TRAUMA INFANTIL
Aspectos neurobiológicos

LA "RESONANCIA" DEL ALMA

DIAGNÓSTICOS PRESUNTIVOS

- SÍNCOPE VASO-VAGAL RECURRENTE
- MIGRAÑA
- OTROS SSRD
- ANSIEDAD GENERALIZADA

PROBLEMAS DEL ALMA

TRATAMIENTO

- MEDIDAS HIGIÉNICO-DIETÉTICAS
- PSICOEDUCACIÓN
- ATENOLOL
- PSICOTERAPIA –en su provincia de origen-

¿Y LA RESONANCIA?

EVOLUCIÓN

AJUSTES DE LA MEDICACIÓN

¿Y LA RESONANCIA?

MARCANDO LA FALTA

¿Y LA PSICOTERAPIA?

EL DIABLO METIÓ LA COLA

INFORME DE LA RESONANCIA

Descenso de la Oliva Cerebelosa de 10 mm
(en el límite de la normalidad)
Correlacionar con clínica y PESS

¿Malformación de Arnold Chiari?

ATENEO DE NEUROCIRUGIA

PESS DLN

PERO TIENE CLÍNICA

EMPATE

SIN CAMBIOS CLÍNICOS

CIRUGÍA

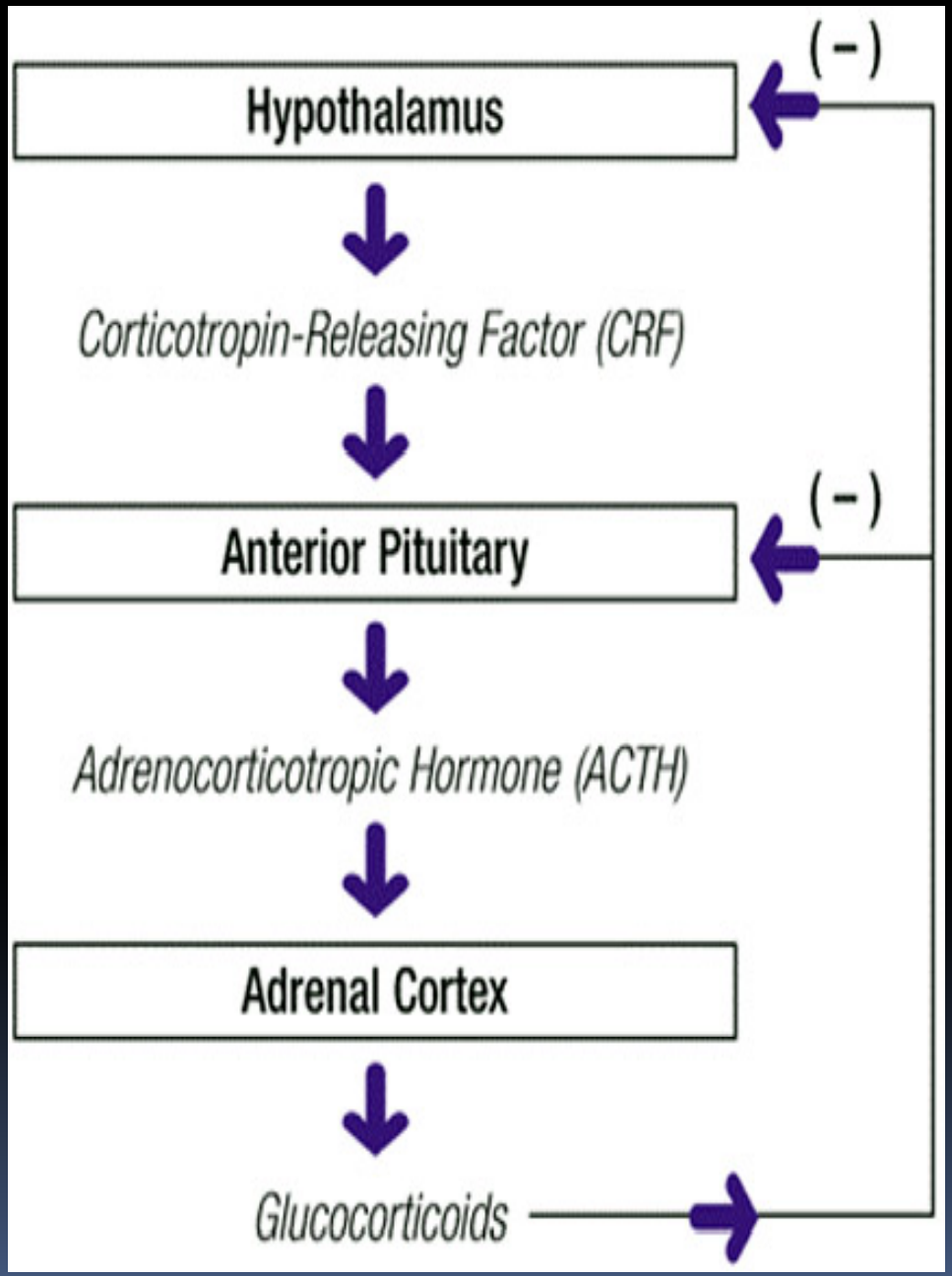

¡¡MILAGRO !!!...
¡¡MILAGRO !!!...
PARA VER EL MILAGRO BASTA DESPLAZAR LA PÁGINA HACIA LA IZQUIERDA HASTA QUE EL MISMO APAREZCA ENCUADRADO DENTRO DE LA ESCENA. SI UD. NO LO LOGRA ES PORQUE PONE POCA FE; ¡INSISTA.
QUINO

