RAD

Research Appreciation Day

2024 Virtual Conference

POSTER DESIGN PREP SESSION

TUESDAY, FEB 20, 2024

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What is a Research Poster?

A research poster is a professional display of research, often used during professional conferences or academic presentations.

PIGS IN SPACE: EFFECT OF ZERO GRAVITY AND AD LIBITUM FEEDING ON WEIGHT GAIN IN CAVIA PORCELLUS

Colin B. Purrington*
6673 College Avenue, Swarthmore, PA 19081 USA

ABSTRACT:

One ignored benefit of space travel is a potential elimination of obesity, a chronic problem for a growing majority in many parts of the world. In theory, when an individual is in a condition of zero gravity, weight is eliminated. Indeed, in space one could conceivably follow ad libitum feeding and never even gain an gram, and the only side effect would be the need to upgrade one's stretchy pants("exercise pants"). But because many diet schemes start as very good theories only to be found to be rather harmful, we tested our predictions with a long-term experiment in a colony of Guinea pigs (Cavia porcellus) maintained on the International Space Station. Individuals were housed separately and given unlimited amounts of high-calorie food pellets. Fresh fruits and vegetables were not available in space so were not offered. Every 30 days, each Guinea pig was weighed. After 5 years, we found that individuals, on average, weighed nothing. In addition to weighing nothing, no weight appeared to be gained over the duration of the protocol. If space continues to be gravity-free, and we believe that assumption is sound, we believe that sending the overweight — and those at risk for overweight — to space would be a lasting cure.

INTRODUCTION:

The current obesity epidemic started in the early 1960s with the invention and proliferation of elastane and related stretchy fibers, which released wearers from the rigid constraints of clothes and permitted monthly weight gain without the need to buy new outfits. Indeed, exercise today for hundreds of million people involve only the act of wearing stretchy pants in public, presumably because the constrictive pressure forces fat molecules to adopt a more compact tertiary structure (Xavier 1965).

Luckily, at the same time that fabrics became stretchy, the race to the moon between the United States and Russia yielded a useful fact: gravity in outer space is minimal to nonexistent. When gravity is zero, objects cease to have weight. Indeed, early astronauts and cosmonauts had to secure themselves to their ships with seat belts and sticky boots. The potential application to weight loss was noted immediately, but at the time travel to space was prohibitively expensive and thus the issue was not seriously pursued. Now, however, multiple companies are developing cheap extra-orbital travel options for normal consumers, and potential travelers are also creating news ways to pay for products and services that they cannot actually afford. Together, these factors open the possibility that moving to space could cure overweight syndrome quickly and permanently for a large number of humans.

We studied this potential by following weight gain in Guinea pigs, known on Earth as fond of ad libitum feeding. Guinea pigs were long envisioned to be the "Guinea pigs" of space research, too, so they seemed like the obvious choice. Studies on humans are of course desirable, but we feel this current study will be critical in acquiring the attention of granting agencies.

MATERIALS AND METHODS:

One hundred male and one hundred female Guinea pigs (Cavia porcellus) were transported to the International Space Laboratory in 2010. Each pig was housed separately and deprived of exercise wheels and fresh fruits and vegetables for 48 months. Each month, pigs were individually weighed by duct-taping them to an electronic balance sensitive to 0.0001 grams. Back on Earth, an identical cohort was similarly maintained and weighed. Data was analyzed by statistics.

RESULTS:

Mean weight of pigs in space was 0.0000 +/- 0.0002 g. Some individuals weighed less than zero, some more, but these variations were due to reaction to the duct tape, we believe, which caused them to be alarmed push briefly against the force plate in the balance. Individuals on the Earth, the control cohort, gained about 240 g/month ($p = 0.0002$). Males and females gained a similar amount of weight on Earth (no main of effect of sex), and size at any point during the study was related to starting size (which was used as a covariate in the ANCOVA). Both Earth and space pigs developed substantial dewlaps (double chins) and were lethargic at the conclusion of the study.

CONCLUSIONS:

Our view that weight and weight gain would be zero in space was confirmed. Although we have not replicated this experiment on larger animals or primates, we are confident that our result would be mirrored in other model organisms. We are currently in the process of obtaining necessary human trial permissions, and should have our planned experiment initiated within 80 years, pending expedited review by local and Federal IRBs.

ACKNOWLEDGEMENTS:

I am grateful for generous support from the National Research Foundation, Black Hole Diet Plans, and the High Fructose Sugar Association. Transport flights were funded by SPACE-EXES, the consortium of wives divorced from insanely wealthy space-flight startups. I am also grateful for comments on early drafts by Mañana Athletic Club, Corpus Christi, USA. Finally, sincere thanks to the Cuy Foundation for generously donating animal care after the conclusion of the study.

