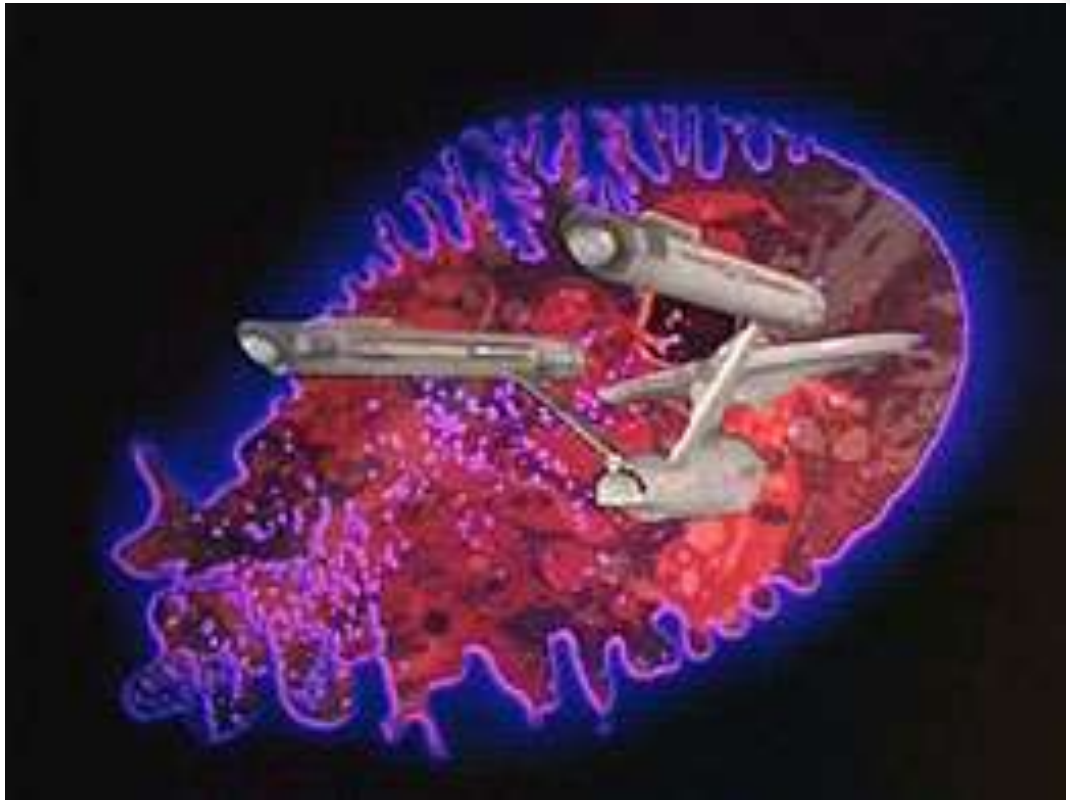
Welcome to section 4

Biology 580
CRN: 32081-Lecture
CRN: 24444-Lab
Spring, 2010

CELLULAR NEUROSCIENCE





Instructors	Department
Ethan Gahtan	Psychology
Bruce A. O'Gara	Biology
Jacob Varkey	Biology
Borbala Mazzag	Mathematics

CELLULAR NEUROSCIENCE – WELCOME TO SECTION 4!

Agenda

1. Note #1: Thanks to Varkey, O'Gara and Mazzag!!!!!!!!
2. Note #2: flash drive with swimmy propaganda on it
3. Reintroduction to the cellular neuroscience mood
4. Organization of section 4 lecture topics by emotion
5. Organization of section 4 material on Moodle
6. Levels of analysis worksheet due every class (to fill in, but not necessarily to hand in)
7. Assignment of students to papers (no powerpoint presentation required- simply know your paper, including all elements of all figures, well enough to explain them if called upon. This will likely require referencing additional sources of information)
8. We will also work together to record a list of important terms and concepts that come up in each class
9. Begin Article #1 on neurobiology of aggression with emphasis on cellular mechanisms
10. Begin article for lab on mechanisms of trigeminal 'spicy heat' transduction
11. Lab meets in BSS 420, then breaks out into groups

Section Themes: Lecture: cells to behavior. Lab: Optical methods

Apr 19	BSS 211	Aggression: Steroids, cells, cognition, behavior	Steroid mechanisms in aggressive behavior	
LAB	BSS 416	Trigeminal chemosensation mechanisms- Signaling through TRP and P2X receptors	TRP receptors article Purine Receptors article	In-class work groups design and begin lab demo
Apr 21	BSS 211	Love and Peptides	A. Love article 1 B. Love article 2	
Apr 26	BSS 211	Fear and amygdala mechanisms	C. Fear article	
LAB		Continue trigeminal chemosensation lab demo and/or present results		
Apr 28	BSS 211	Hunger: Leptin signaling	D. Leptin signaling and obesity review	
May 3	BSS 211	Depression: neurogenesis	E. Antidepressant drugs and neurogenesis	
LAB		Imaging synapses in vivo with GFP fusion proteins transgenic zebrafish	F. Synaptophysin review	In-class Worksheet
May5	BSS 211	Hallucination	G. Serotonin-glutamate receptor interactions	reading: PMID: 18297054 post pending
May 10	BSS 211	Section review and quiz activity event		
LAB		Optical Physiology- focus on Calcium imaging Visualize calcium responses in zebrafish reticulospinal neurons	Roger Y. Tsien Nobel speech H. Calcium indicator review (MR-E5)	