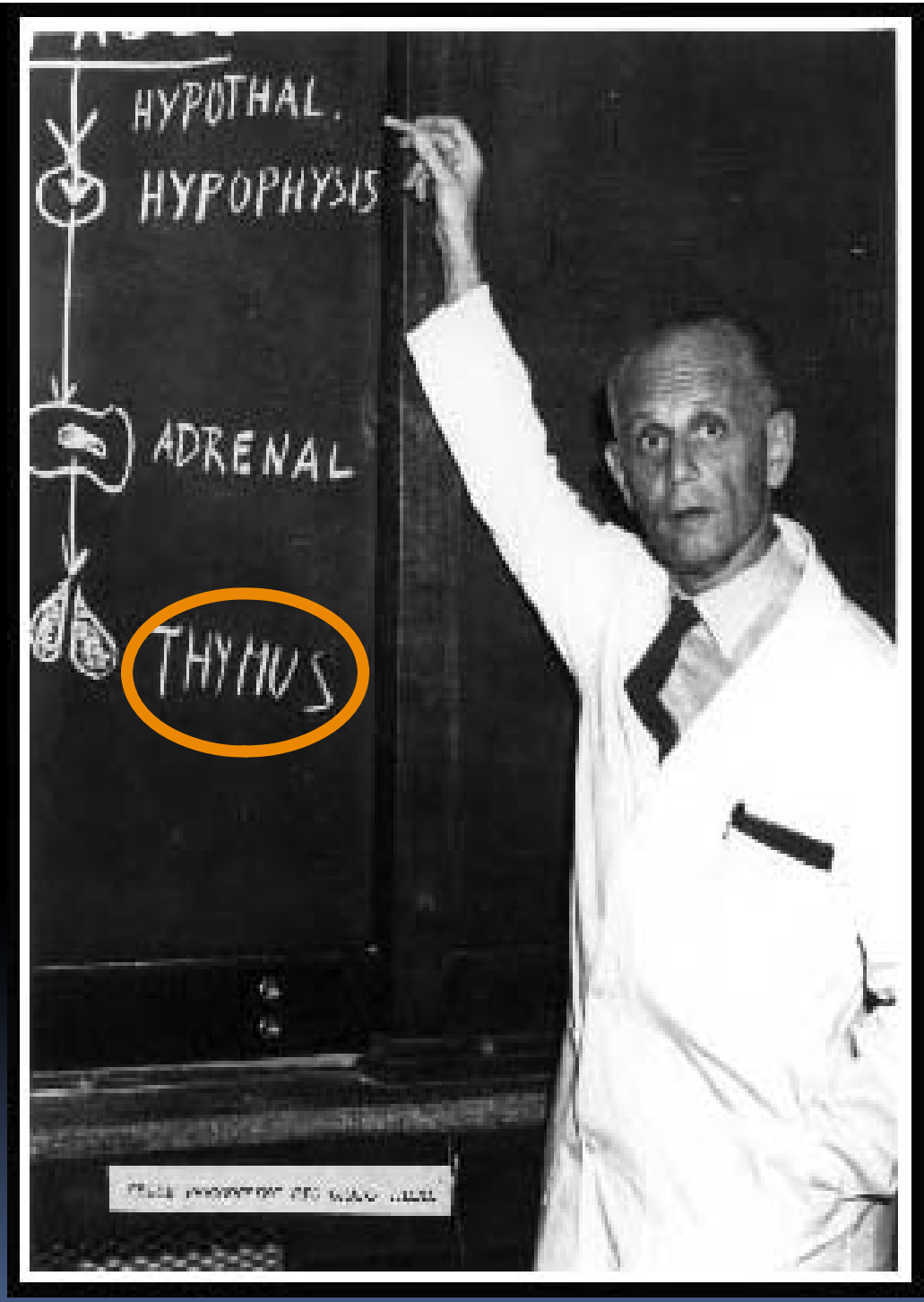

Y PASARON UNOS MESES
Veganana

Y PASARON LOS AÑOS

OTRA MIRADA CIENTÍFICA

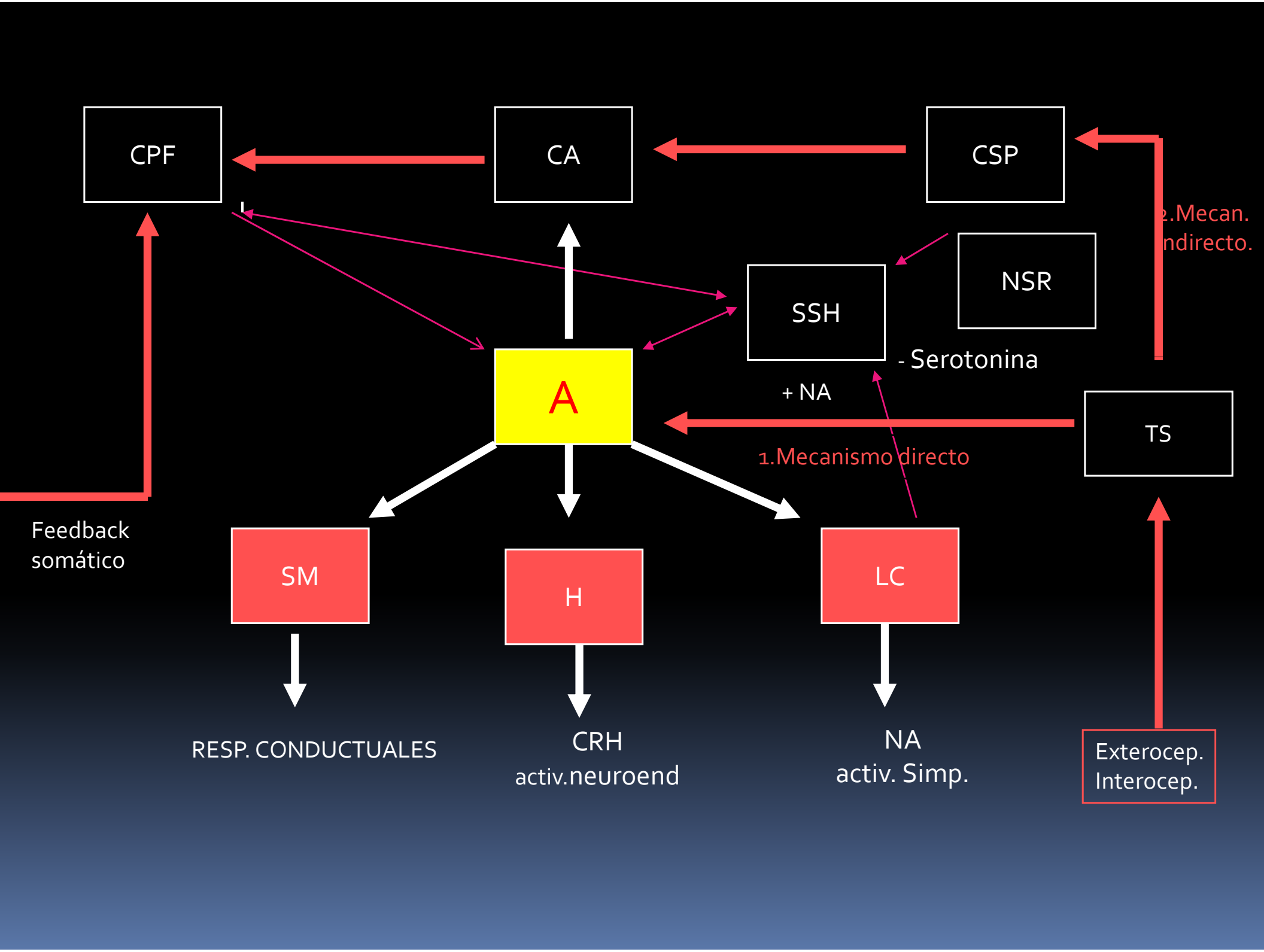
LA RESPUESTA GENERAL DE ADAPTACIÓN


(-)
Hypothalamus
Corticotropin-Releasing Factor (CRF)
(-)
Anterior Pituitary
Adrenocorticotropic Hormone (ACTH)
