به نام آنکه ما را زندگی داد وزان پس مژده پایندگی داد

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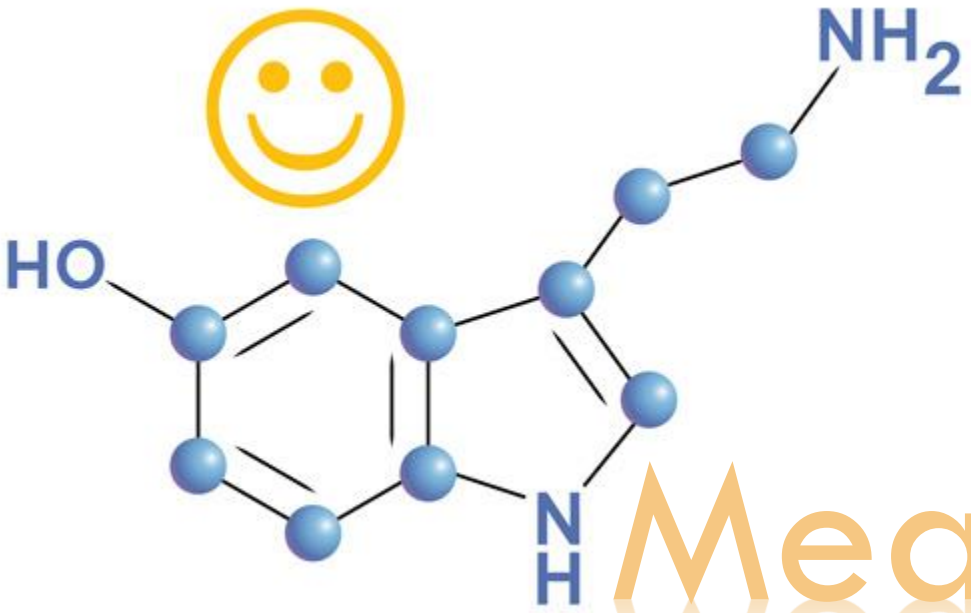
UNIVERSITY: ISLAMIC AZAD UNIVERSITY OF CENTRAL TEHRAN BRANCH

Serotonin



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Measurement of Serotonin in the body

1. What is Serotonin?

2. What factors increase Serotonin in the body?

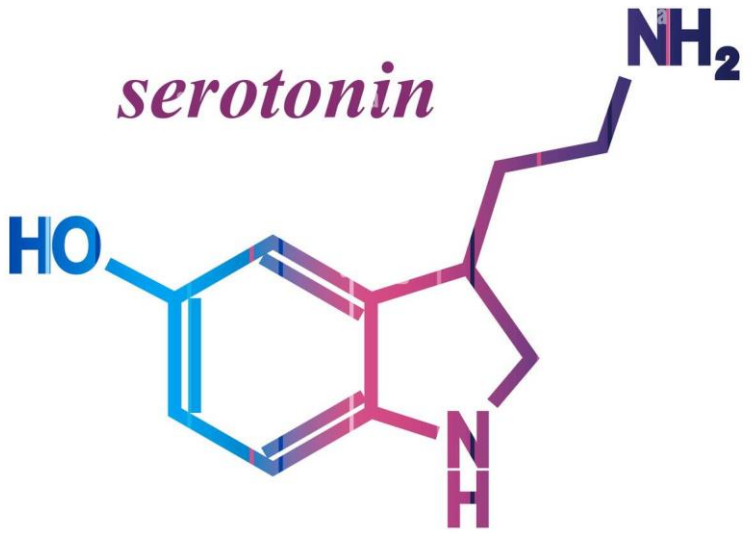
3. How we can measure the Serotonin in the body?

1. WHAT IS SEROTONIN?

Maurice M. Rapport, Arda Green and Irvine Page

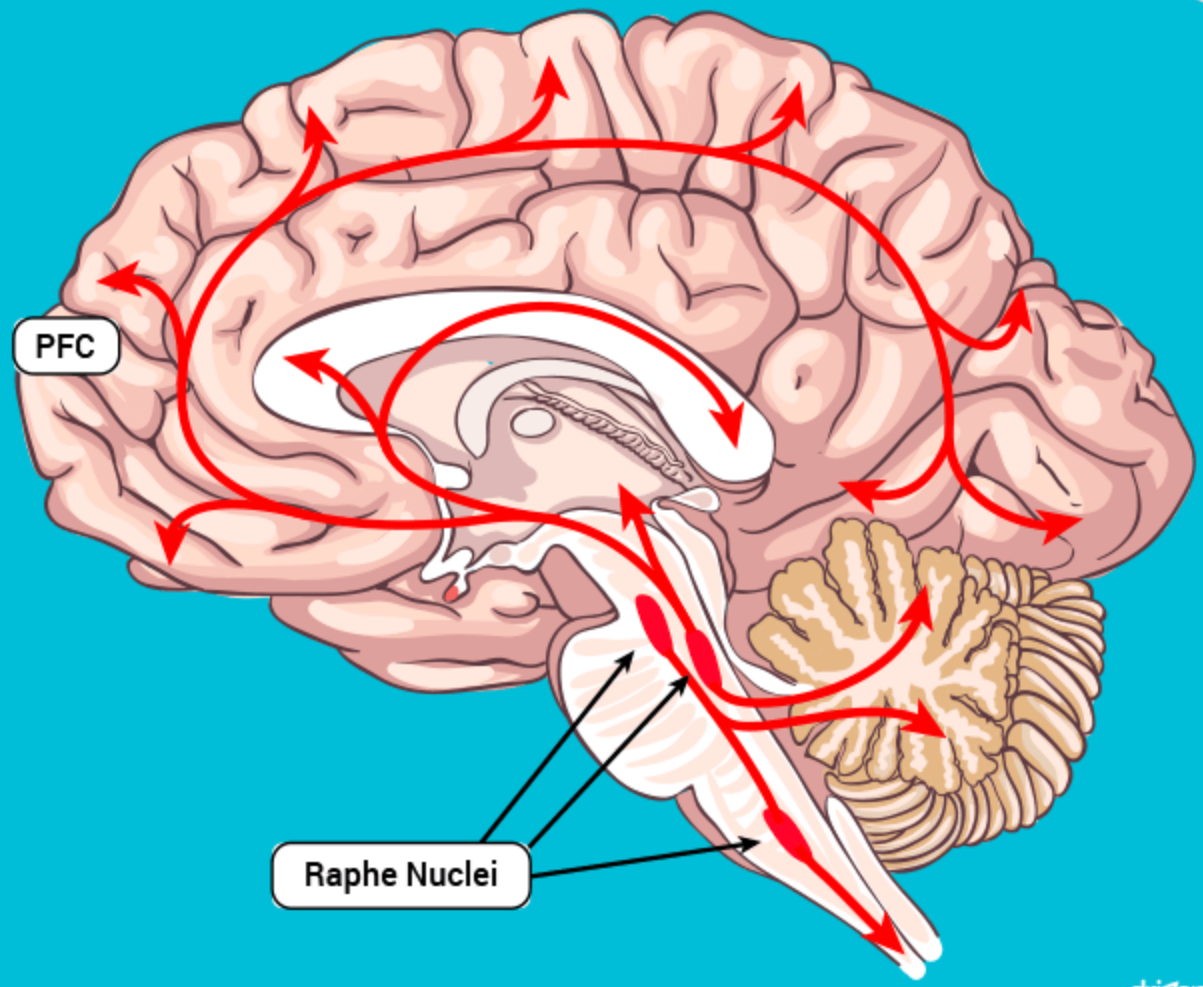
5-hydroxytryptamine (5-HT)