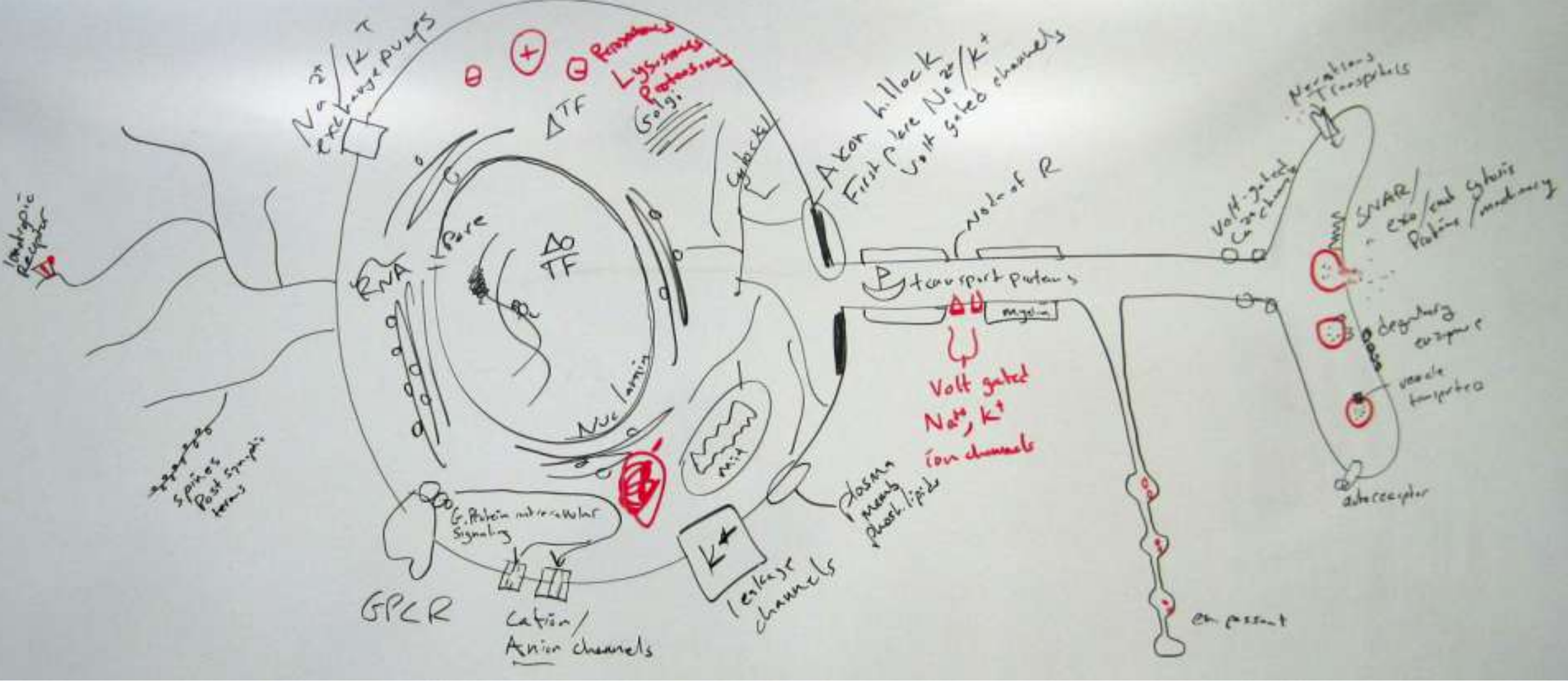
Course review and evaluation

May 12	BSS 211			

Section 4 at a glance

NOTES:
Readings are hyperlinked through calendar

Need to sign up for papers A-H


Na+/K+ exchange pumps
Lysosomes
Golgi
ΔTF
Axon hillock
First place Na+/K+ volt gated channels
Node of R
RNA
Pore
Nuc lamina
Mit
transport proteins
myelin
Volt gated Na+, K+ ion channels
plasma memb phospholipids
G-Protein intracellular Signaling
GPCR
Cation/ Anion channels
leakage channels
spines Post synaptic terms
SNARE
degrading enzymes
vesicle transporters
autoreceptor
en passant

The cellular level of analysis in neuroscience:

Events in or on a neuron that are important to its function and the operation of neural systems

Cellular level events include the following (can we add to this list?). Operations of:

Membrane channels
Receptors
Signaling molecules
Organelles
Morphology
Size
Gene regulation
Expressed proteins
Epigenetic states
Development
Projection patterns
Neural system it operates in

What is the dictionary definition of “cellular neuroscience”?

Category:Cellular neuroscience

From Wikipedia, the free encyclopedia

The study of neurons at a cellular level including morphology and physiological properties of single neurons.

Pages in category "Cellular neuroscience"

The following 10 pages are in this category, out of 10 total. This list may not reflect recent changes (learn more).

A

- Action potential

C

- Calcium concentration microdomains
- Cellular neuroscience
- Chemical synapse

D

- Dendrite

I

- IKK2

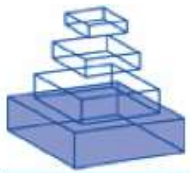
S

- Sholl analysis
- Soliton model

S cont.

- Synapse
- Synaptotropic hypothesis

Bottom line: cells and subcellular processes



frontiers IN CELLULAR NEUROSCIENCE

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Frontiers in Science › Frontiers In Cellular Neuroscience

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About the Journal

FACTS

Specialty Editor-In-Chief: Alexander Borst, Max Planck Institute of Neurobiology, Germany
Abbreviation: Front. Cell. Neurosci.
Print ISSN:1662-5102
NLM ID: 101477935
Indexed in: Pubmed Central, PubMed and Google Scholar

MISSION STATEMENT

Frontiers in Cellular Neuroscience is a first-tier electronic journal devoted to better understanding the cellular mechanisms underlying the functions of the cells composing the nervous system (neural and non-neuronal) across all species. The past years have seen exciting progress in this area because of the merging of traditionally separate fields such as anatomy, physiology and molecular genetics. We welcome submissions of multidisciplinary studies of cellular function in vertebrates and invertebrates, in cell culture, *in vivo*, or using slices, and involving genetically amenable species, such as *Drosophila*, *C. elegans*, mice and zebra fish, as well as any other species suited to elucidate fundamental principles of cellular function in neurons. Studies on basic neural functions, such as the generation of action potentials, synaptic transmission, biophysical and biochemical aspects of receptor activation, synaptic plasticity, intra and inter cell signaling related to the emergent functions of cells, glial-neuronal signaling and synaptic and dendritic integration, are welcome. Frontiers in Cellular Neuroscience also publishes research on the morphology of cells and how these morphologies relate to the emergent functions of neurons. Our journal also publishes research focused on the developing adult and ageing cell, as well as cellular changes in diseases. While the journal's primary focus is on experimental studies, we also welcome the addition of computational models to further explore experimental findings.

Bottom line: mostly subcellular processes but some allowance for cell-cell signaling processes

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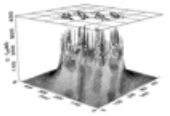
Cellular Neuroscience



transduction mechanisms and modulation

The cellular neuroscience group is united by an interest in biophysical, biochemical, and molecular processes that govern the function of a variety of sensory receptors and neurons. Intracellular recording, patch-clamp, and voltage-clamp techniques as well as pharmacology and Ca2+ imaging are applied to a wide range of model systems including hair cells in amphibia, chemosensory neurons in the nematode, photoreceptors in the horseshoe crab and fruit fly, muscle cells of the tobacco moth heart, pyramidal cells of the rat hippocampus, and olfactory neurons in salmon.