Adrenal Cortex
Glucocorticoids


HYPOTHAL.
HYPOPHYSIS
ADRENAL
THYMUS

Hans Selye

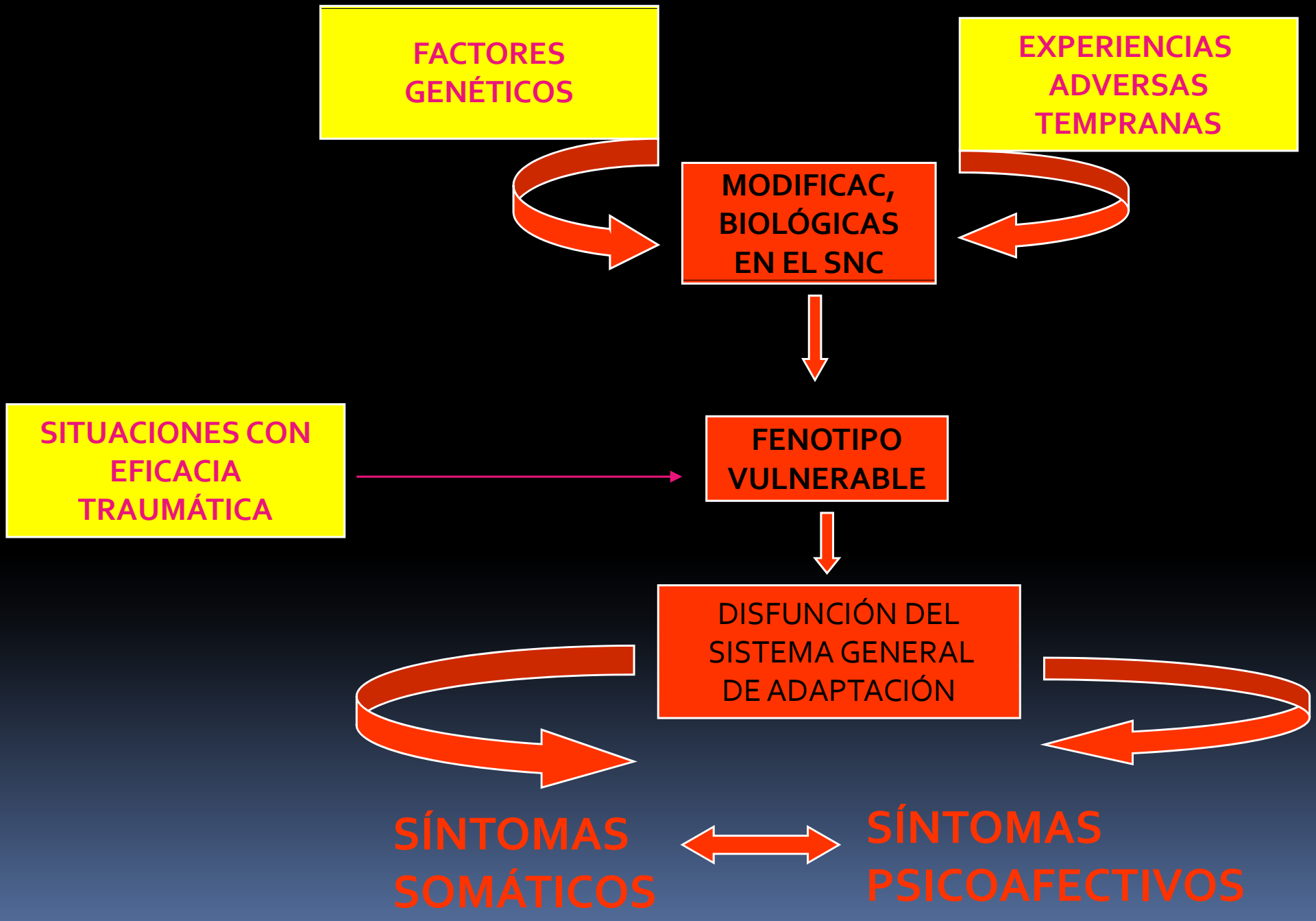
LA AMÍGDALA


CPF
CA
CSP
2.Mecan.
ndirecto.
NSR
SSH
- Serotonina
+ NA
A
TS
1.Mecanismo directo
Feedback
somático
SM
H
LC
RESP. CONDUCTUALES
CRH
activ.neuroend
NA
activ. Simp.
Exterocep.
Interocep.