$C_{10}H_{12}N_2O$



SEROTONIN PATHWAYS

Distribution of serotonin from **raphe nuclei** through cortext and into the cerebellum.



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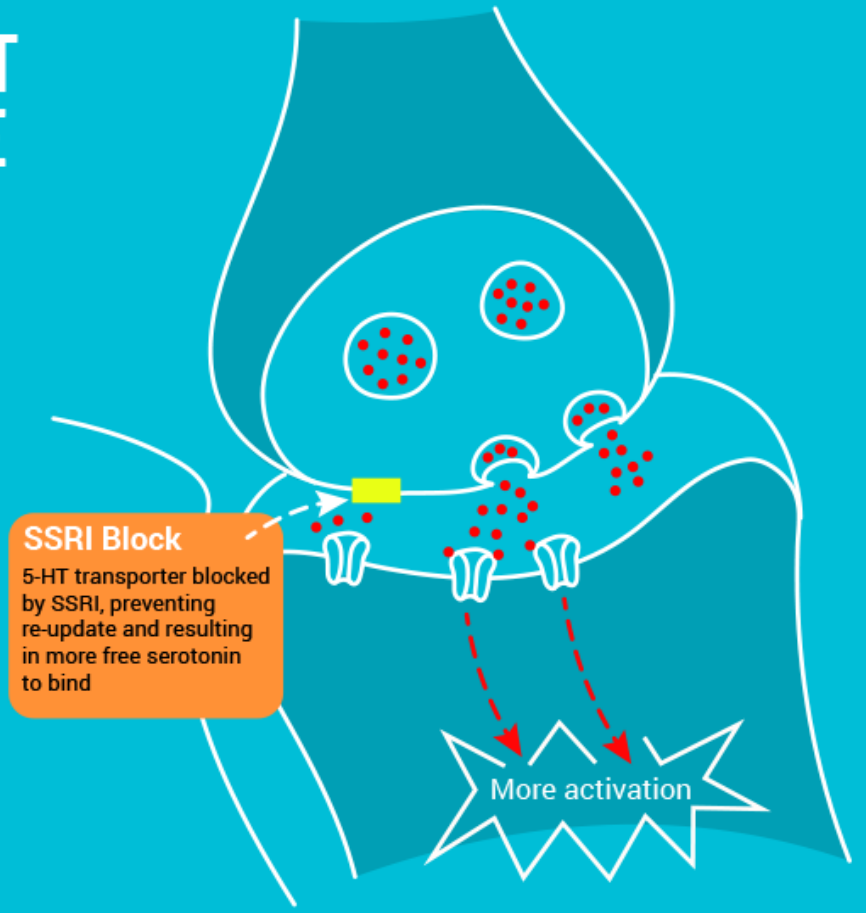
SSRI ACTION TO INHIBIT SEROTONIN RE-UPTAKE

Selective serotonin re-uptake inhibitors actively **block re-uptake transporters** that would have otherwise moved released serotonin back into the pre-synaptic terminal.

Due to this, there is more serotonin available in the synaptic cleft for binding with receptors, resulting in a **stronger activation** of the post-synaptic neuron.



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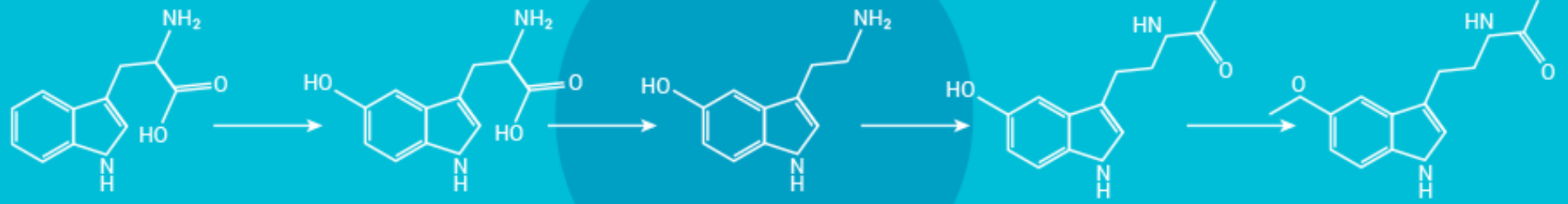
SEROTONIN CYCLE

Tryptophan → 5-Hydroxytryptophan → Serotonin → N-Acetylserotonin → Melatonin



Basic building block needed for serotonin

Sources:

Eggs, fish, soybeans, cheese, seeds, poultry, meats, milk

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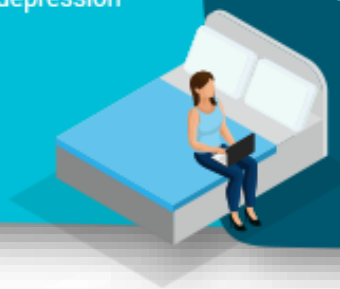
Potential use as antidepressant. Under investigation

Wide impacts:

- Mood
- Cognition
- Sleep
- Memory

Potential use for age-associated cognitive decline and depression



Functions:

- Circadian rhythm, sleep-wake cycles
- Immune function
- Antioxidant

driven

2. WHAT FACTORS INCREASE SEROTONIN IN THE BODY?

1. HEALTHY EATING

Protein foods:
Eat fish, meats, eggs, cheese, and other foods high in tryptophan

2. GOOD SLEEP

Set yourself up for deep sleep to increase serotonin release

3. MORE LIGHT

More sunlight during the day helps to increase release of serotonin

4. EXERCISE REGULARLY

4 times per week is a good amount to keep healthy in both body and mind

HOW WE CAN MEASURE THE SEROTONIN IN THE BODY?

A Comparison of the Subsecond Dynamics of Neurotransmission of Dopamine and Serotonin

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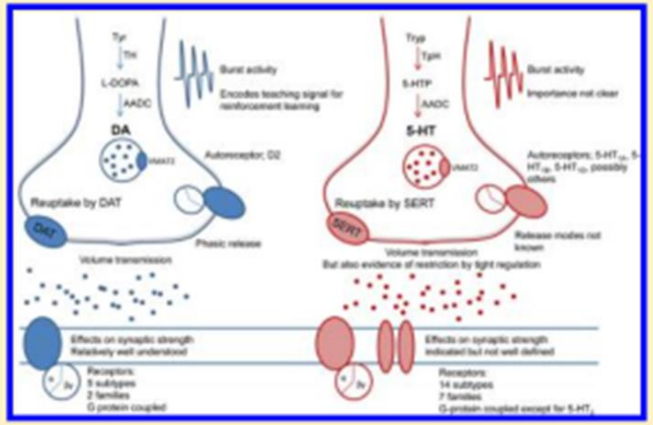
ABSTRACT: The neuromodulators dopamine (DA) and serotonin (5-hydroxytryptamine; 5-HT) are similar in a number of ways. Both monoamines can act by volume transmission at metabotropic receptors to modulate synaptic transmission in brain circuits. Presynaptic regulation of 5-HT and DA is governed by parallel processes, and behaviorally, both exert control over emotional processing. However, differences are also apparent: more than twice as many 5-HT receptor subtypes mediate postsynaptic effects than DA receptors and different presynaptic regulation is also emerging. Monoamines are amenable to real-time electrochemical detection using fast scan cyclic voltammetry (FSCV), which allows resolution of the subsecond dynamics of release and reuptake in response to a single action potential. This approach has greatly enriched understanding of DA transmission and has facilitated an integrated view of how DA mediates behavioral control. However, technical challenges are associated with FSCV measurement of 5-HT and understanding of 5-HT transmission at subsecond resolution has not advanced at the same rate. As a result, how the actions of 5-HT at the level of the synapse translate into behavior is poorly understood. Recent technical advances may aid the study of 5-HT in real-time. It is timely, therefore, to compare and contrast what is currently understood of the subsecond characteristics of transmission for DA and 5-HT. In doing so, a number of areas are highlighted as being worthy of exploration for 5-HT.



KEYWORDS: *Serotonin, dopamine, comparison, electrochemistry, electrophysiology, fast scan cyclic voltammetry*

Electrochemical Measurement of Endogenous Serotonin Release from Human Blood Platelets

