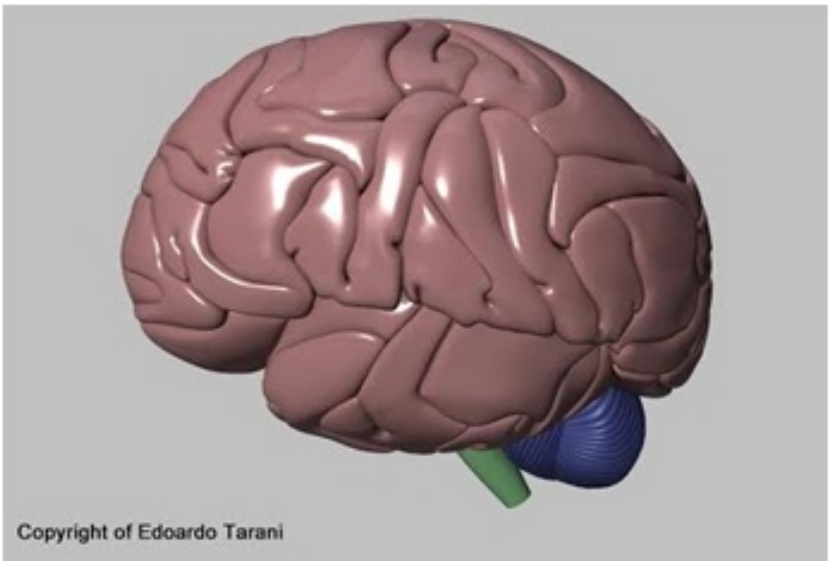
Centro di Neuroscienze Comportamentali
Psicoterapia, Psicosessuologia, Neuropsicologia e Psicologia Forense
Roma



Un “mondo” nella scatola cranica

Uno sguardo nel cervello

A cura di

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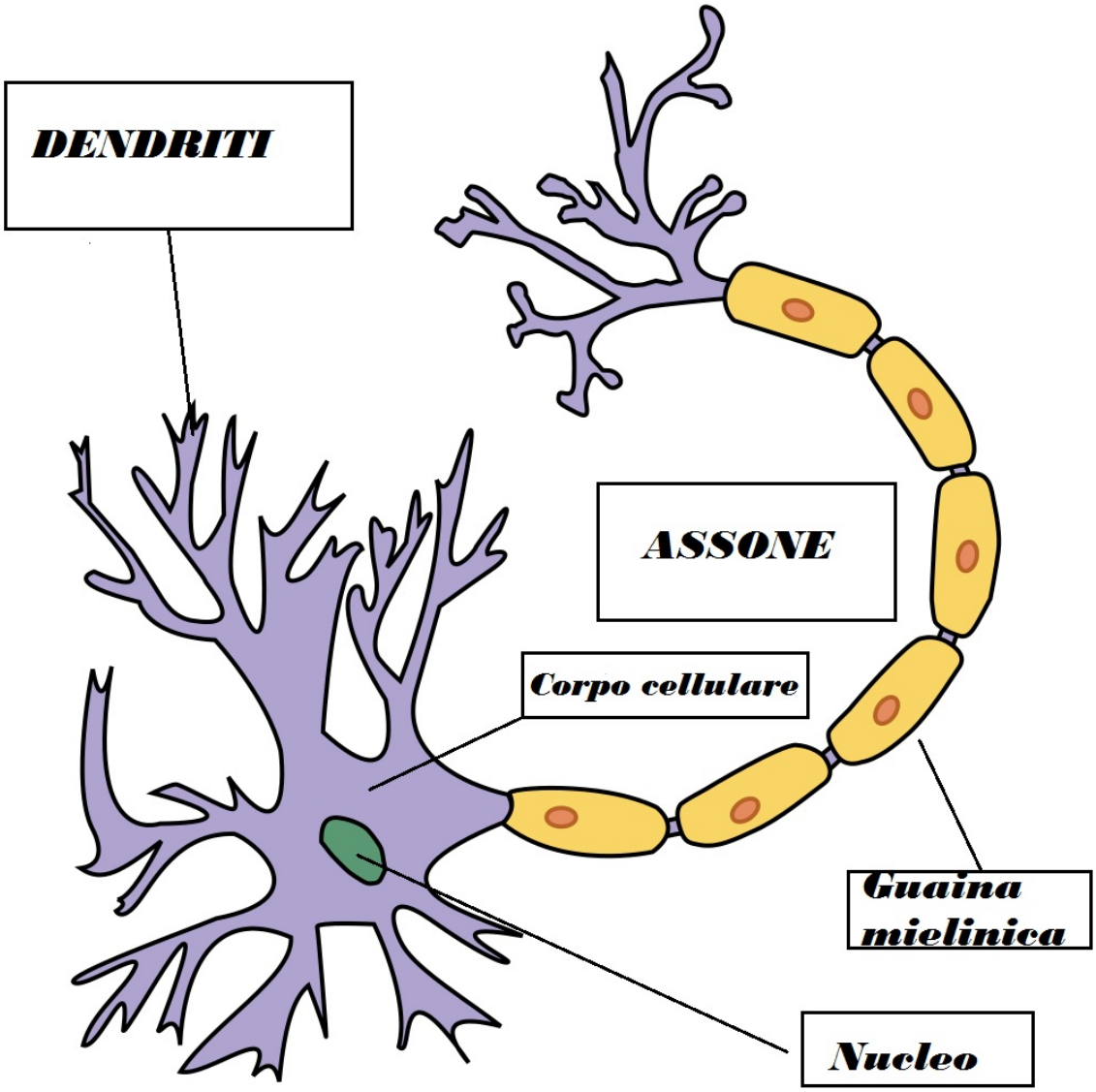
Indice