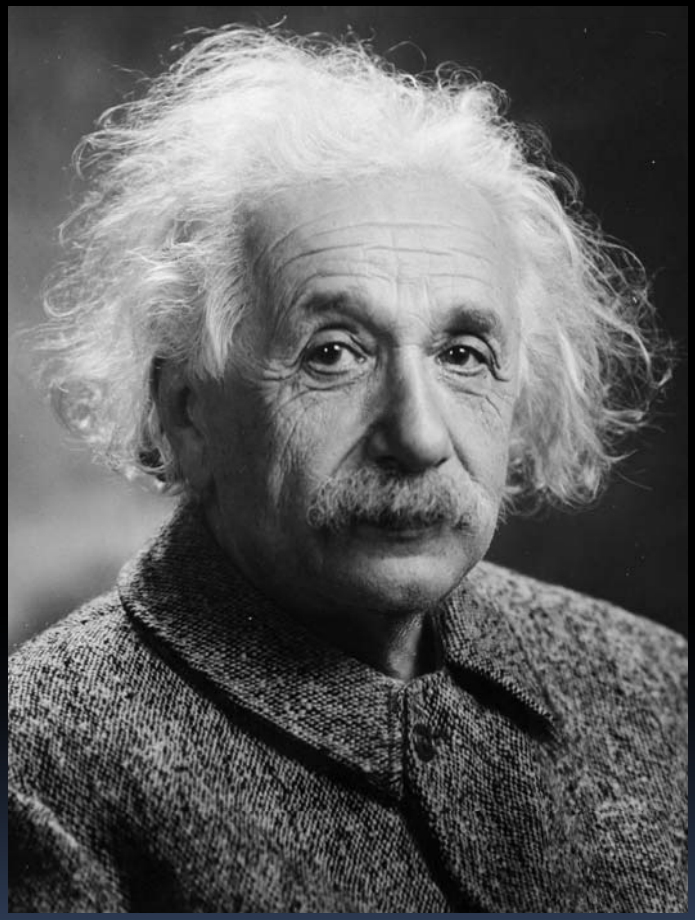
EXPERIENCIAS ADVERSAS Y DESADAPTACIÓN DE LA RESPUESTA GENERAL DE ADAPTACIÓN

- Vulnerabilidad genética
- Vulnerabilidad adquirida
- Situación no resuelta
- Superación de los recursos defensivos
- Inadecuado sostén vincular

MECANISMO FISIOPATOLÓGICO



¿una "imagen" vale
más que mil palabras?