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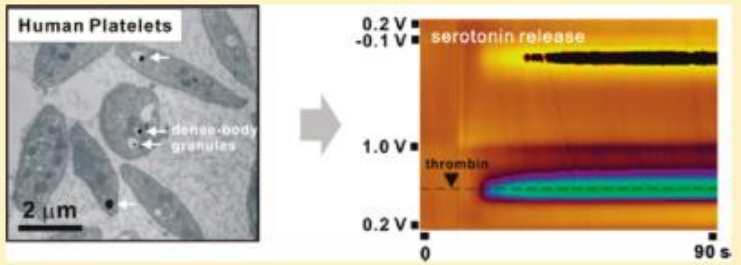
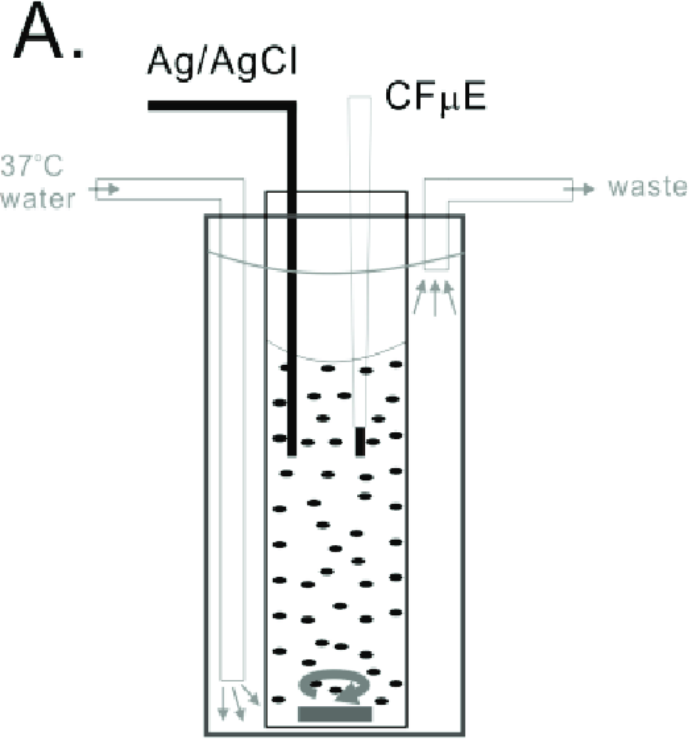
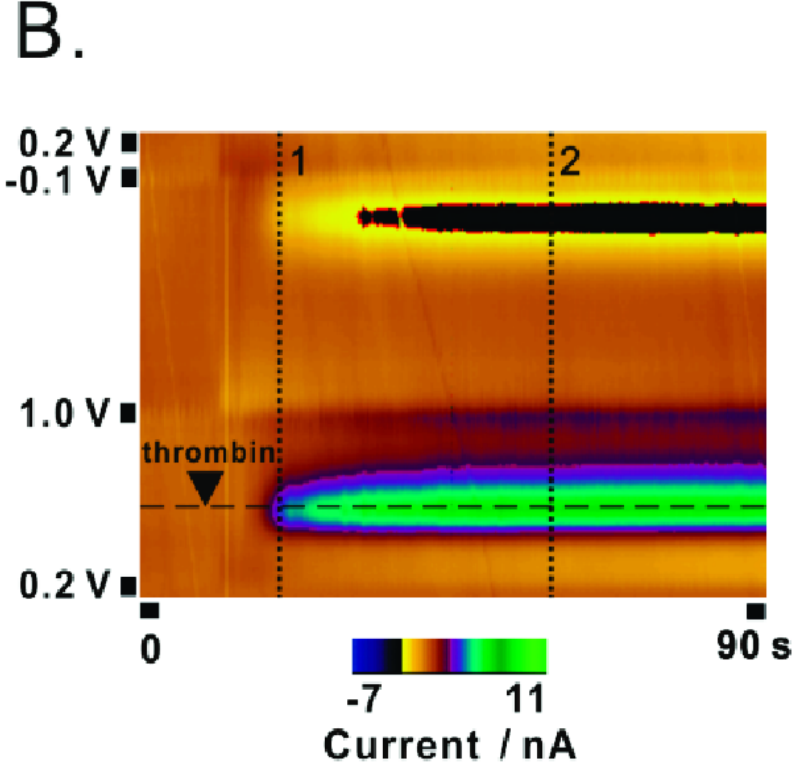
odstream is tightly latelets based on es, each sequester- nse-body granules le responsible for rs. Upon platelet se small molecules Therefore, techni- ontent release are erties of platelet secretion and aggregation. Existing techniques lack adequate time enous reagents for real-time measurement of granule content release. Herein, we ethod based on the endogenous electroactive chemical messenger serotonin (5- e measurement of dense-body granule secretion from platelet suspensions; fast-scan er microelectrodes was chosen on the basis of its excellent temporal resolution, high ctrochemical signature cyclic voltammograms for molecular identification. Real-time uman platelet suspensions was successfully measured, and the amount and time course agree well with data obtained from single platelet measurements, thus confirming n. Furthermore, this electrochemical method was applied to study the stimulation- torage and release dynamics with applied pharmacological agents, and chemical Pudlak Syndrome (HPS) platelets, and the potential of this method to reveal secretion ts has clearly been demonstrated.

