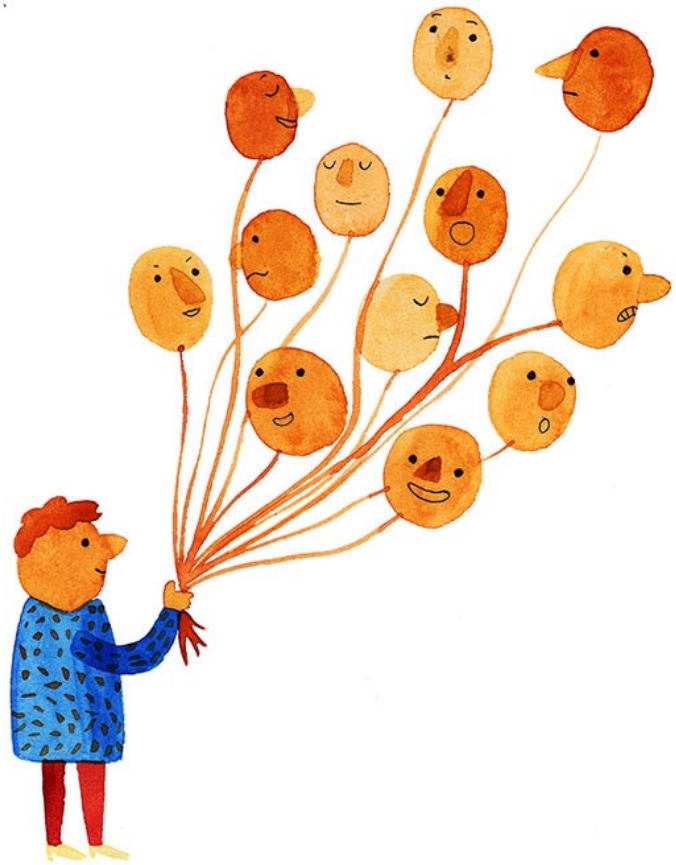
EMOZIONI DI TESTA O EMOZIONI DI PANCIA?

COME QUELLO CHE MANGIAMO CI PUO' RENDERE FELICI

Prof Lorenzo Pascazio

CHE COSA SONO LE EMOZIONI?



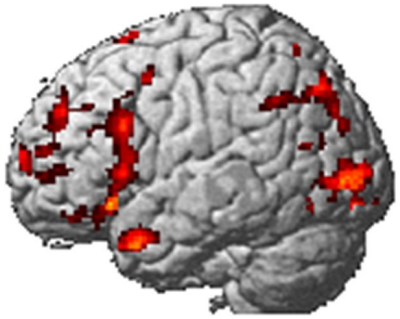
UN'EMOZIONE E' UN PROCESSO CHE AVVIENE NEL NOSTRO ORGANISMO CHE SI VERIFICA IN RISPOSTA AD UN EVENTO

E' UN PROCESSO DINAMICO ED ADATTIVO…

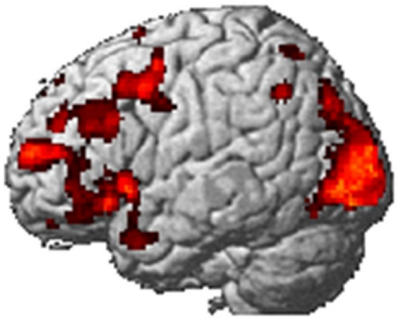
…ED E’ MOLTO PRECOCE PERCHE’ SERVE PER LA SOPRAVVIVENZA

LE EMOZIONI SONO UNA RISPOSTA AD UNO STIMOLO DELL' AMBIENTE E GENERANO:

- ESPERIENZE SOGGETTIVE (SENTIMENTI)
- COMPORTAMENTI ESPRESSIVI (POSTURA E MOVIMENTO DEL CORPO)
- CAMBIAMENTI FISIOLOGICI (ATTIVAZIONE DEL SISTEMA NERVOSO AUTONOMO, REAZIONI ORMONALI ED ELETTROCORTICALI)

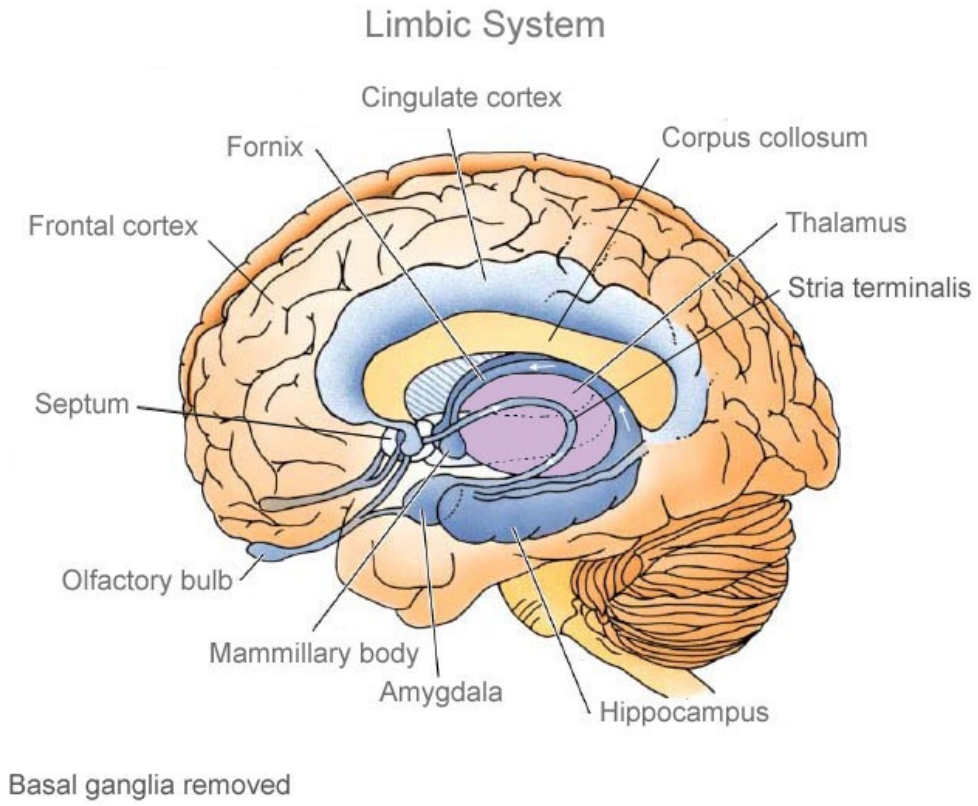


HAPPY



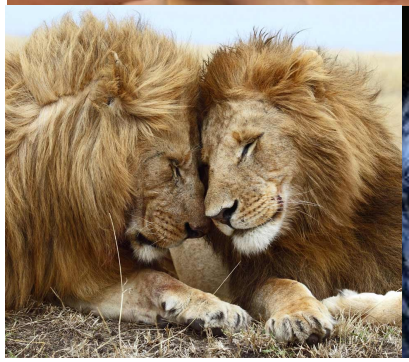
SAD

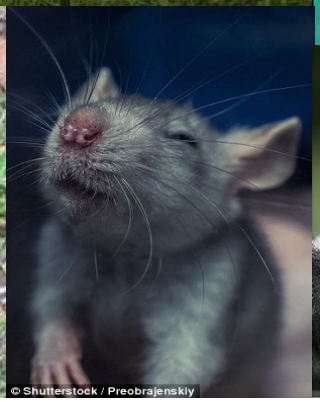
LE STRUTTURE DEL CERVELLO IN CUI ORIGINANO LE EMOZIONI



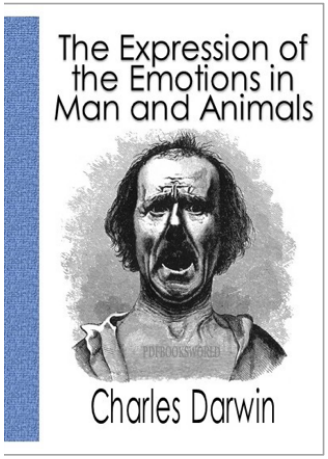
UOMINI ED ANIMALI PROVANO LE STESSE EMOZIONI?



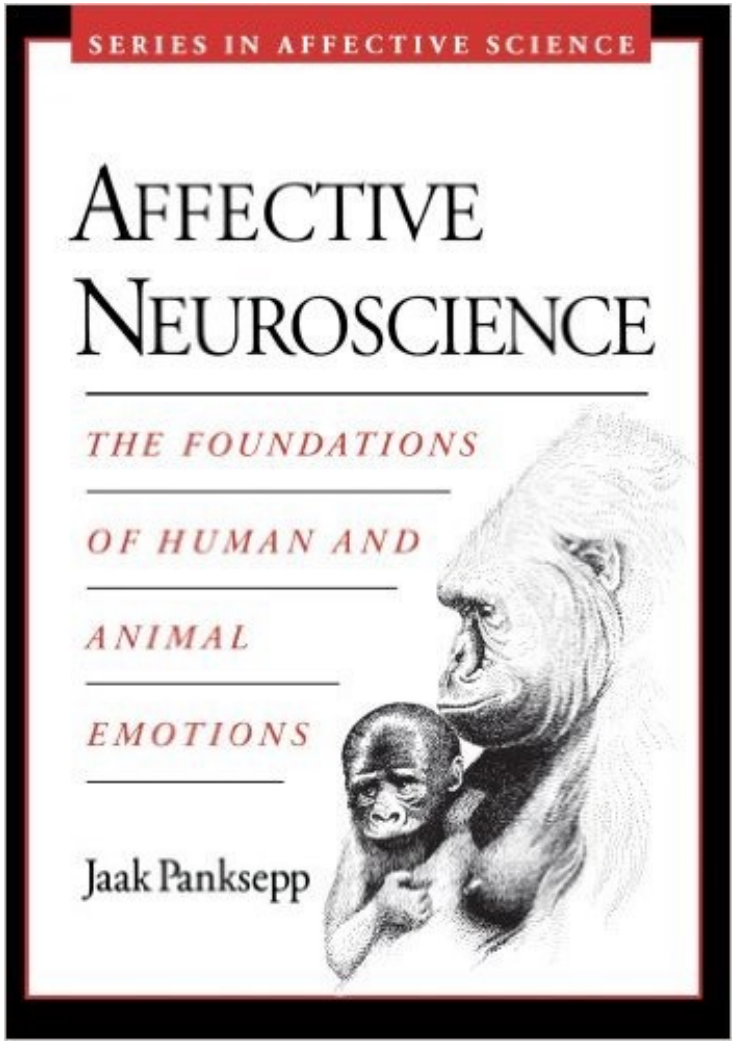



In 1872, Darwin published *The Expression of the Emotions in Man and Animals*, in which he argued that all humans, and even other animals, sh
similar beh
evolutionar
across cul
view at th
agree that
humans, r
surprise, di