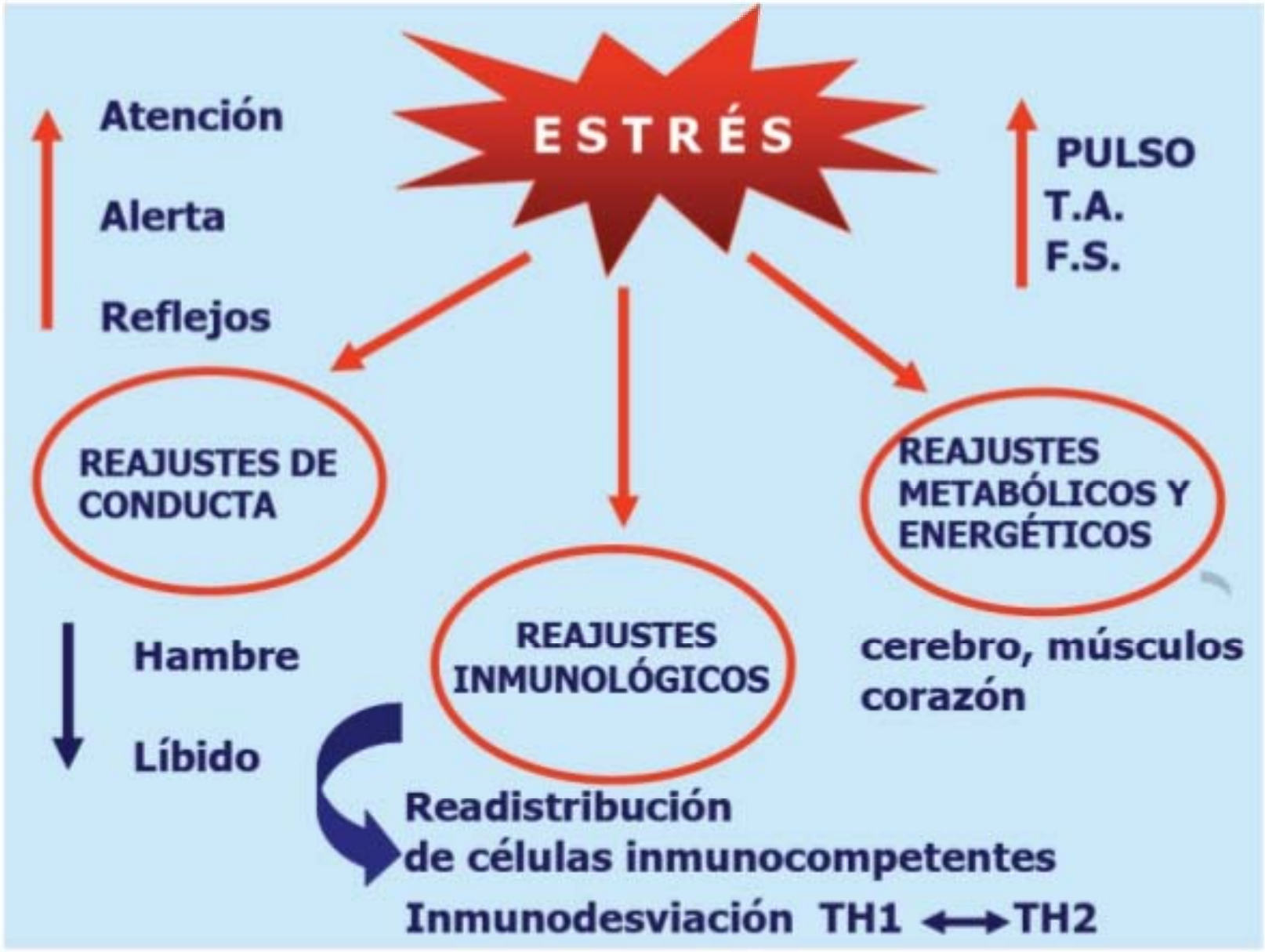


TODO ES RELATIVO

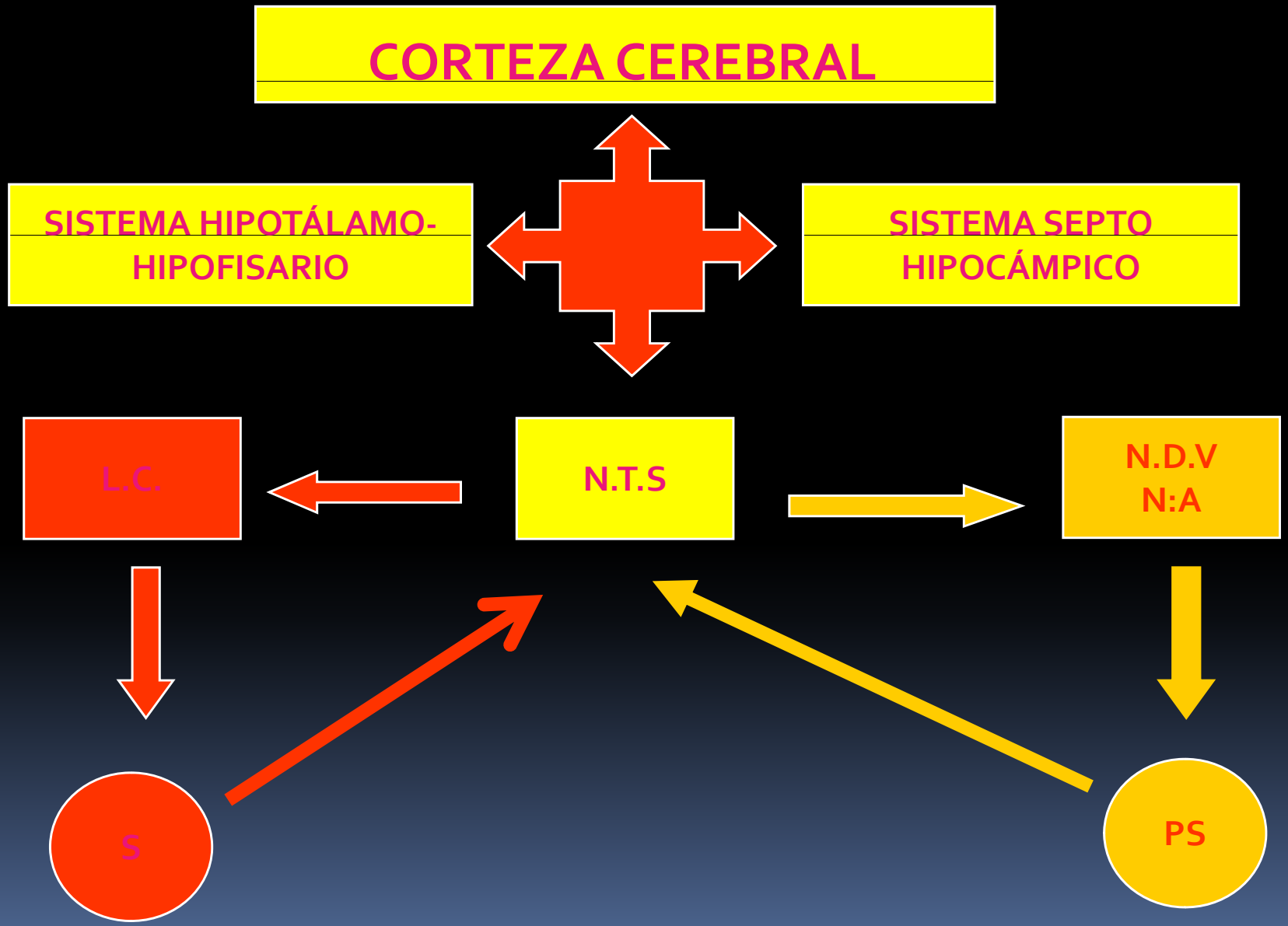
MUCHAS GRACIAS

EVIDENCIAS PNIE

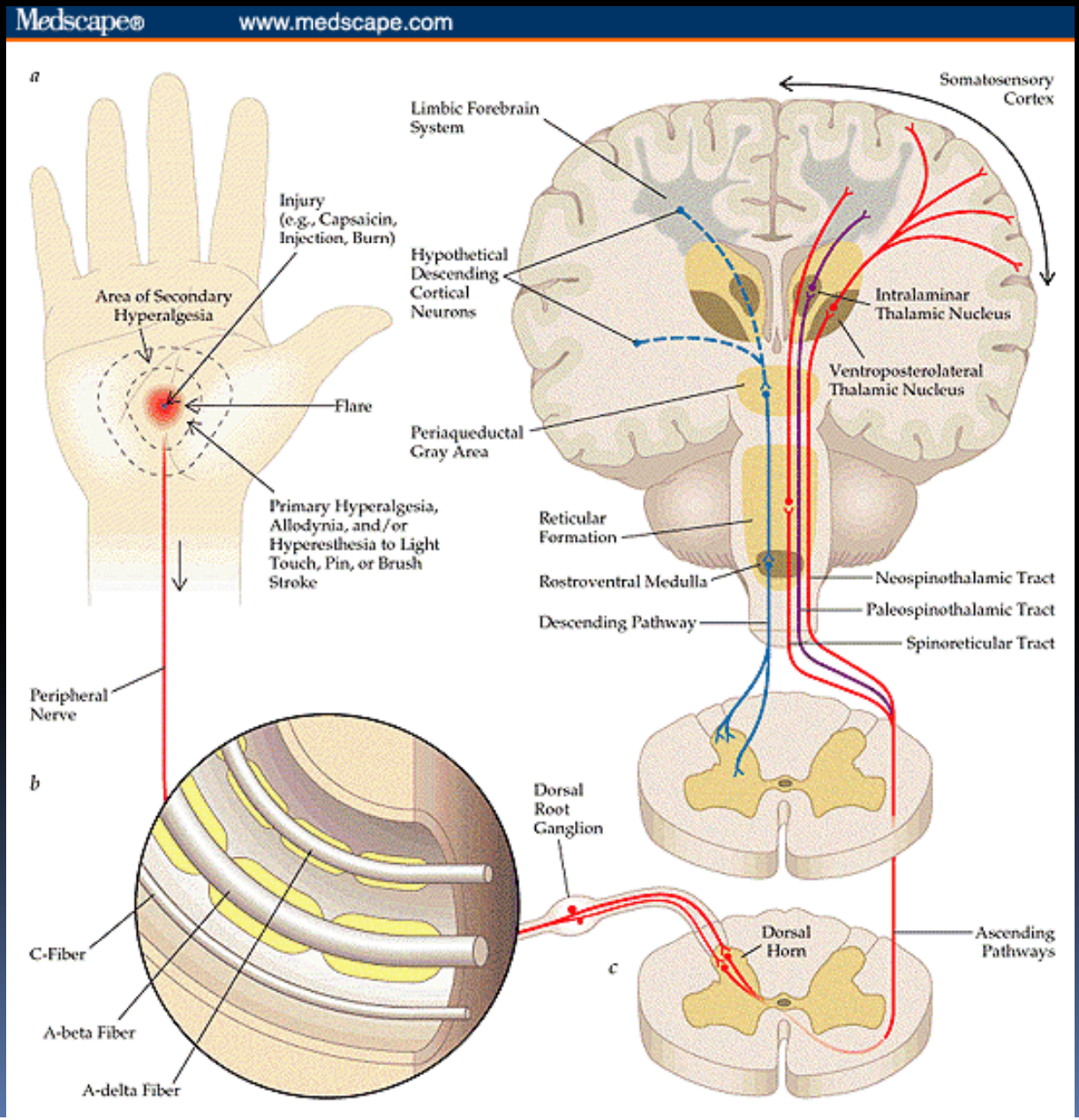
- Comunicación bidireccional entre el Cerebro y el Sistema Inmune.
- Inervación directa de los órganos inmunes.
- Neuroendocrinomodulación de la Inmunidad.
- Lesiones en áreas cerebrales producen alteraciones en respuesta inmune.
- Lateralización interhemisférica, hemisferio izquierdo inmunoestimulador.
- Citoquinas modifican actividad eléctrica y neuroquímica del Cerebro.
- Receptores de Hormonas en Cerebro y Sistema Inmune.
- Células Inmunes producen Neuropéptidos y Hormonas.
- Neuronas producen Citoquinas y Hormonas.
- Emociones relacionadas a modulación Neuroinmunoendócrina.