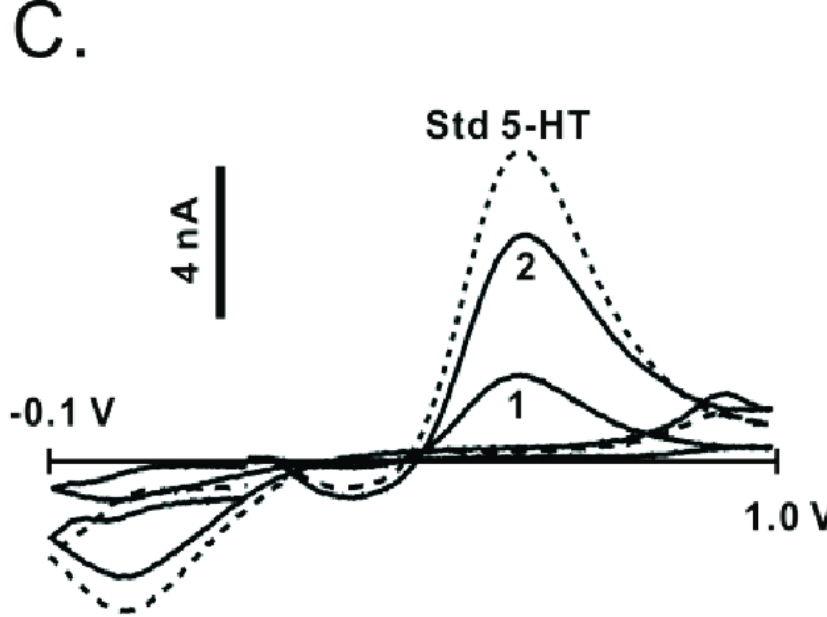


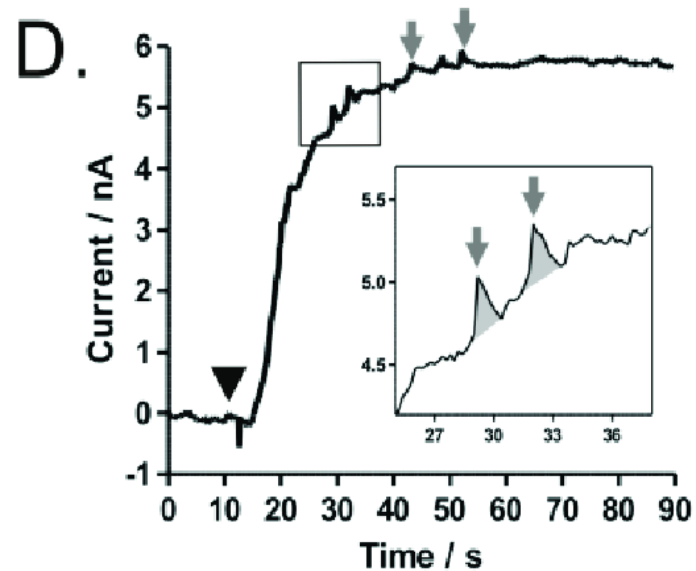
EXPERIMENTAL SECTION

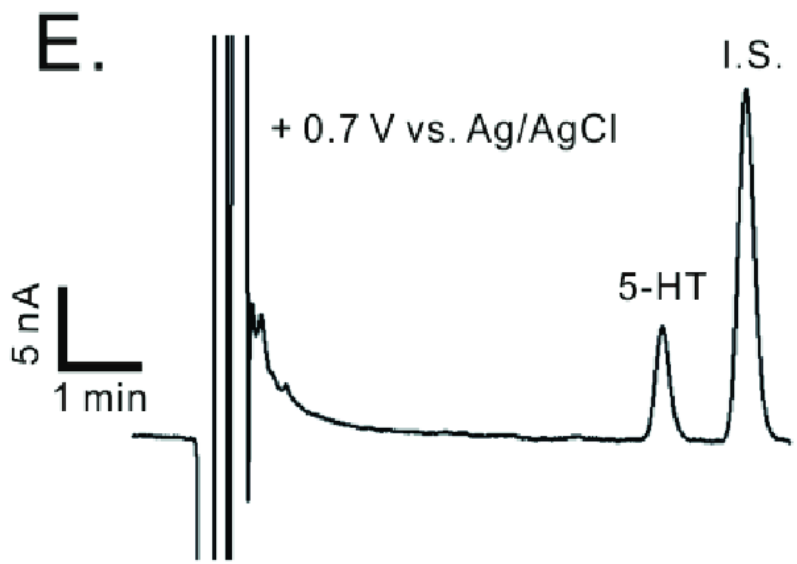
1. Preparation of Washed Human Platelets
2. Fabrication of Carbon-Fiber Microelectrodes
3. Measurements of Serotonin Release from Human Platelet Suspension
4. Pharmacological Manipulations
5. Measurement of Serotonin Release from Single Human Platelets
6. HPLC Analysis
7. Transmission Electron Microscopy Analysis
8. Data Analysis