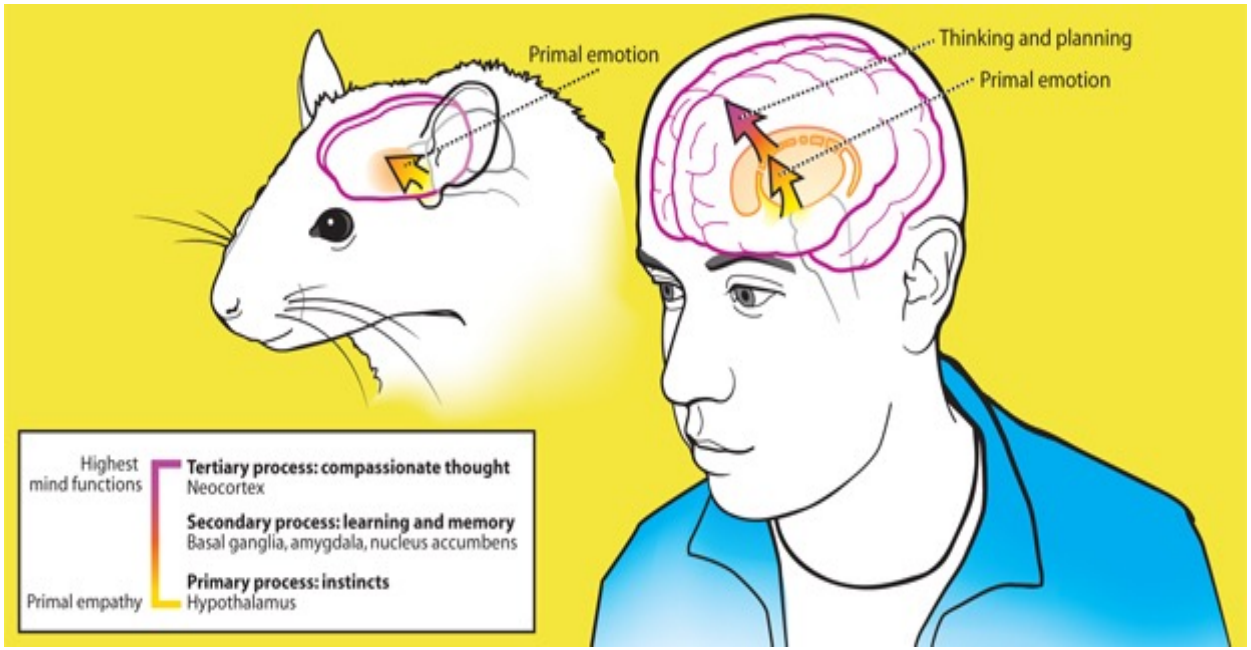


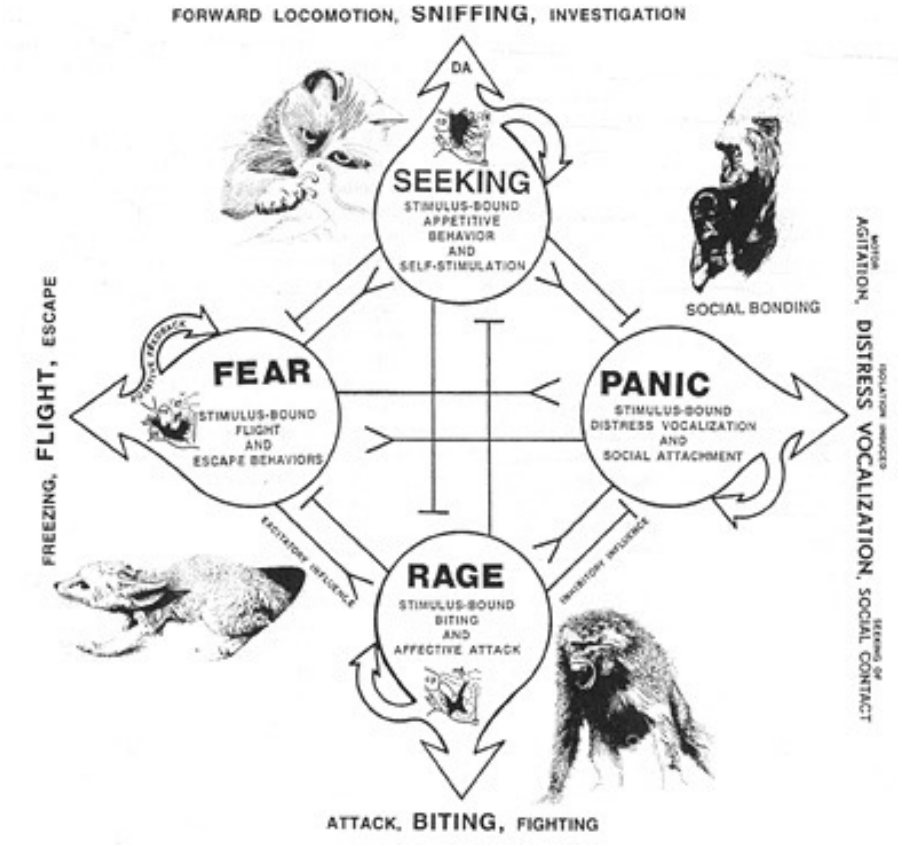
Tutti gli esseri umani, compresi gli animali, mostrano emozioni attraverso comportamento simili…si è concordi che certe emozioni sono universali e presenti in tutti gli esseri umani, senza distinzione di cultura, e sono: rabbia, paura, sorpresa, disgusto, felicità e tristezza.

JAAK PANKSEPP (1943-2017)



UOMINI E ANIMALI CONDIVIDONO LE STESSE EMOZIONI DI BASE CHE ORIGINANO DALLE MEDESIME AREE DEL CERVELLO





EMOZIONI DI BASE

I MECCANISMI EMOZIONALI DI BASE RACCHIUDONO EMOZIONI CHE COSTITUISCONO IL SUBSTRATO EVOLUTIVO COMUNE A TUTTI GLI ESSERE VIVENTI E POSSIEDONO UNA BASE FISIOLOGICA SPECIFICA NEL SISTEMA NERVOSO CENTRALE (Panksepp 1998).

EMOZIONI DI BASE E CIRCUITI NEURONALI

- I SISTEMI EMOZIONALI DI BASE FUNGONO DA SEGNALATORI DELLO STATO EMOTIVO INTERIORE (Solms & Turnbull, 2002).

PANKSEPP IDENTIFICA SEI SISTEMI EMOZIONALI DI BASE:

- **FEAR** → SISTEMA DELLA PAURA
- **ANGER** → SISTEMA DELLA RABBIA
- **SEPARATION DISTRESS** → ANSIA DA SEPARAZIONE
- **SEEK** → SISTEMA DELLA RICERCA
- **PLAY** → SISTEMA DEL GIOCO E DELL'APPRENDIMENTO
- **CARE** → SISTEMA DELLE CURE

OGNUNO SOTTENDE A SPECIFICI CIRCUITI NEURONALI.

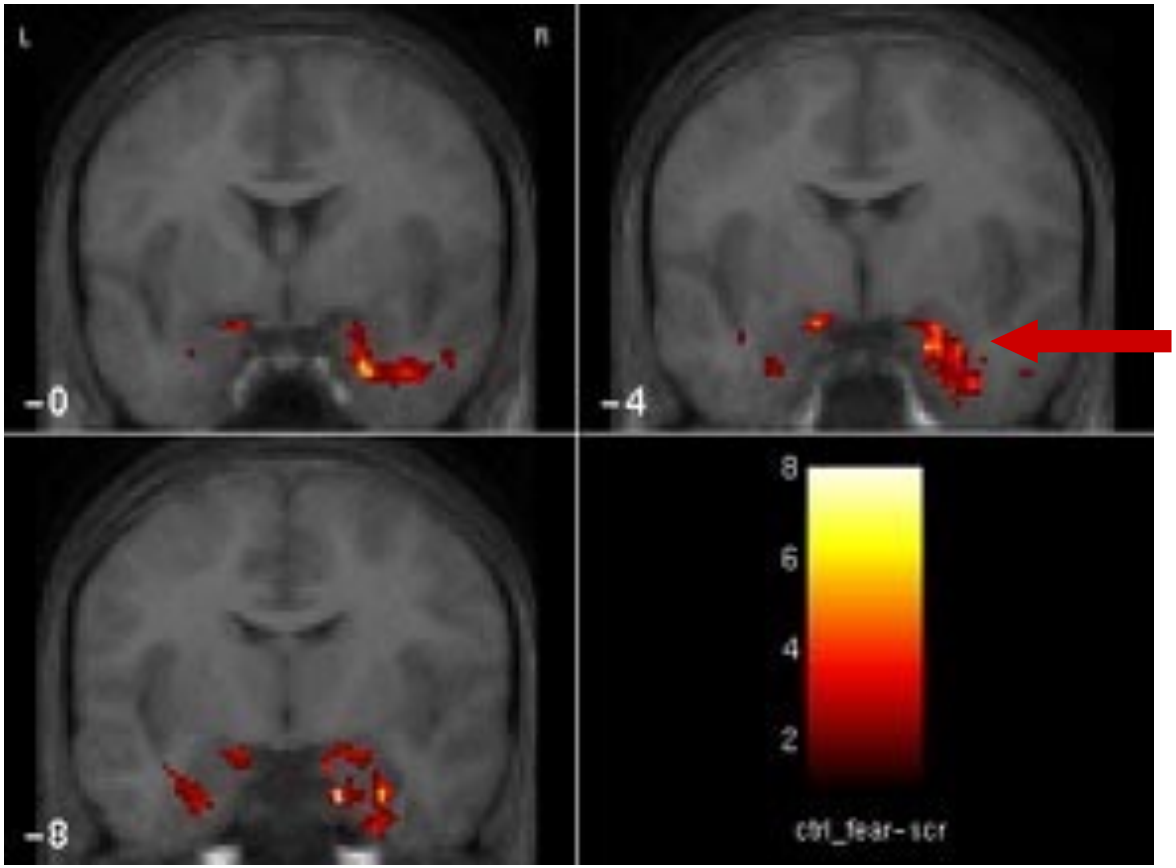
FEAR

CORRISPONDE A SENSAZIONI DI PAURA E ANSIA

AMIGDALA E PROIEZIONI ALL'IPOTALAMO ANTERIORE, MEDIALE E AL GRIGIO PERIACQUEDUTTALE DORSALE
ACIDO GLUTAMMICO (+), DBI (recettore benzodiazepine), CRH (+) (corticotropine), CCK (colecistochinina – endocannabinoide),

fMRI ed emozioni

Aree specifiche per emozione



Stimolo di paura

Amigdala

ANGER

SENTIMENTI DI RABBIA, SI MANIFESTANO ANCHE ATTRAVERSO L'IRRITABILITÀ E LA FRUSTRAZIONE

NUCLEI MEDIALI DELL'AMIGDALA, STRIA TERMINALE, IPOTALAMO E GRIGIO PERIACQUEDOTTALE

ACETILCOLINA (+), ACIDO GLUTAMMICO (+), VASOPRESSINA, SOSTANZA P (+)