LITERATURE CITED:

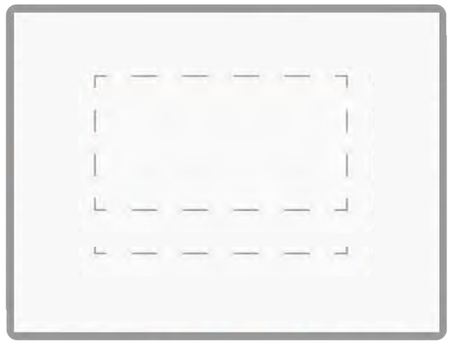
NASA. 1982. Project STS-XX: Guinea Pigs. Leaked internal memo.

Sekulić, S.R., D. D. Lukač, and N. M. Naumović. 2005. The Fetus Cannot Exercise Like An Astronaut: Gravity Loading Is Necessary For The Physiological Development During Second Half Of Pregnancy. Medical Hypotheses. 64:221-228

Xavier, M. 1965. Elastane Purchases Accelerate Weight Gain In Case-control Study. Journal of Obesity. 2:23-40.

Recommended Program:

PowerPoint



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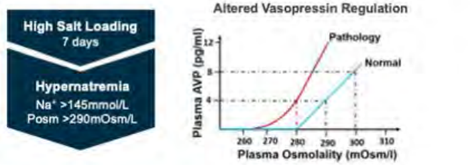
High Salt Loading Increases Brain Derived Neurotrophic Factor in Supraoptic Vasopressin Neurons

Kirthikaa Balapattabi, Joel T Little and J. Thomas Cunningham, Ph.D.
Department of Physiology & Anatomy, University of North Texas Health Science Center, Fort Worth, TX

hsc
THE UNIVERSITY of NORTH TEXAS HEALTH SCIENCE CENTER at FORT WORTH

BACKGROUND

Altered synaptic homeostasis of the vasopressin (AVP) neurons contributes to elevated circulating AVP and increased in blood pressure during high salt loading (SL). Previous studies show that SL impairs baroreceptor mediated GABAA inhibition of rat AVP neurons through Brain-Derived Neurotrophic Factor (BDNF). BDNF increases intracellular chloride through tyrosine receptor kinase B (TrKB) receptor activation and KCC2 downregulation causing sustained release of AVP. However, the source of BDNF is not known.



HYPOTHESIS

BDNF from the Supraoptic Nucleus (SON) contributes to increased vasopressin release in high SL rats

METHODS

Rats were bilaterally injected in the SON (300 nL each side) with an AAV2 serotype conjugated with a shRNA directed against BDNF, a mCherry reporter and a U6 promoter (shBDNF). Another group of rats received bilateral SON injections of equal titre and amount of AAV2 conjugated with a scrambled (SCR) sequence of shRNA as controls. The vectors were injected at a titre of 1.0 × 1013 GC mL−1. Each rat was anaesthetised with isoflurane (2%–3%) and placed in a stereotaxic frame. Their skulls were exposed and levelled between lambda and bregma.

RESULTS

mRNA Expression

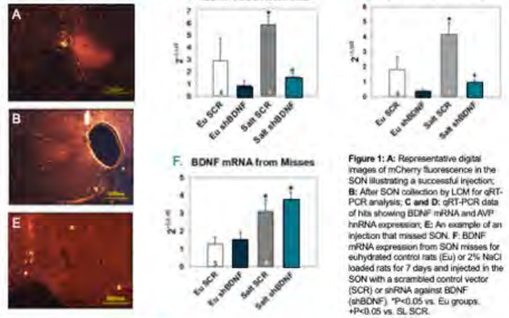


Figure 1: A: Representative digital images of mCherry fluorescence in the SON illustrating a successful injection; B: After SON collection by LCM for qRT-PCR analysis; C and D: qRT-PCR data of hits showing BDNF mRNA and AVP hnRNA expression; E: An example of an injection that missed SON. F: BDNF mRNA expression from SON misses for euhydrated control rats (Eu) or 2% NaCl loaded rats for 7 days and injected in the SON with a scrambled control vector (SCR) or shRNA against BDNF (shBDNF). *P<0.05 vs. Eu groups. +P<0.05 vs. SL SCR.

Protein Expression

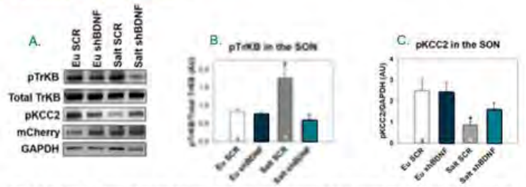


Figure 2: A: Sample Western blot images showing changes in protein expression of SON punches and quantification bar graphs for A: Phosphorylated TrKB/ Total TrKB, B: Phosphorylated KCC2 from euhydrated controls (Eu) and 2% salt loaded (SL) rats injected in the SON with a control vector (SCR) or shRNA BDNF (shBDNF). *P<0.05 vs. Eu groups. +P<0.05 vs. Salt SCR.