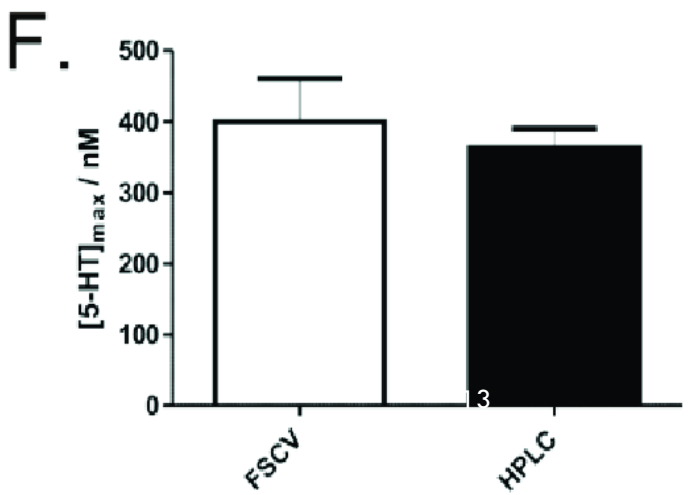

A.
Ag/AgCl
CFμE
37°C
water
waste

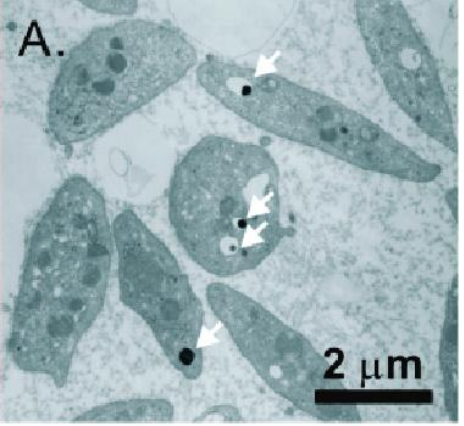

B.
0.2 V
-0.1 V
1.0 V
0.2 V
1
2
thrombin
0
90 s
-7
11
Current / nA

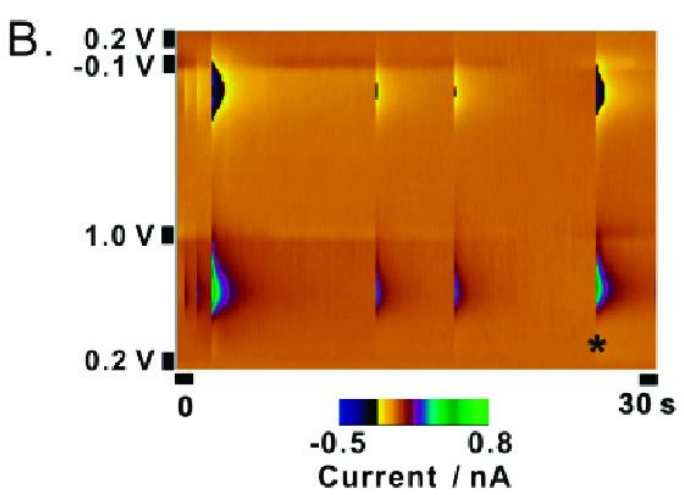

C.
Std 5-HT
4 nA
2
1
-0.1 V
1.0 V

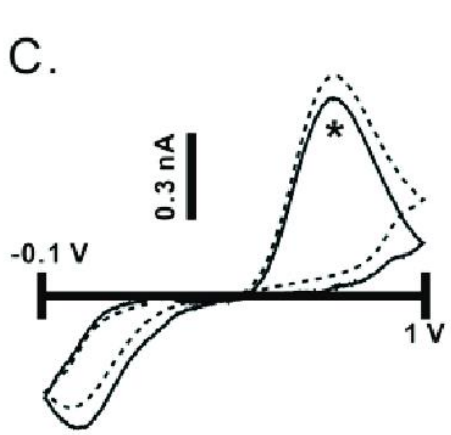

D.
Current / nA
Time / s
0
10
20
30
40
50
60
70
80
90
5.5
5.0
4.5
27
30
33
36

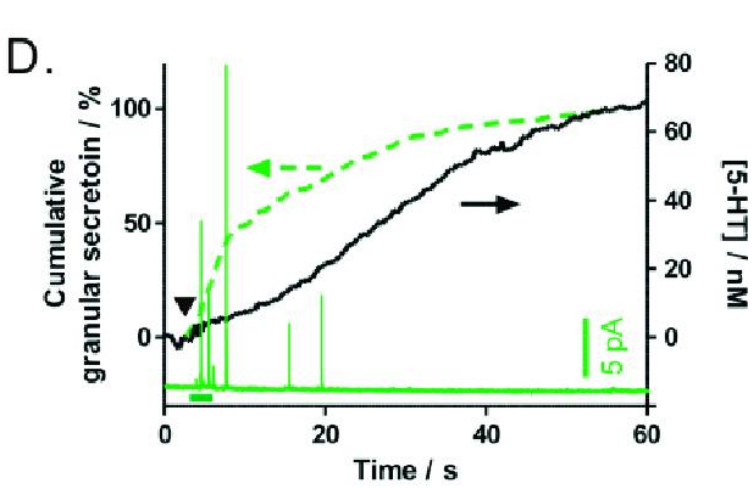

E.
+ 0.7 V vs. Ag/AgCl
I.S.
5-HT
5 nA
1 min

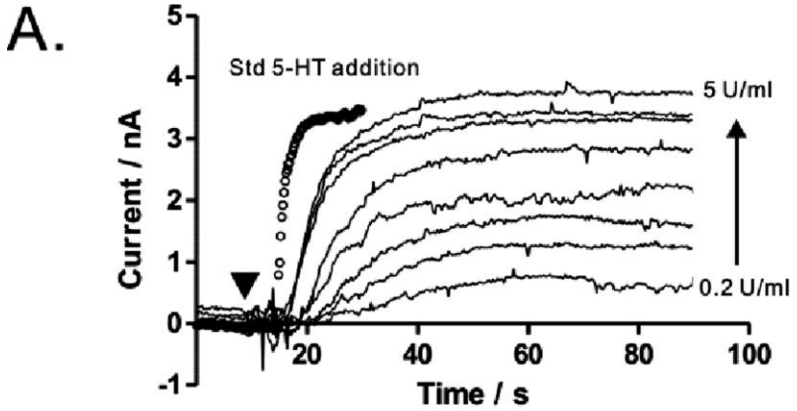

F.
500
400
300
200
100
0
[5-HT]max / nM
FSCV
HPLC

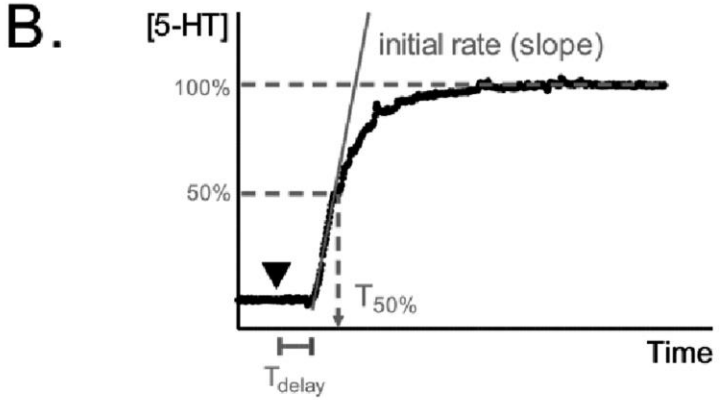

A.
2 μm

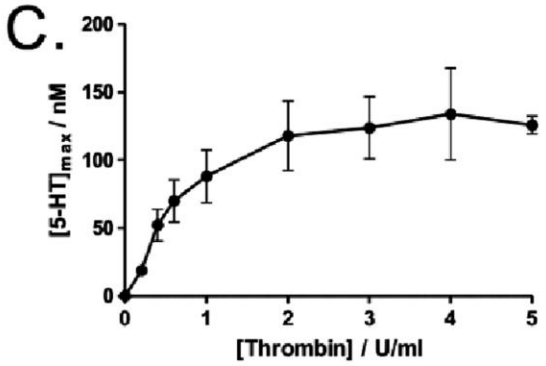

B.
0.2 V
-0.1 V
1.0 V
0.2 V
0
30 s
-0.5
0.8
Current / nA

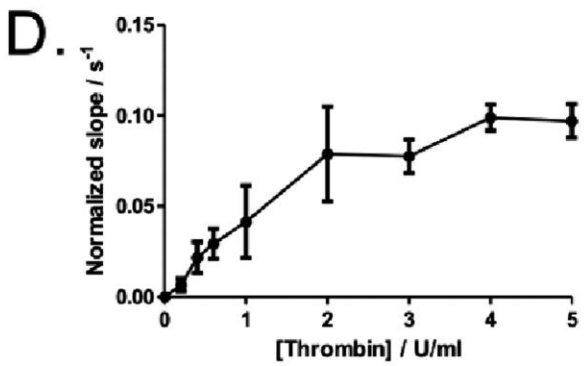

C.
0.3 nA
-0.1 V
1 V

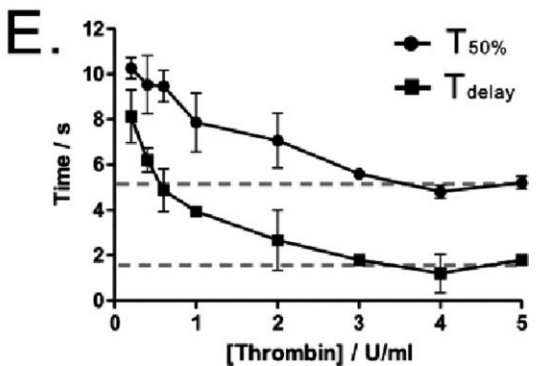

D.
Cumulative granular secretoin / %
[5-HT] / nM
5 pA
Time / s

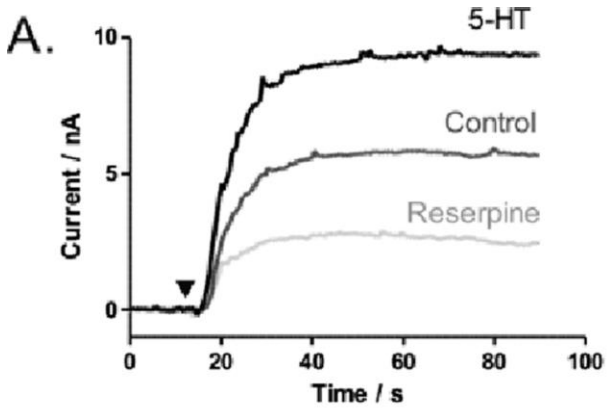

A.
Std 5-HT addition
5 U/ml
0.2 U/ml
Current / nA
Time / s

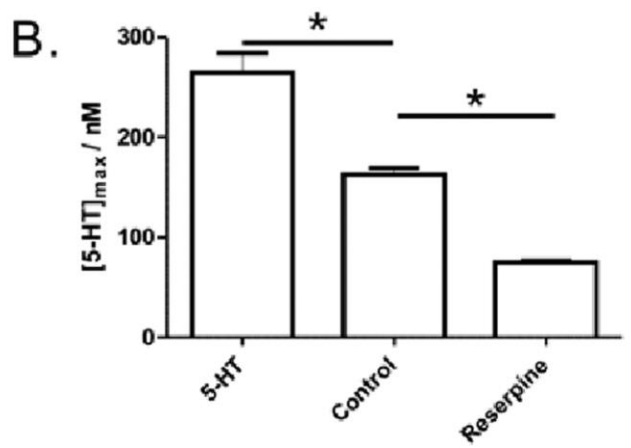

B.
[5-HT]
initial rate (slope)
100%
50%
T50%
Tdelay
Time

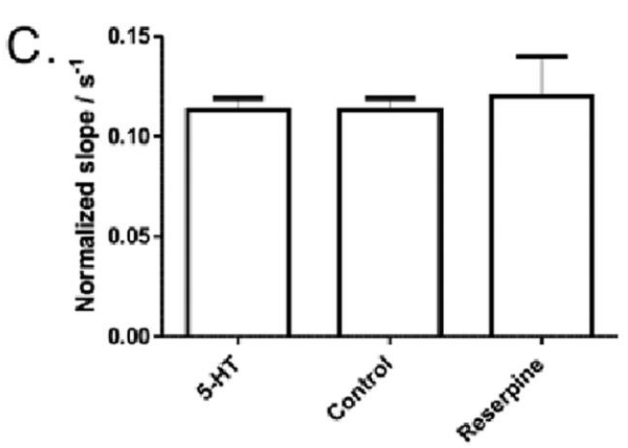

C.
[5-HT]max / nM
[Thrombin] / U/ml

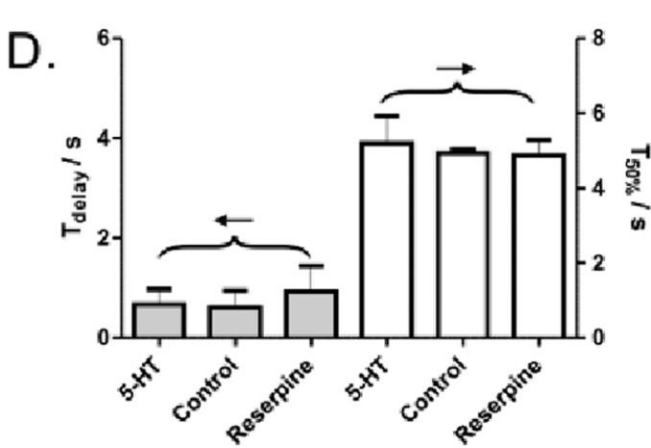

D.
Normalized slope / s-1
[Thrombin] / U/ml

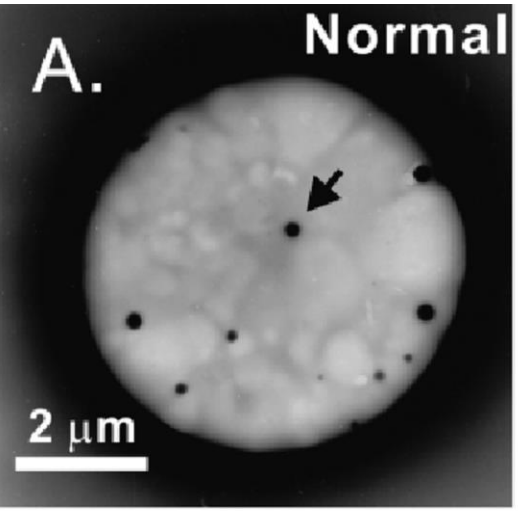

E.
T50%
Tdelay
Time / s
[Thrombin] / U/ml

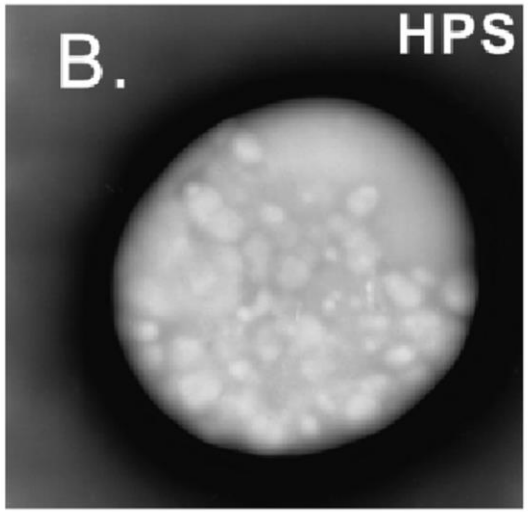

A.
5-HT
Control
Reserpine
Current / nA
Time / s

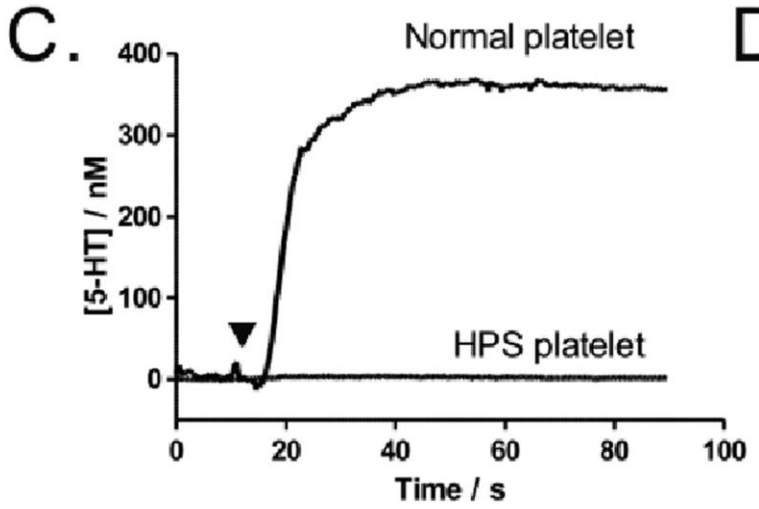