Much of the work in this group focuses on the activity and regulation of membrane currents and ion channels. Common themes in this research include the relationship between voltage-gated ion channels and whole-cell electrical properties (Lockery, O'Day, Roberts), ion currents in wild-type and mutant animals (Lockery, O'Day), the spatial distribution of voltage and ligand-gated channels (Roberts, Barbara Gordon-Lickey, Marvin Gordon-Lickey), and the effects of peptides and second-messenger systems on membrane currents (O'Day, Roberts, Tublitz).

A second focus is on the cellular mechanisms of sensory transduction. Several groups investigate the physiology of excitatory responses to sensory inputs in taste and olfaction (Lockery, Roberts, Takahashi), vision (O'Day), and hearing (Roberts). Other groups are concerned with how the response to sensory input is modified by experience through sensory adaptation. Topics here include the covalent modification of primary receptor molecules (Dahlquist), and the biochemical feedback pathways contributing to adaptation in photoreceptors (O'Day).

Bottom line: subcellular processes

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Molecular and Cellular Neuroscience

ISSN: 1044-7431
Imprint: ACADEMIC PRESS

Actions

Molecular and Cellular Neuroscience publishes original research of exceptional significance from those areas of the neurosciences indicated by the broadest interpretation of the journal's title. In particular, the journal focuses on synaptic maintenance and organization, neuron-glia communication and regenerative neurobiology. As part of the submission process, authors are asked to state why they consider their paper is of such significance. Furthermore, since rapid peer-review and publication of such research is of paramount importance, extended cycles of article revision and re-review will not be entered into; it is anticipated that authors will fully address all referees' comments during the course of a single revision of their original manuscript.

US National Institutes of Health (NIH) voluntary posting ("Public Access") policy

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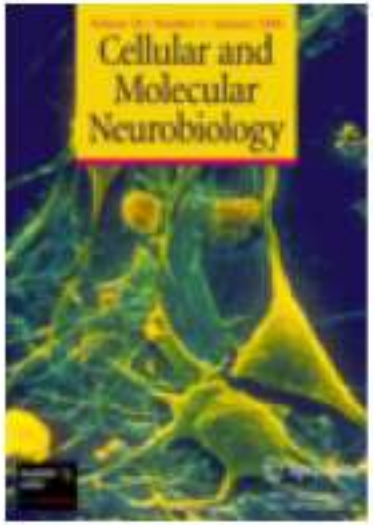
Hide Aims & Scope

Bottom line:

1. broad definition includes/highlights cell-cell interactions.
2. Our journal is hot sh*t.



Cellular and Molecular Neurobiology
Editor: J.M. Saavedra
ISSN: 0272-4340 (print version)
ISSN: 1573-6830 (electronic version)
Journal no. 10571
Springer US
Online version available
Online First articles available

Description

Bottom line:
Single cells and subcellular processes

Cellular and Molecular Neurobiology publishes original research concerned with the analysis of neuronal and brain function at the cellular and subcellular levels. This superb bimonthly offers timely, peer-reviewed articles that describe anatomic, genetic, physiologic, pharmacologic, and biochemical approaches to the study of neuronal function and the analysis of elementary mechanisms. Studies are presented on isolated mammalian tissues and intact animals, with investigations aimed at the molecular mechanisms or neuronal responses at the level of single cells. The journal also presents studies of the effects of neurons on other organ systems, such as analysis of the electrical or biochemical response to neurotransmitters or neurohormones on smooth muscle or gland cells. All articles are rigorously appraised by at least two referees.

The Ph.D. Program in Behavioral and Cellular Neuroscience at Texas A&M University

Behavioral neuroscience represents an exciting and rapidly expanding field. Graduate training in Behavioral Neuroscience at Texas A&M University provides students with the opportunity to develop a close collaborative relationship with a primary advisor. Behavioral Neuroscience faculty and graduate students meet regularly to discuss current and ongoing research. An early emphasis on laboratory research allows graduate students in Behavioral Neuroscience the opportunity to rapidly participate in the exciting process of disseminating their research findings to the larger scientific community. Graduate students in the Behavioral Neuroscience program routinely receive research awards from the Office of Graduate Studies to provide additional support for their thesis and dissertation research. Students also receive travel awards regularly from the Psychology Department and Faculty of Neuroscience that allow for attendance and participation at prestigious scientific conferences. As a result, students typically graduate having published several scientific papers in addition to their Ph.D. dissertation. It is not surprising, then, that graduates in Behavioral Neuroscience at TAMU have obtained faculty positions at other first-rate academic institutions (e.g., University of Arizona, University of Missouri, Kent State University), as well as prestigious post-doctoral fellowships (e.g., Columbia University, Yale, and UCLA).

Bottom line:

1. Broad definition… even *psychologists* are involved in cellular neuroscience
2. Cellular neuroscience is even relevant to understanding *behavior*
3. Our graduate program is hot sh*t

Bottom line:

1. **Broad definition: subcellular events, but at the service cell-cell interactions**
2. **Cellular neuroscience is even relevant to SOCIAL behavior**

Social studies

DOI:
10.1038/nrn2120

The transmembrane glycoprotein CD38, found on many immune cells, is also expressed in the brain, although its function there has remained elusive. Jin, Liu *et al.* have now shown that CD38 is required in mice for the regulation of social behaviour by oxytocin.

Female mice in which *Cd38* was knocked out displayed disrupted maternal behaviour and their male counterparts showed reduced social memory, resembling the phenotype of oxytocin- and oxytocin receptor-knockout mice. Investigating a possible link between CD38 and oxytocin, the researchers found that, compared with wild-type animals, $Cd38^{-/-}$ mice had reduced plasma and cerebrospinal fluid (CSF) oxytocin levels, but elevated levels in the hypothalamus and pituitary. Furthermore, the neurovascular contact zone in the pituitary of $Cd38^{-/-}$ mice contained many dense core vesicles containing oxytocin, indicating that although the hormone was produced and packaged into vesicles in $Cd38^{-/-}$ mice, it was not released. Indeed, the behavioural phenotype of $Cd38^{-/-}$ mice could be normalized by subcutaneous oxytocin injections.

The researchers infused a lentivirus carrying the human *CD38* gene into the third ventricle of $Cd38^{-/-}$ mice, which resulted in normalization of plasma and CSF oxytocin levels. It also normalized their social memory, indicating that the mechanisms underlying social behaviour require CD38-dependent oxytocin secretion.