Plasma Measurements

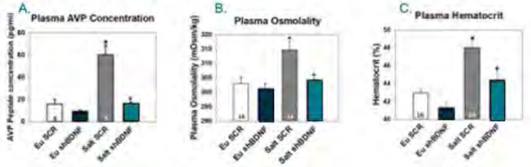
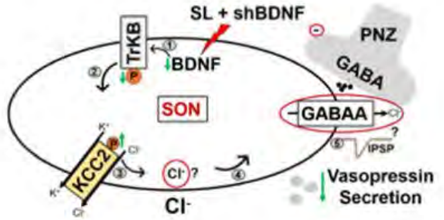


Figure 3: Changes in various plasma parameters A: Plasma AVP Concentration; B: Plasma Osmolality; and C: Hematocrit from euhydrated controls (Eu) and 2% salt loaded (SL) rats injected in the SON with a control vector (SCR) or shRNA BDNF (shBDNF). *P<0.05 vs. Control groups. +P<0.05 vs. Salt SCR.

CONCLUSION

The results indicate that BDNF produced in the SON is necessary for increased vasopressin secretion during high salt loading.



FUTURE DIRECTION

The present study advances our understanding about the pathophysiology of AVP neurone regulation. Identifying the source of BDNF underlying the changes in postsynaptic inhibition of AVP neurones in response to salt loading may result in novel strategies for reducing AVP secretion in other pathological states, such as heart and liver failure.

- To determine changes in intracellular chloride concentration [Cl]I using ClopHensorN
- To determine possible role of BDNF-TrKB signaling in elevated AVP release associated with liver cirrhosis

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Poster Elements

Title

Sub-Title

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color

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Typography

Section

No more than two font types.

- Serif fonts use extra strokes or "feet"
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- **Type title:** in ALL CAPS
- **Type authors:** in Upper And Lower Case

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Author Name

Sub-Title

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- **Type sub-title:** in ALL CAPS

SUB-TITLE

Body Text

- **Font Size:** should be 24-32 points, including captions
- **Type body:** in sentence style.

The quick, brown fox jumps over a lazy dog.

Typography - Summary

- Keep it simple, no more than two fonts —generally one for the titles and one for the body text.

- When selecting a font you can’t go wrong with the classics: Arial, Times New Roman or Helvetica.

Balance

Section

High Salt Loading Increases Brain Derived Neurotrophic Factor in Supraoptic Vasopressin Neurons

Kirthikaa Balapattabi, Joel T Little and J. Thomas Cunningham, Ph.D.
Department of Physiology & Anatomy, University of North Texas Health Science Center, Fort Worth, TX

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THE UNIVERSITY of NORTH TEXAS HEALTH SCIENCE CENTER at FORT WORTH

BACKGROUND

Arginine vasopressin (AVP) is a peptide hormone that is synthesised in magnocellular neurosecretory cells (MNCs) present in the supraoptic nucleus (SON) and paraventricular through the hypothalamic-hypophyseal tract from hypothalamus to the posterior pituitary nucleus (PVN) of the hypothalamus. Arginine vasopressin is axonally transported where it is released into systemic circulation. The release of AVP is regulated primarily by plasma osmolality, blood pressure and blood volume. Increased plasma osmolality activates hypothalamic MNCs increasing circulating AVP which acts at the kidneys to increase water reabsorption and maintain homeostasis. Conversely, decreased plasma osmolality is associated with inhibition of AVP MNCs, reduced plasma AVP and diuresis. A linear relationship between plasma AVP and osmolality is observed during normal conditions and requires highly coordinated excitatory and inhibitory postsynaptic responses.

HYPOTHESIS

BDNF from the Supraoptic Nucleus (SON) contributes to increased vasopressin release in high SL rats. Altered synaptic homeostasis of vasopressinergic MNCs could contribute to disease. For example, congestive in these diseases. Despite the vast amount of knowledge available about the regulation conditions in which circulating AVP is abnormally elevated. heart failure and decompensated cirrhosis can be associated with dilutional hyponatraemia. The development of hyponatraemia is associated with increased morbidity and mortality of AVP neurones, the pathophysiology of inappropriate AVP secretion remains unknown. Increased AVP release into systemic circulation from MNCs despite elevated mean arterial pressure (MAP) is reported during high salt loading (SL) (ie, giving rats only

METHODS

Rats were bilaterally injected in the SON (300 nL each side) with an AAV2 serotype conjugated with a shRNA directed against BDNF, a mCherry reporter and a U6 promoter (shBDNF). Another group of rats received bilateral SON injections of equal titre and amount of AAV2 conjugated with a scrambled (SCR) sequence of shRNA as controls. The vectors were injected at a titre of 1.0 × 1013 GC mL−1. Each rat was anaesthetised with isoflurane (2%–3%) and placed in a stereotaxic frame. Their skulls were exposed and levelled between lambda and bregma.