Introduzione

E’ dal nostro cervello, insieme di almeno 100 miliardi di cellule nervose (neuroni) interconnesse, che hanno origine bisogni (fame/sete, sonno/veglia, accoppiamento/procreazione), percezioni, emozioni, pensieri e azioni. Detto organo è avvolto, come un mantello, dalla corteccia cerebrale (strato di sostanza grigia).

Nel corso dell’evoluzione, questo manto si è accartocciato permettendo di aumentare la superficie cerebrale senza alterarne il volume, grazie alle circonvoluzioni o giri (creste esterne) e ai solchi (pieghe interne). Questi ripiegamenti (giri e solchi) hanno permesso di sviluppare le nostre capacità cognitive (funzioni superiori). Il complesso strato di sostanza grigia, composto da corpi di neuroni,

si differenzia dalla sostanza bianca formata dagli assoni mielinizzati (prolungamenti in uscita), riuniti in fasci.



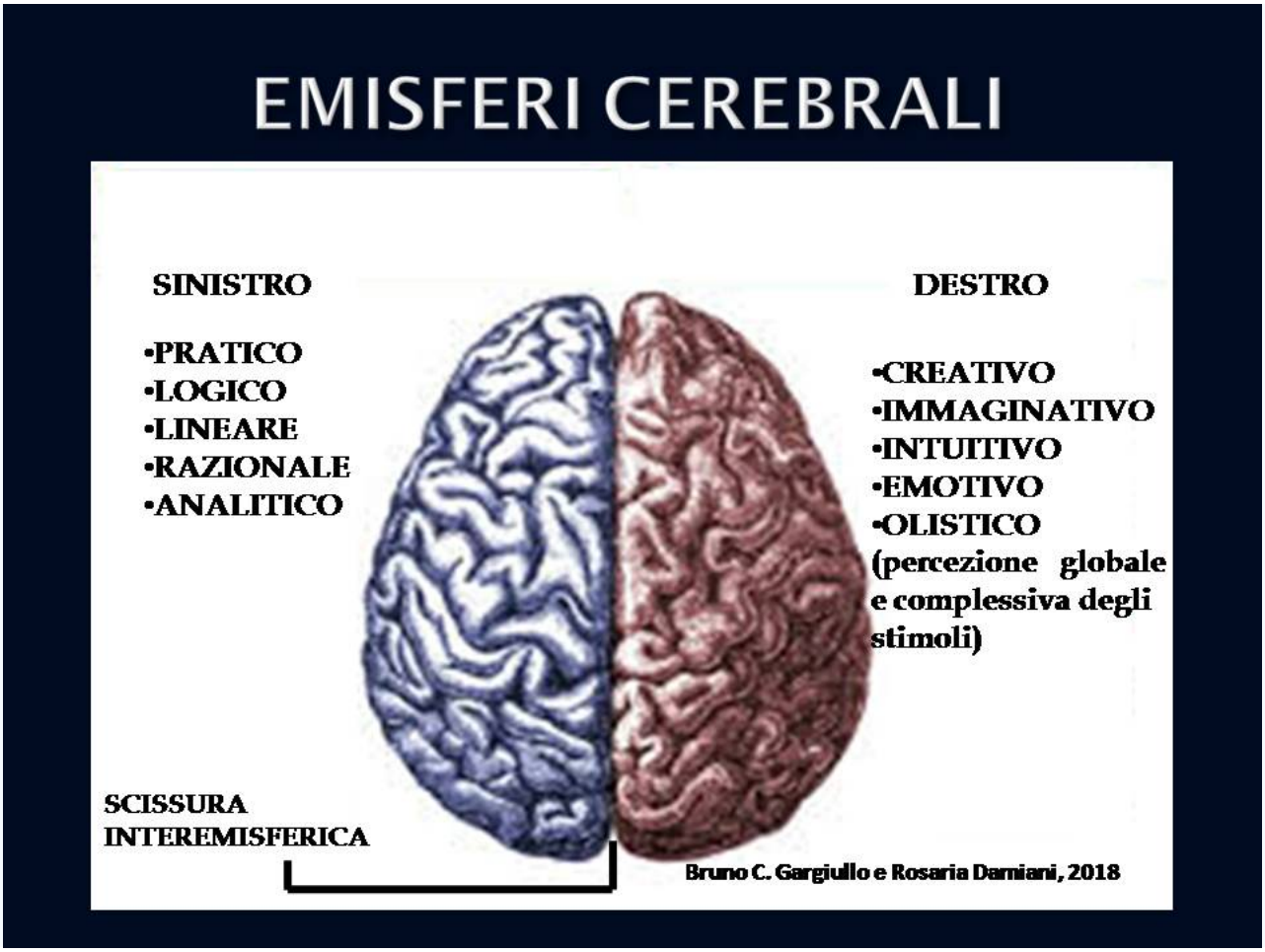
L'essenza della nostra identità è racchiusa nella peculiarità di queste connessioni cerebrali e nell'interazione tra organismo ed ambiente.

Sembra incredibile che da questo organo, che pesa circa 1400 grammi, il 2% in media del nostro peso corporeo, possano dipendere «*… le gioie, i piaceri, le risa e gli svaghi, e i dispiaceri e le angosce, lo scoramento e le afflizioni*» (Ippocrate – La malattia sacra – "De morbo sacro" – a cura di Roselli A., Marsilio Editore, 1996).

Vale la pena rammentare che il nostro cervello è continuamente esposto al flusso delle stimolazioni ambientali che influenzano, ad esempio, le nostre capacità di percepire gli stimoli, di compiere movimenti, di pensare, di apprendere, di ricordare e di pianificare strategie comportamentali.

Nel corso della nostra vita, questo instancabile organo, nonostante sia contenuto all'interno di una scatola cranica ed avvolto da tre strati protettivi (partendo dall'esterno verso l'interno, dura madre, aracnoide e pia madre), si modifica continuamente (sia strutturalmente che funzionalmente) mediante un processo definito "neuroplasticità" (Gogtay N., Thompson P.M., 2010).