ESTRÉS
Atención
Alerta
Reflejos
PULSO
T.A.
F.S.
REAJUSTES DE CONDUCTA
REAJUSTES METABÓLICOS Y ENERGÉTICOS
REAJUSTES INMUNOLÓGICOS
Hambre
Líbido
cerebro, músculos corazón
Readistribución de células inmunocompetentes
Inmunodesviación TH1 ⟷ TH2

ORGANIZACIÓN JERÁRQUICA DEL SNA



VÍAS PARA EL PROCES. DEL DOLOR
VÍAS PARA EL PROCES. DE LAS EMOCIONES



B E McEwen:
***Dialogues Clin Neurosci*.**
2006; 8:367-381.

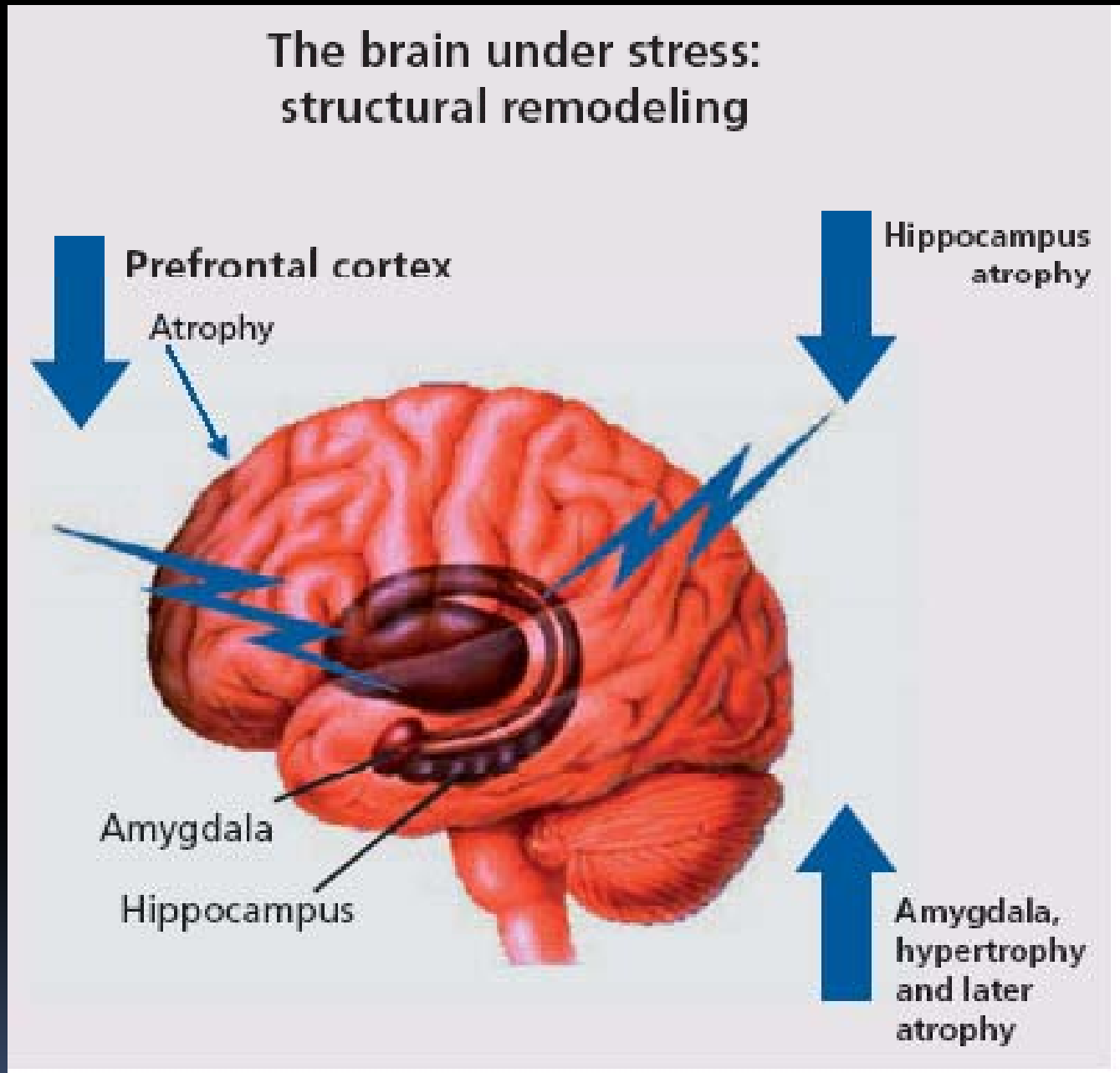


Figure 4. Brain regions that are involved in perception and response to stress, and which show structural remodeling as a result of stress.