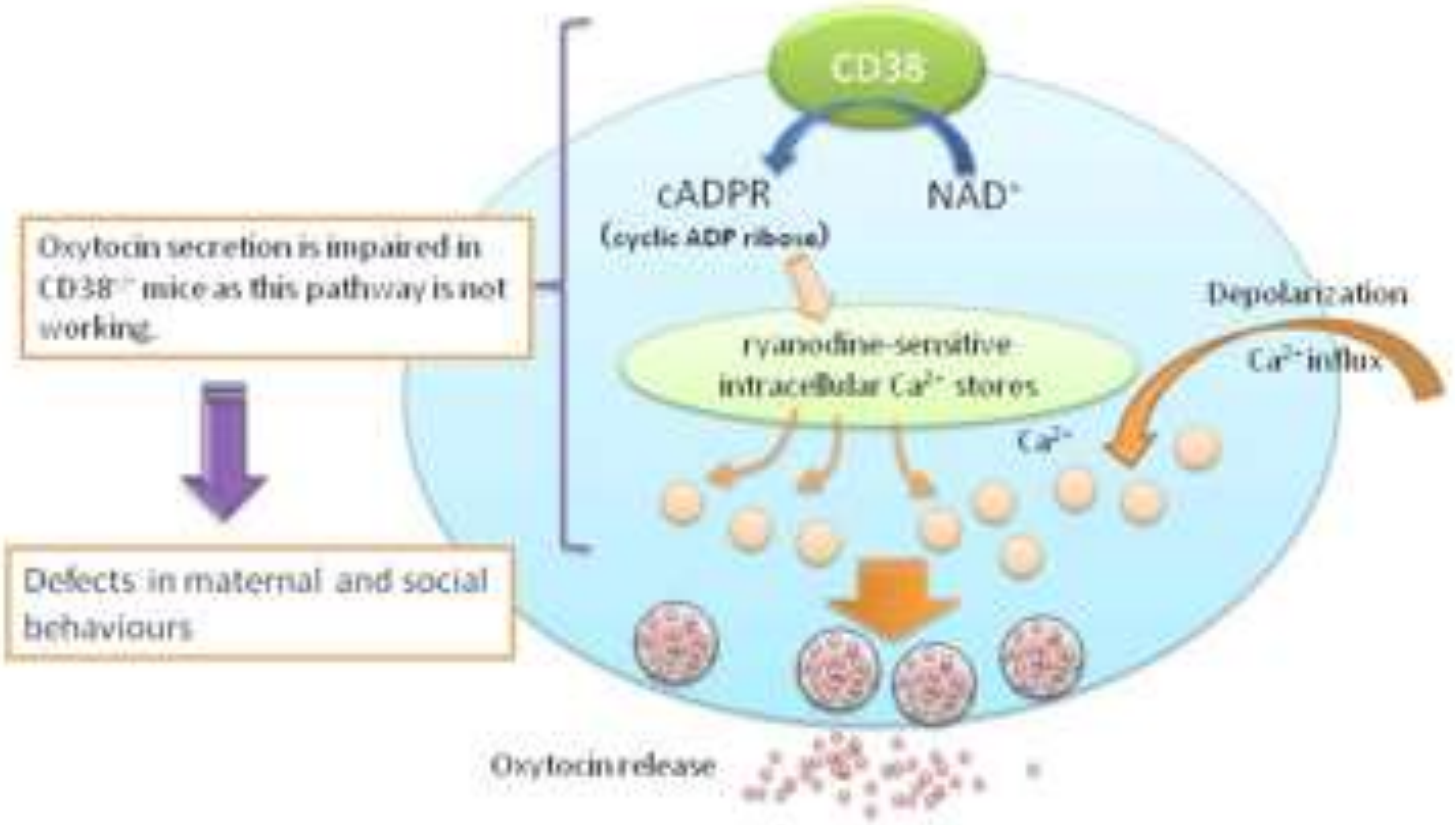
Jin, Liu *et al.* next investigated how CD38 regulates oxytocin secretion. CD38 is known to catalyse the extracellular generation of cyclic ADP ribose (cADPR) and transport it across the cell membrane, where it subsequently mobilizes Ca^{2+} from intracellular stores. Ca^{2+} signalling is necessary for oxytocin secretion, but the mechanisms by which this happens are unknown. In $Cd38^{-/-}$ mice, extracellular cADPR could not stimulate oxytocin release in pituitary nerve endings like it does in wild-type mice, unless the nerve endings were first made permeable with digitonin. Furthermore, depolarization-induced increases in intracellular Ca^{2+} were lower in pituitary nerve endings from $Cd38^{-/-}$ mice, indicating that intracellular Ca^{2+} is mobilized less efficiently than in wild-type animals. Finally, Ca^{2+}-dependent oxytocin secretion in response to depolarization was reduced in hypothalamic neurons and axon terminals from $Cd38^{-/-}$ mice compared with those from wild-type mice. Together, these data showed that the dual functions of CD38 as a transporter and an enzyme are necessary for normal Ca^{2+}-induced oxytocin release.

These findings shed light on the function of CD38 in the brain and, potentially, in human diseases associated with abnormal social behaviour such as autism.

Leonie Welberg

ORIGINAL RESEARCH PAPER Jin, D., Liu, H.-X. et al. CD38 is critical for social behaviour by regulating oxytocin secretion. *Nature* **446**, 41–45 (2007)

CD38 is a transporter and enzyme that catalyzes formation of intracellular Ca++ signaling molecules required for release of oxytocin



Cellular mechanisms regulating OT release from posterior pituitary nerve endings

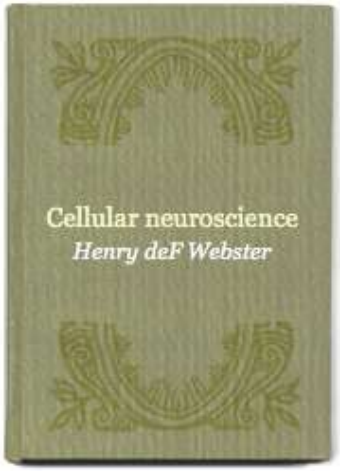
http://int.physiology.jp/image/4666/file.png

Cellular neuroscience

projects and images, 1957-1997

by Henry deF Webster

Published in 2006, Gateway Press, Inc. (Baltimore, MD)



CHANGE COVER

Contributions:	National Institutes of Health (U.S.), National Institute of Neurological Disorders and Stroke (U.S.)
works:	Cellular neuroscience
By statement:	Henry de Forest Webster.
Language:	English
Pagination:	x, 139 p. :
LCCN:	2006932016
Dewey:	612.8092
LC:	QP355.2 .W45 2006
Genre:	Biography.
Subject:	Webster, Henry deF. Neuroscientists — Biography. Neurosciences.

DESCRIPTION

Notes: Includes bibliographical references (p. [95]).

Dr. Webster writes about his career in neuroscience research, primarily during his tenure at the National Institutes of Health. He retired as chief of the Laboratory of Experimental Neuropathology, National Institute of Neurological Disorders and Stroke, in 1997.

Bottom line: Someone named their personal memoire 'cellular neuroscience'!