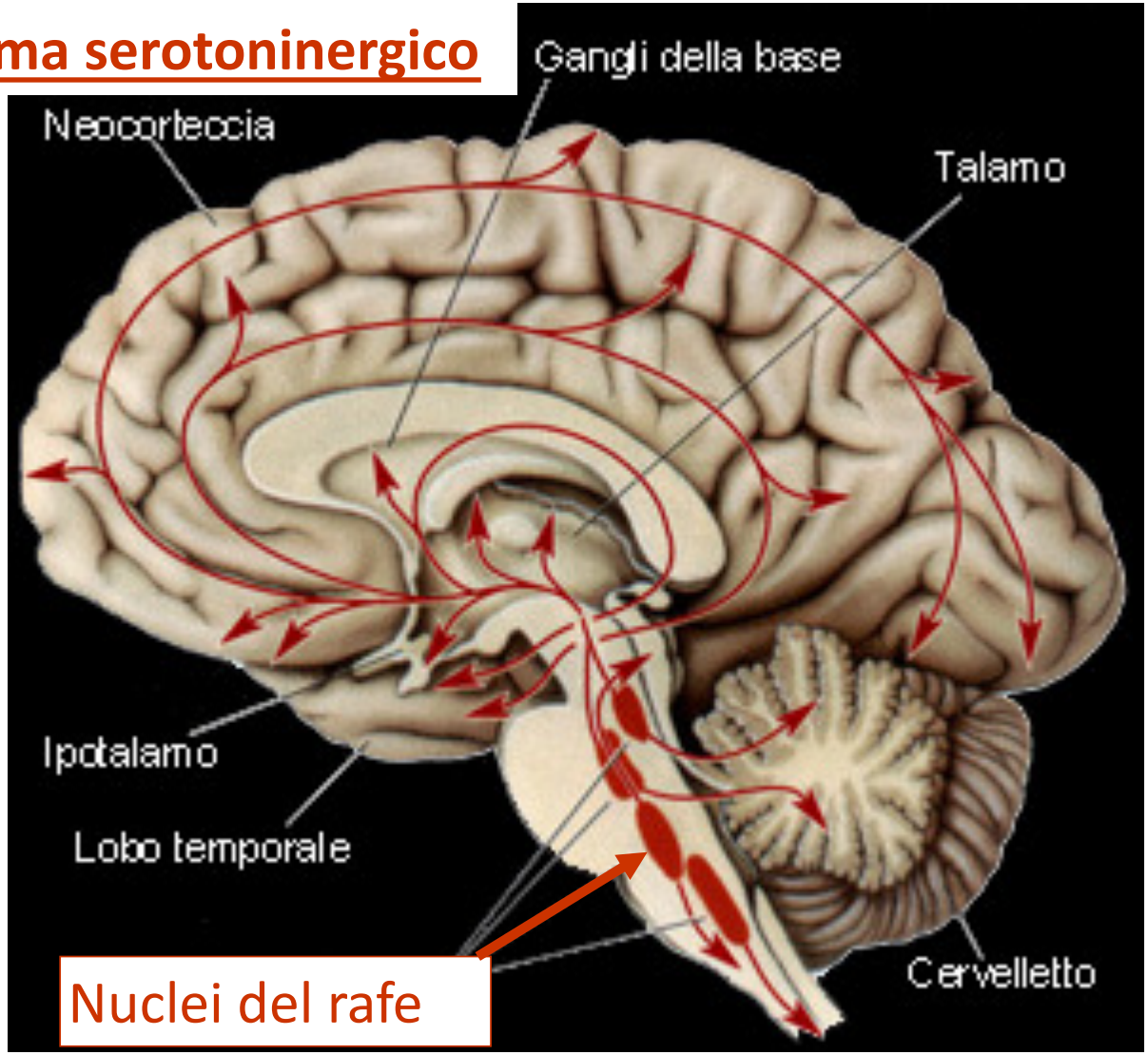
SEPARATION DISTRESS

ANSIA DA SEPARAZIONE,
È LEGATO AD ASPETTI
CHE
IMPLICANO IL DISTACCO
E A SENTIMENTI
DEPRESSIVI

→ CINGOLO ANTERIORE, TALAMO DORSOMEDIALE BED NUCLEUS STRIA TERMINALE, GRIGIO PERIACQUEDOTTALE

→ **OPPIOIDI** (+/-), **OSSITOCINA (-), PROLATTINA (-), ACIDO GLUTAMMICO (+), CRF O CORTICOTROPINA (+)** (ELEVATA NEI SUICIDI).

Sistema serotoninergico



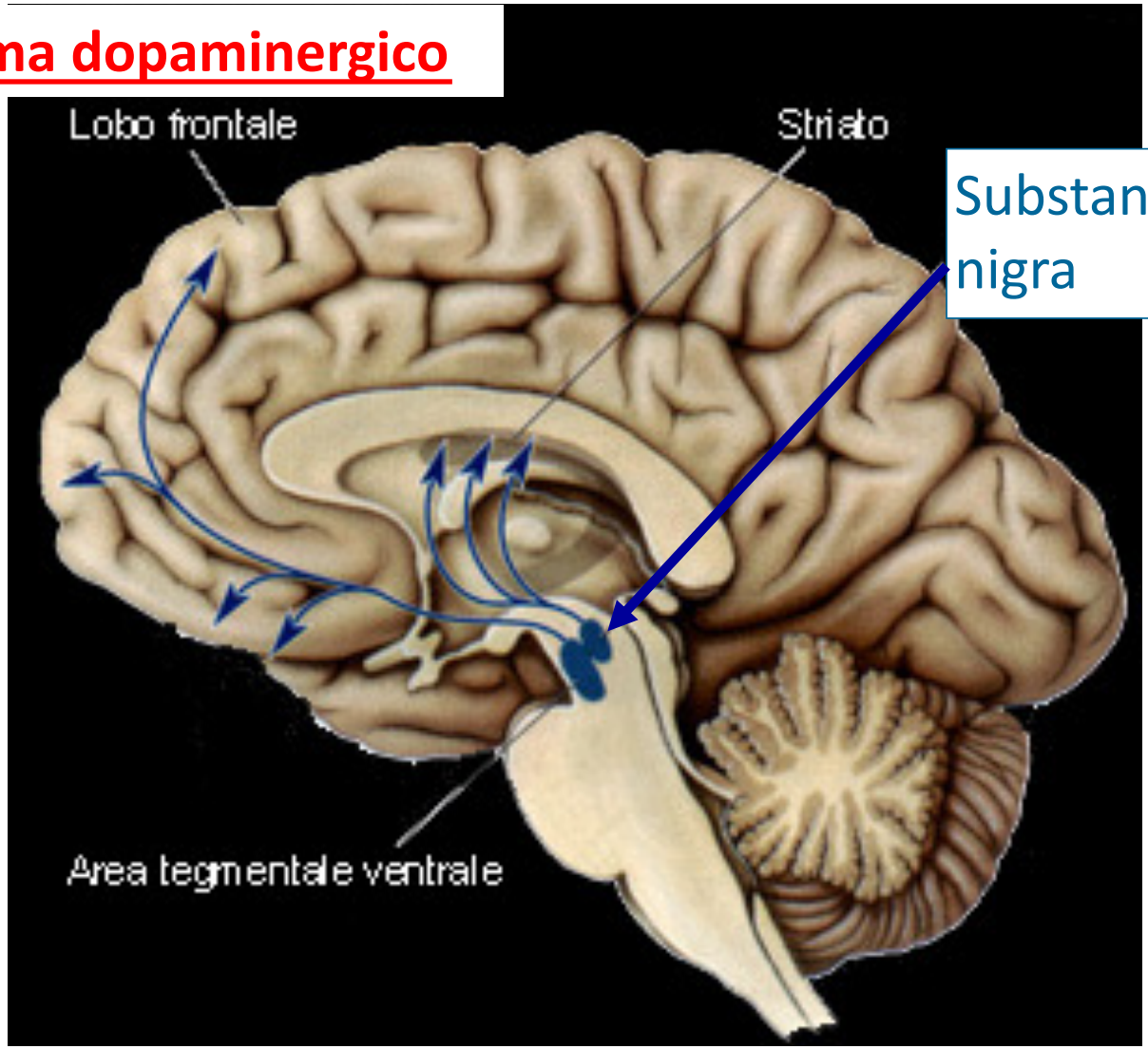
SEEKING

ALLA BASE DELLA CURIOSITÀ E VOLONTA' DI ESPLORAZIONE DELL'AMBIENTE CIRCOSTANTE MA NON SOLO…

NUCLEO ACCUMBENS AREA VENTRALE TEGMENTALE, AREE MESOLIMBICHE, IPOTALAMO LATERALE

DOPAMINA (+), ACIDO GLUTAMMICO (+), OPPIOIDI (+), OREXINA (+)

Sistema dopaminergico



PLAY

HA UN RUOLO IMPORTANTE NEI PROCESSI DI APPRENDIMENTO E DI SVILUPPO E NELL' INTEGRAZIONE SOCIALE

DIENCEFALO DORSO MEDIALE , AREA PARAFASCICOLARE, GRIGIO PERIACQUEDUTTALE

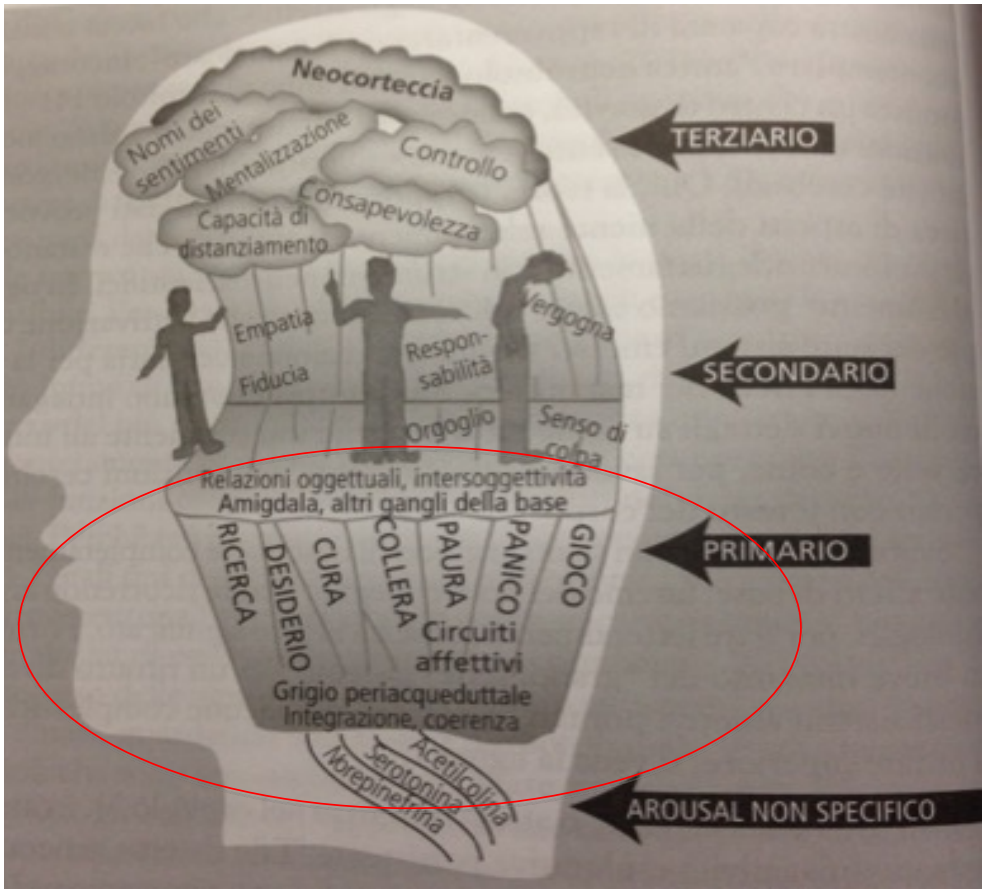
FACILITATORI: OPPIOIDI (+/-), ACIDO GLUTAMMICO (+), ACETILCOLINA (+), CANNABINOIDI
EFFETTO INIBITORE: SEROTONINA, NORADRENALINA E GABA

CARE

È ASSOCIATO AD ATTEGGIAMENTI DI ACCUDIMENTO E ALLA BASE DEGLI ATTEGGIAMENTI EMPATICI

→ **CINGOLO ANTERIORE, NUCLEO VENTRALE DELLA STRIA TERMINALE (BNST),AREA PREOTTICA, AREA TEGMENTALE VENTRALE, GRIGIO PERIACQUEDOTTALE**