Hindawi
BioMed Research International
Volume 2018, Article ID 8142631, 11 pages
https://doi.org/10.1155/2018/8142631

Research Article

Negative Mood States Correlate with Laterobasal Amygdala in Collegiate Football Players

Chen *et al. The Journal of Headache and Pain* (2017) 18:7
DOI 10.1186/s10194-017-0722-5

The Journal of Headache and Pain

RESEARCH ARTICLE Open Access

Altered functional connectivity of amygdala underlying the neuromechanism of migraine pathogenesis

Zhiye Chen[1,2,3], Xiaoyan Chen[2], Mengqi Liu[1,3], Zhao Dong[2], Lin Ma[1*] and Shengyuan Yu[2*]

Cogn Affect Behav Neurosci (2017) 17:858–868
DOI 10.3758/s13415-017-0518-8

Experimental manipulation of infant temperament affects amygdala functional connectivity

Madelon M. E. Riem[1,2,3] · Marinus H. Van Ijzendoorn[2,3] · Christine E. Parsons[4] · Katherine S. Young[5,6] · Pietro De Carli[7] · Morten L. Kringelbach[5] · Marian J. Bakermans-Kranenburg[2,3]

DOI: 10.1111/bdi.12516

ORIGINAL ARTICLE

WILEY BIPOLAR DISORDERS

Amygdala and hippocampus volumes are differently affected by childhood trauma in patients with bipolar disorders and healthy controls

Delfina Janiri[1,*] | Gabriele Sani[2,3,4,*] | Pietro De Rossi[2,3,5] | Fabrizio Piras[6,7] | Mariangela Iorio[6] | Nerisa Banaj[6] | Giulia Giuseppin[8] | Edoardo Spinazzola[1] | Matteo Maggiora[1] | Elisa Ambrosi[6,9] | Alessio Simonetti[3,9] | Gianfranco Spalletta[6,9]

Received: 22 February 2017 | Revised: 22 May 2017 | Accepted: 20 June 2017

DOI: 10.1002/hipo.22755

RESEARCH ARTICLE

WILEY

Cannabinoids prevent the differential long-term effects of exposure to severe stress on hippocampal- and amygdala-dependent memory and plasticity

Noa Shoshan | Amir Segev | Hila Abush | Tomer Mizrachi Zer-Aviv | Irit Akirav

EJN European Journal of Neuroscience

FENS Federation of European Neuroscience Societies

European Journal of Neuroscience, Vol. 46, pp. 2149–2160, 2017

doi:10.1111/ejn.13662

MOLECULAR AND SYNAPTIC MECHANISMS

Essential role of endogenous calcitonin gene-related peptide in pain-associated plasticity in the central amygdala

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[4]Department of Molecular Cell Biology, Graduate School of Medicine, The University of Tokyo, Tokyo, Japan

Abstract

The role of the neuropeptide calcitonin gene-related peptide (CGRP) is well established in nociceptive behaviors. CGRP is highly expressed in the projection pathway from the parabrachial nucleus to the laterocapsular region of the central amygdala (CeC), which plays a critical role in relaying nociceptive information. The CeC is a key structure in pain behavior because it integrates and modulates nociceptive information along with other sensory signals. Previous studies have demonstrated that blockade of the amygdalar CGRP-signaling cascade attenuates nociceptive behaviors in pain models, while CGRP application facilitates amygdalar synaptic transmission and induces pain behaviors. Despite these lines of evidence, it remains unclear whether endogenous CGRP is involved in the development of nociceptive behaviors accompanied with amygdalar plasticity in a peripheral inflammation model *in vivo*. To directly address this, we utilized a previously generated CGRP knockout (KO) mouse to longitudinally study formalin-induced plasticity and nociceptive behavior. We found that synaptic potentiation in the right PB-CeC pathway that was observed in wild-type mice was drastically attenuated in the CGRP KO mice 6 h post-inflammation, when acute nociceptive behavior was no longer observed. Furthermore, the bilateral tactile allodynia 6 h post-inflammation was significantly decreased in the CGRP KO mice. In contrast, the acute nociceptive behavior immediately after the formalin injection was reduced only at 20–25 min post-injection in the CGRP KO mice. These results suggest that endogenous CGRP contributes to peripheral inflammation-induced synaptic plasticity in the amygdala, and this plasticity may underlie the exaggerated nociception–emotion linkage in pain chronification.

ARTÍCULO

Hippocampal and amygdala volumes in adults with posttraumatic stress disorder secondary to childhood abuse or maltreatment: A systematic review

Fatima Ahmed-Leitao, Georgina Spies, Leigh van den Heuvel y Soraya Seedat

Psychiatry Research: Neuroimaging, 2016-10-30, Volumen 256, Páginas 33-43, Copyright © 2016 Elsevier Ireland Ltd

GENETICA Y EPIGENETICA

Genes, Brain and Behavior

Official publication of the International Behavioural and Neural Genetics Society

Genes, Brain and Behavior (2012) ***11:*** *869–878*

doi: 10.1111/j.1601-183X.2012.00837.x

FKBP5 and emotional neglect interact to predict individual differences in amygdala reactivity

Individual variation in physiological responsiveness to stress mediates risk for mental illness and is influenced by both experiential and genetic factors. Common polymorphisms in the human gene for FK506 binding protein 5 (*FKBP5*), which is involved in transcriptional regulation of the hypothalamic–pituitary–adrenal (HPA) axis, have been shown to interact with childhood abuse and trauma to predict stress-related psychopathology. In the current study, we examined if such gene–environment interaction effects may be related to variability in the threat-related reactivity of the amygdala, which plays a critical role in mediating physiological and behavioral adaptations to stress including modulation of the HPA axis. To this end, 139 healthy Caucasian youth completed a blood oxygen level-dependent functional magnetic resonance imaging probe of amygdala reactivity and self-report assessments of emotional neglect (EN) and other forms of maltreatment. These individuals were genotyped for 6 *FKBP5* polymorphisms (rs7748266, rs1360780, rs9296158, rs3800373, rs9470080 and rs9394309) previously associated with psychopathology and/or HPA axis function. Interactions between each SNP and EN emerged such that risk alleles predicted relatively increased dorsal amygdala reactivity in the context of higher EN, even after correcting for multiple testing. Two different haplotype analyses confirmed this relationship as haplotypes with risk alleles also exhibited increased amygdala reactivity in the context of higher EN. Our results suggest that increased threat-related amygdala reactivity may represent a mechanism linking psychopathology to interactions between common genetic variants affecting HPA axis function and childhood trauma.