CONCLUSION IN THE EXERCISE OF DEFINING CELLULAR NEUROSCIENCE :

NO NEURON IS AN ISLAND

Article 1: Aggression

Adolescent exposure to anabolic/androgenic steroids and the neurobiology of offensive aggression: A hypothalamic neural model based on findings in pubertal Syrian hamsters

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GABA
Glutamate
Hypothalamus
Hamster

ABSTRACT

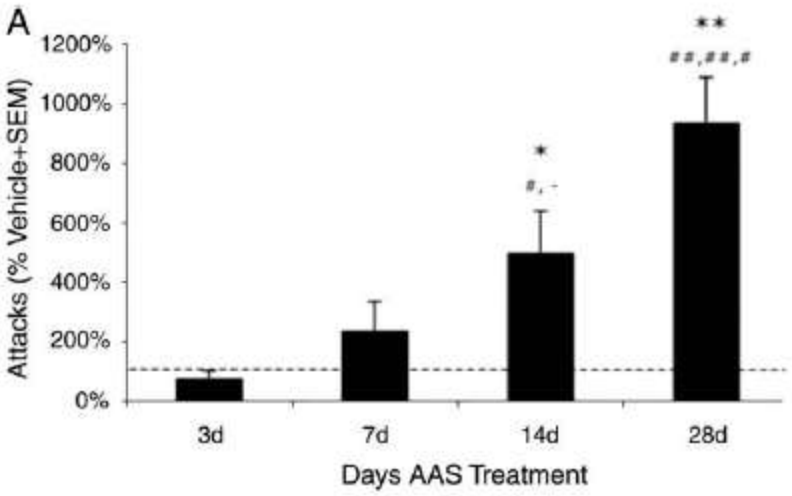
Considerable public attention has been focused on the issue of youth violence, particularly that associated with drug use. It is documented that anabolic steroid use by teenagers is associated with a higher incidence of aggressive behavior and serious violence, yet little is known about how these drugs produce the aggressive phenotype. Here we discuss work from our laboratory on the relationship between the development and activity of select neurotransmitter systems in the anterior hypothalamus and anabolic steroid-induced offensive aggression using pubertal male Syrian hamsters (*Mesocricetus auratus*) as an adolescent animal model, with the express goal of synthesizing these data into an cogent neural model of the developmental adaptations that may underlie anabolic steroid-induced aggressive behavior. Notably, alterations in each of the neural systems identified as important components of the anabolic steroid-induced aggressive response occurred in a sub-division of the anterior hypothalamic brain region we identified as the hamster equivalent of the latero-anterior hypothalamus, indicating that this sub-region of the hypothalamus is an important site of convergence for anabolic steroid-induced neural adaptations that precipitate offensive aggression. Based on these findings we present in this review a neural model to explain the neurochemical regulation of anabolic steroid-induced offensive aggression showing the hypothetical interaction between the arginine vasopressin, serotonin, dopamine, γ-aminobutyric acid, and glutamate neural systems in the anterior hypothalamic brain region.

WHY HAMSTERS?

Because

1. They have a definite adolescence period
2. They display offensive aggression as a normal part of adolescent behavior- this is attributed to **adaptations** to mating challenges
3. Extensive research shows that steroids exposure during **development** modulates offensive aggression
4. Brain structures and chemicals implicated in aggression control are expected to be homologous in humans

http://www.youtube.com/watch?v=xO7IlFIgZAc



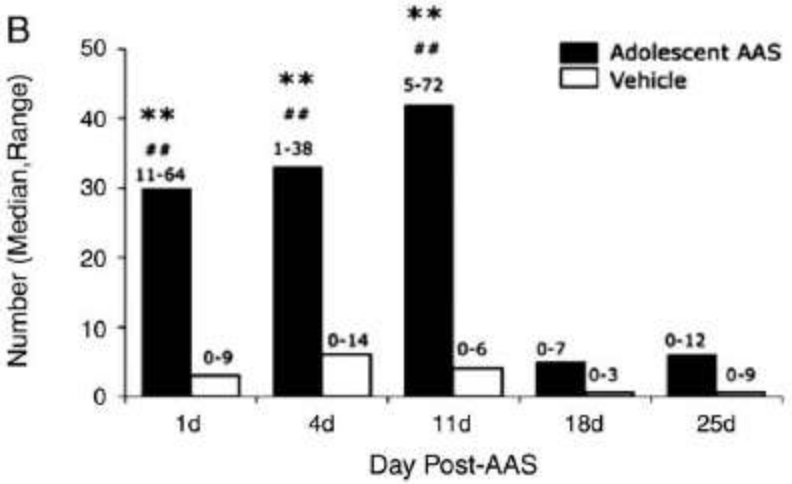


Fig. 1. Adolescent AAS exposure increases offensive aggression in pubertal Syrian hamsters. (A) Comparison of offensive aggression during the course of adolescent AAS treatment from (Grimes et al., 2007). Comparisons to vehicle-treated controls: *$p<0.05$, **$p<0.01$. Within-group comparisons of adolescent AAS-treated animals at 3 and 7 days versus 14 days of exposure and 3, 7, and 14 days versus 28 days of exposure, respectively: #$p<0.05$, ##$p<0.01$. Mann–Whitney *U* tests (two-tailed). (B) Comparison of offensive aggression during withdrawal from adolescent AAS treatment from (Grimes and Melloni, 2006; Grimes et al., 2006a). Comparisons to controls: **$p<0.01$. Within-group comparisons of adolescent AAS-treated animals at 1, 4 and 11 days versus 18 and 25 days post-AAS exposure, respectively: ##$p<0.01$. Mann–Whitney *U* tests (two-tailed).

Exposure to AAS during puberty increases offensive aggression in a dose dependent manner, suggest a receptor (cell biological) mediated effect

The effects of pubertal AAS exposure last for days after treatment cessation, suggesting cellular level neuroplastic changes (more later)