OSSITOCINA (+), PROLATTTINA (+), DOPAMINA (+), OPPIOIDI (+/-)

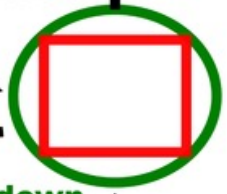


Two-Way or "Circular" Causation

Tertiary-Process Cognitions
Largely Neocortical

Top-down
Cognitive
Regulation

Bottom-Up Influences on
Ruminations and Thoughts



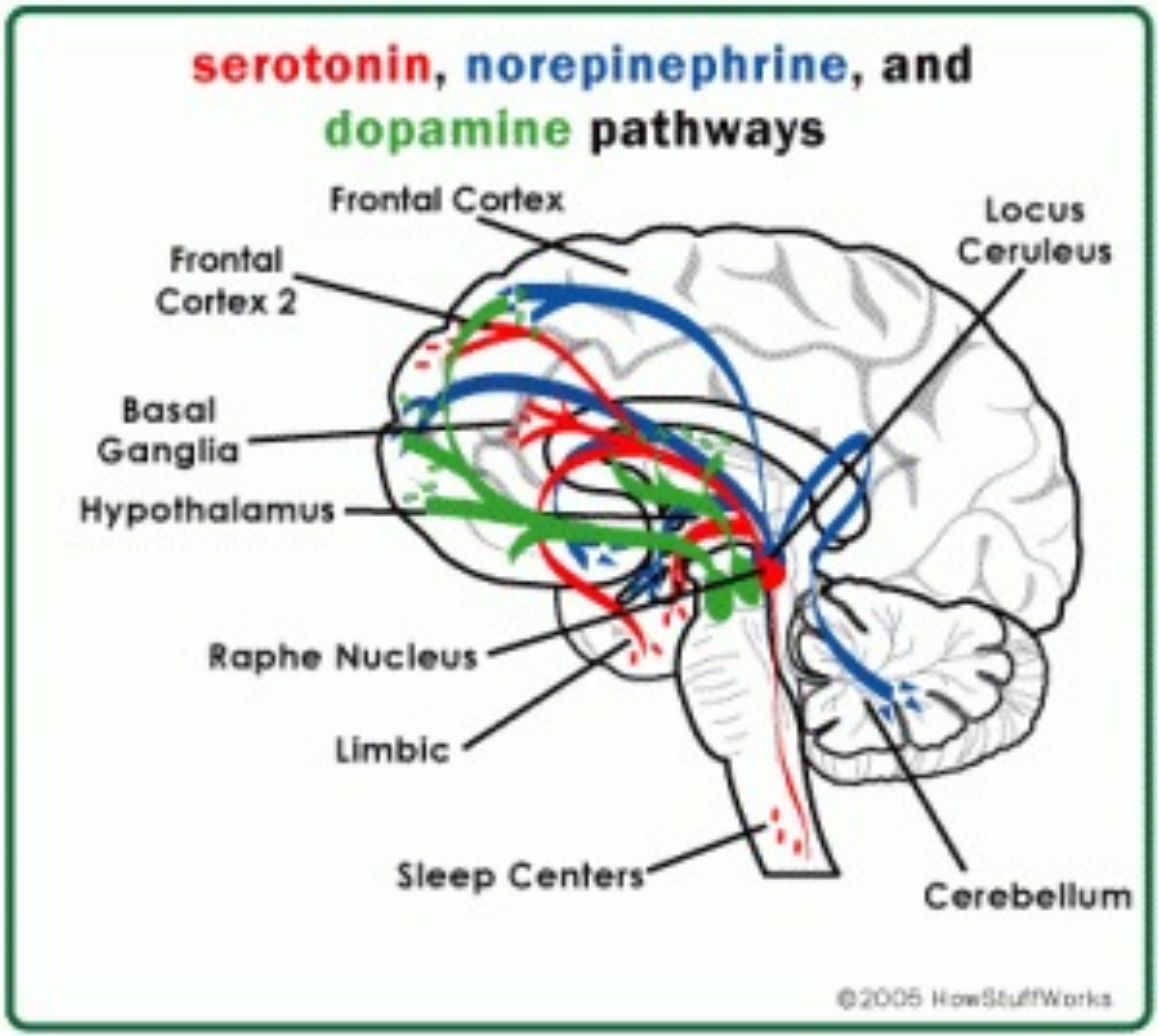
Secondary-Process Learning
Largely Upper Limbic

Top-down
Conditioned
Responses

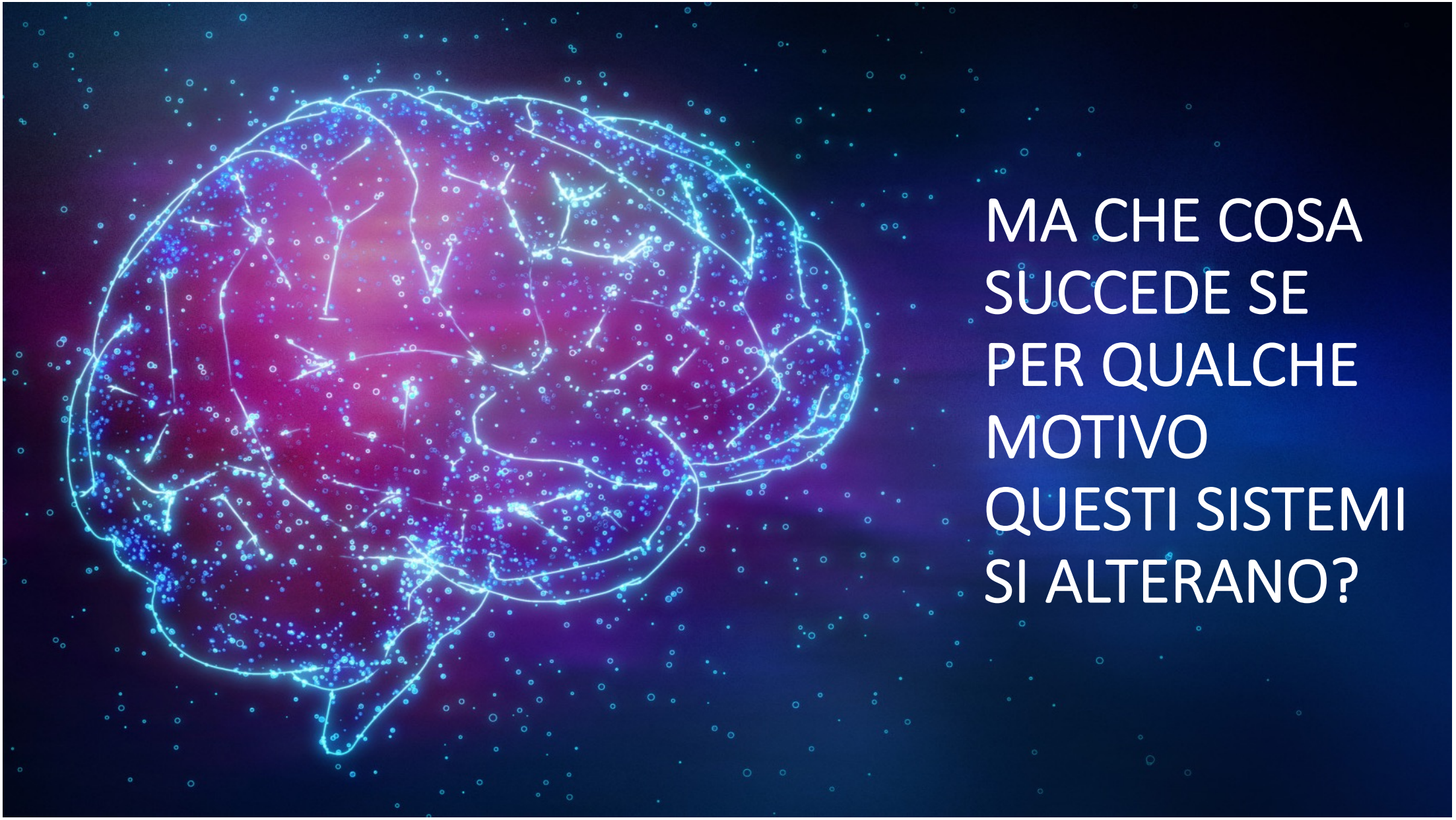
Bottom-Up Learning
and Development

Primary-Process Emotions
Affects Deeply Subcortical

Nested BrainMind Hierarchies

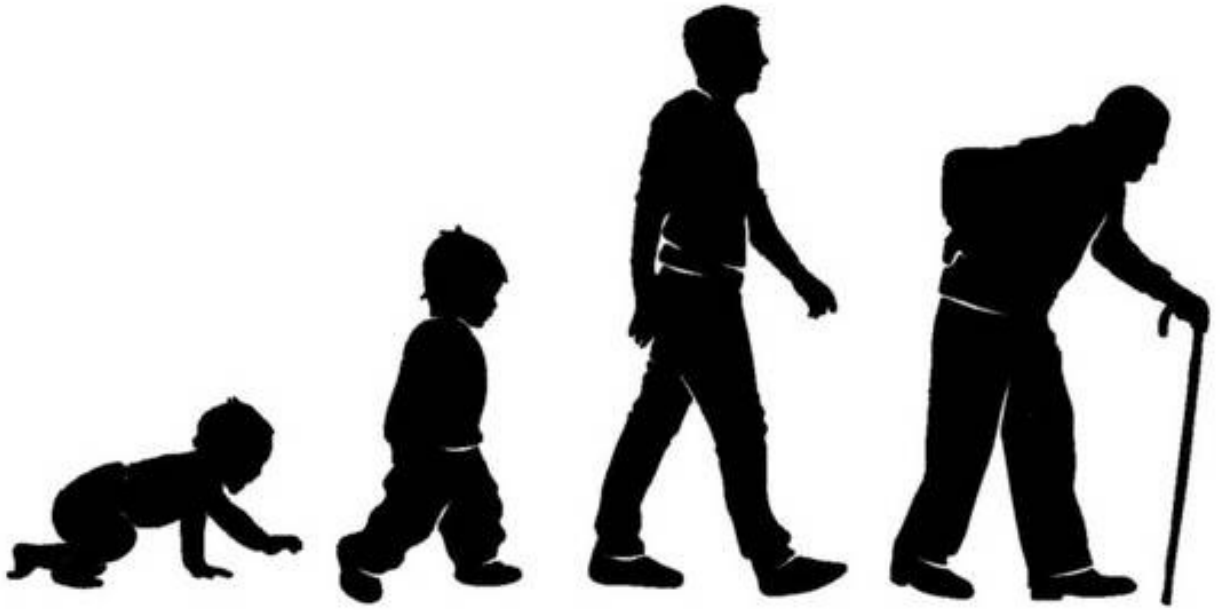

serotonin, norepinephrine, and dopamine pathways
Frontal Cortex
Frontal Cortex 2
Basal Ganglia
Hypothalamus
Raphe Nucleus
Limbic
Sleep Centers
Locus Ceruleus
Cerebellum
©2005 HowStuffWorks


• LE EMOZIONI ORIGINANO DALL'ATTIVAZIONE O DALLA DISATTIVAZIONE DI SPECIFICI CIRCUITI DI NEURONI NEL CERVELLO
• LE EMOZIONI INFLUENZANO I NOSTRI COMPORTAMENTI


MA CHE COSA
SUCCEDE SE
PER QUALCHE
MOTIVO
QUESTI SISTEMI
SI ALTERANO?