RESULTS

Using LCM, we verified the accuracy of the stereotaxic injections by visualising the mCherry reporter (Figure 1A) and collected the SONs to measure changes in the BDNF mRNA and AVP hnRNA expression using quantitative RT-PCR by the 2−ΔΔCt method. Rats that did not have successful virus injections in the SON were analysed separately. One-way ANOVA revealed significant difference between the groups in BDNF ($F_{3,23} = 5.78$, $P < 0.05$) and AVP ($F_{3,23} = 8.83$, $P < 0.05$) gene expression. Bonferroni post-hoc analysis showed that salt loading significantly increased BDNF and AVP gene expression in the SON of rats injected with SCR compared to the euhydrated rats (Bonferroni t tests, all $P < 0.05$) (Figure 1). Post-hoc multiple comparison of mRNA levels between Salt SCR and Salt shBDNF groups showed that SON injections of shBDNF significantly blocked the increases in BDNF mRNA (Bonferroni $t = 3.310$, $P < 0.05$) and AVP hnRNA (Bonferroni $t = 4.09$, $P < 0.05$) in the SON of salt loaded rats. In rats with injections of shBDNF outside of the SON, salt loading significantly increased BDNF mRNA in the SON ($F_{3,12} = 7.33$, $P < 0.05$) (Figure 2) and the increase produced by salt loading was not different compared to the Salt SCR group (Bonferroni $t = 0.37$, $P > 0.05$) (Figure 2).

We used mCherry expression to verify the specificity of stereotaxic injections (Figure 3). One-way ANOVA analysis revealed significant difference between the groups in TrkB phosphorylation ($F_{3,23} = 6.778$, $P < 0.05$) and KCC2 phosphorylation ($F_{3,24} = 4.546$, $P < 0.05$). Seven days of 2% salt loading significantly increased TrkB phosphorylation without affecting total TrkB expression and decreased phosphorylation of KCC2 (Bonferroni t tests, all $P < 0.05$) (Figure 3) in the SON of rats injected with the control vector compared to the euhydrated rats. Virally mediated BDNF knockdown in the SON of salt loaded rats significantly prevented the increase in TrkB phosphorylation and decrease in KCC2 phosphorylation compared to euhydrated rats (Bonferroni t tests, all $P < 0.05$) (Figure 3). One to two rats in each group did not have successful virus injections in the SON, as confirmed at the end of the experiment using either LCM/quantitative RT-PCR or western blot analysis, and the results were excluded from the data analysis in all of the subsequent experiments.

CONCLUSION

AVP MNCs are regulated by negative-feedback from arterial baroreceptors mediated by Circulating AVP is primarily determined by the activity of MNCs located in the PVN and this baroreceptor inhibition by altering chloride homeostasis of MNCs through a BDNF-GABAA inhibition. Previous studies have established that chronic salt loading can impair TrkB signalling mechanism. changes in the regulatory mechanism to obtain a complete understanding of the pathophysiology of inappropriate AVP release. The results of the present study show that the SON is the source of BDNF resulting in an inappropriate release of AVP in salt loaded rats. SON of the hypothalamus. related to the decrease in circulating AVP, although this was not tested. Elevation of It is important to identify the source of BDNF leading to In addition, MNCs are reported to express both BDNF and Because SON is less heterogeneous than PVN and we previously observed salt. Knocking down BDNF in the SON of salt loaded rats significantly attenuated the increases in AVP hnRNA in the SON and circulating AVP normally associated with salt loading.

FUTURE DIRECTION

The present study advances our understanding about the pathophysiology of AVP neurone regulation. Identifying the source of BDNF underlying the changes in postsynaptic inhibition of AVP neurones in response to salt loading may result in novel strategies for reducing AVP secretion in other pathological states, such as heart and liver failure.

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Un-Balanced Poster

High Salt Loading Increases Brain Derived Neurotrophic Factor in Supraoptic Vasopressin Neurons

Kirthikaa Balapattabi, Joel T Little and J. Thomas Cunningham, Ph.D.
Department of Physiology & Anatomy, University of North Texas Health Science Center, Fort Worth, TX

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BACKGROUND

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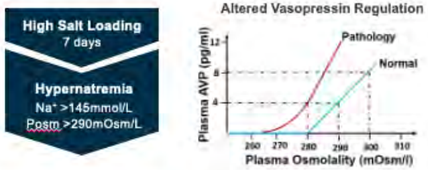

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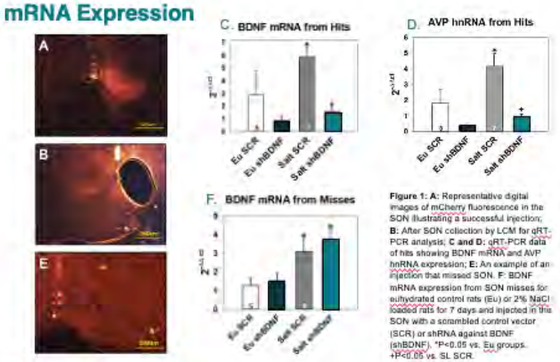