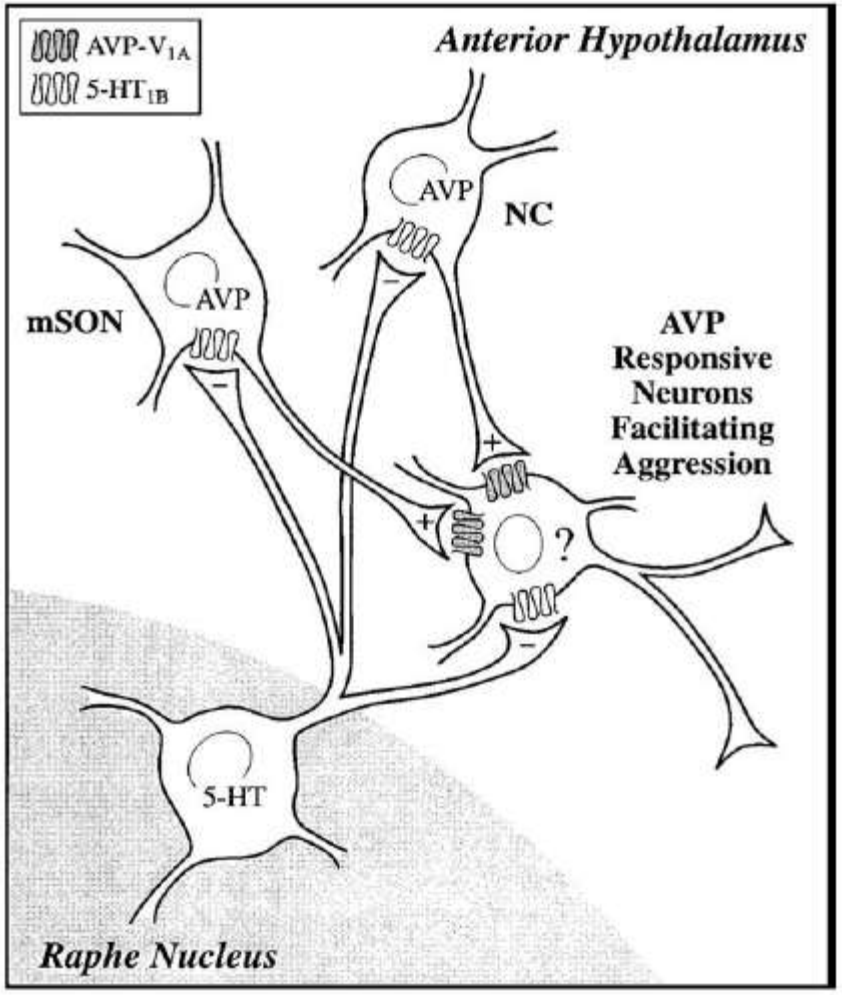


Fig. 2. The first model of the neurochemical regulation of offensive aggression from (Ferris et al., 1997) showing the hypothetical interaction between serotonin (5HT) with the arginine vasopressin (AVP) system in the anterior hypothalamus (AH). 5HT fibers originating from neurons in the medial and dorsal raphe nucleus innervate populations of AVP neurons in the AH localized to the medial supraoptic nucleus (mSON) and nucleus circularis (NC). Theses AVP neurons have been identified as potential sources of AVP innervation to the AH brain region involved in agonistic behavior. These AVP neurons together with 5HT neurons from the raphe nucleus impinge on neurons in the AH involved in the facilitation of aggression. The identity (?) of these post-synaptic neurons is unknown. 5HT is inhibitory (−), working through 5HT1B receptors (and/or 5HT1A receptors—not shown), whereas AVP is excitatory (+), working through AVP V1A receptors.

Modeling is understanding

Or is it?

This is a first generation model of the neural pathways and chemicals controlling offensive aggression

How does this 'model' relate to modeling in the computations sense? Would it be useful to translate this neural circuit schematic into a computation model?

The goal of this paper was to summarize decades of work on this system into a new, updated model. Is this 'modeling' effort useful?

Does the model help to predict things, e.g., drug effects (antidepressants, steroids)

Modeling and understanding begins with mapping effects.

What levels of analysis do these AAS effects belong to?

Table 1
Adolescent AAS-induced alterations in the neurobiology of offensive aggression.

Neural system(s) and experimental finding		Brain area					Reference
		AH	LAH	VLH	MeA	LS	
AVP	⇑ AVP afferent fibers/terminals		√				Harrison et al., 2000; Grimes et al., 2006a, 2007
	⇑ AVP peptide levels	√	√				Harrison et al., 2000
	⇑ AVP release	√	√				
	⇑ AVP V1A receptor binding			√		√	DeLeon et al., 2002
5HT	⇓ 5HT afferent fibers/terminals	√	√	√	√		Grimes and Melloni, 2002, 2006
	⇓ 5HT1A receptors (somata)	√	√				Ricci et al., 2006
	⇑ 5HT1B receptors (somata)		√	√	√		Grimes and Melloni, 2005
	⇓ 5HT1B receptors (punctate)	√	√	√	√		Grimes and Melloni, 2005
	⇑ 5HT2A receptors (somata)		√				Schwartzer et al., 2009b
	⇓ 5HT afferents onto AVP afferents		√				
AVP/5HT	⇓ 5HT1A receptors on AVP neurons		√				
	⇓ 5HT1B receptors on AVP neurons		√				
DA	⇑ DA neurons	√	√				Ricci et al., 2009
	⇑ DA afferent fibers/terminals	√	√				Ricci et al., 2009
	⇑ DA D2 receptors (somata)		√				Ricci et al., 2009; Schwartzer et al., 2009c
	⇓ DA D2 receptors (somata)			√			Ricci et al., 2009
	⇑ DA/AVP co-expression		√				
GABA	⇑ GABA afferent fibers/terminals	√	√	√	√		Grimes et al., 2003
	⇑ GABA neurons		√				Schwartzer et al., 2009c
	⇓ GABA A⟨1 receptors (punctate)		√				Schwartzer et al., 2009c
	⇑ GABA A⟨1 receptors (punctate)				√		Schwartzer et al., 2009c
DA/GABA	⇑ DA D2 receptors on GABA neurons		√				Schwartzer et al., 2009c
FOS	⇑ FOS (somata)		√			√	Ricci et al., 2007b
GLU	⇑ GLU neurons		√		√	√	Fischer et al., 2007; Carrillo et al., 2009
	⇓ GLU efferent projections to VLH		√				Carrillo et al., 2009
	⇑ GluR1 receptor (somata)			√			Fischer et al., 2007
FOS/GLU	⇑ FOS/GLU co-expression		√			√	Carrillo et al., 2009

Mapping effects of steroids to cellular events in neurons using in vivo microdialysis

Prior AAS exposure leads to greater AVP release in response to social threat.

Leads to questions: (1) what is the mechanism relating AAS to AVP? (2) what is the relationship between AVP release and aggression?