Basic Emotional System (see Panksepp 1998a)	Emergent Emotions	Emotional Disorders
SEEKING (+ & –)	Interest Frustration Craving	Obsessive Compulsive Paranoid Schizophrenia Addictive Personalities
RAGE (– & +)	Anger Irritability Contempt Hatred	Aggression Psychopathic tendencies Personality Disorders
FEAR (–)	Simple anxiety Worry Psychic trauma	Generalized Anxiety Disorders Phobias PTSD variants
PANIC (–)	Separation distress Sadness Guilt/Shame Shyness Embarrassment	Panic Attacks Pathological Grief Depression Agoraphobia Social Phobias
PLAY (+)	Joy and glee Happy playfulness	Mania ADHD
LUST (+ & –)	Erotic feelings Jealousy	Fetishes Sexual Addictions
CARE (+)	Nurturance Love Attraction	Dependency Disorders Autistic aloofness Attachment Disorders
The SELF—a substrate for Core Consciousness (see Panksepp 1998b).	A mechanism for all Emotional Feelings	Multiple Personality Disorders?

E PIU’ A LUNGO TERMINE….



La depressione può essere un fattore di rischio per il successivo sviluppo di demenza e in alcune condizioni può essere un sintomo prodromico. E'importante riconoscerlo ed individuare un trattamento efficace.

Contribution of Depression to Cognitive Impairment and Dementia in Older Adults

Guy G. Potter, PhD, and David C. Steffens, MD, MHS

Background: The objective of this review is to provide information for clinicians regarding current research and opinions on the association of depression to conditions of cognitive impairment and dementia. We also intend to integrate this current research and thinking into strategies for the assessment and treatment of depression in the context of cognitive impairment.

Review Summary: Depression is highly prevalent in mild cognitive impairment and most dementias. It may be a risk factor for the subsequent development of dementia and in some conditions may be a prodromal symptom. It is important to detect and effectively treat depression because the comorbidity of depression and cognitive impairment is associated with greater cognitive and functional decline and higher rates of institutionalization. Depression often can be differentiated from Alzheimer disease and other dementias based on characteristics of clinical history and presentation. Screening of depression and cognitive impairment will help characterize the presence and severity of these conditions, but limitations in screening approaches may necessitate comprehensive assessment in complex cases where differential diagnosis is important to treatment planning.

Conclusion: Although depression and cognitive impairment are important issues in the treatment of older adults, there are particular risks when they occur together. Appropriate assessment and screening can help guide the clinician to appropriate and timely interventions. Pharmacologic and nonpharmacologic treatment approaches are both efficacious in reducing depression in cognitive impairment and dementia.

Key Words: depression, cognitive impairment, dementia,

Cognitive impairment is estimated to exist in 17%–36% of adults over the age of 65 in the United States[1] and in many cases evolves to dementia.[2] Significant depressive symptoms are present in 15%–30% of older adults and are often comorbid with cognitive impairment and dementia.[3,4] Depression co-occurring with cognitive impairment or dementia has an additive effect on adverse outcomes for physical health, functional status, and mortality.[5] Debate continues about whether depression in either early or late life is a risk factor for dementia[6–8] or whether it is more accurately characterized as a prodrome of this condition.[9,10] Complicating this debate is the fact that depression among older adults often causes cognitive impairment in its own right.[11,12] Given that the personal and public costs of both depression and cognitive impairment are likely to increase along with the growing proportion of aged individuals in populations of the United States and other developed countries, it is important for clinicians to develop skills to detect and treat cognitive impairment and depression whether they occur separately or together.

Depression co-occurring with cognitive impairment or dementia has an additive effect on adverse outcomes for physical health, functional status, and mortality.

Is Depression a Risk Factor for Dementia or Cognitive Decline?

A Review

A.F. Jorm

NHMRC Psychiatric Epidemiology Research Centre, Australian National University, Canberra, Australia

Key Words
Depression · Dementia · Alzheimer's disease · Vascular dementia · Cognitive decline · Risk factors

Abstract
Background: It is generally accepted that depression can be associated with significant cognitive deficits and that depression can be comorbid with dementia. ***Objective:*** This review seeks to go further and ask whether depression earlier in life can be a risk factor for subsequent dementia or for cognitive decline. ***Methods:*** A review was made of the epidemiological evidence from case-control and prospective studies that depression is a risk factor. The literature was also reviewed in relation to six for hypotheses 1 and 2. The other hypotheses have limited support, but warrant further research. ***Conclusion:*** There is sufficient evidence to take seriously the possibility that depression is a risk factor for dementia and cognitive decline. Further work is needed to examine depression as a prodrome of vascular dementia, depression as an early reaction to perceived cognitive decline, the effects of depression on the threshold for manifesting dementia, and depression as a source of hippocampal damage through a glucocorticoid cascade.

Introduction

CI SONO SUFFICIENTI EVIDENZE DA CONSIDERARE LA POSSIBILITA' CHE LA DEPRESSIONE SIA UN FATTORE DI RISCHIO DELLA DEMENZA E DEL DECLINO COGNITIVO!!

Does Anxiety Affect Risk of Dementia? Findings From the Caerphilly Prospective Study

JOHN GALLACHER, BSc, PhD, ANTHONY BAYER, MB BCh, MARK FISH, MB BCh, JANET PICKERING, BSc, MSc, SOFIA PEDRO, BSc, MSc, FRANK DUNSTAN, MA, PhD, SHAH EBRAHIM, MB BCh, MD, AND YOAV BEN-SHLOMO, MB BCh, PhD

Objective: To examine the association of anxiety with incident dementia and cognitive impairment not dementia (CIND). **Methods:** We conducted a prospective study of men aged 48 to 67 years at baseline anxiety assessment; we measured cognition 17 years later. We studied 1481 men who were either eligible for examination or were known to have dementia. Trait Anxiety was assessed using the Spielberger State Trait Anxiety Inventory. Psychological distress was assessed using the 30-item general health questionnaire. Cognitive screening was followed by a clinical examination. Medical notes and death certificates of those not seen were also examined. Outcomes were CIND and Diagnostic and Statistical Manual of Mental Disorders, 4th Edition (DSM-IV) dementia. **Results:** Of 1160 men who were cognitively screened, 174 cases of CIND and 69 cases of dementia were identified. A further 21 cases of dementia were identified from medical records. After adjustment for age, vascular risk factors and premorbid cognitive function associations with higher anxiety (31st–95th centile) were for CIND odds ratio (OR) 2.31 (95% Confidence Interval (CI) = 1.20–4.44) and for dementia OR 2.37 (95% CI = 0.98–5.71). These associations were slightly stronger for nonvascular (OR = 2.45; 95% CI = 1.28–4.68) than for vascular impairment (OR = 1.94; 95% CI = 0.77–4.89). Analyses of change in cognitive performance, assessed by the Cambridge Cognitive Examination of the Elderly subscales found some evidence for decline in learning memory with higher anxiety score ($b_{age\ adj}$ = −0.291 (−0.551, −0.032), but not for any other subscale. **Conclusions:** Anxiety is a risk factor for CIND and dementia. The extent to which the association is independent of depression and whether or not it is causal requires further study. **Key words:** anxiety, dementia, CIND, depression, cognitive decline, vascular dementia.

Psychosomatic Medicine 71:659–666 (2009)
0033-3174/09/7106-0659

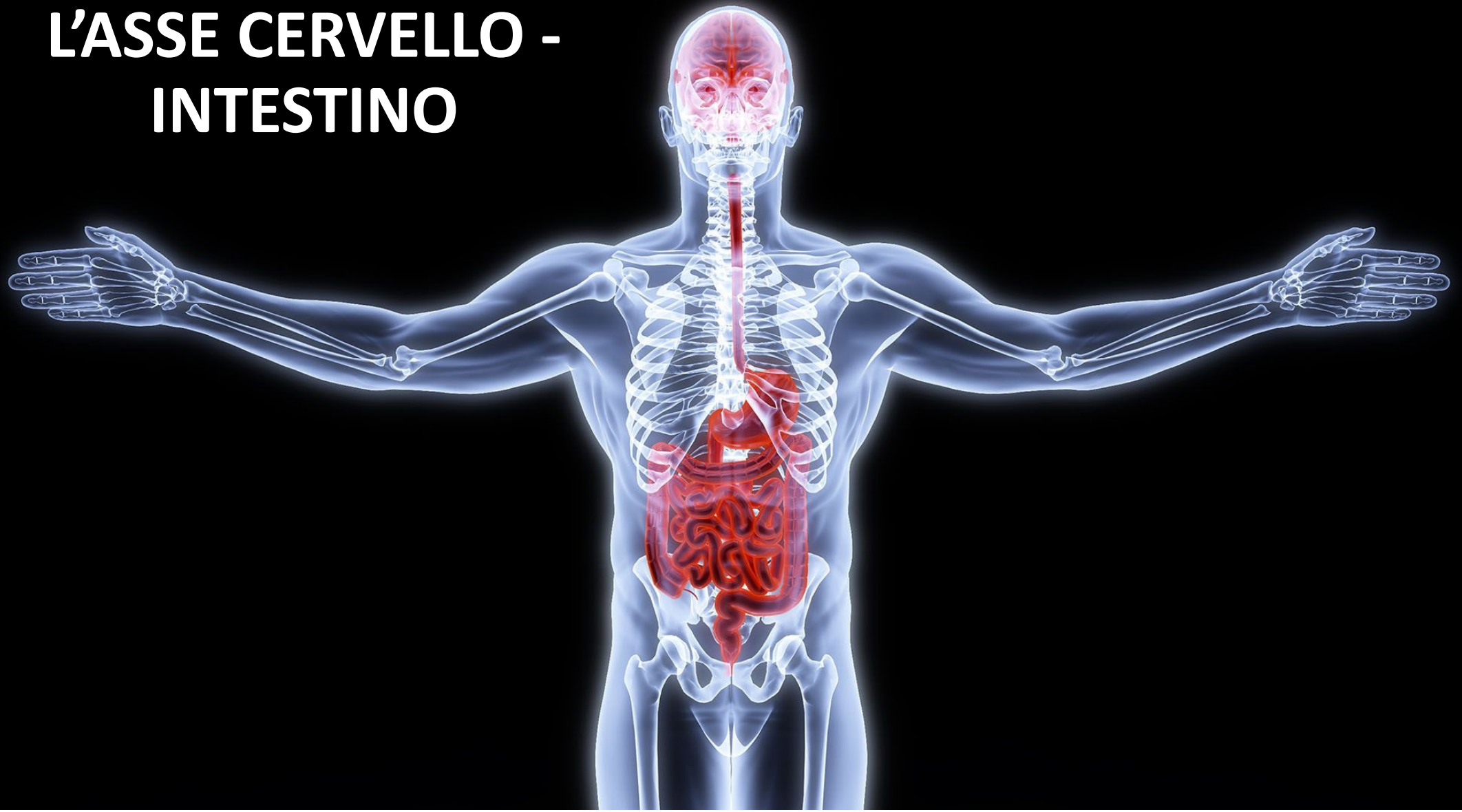
È stata valutata l'eventuale presenza di ansia in una popolazione tra i 48 e i 67 anni. A distanza di 17 anni è stata valutata l'associazione tra la presenza di ansia e l'incidenza di deficit cognitivo o di demenza.