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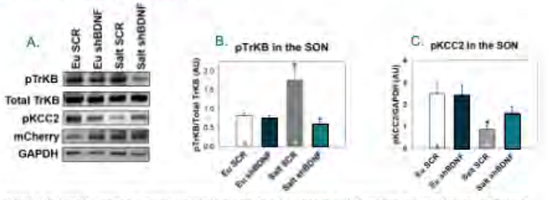

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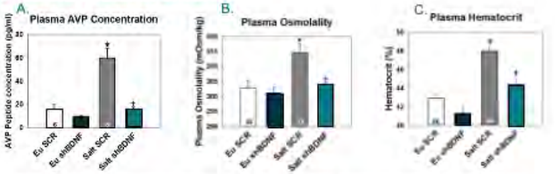

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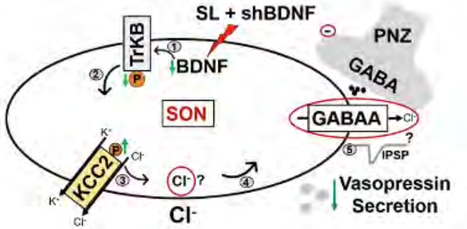

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Balanced Poster

Image Best Practices

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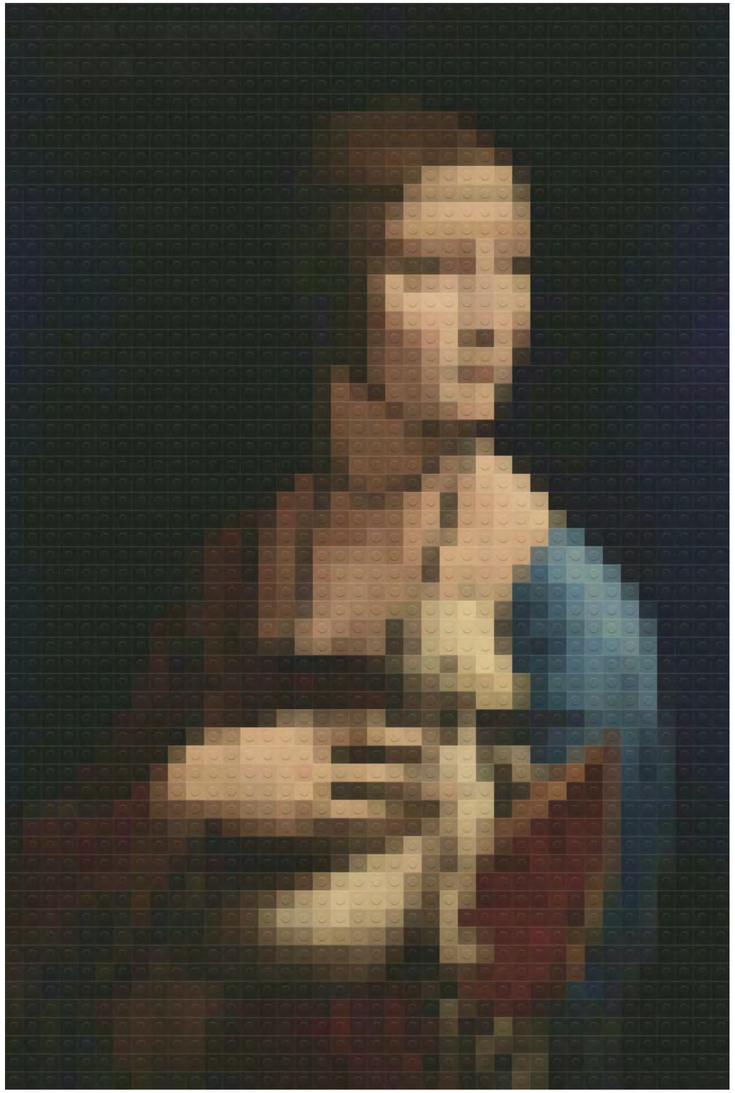

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Image Resources - Generic

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- Stocksy - Resource for art forward stock photos and footage. (not free!)