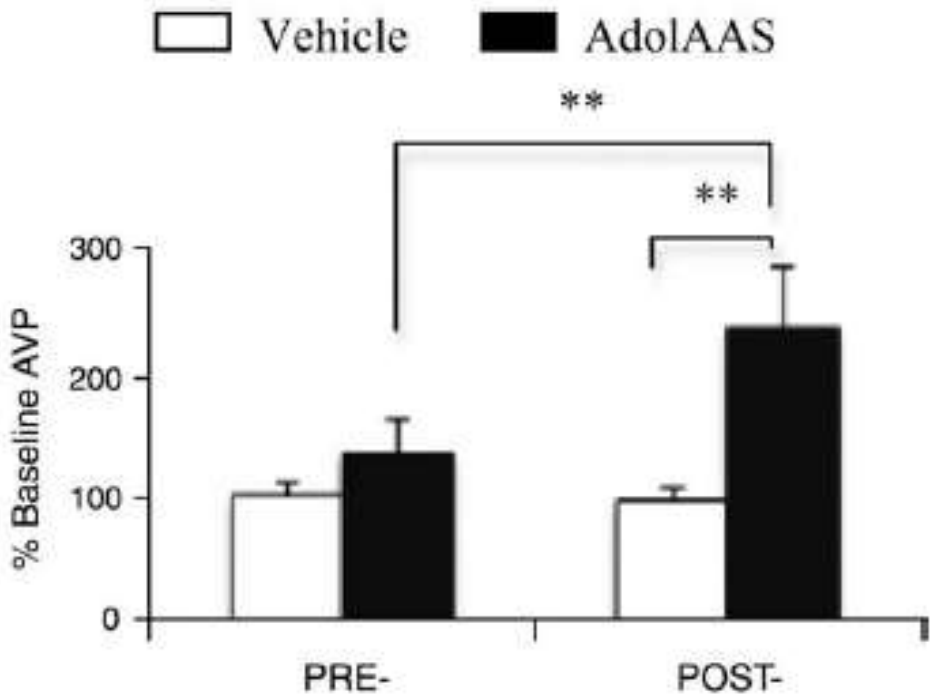


Fig. 3. Adolescent AAS exposure increases AVP release in the AH of pubertal Syrian hamsters. AH AVP release sampled using *in vivo* microdialysis every 30 min for 1 h prior to–(PRE) and following–(POST) exposure to intruders during resident/intruder testing in adolescent AAS- and vehicle-treated residents. Residents were separated from intruders by a wire mesh to prevent problems with the dialysis tether that often occur during testing. Even under these less than intense agonistic conditions, an increase in "intruder-evoked" release of AH AVP was seen in adolescent AAS-exposed residents (POST). $^{**}p<0.01$, Student's *t*-tests followed by ANOVA.

Circuit, developmental, and cellular levels of analysis

Circuit: AAS alters connectivity between 5HT and AVP neurons in the hypothalamus

Developmental: Effects are evident days or weeks after AAS treatment in adolescence, while functional system is developing

Cellular: different effects of AAS on 5HT1A (decreased) versus 5HT1B (increased) receptor expressing neurons in NC

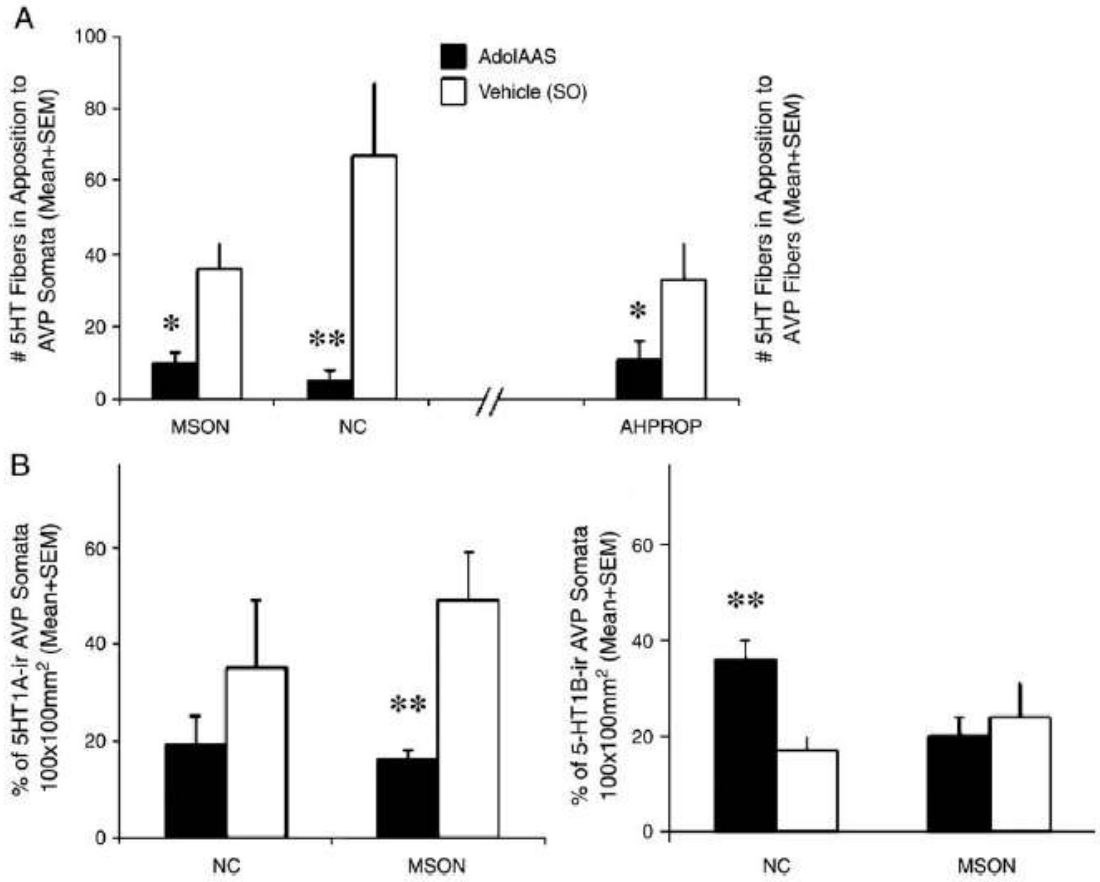


Fig. 4. Adolescent AAS exposure alters the connectivity of the AVP/5HT neural systems in the AH of pubertal Syrian hamsters. (A) Proximity of 5HT afferent fibers to AH AVP neurons in the mSON and NC and fibers the AH proper, respectively using double-label immunohistochemistry. Adolescent AAS-treated hamsters had fewer 5HT afferent terminals in close apposition to AVP somata in the mSON and NC (left graph) and AVP afferent fibers in the AH proper (right graph) compared to vehicle-treated controls. (B) Localization of 5HT1A and 5HT1B receptors onto AH AVP neurons using double-label immunohistochemistry. Adolescent AAS exposure decreased the percentage of 5HT1A-containing AVP neurons in the mSON (left graph), and increased the percentage of 5HT1B-containing AVP neurons in the NC (right graph).