ANSIA E' UN FATTORE DI RISCHIO PER LA DEMENZA

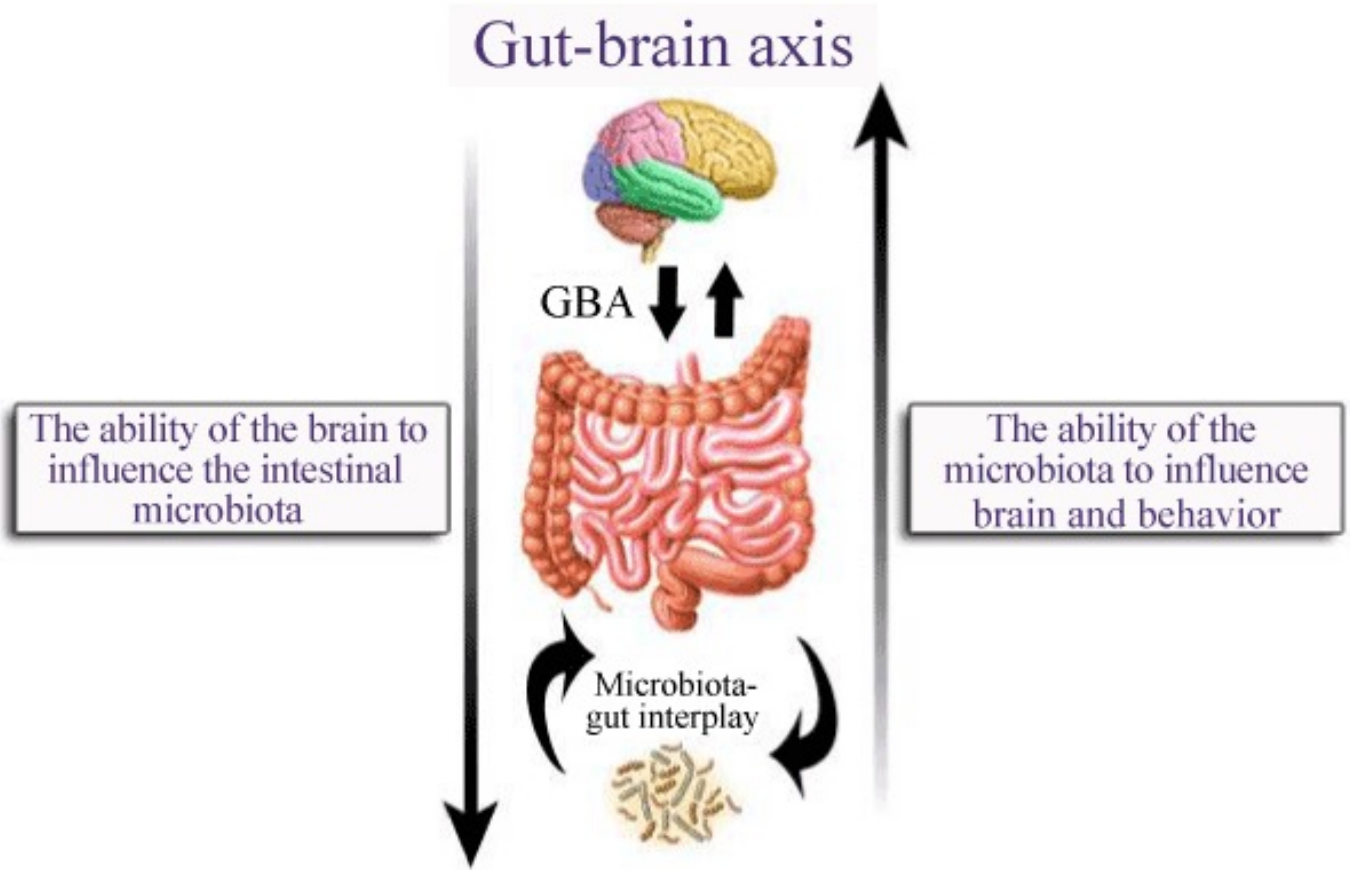
MA PERCHE' STIAMO PARLANDO DI EMOZIONI?

EMOZIONI DI TESTA O EMOZIONI DI PANCIA?

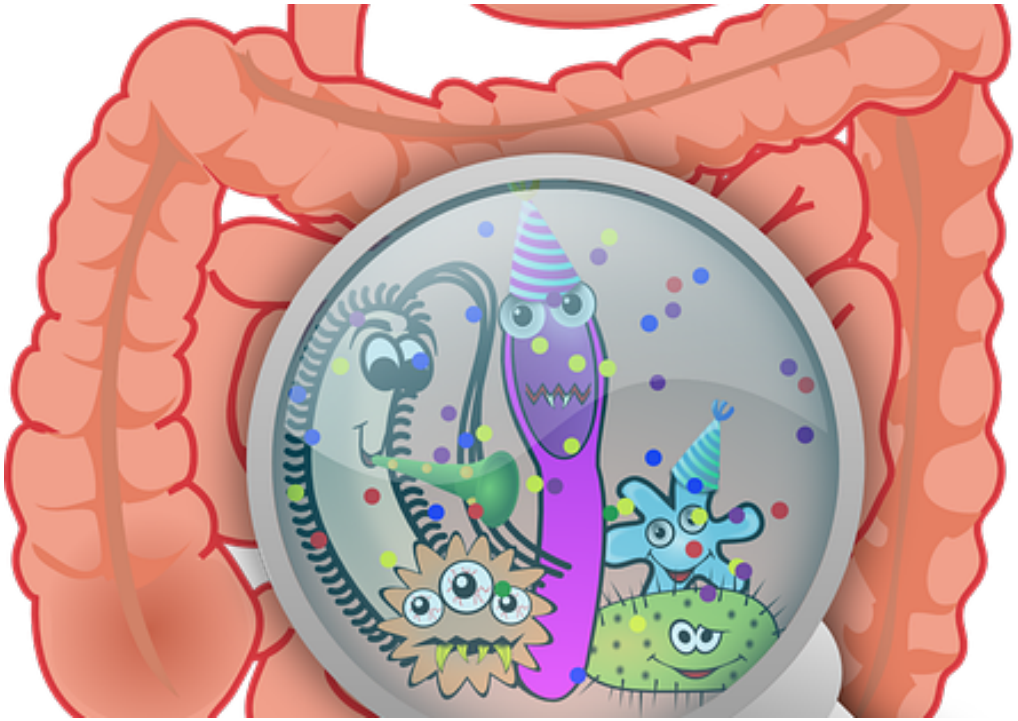
L’ASSE CERVELLO - INTESTINO



L’ASSE CERVELLO-INTESTINO



MA CHE COS'E' IL MICROBIOTA?



IL MICROBIOTA E' L'INSIEME DEI GERMI PRESENTI FISIOLOGICAMENTE NELL'INTESTINO

SPECIAL

See all specials

HUMAN MICROBIOTA

The human body is colonized by a vast number of microbes, collectively referred to as the human microbiota. The link between these microbes and our health is the focus of a growing number of research initiatives, and new insights are emerging rapidly, some of which we are proud to present in this special collection.

Credit: Joana Ricou / Steven H. Lee / Studio Graphiko

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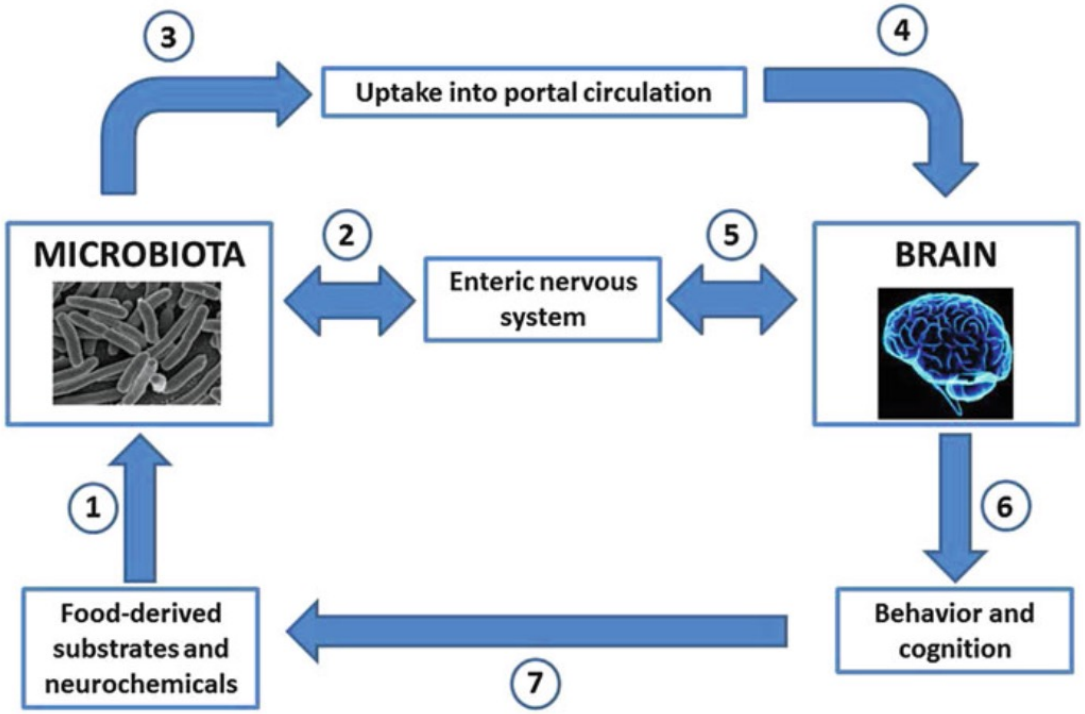
Computational Molecular Evolution
08 May 2017 — 19 May 2017
Hinxton, Cambridge, United Kingdom

Biopharma Innovation Cup 2017
18 June 2017 — 23 June 2017
Germany

IL MICROBIOTA - 1

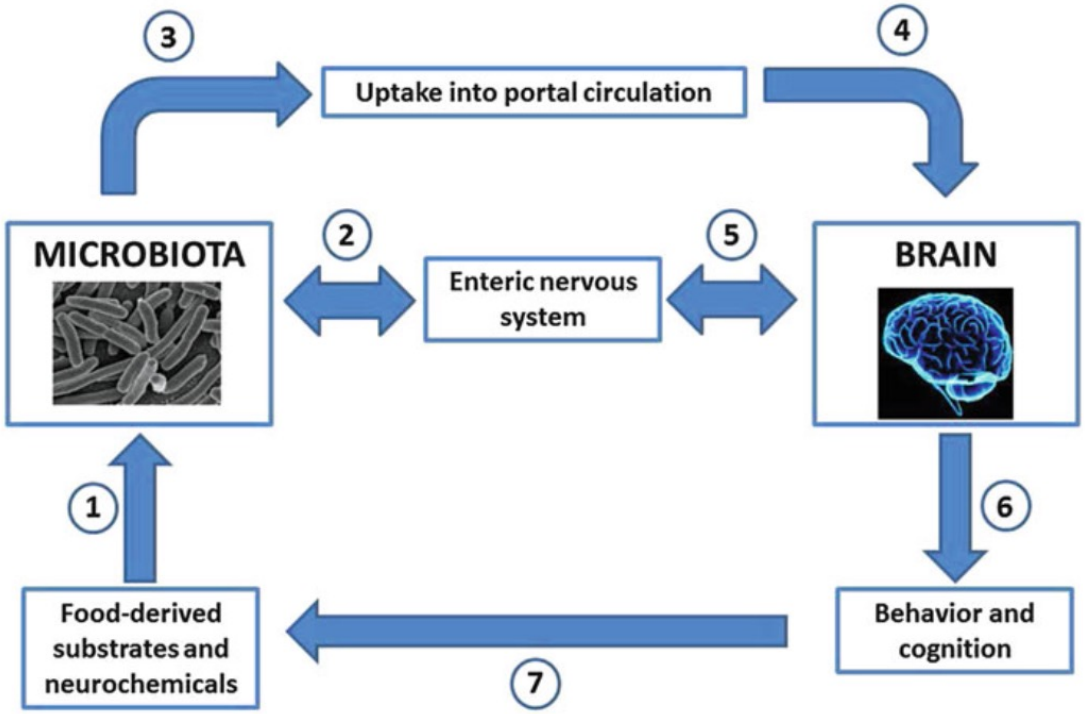


Tratto da: “Microbial Endocrinology: An Ongoing Personal Journey”
Mark Lyte

1.