B.
*
*
[5-HT]max / nM
5-HT
Control
Reserpine

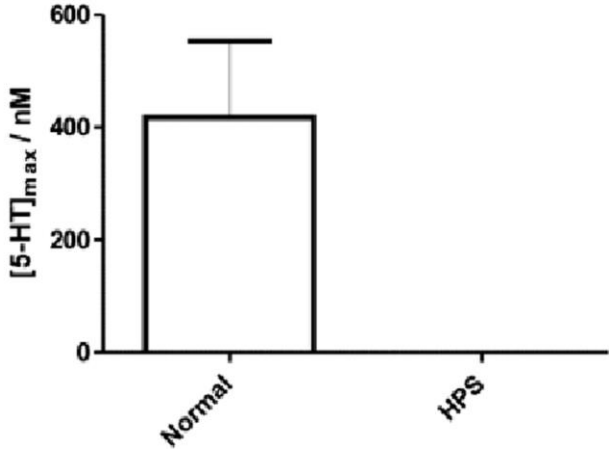

C.
Normalized slope / s-1
5-HT
Control
Reserpine


D.
Tdelay / s
T50% / s
5-HT
Control
Reserpine
5-HT
Control
Reserpine


A.
Normal
2 µm


B.
HPS


C.
Normal platelet
HPS platelet
[5-HT] / nM
Time / s


D.
[5-HT]max / nM
Normal
HPS

Postnatal Growth Defects in Mice with Constitutive Depletion of Central Serotonin

Nicolas Narboux-Nême,[†,‡,▽] Gaelle Angenard,[†,‡,▽] Valentina Mosienko,[§,▽] Friederike Klempin,[§] Pothitos M. Pitychoutis,[†,‡] Evan Deneris,[#] Michael Bader,[§] Bruno Giros,[‡,∥,⊥] Natalia Alenina,[§] and Patricia Gaspar*[,†,‡]

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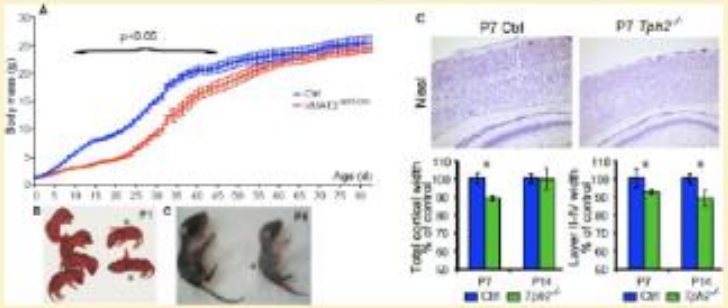
Supporting Information

ABSTRACT: Although the trophic actions of serotonin (5-HT) are well established, only few developmental defects have been reported in mouse strains with constitutive hyposerotonergia. We analyzed postnatal growth and cortical development in three different mutant mouse strains with constitutive reductions in central 5-HT levels. We compared two previously published mouse strains with severe (−95%) depletions of 5-HT, the tryptophan hydroxylase (Tph) $2^{-/-}$ mouse line and $VMAT2^{sert\text{-}cre}$ mice, with a new strain, in which VMAT2 deletion is driven by *Pet1* ($VMAT2^{pet1\text{-}cre}$) in 5-HT raphe neurons leading to partial (−75%) reduction in brain 5-HT levels. We