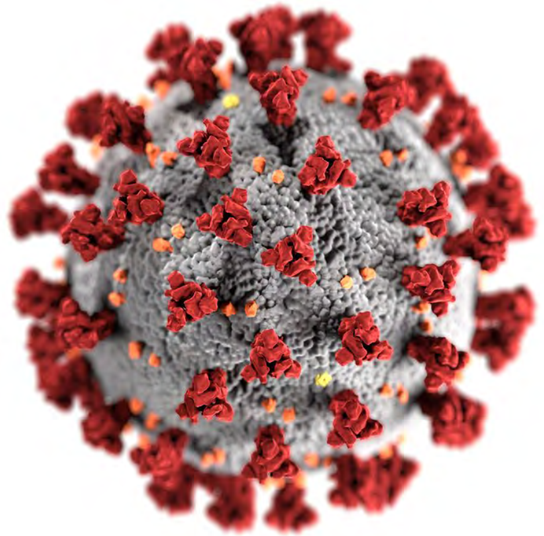
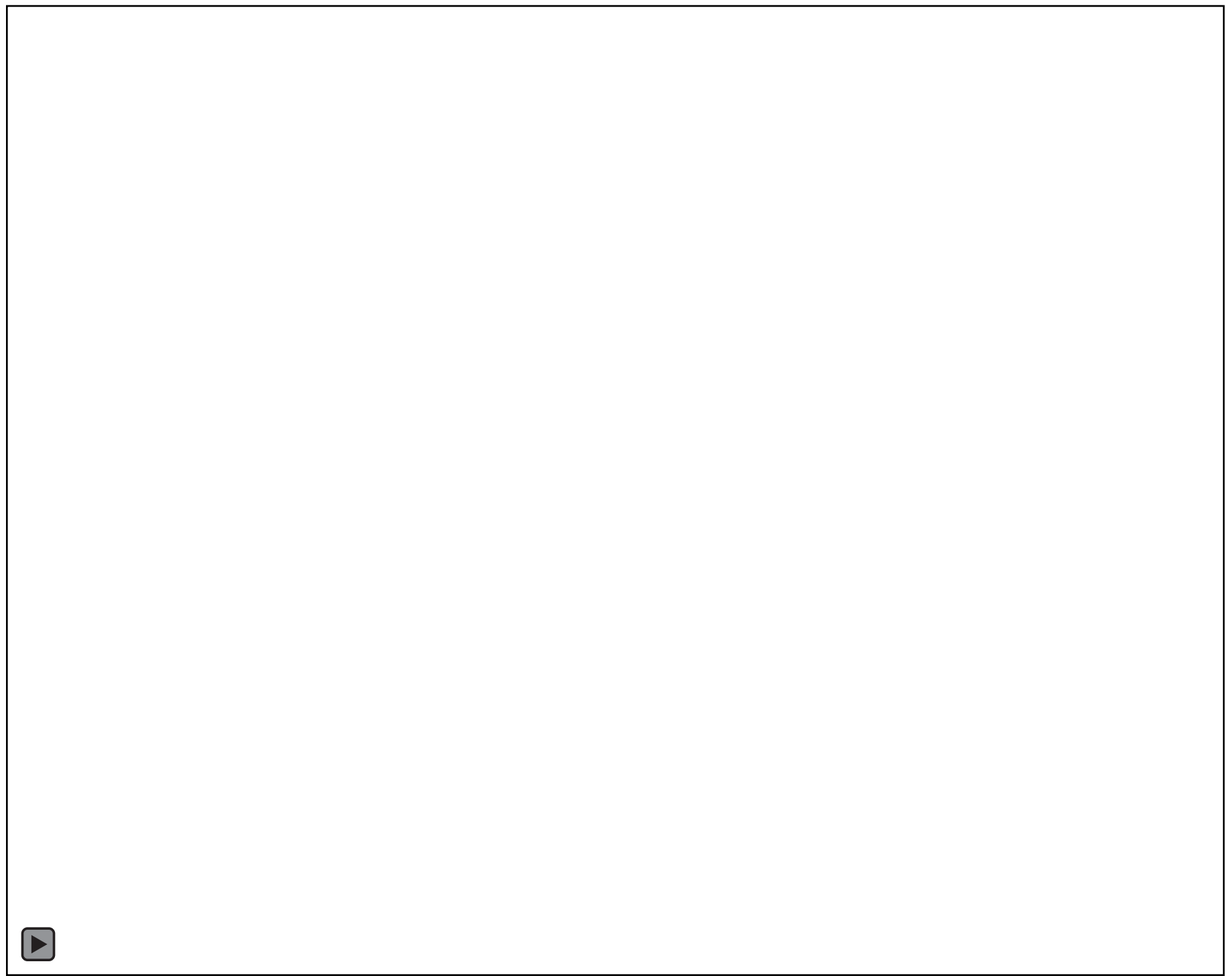


Image Resources - Biomedical

- Open-I - A resource of the National Library of Medicine, the website is an open access biomedical image search engine.
- BioRender - Tool to help scientists create and share beautiful, professional scientific figures.



Balance - Summary

- Your poster should be more than just words.
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- Avoid copyright materials on your poster.
- Always give credit to the photographer or cite the source.