IL MICROBIOTA PRESENTE NELL’INTERSTINO E’ IN GRADO DI PRODURRE MOLECOLE NEUROCHIMICHE UGUALI A QUELLE UTILIZZATE DAL NOSTRO SISTEMA NERVOSO PER COMUNICARE, SIA A PARTIRE DAL CIBO INGERITO DALL’OSPITE SIA DI RISPONDERE ALLE COMPONENTI NEUROATTIVE DEL CIBO STESSO, CHE DI RISPONDERE ALLE MOLECOLE NEUROCHIMICHE SECRETE DALL’INTESTINO STESSO.

IL MICROBIOTA - 2

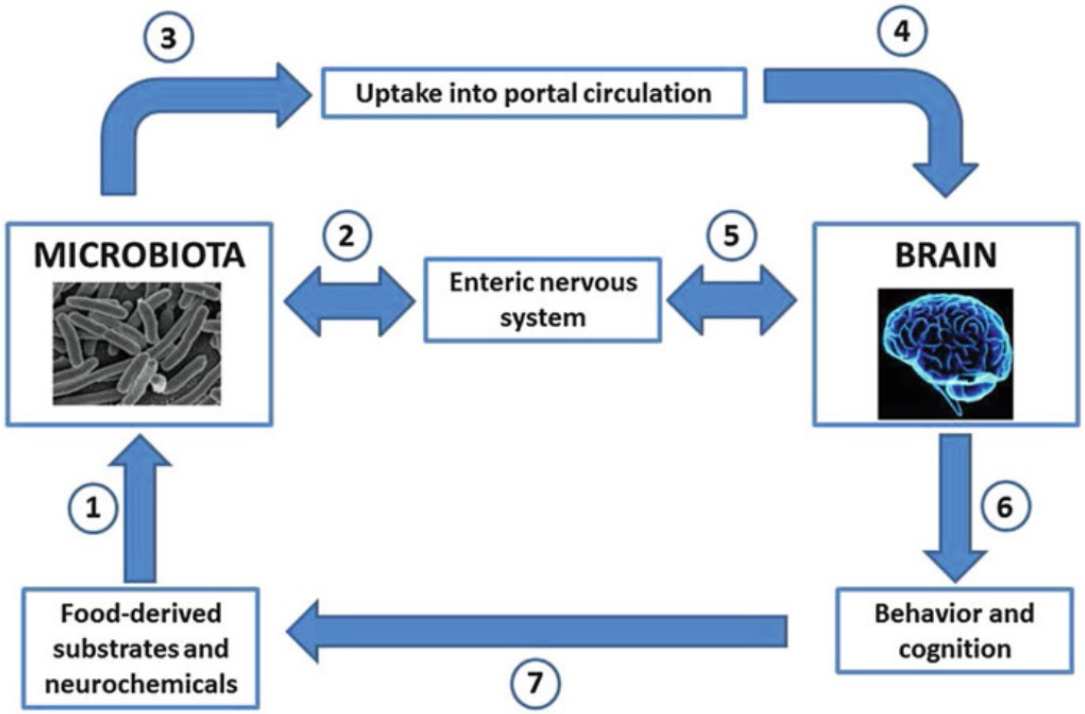


Tratto da: "Microbial Endocrinology: An Ongoing Personal Journey"
Mark Lyte

2.

LE SOSTANZE NEUROCHIMICHE PRODOTTE DAL MICROBIOTA ALL'INTERNO DELL'INTESTINO AGISCONO SUL OSPITE ATTRAVERSO DUE VIE:

O VENGONO TRASFERITE DIRETTAMENTE ALL'INTERNO DEL CIRCOLO PORTALE ATTRAVERSO CUI VENGONO PORTATI AD AGIRE DIRETTAMENTE SUL CERVELLO, O AGISCONO DIRETTAMENTE SUI RECETTORI DEL SISTEMA NERVOSO CENTRALE CHE SI TROVANO ALL'INTERNO DEL LUME INTESTINALE.



Tratto da: "Microbial Endocrinology: An Ongoing Personal Journey"
Mark Lyte

3.

I NEUROTRASMETTITORI PRODOTTI DAL MICROBIOTA AGISCONO A LIVELLO DEL SISTEMA NERVOSO CENTRALE E POSSONO PRODURRE DELLE ALTERAZIONI DEL COMPORTAMENTO O DELLA COGNITIVITA' COME ANCHE INFLUENZARE LE PREFERENZE ALIMENTARI E L' APPETITO

Lyte M (2010b) The microbial organ in the gut as a driver of homeostasis and disease. Med Hypotheses 74:634–638
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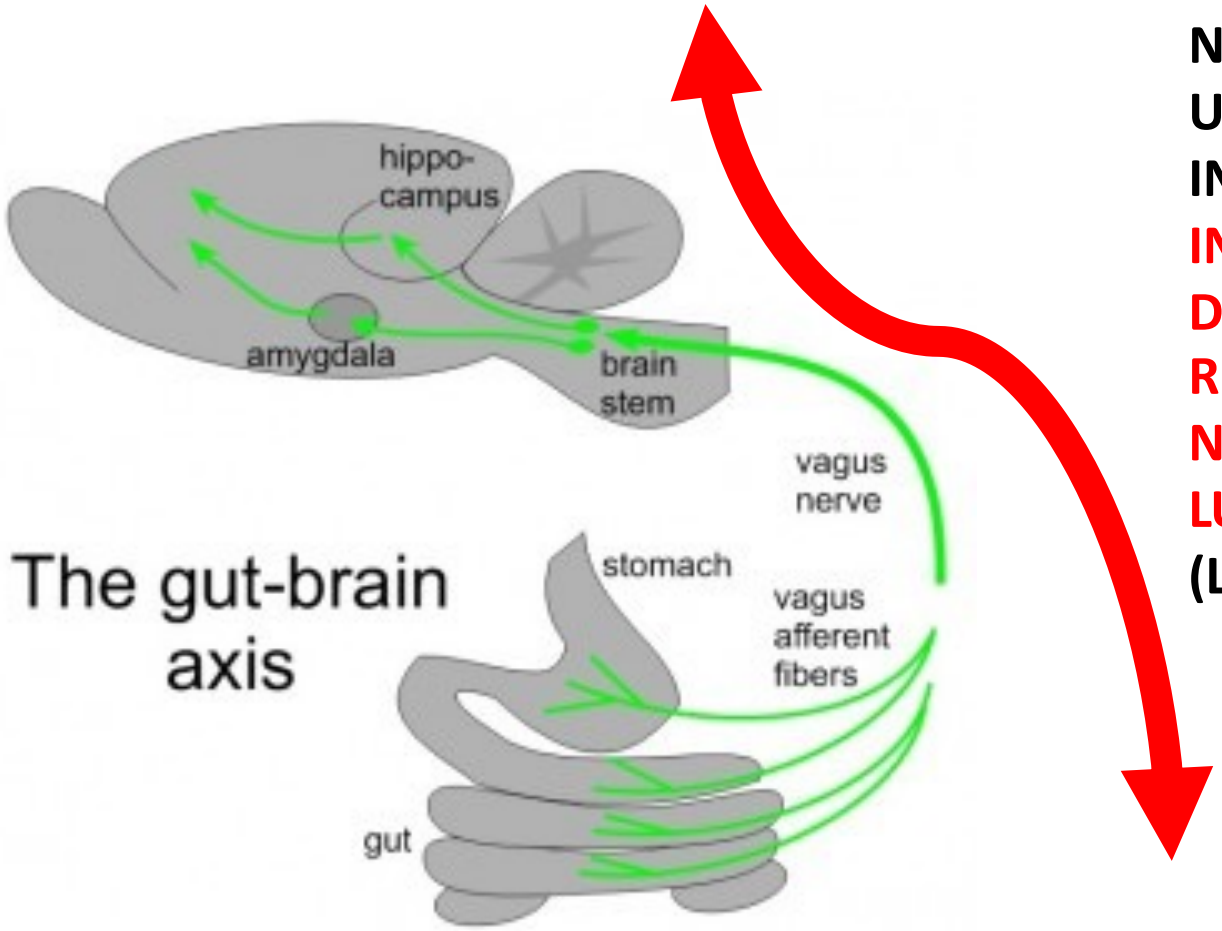
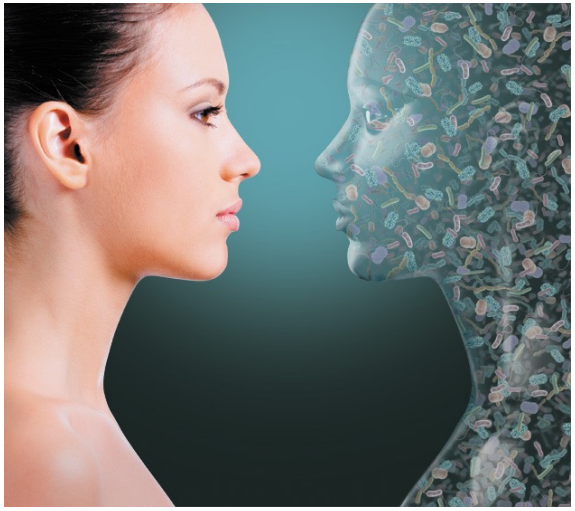
Norris V, Molina F, Gewirtz AT (2013) Hypothesis: bacteria control host appetites. J Bacteriol 195:411–416
Alcock J, Maley CC, Aktipis CA (2014) Is eating behavior manipulated by the gastrointestinal microbiota? Evolutionary pressures and potential mechanisms. Bioessays 36:940–949

E’ DA MOLTO TEMPO NOTO CHE CIRCA IL 50% DELLA DOPAMINA E LA QUASI TOTALITA’ DELLA SEROTONINA ORIGINANO DALL’INTESTINO, DOVE QUESTE DUE MOLECOLE REGOLANO IL SENSO DI SAZIETA’ E DI FAME ED I PROCESSI DIGESTIVI