find that normal embryonic growth and postnatal growth retardation are common features of all these mouse strains. Postnatal growth retardation varied from mild to severe according to the extent of the brain 5-HT reduction and gender. Normal growth was reinstated in $VMAT2^{sert\text{-}cre}$ mice by reconstituting central 5-HT stores. Growth abnormalities could not be linked to altered food intake or temperature control. Morphological study of the cerebral cortex over postnatal development showed a delayed maturation of the upper cortical layers in the $VMAT2^{sert\text{-}cre}$ and $Tph2^{-/-}$ mice, but not in the $VMAT2^{pet1\text{-}cre}$ mice. No changes in layer-specific gene expression or morphological alterations of barrel cortex development were found. Overall, these observations sustain the notion that central 5-HT signaling is required for the preweaning growth spurt of mouse pups. Brain development appeared to be immune to severe central 5-HT depletion for its overall growth during prenatal life, whereas reduced brain growth and delayed cortical maturation development occurred during postnatal life. Reduced developmental 5-HT signaling during postnatal development might modulate the function and fine structure of neural circuits in ways that affect adult behavior.



KEYWORDS: *Cerebral cortex, development, somatic growth, knockout mice, vesicular monoamine transporter, tryptophan hydroxylase, cux1*

THANKS FOR YOUR ATTENTION