Color

Section

Color - Summary

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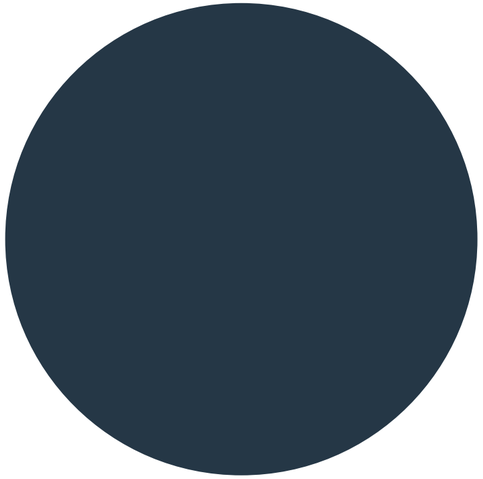

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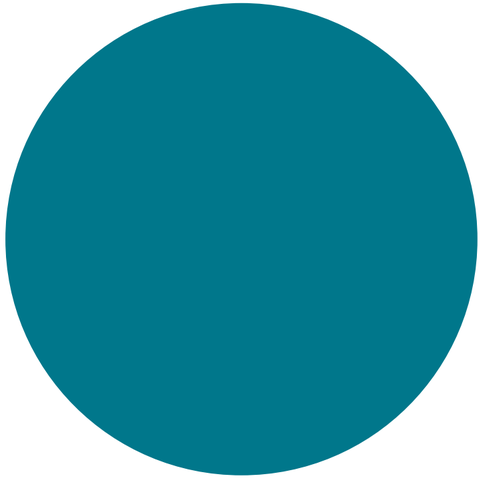

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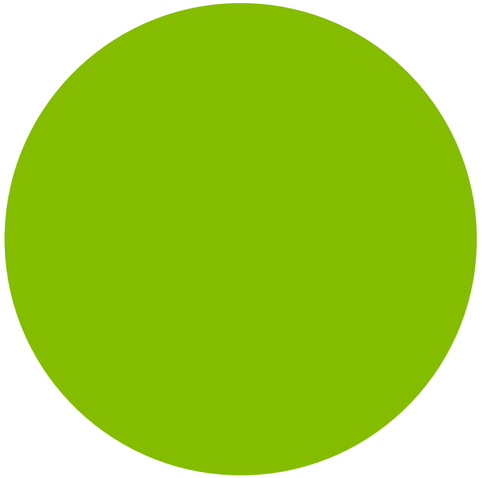

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Layout

Section

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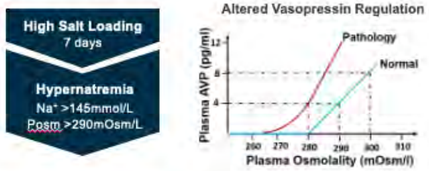

HYPOTHESIS

BDNF from the Supraoptic Nucleus (SON) contributes to increased vasopressin release in high SL rats

METHODS

Rats were l… the SON (300 nL each side) with an AAV2.… …ed with a shRNA directed against BDNF, a mC… …and a U6 promoter (shBDNF). Another group of … bilateral SON injections of equal titre and amount of …onjugated with a scrambled (SCR) sequence of shRNA as controls. The vectors were injected at a titre of 1.0 × 1013 GC mL−1. Each rat was anaesthetised with isoflurane (2%–3%) and placed in a stereotaxic frame. Their skulls were exposed and levelled between lambda and bregma.

RESULTS

mRNA Expression

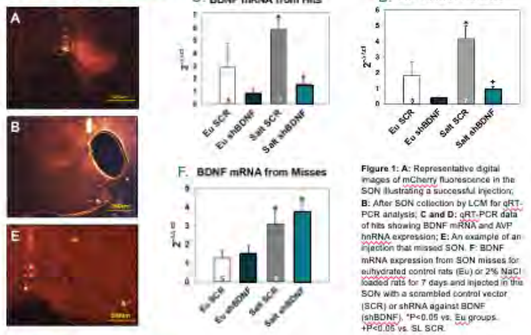

Figure 1: A: Representative digital images of mCherry fluorescence in the SON illustrating a successful injection; **B:** After SON collection by LCM for qRT-PCR analysis; **C and D:** qRT-PCR data of hits showing BDNF mRNA and AVP hnRNA expression; **E:** An example of an injection that missed SON. **F:** BDNF mRNA expression from SON misses for euhydrated control rats (Eu) or 2% NaCl loaded rats for 7 days and injected in the SON with a scrambled control vector (SCR) or shRNA against BDNF (shBDNF). *P<0.05 vs. Eu groups. +P<0.05 vs. SL SCR.

Protein Expression

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Plasma Measurements

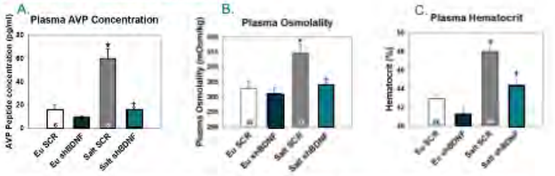
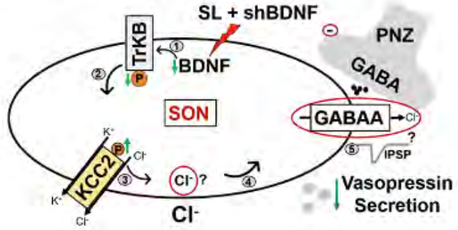


Figure 3: Changes in various plasma parameters **A:** Plasma AVP Concentration; **B:** Plasma Osmolality; and **C:** Hematocrit from euhydrated controls (Eu) and 2% salt loaded (SL) rats injected in the SON with a control vector (SCR) or shRNA BDNF (shBDNF). *P<0.05 vs. Control groups. +P<0.05 vs. Salt SCR.