TUTTAVIA SOLO RECENTEMENTE E’ STATO RESO NOTO IL RUOLO DELLA FLORA BATTERICA INTESTINALE NELLA LORO SECREZIONE



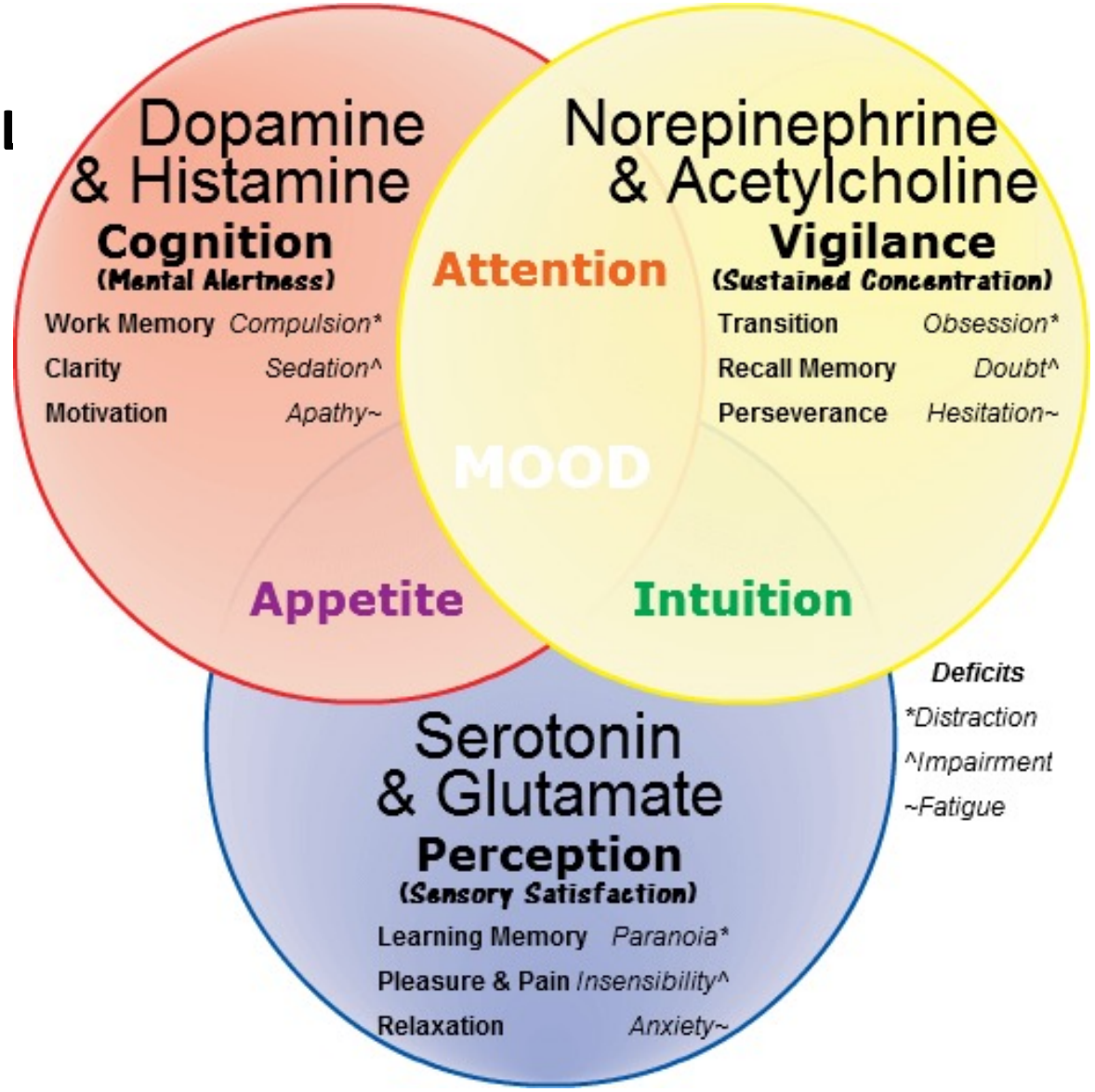
NON SI TRATTA DI UN PERCORSO UNIVOCO INTESTINO-CERVELLO, INFATTI IL CERVELLO PUO' INFLUENZARE LA COMPOSIZIONE DEL MICROBIOTA ATTRAVERSO IL RILASCIO DI SOSTANZE NEUROCHIMICHE ALL'INTERNO DEL LUME INTESTINALE
(Lyte, 2013)



I MICROORGANISMI PRESENTI NEI NOSTRO INTESTINO SECERNONO LE STESSE SOSTANZE CHE I NEURONI UTLIZZANO PER COMUNICARE FRA LORO E CHE REGOLANO L'UMORE:

- DOPAMINA
- SEROTONINA
- ACIDO GAMMA AMINOBUTIRRICO (GABA)

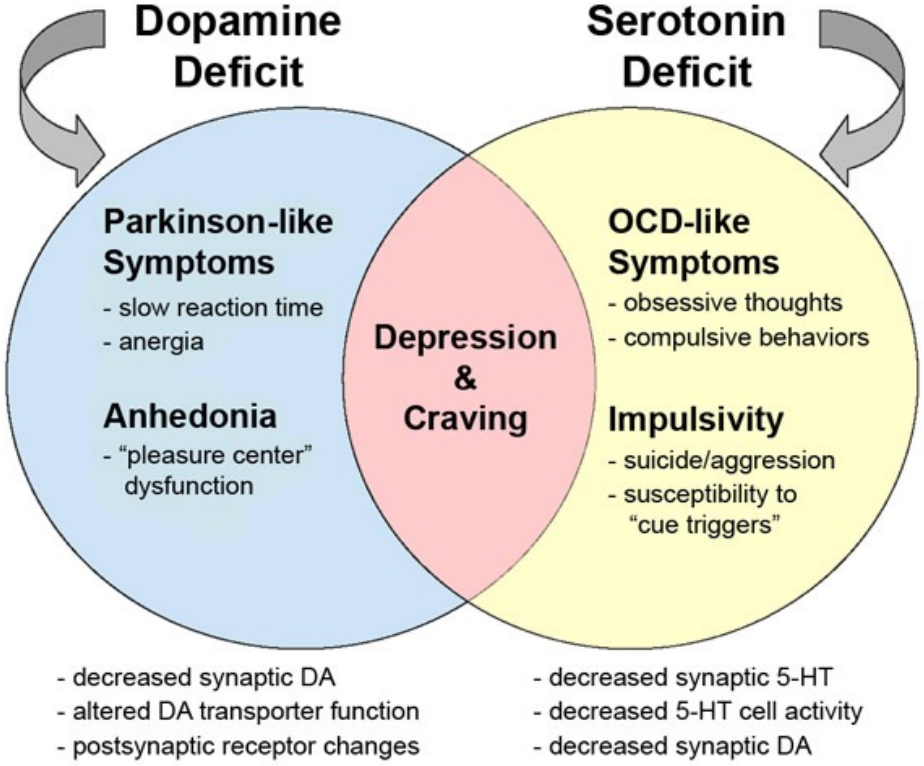
FARMACI COME IL VALIUM E LO XANAX AGISCONO SUI RECETTORI GABA



NEI DISTURBI INFIAMMATORI INTESTINALI:

INDIVIDUI CON ALTERATA BIODIVERSITA' DELLA FLORA BATTERICA INTESTINALE HANNO EVIDENZIATO UN'ALTERAZIONE DELLE FUNZIONI EMOTIVE CHE SI TRADUCEVANO IN UN AUMENTO DI DEPRESSIONE ED ANSIA

Blanchard EB, Scharff L, Schwarz SP, Suls JM, Barlow DH (1990) The role of anxiety and depression in the irritable bowel syndrome. Behav Res Ther 28: 401–405.



DIVERSI SONO I FATTORI CHE INFLUENZANO IL MICROBIOTA E UNO DEI PRINCIPALI E' LA DIETA!

MICROBIAL ECOLOGY in Health and Disease

COACTION PUBLISHING

ENGIHR SUPPLEMENT

Contribution of diet to the composition of the human gut microbiota

Daniela Graf[1], Raffaella Di Cagno[2], Frida Fåk[3], Harry J. Flint[4], Margareta Nyman[3], Maria Saarela[5] and Bernhard Watzl[1]*

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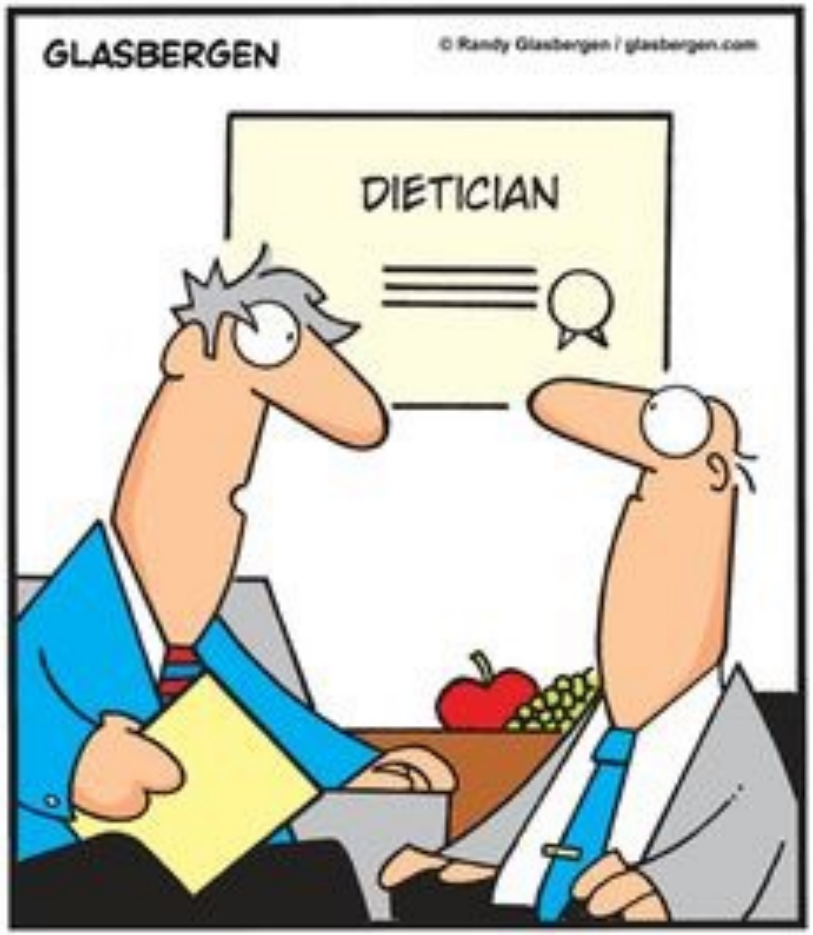
MA PER QUESTO ASPETTO VI RIMANDO AI COLLEGHI!



"Tobacco is a green, leafy plant...but a cigarette does not count as salad!"

CHE COSA SI PUO' FARE DAL PUNTO DI VISTA PSICOLOGICO?

- **SI POSSONO INDAGARE LE EMOZIONI FONDAMENTALI DELLA PERSONA, MISURARE I DISTURBI DELL'UMORE E I LIVELLI DI ANSIA.**
- **E' POSSIBILE FORNIRE AL CLINICO GLI ELEMENTI NEUROCOGNITIVI PER PERMETTERGLI DI INTERVENIRE CON APPOSITI STRUMENTI NUTRIZIONALI E FISIOLOGICI PRIMA CHE LA MALATTIA SI SVILUPPI**
- **SI PUO'RIVALUTARE NEL TEMPO IL PAZIENTE EVIDENZIANDO I BENEFICI DEL TRATTAMENTO E PERMETTENDOGLI COSI DI AGIRE Più SULLA PREVENZIONE CHE SULLA CURA DELLE MALATTIE PSICHICHE**
- **SI POSSONO EVIDENZIARE I PRIMI SCOSTAMENTI DELLA COGNITIVITA' DALLA NORMA EFFETTUANDO L'UNICA PREVENZIONE ATTUALMENTE POSSIBILE PER LO SVILUPPO DELLE DEMENZE**