Psychoneuroendocrinology 85 (2017) 115–122

Contents lists available at ScienceDirect

Psychoneuroendocrinology

journal homepage: www.elsevier.com/locate/psyneuen

Emotional maltreatment is associated with atypical responding to stimulation of endogenous oxytocin release through mechanically-delivered massage in males☆

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frontiers in Neuroscience

ORIGINAL RESEARCH
published: 31 July 2018
doi: 10.3389/fnins.2018.00512

Check for updates

Oxytocin Enhancement of Emotional Empathy: Generalization Across Cultures and Effects on Amygdala Activity

Yayuan Geng[1], Weihua Zhao[1], Feng Zhou[1], Xiaole Ma[1], Shuxia Yao[1], Rene Hurlemann[2,3], Benjamin Becker[1] and Keith M. Kendrick[1]**

[1] The Clinical Hospital of the Chengdu Brain Science Institute, MOE Key Laboratory for Neuroinformation, University of Electronic Science and Technology of China, Chengdu, China, [2] Department of Psychiatry, University of Bonn, Bonn, Germany, [3] Division of Medical Psychology, University of Bonn, Bonn, Germany*

Abstract | **Full Text**

Accumulating evidence suggests that the neuropeptide oxytocin (OXT) can enhance empathy although it is unclear which specific behavioral and neural aspects are influenced, and whether the effects are modulated by culture, sex, and trait autism. Based on previous findings in Caucasian men, we hypothesized that a single intranasal dose of OXT would specifically enhance emotional empathy (EE) via modulatory effects on the amygdala in an Asian (Chinese) population and explored the modulatory role of sex and trait autism on the effects. We first conducted a double-blind, randomized between-subject design experiment using a modified version of the multifaceted empathy task to determine whether OXT's facilitation of EE can be replicated in Chinese men (n = 60). To further explore neural mechanisms behind and potential sex differences, functional MRI and skin conductance measures were acquired in an independent experiment incorporating men and women (n = 72). OXT enhanced EE across experiments and sex, an effect that was accompanied by reduced amygdala activity and increased skin conductance responses. On the network level OXT enhanced functional coupling of the right amygdala with the insula and posterior cingulate cortex for positive valence stimuli but attenuated coupling for negative valence stimuli. The effect of OXT on amygdala functional connectivity with the insula was modulated by trait autism. Overall, our findings provide further support for the role of OXT in facilitating EE and demonstrate that effects are independent of culture and sex and involve modulatory effects on the amygdala and its interactions with other key empathy regions.

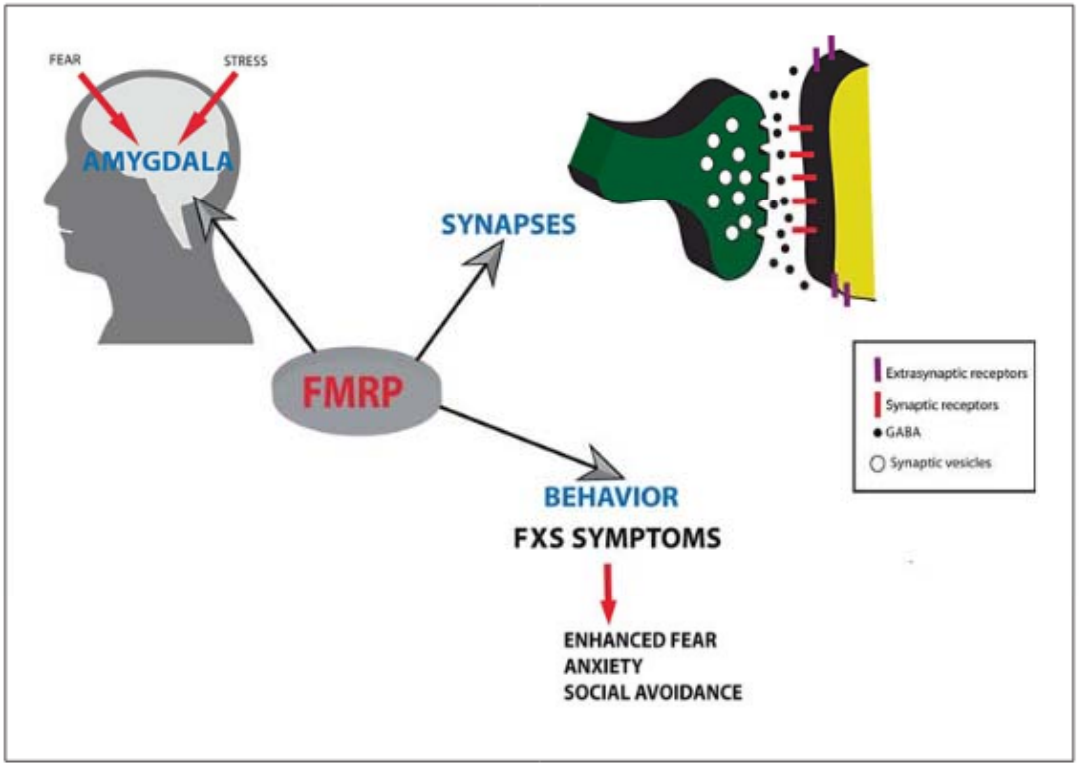

Fig. 2. FMRP is expressed in the amygdala and influences fear and stress response. FMRP is present at the synapse and regulates the translation of numerous proteins important for synaptic structure and function. Absence of FMRP at the synapse affects amygdala circuit function and therefore ultimately behavior.