CONCLUSION

The results indicate that BDNF produced in the SON is necessary for increased vasopressin secretion during high salt loading.



FUTURE DIRECTION

The pres… advances our understanding about the pathophy… VP neurone regulation. Identifying the source of BDNF un… anges in postsynaptic inhibition of AVP neurones i… alt loading may result in novel strategies for reducing… n other pathological states, such as heart and live…

…termine c… …ellular chloride concentration [Cl]I

…F-TrKB signaling in elevated …rrhosis

Ack…
The authors t… A. Wang for th… supp…

High Salt Loading Increases Brain Derived Neurotrophic Factor in Supraoptic Vasopressin Neurons

Kirthikaa Balapattabi, Joel T Little and J. Thomas Cunningham, Ph.D.
Department of Physiology & Anatomy, University of North Texas Health Science Center, Fort Worth, TX

hsc
THE UNIVERSITY of NORTH TEXAS HEALTH SCIENCE CENTER at FORT WORTH

BACKGROUND

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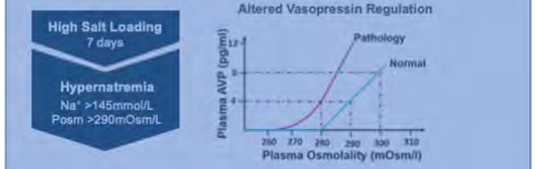

HYPOTHESIS

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METHODS

Rats were bilaterally injected in the SON (300 nL each side) with an AAV2 serotype conjugated with a shRNA directed against BDNF, a mCherry reporter and a U6 promoter (shBDNF). Another group of rats received bilateral SON injections of equal titre and amount of AAV2 conjugated with a scrambled (SCR) sequence of shRNA as controls. The vectors were injected at a titre of 1.0 × 1013 GC mL−1. Each rat was anaesthetised with isoflurane (2%–3%) and placed in a stereotaxic frame. Their skulls were exposed and levelled between lambda and bregma.

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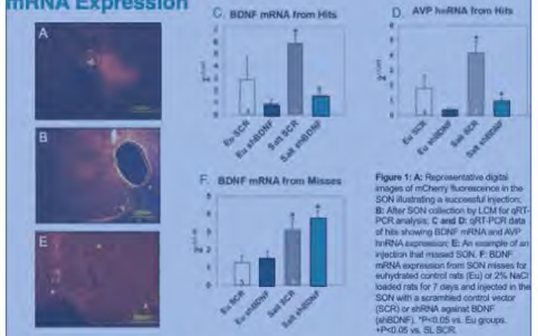

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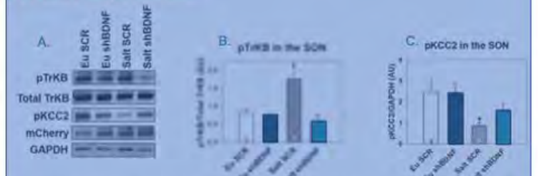

Figure 2: A: Sample Western blot images showing changes in protein expression of SON punches and quantification bar graphs for A: Phosphorylated TrKB/ Total TrKB; B: Phosphorylated KCC2 from euhydrated controls (Eu) and 2% salt loaded (SL) rats injected in the SON with a control vector (SCR) or shRNA BDNF (shBDNF). *P<0.05 vs. Eu groups. +P<0.05 vs. Salt SCR.

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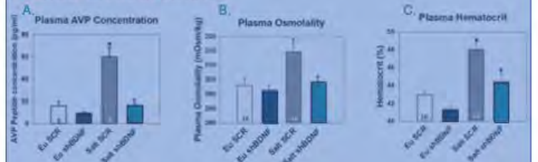

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CONCLUSION

The results indicate that BDNF produced in the SON is necessary for increased vasopressin secretion during high salt loading.

FUTURE DIRECTION

The present study advances our understanding about the pathophysiology of AVP neurone regulation. Identifying the source of BDNF underlying the changes in postsynaptic inhibition of AVP neurones in response to salt loading may result in novel strategies for reducing AVP secretion in other pathological states, such as heart and liver failure.

- To determine changes in intracellular chloride concentration [Cl]I using ClopHensorN
- To determine possible role of BDNF-TrKB signaling in elevated AVP release associated with liver cirrhosis

Acknowledgments

The authors thank M. Bachelor and Dr L. A. Wang for their assistance. This work was supported by R01 HL119458 to JTC and AHA18PRE34060035 to KB.

consistent margins

uniform "gutter"

Layout - Summary

- Do not over crowd poster with information.
- Be conscious of the margins, information too close to the edge may be cut off.

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