A cellular level of analysis of behavior requires knowing what important subcellular elements (such as neurotransmitters) are present on what circuit elements (neurons and pathways)

This indicates that huge amount of knowledge about a neural system is required for a cellular level analysis

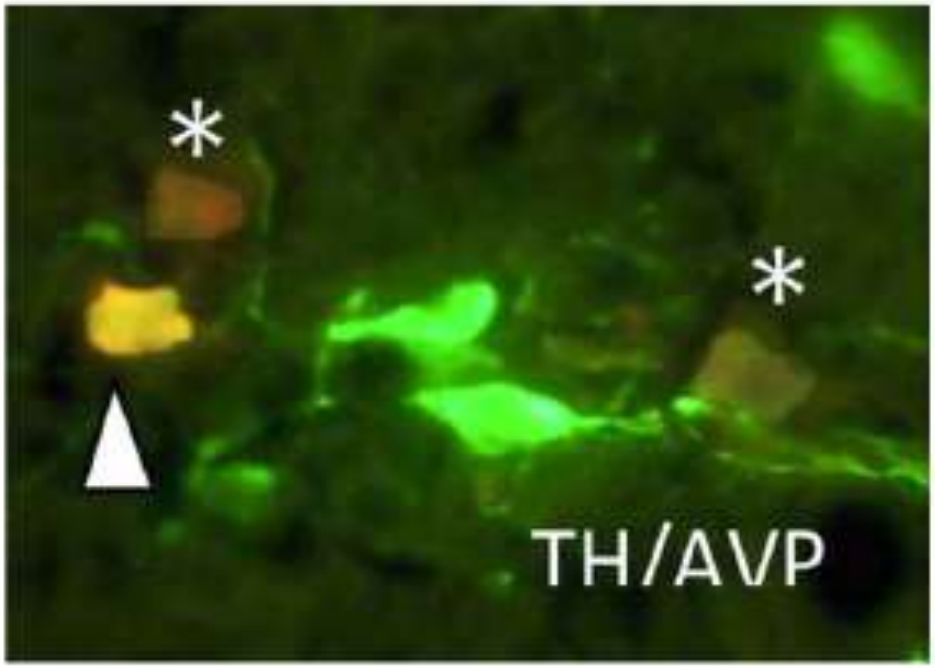


Fig. 5. Photomicrographs of the immunohistochemical localization of TH (i.e., DA) and AVP neurons in the AH using fluorescent double-label immunohistochemistry. DA and AVP containing neurons localize to the NC and mSON, and in both areas DA localized to AVP (yellow and arrowhead) and non-AVP (red and asterisks) neurons while AVP also localized to a separate set of neurons (green) in these regions.

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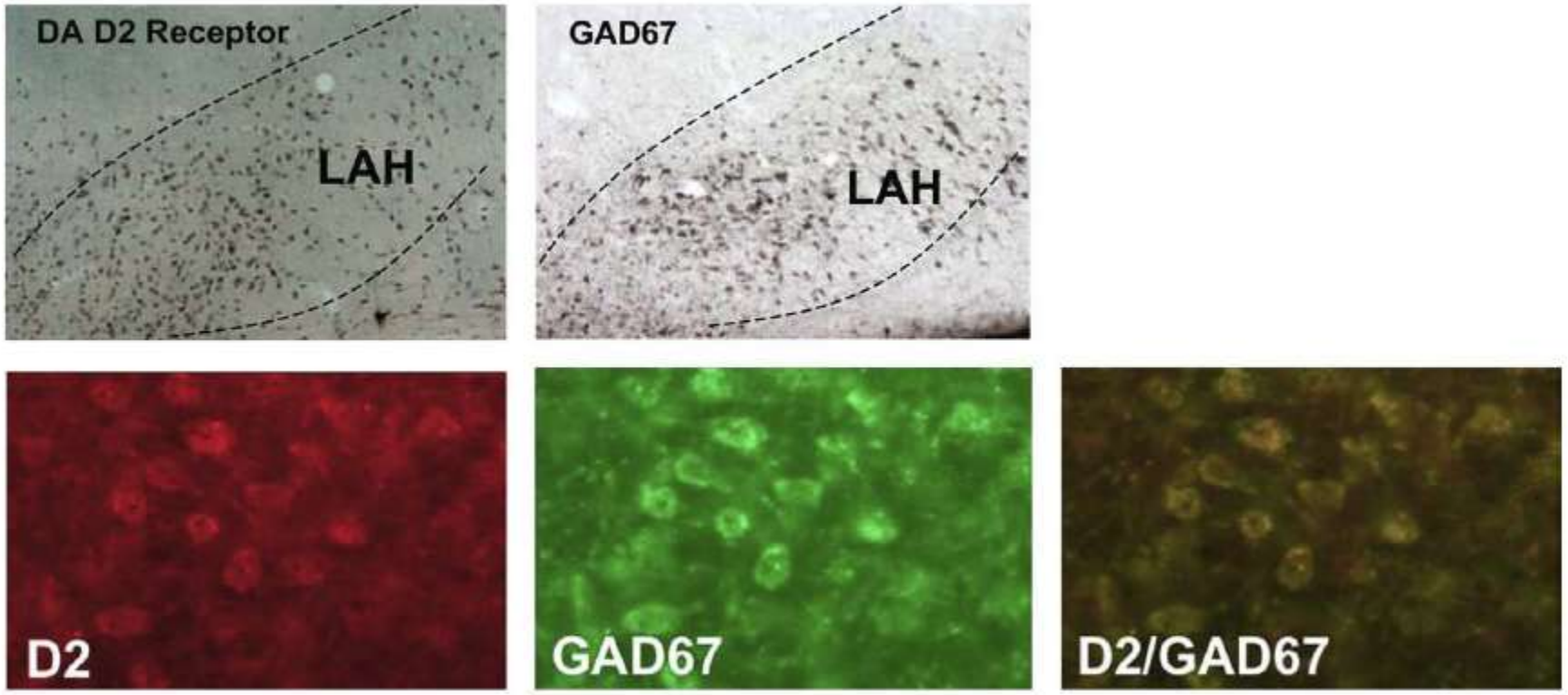

Fig. 6. Photomicrographs demonstrating the expression of DA D2 receptors and GAD67-containing (GABA) neurons in the LAH and the expression of DA D2 receptors on GABA neurons in the LAH using fluorescence double-label immunohistochemistry. (Top row) DA D2 receptors and GABA neurons (GAD67 immunopositive) localize to the LAH. (Bottom row) DA D2 receptors (red in first photo) and GABA neurons (GAD67; green in second photo) localize to the LAH and when combined, DA D2 receptors were observed to co-localize heavily on GABA neurons (yellow in third photo) in the LAH brain region.

Their new model incorporates many levels of analysis, including, prominently, the cellular level