Emisferi cerebrali



Il cervello umano è costituito da due emisferi (sinistro e destro), messi in connessione tra loro dal *corpo calloso*. Le forme di detti emisferi cerebrali derivano dal ripiegamento del sottile mantello (*corteccia cerebrale*) che va ad aumentare la superficie neurale, e a creare così, giri e solchi.
I due emisferi, che comunicano tra loro grazie al corpo calloso, svolgono le seguenti funzioni:

- il **sinistro** è il "centro del pensiero e della risoluzione dei problemi nonchè il centro dominante del discorso e del linguaggio". "Interpreta il nostro comportamento e le nostre risposte, sia

cognitive che emotive, agli stimoli ambientali" (funzione di "interprete" secondo il neuroscienziato M. Gazzaniga, "Come il cervello decodifica il mondo" DR Editore, 2011);

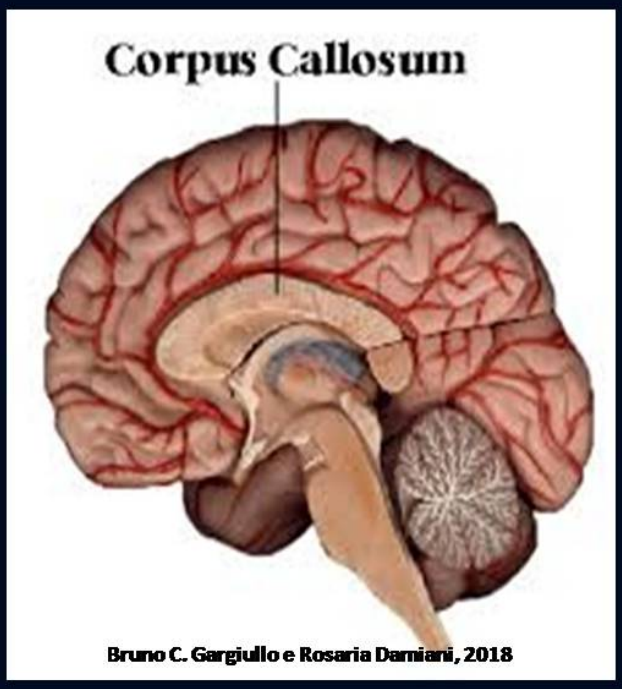
- il **destro** "è, invece, specializzato in alcuni compiti di ricognizione spaziale, quali la determinazione e il riconoscimento tattile di taluni oggetti" (elaborazione degli stimoli visivi e visuo-spaziali) ed è associato al riconoscimento dei volti e all'attribuzione del significato emotivo delle espressioni facciali.

"*In caso di stati emotivi di disturbo, che si tratti di ansia o depressione, euforia o panico, è l'interprete – quindi l'emisfero sinistro – che prova a comprendere le ragioni per cui l'umore o lo stato emotivo del suo possessore è cambiato, anche se i suddetti cambiamenti dipendono dall'emisfero destro. (…) Il paziente percepisce una variazione d'umore e, subito, l'interprete comincia a costruire una teoria sui motivi che hanno scatenato quel determinato disturbo e per farlo – naturalmente – deve concentrarsi sia sugli eventi dell'ambiente esterno che sui risultati dell'elaborazione intellettiva (…) ripescando nell'infanzia o nelle relazioni adulte"* (Ibidem).

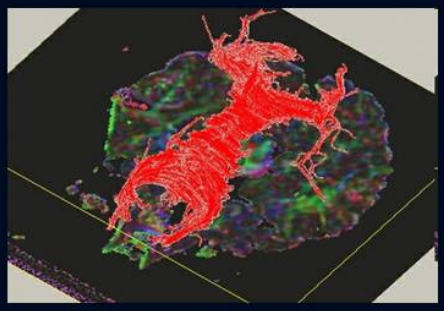
N.B.
Le attività sensoriali e motorie di un lato del corpo sono governate dall'emisfero del lato opposto (es., i movimenti della parte sinistra del corpo sono controllati dai neuroni della corteccia motoria di destra e viceversa).

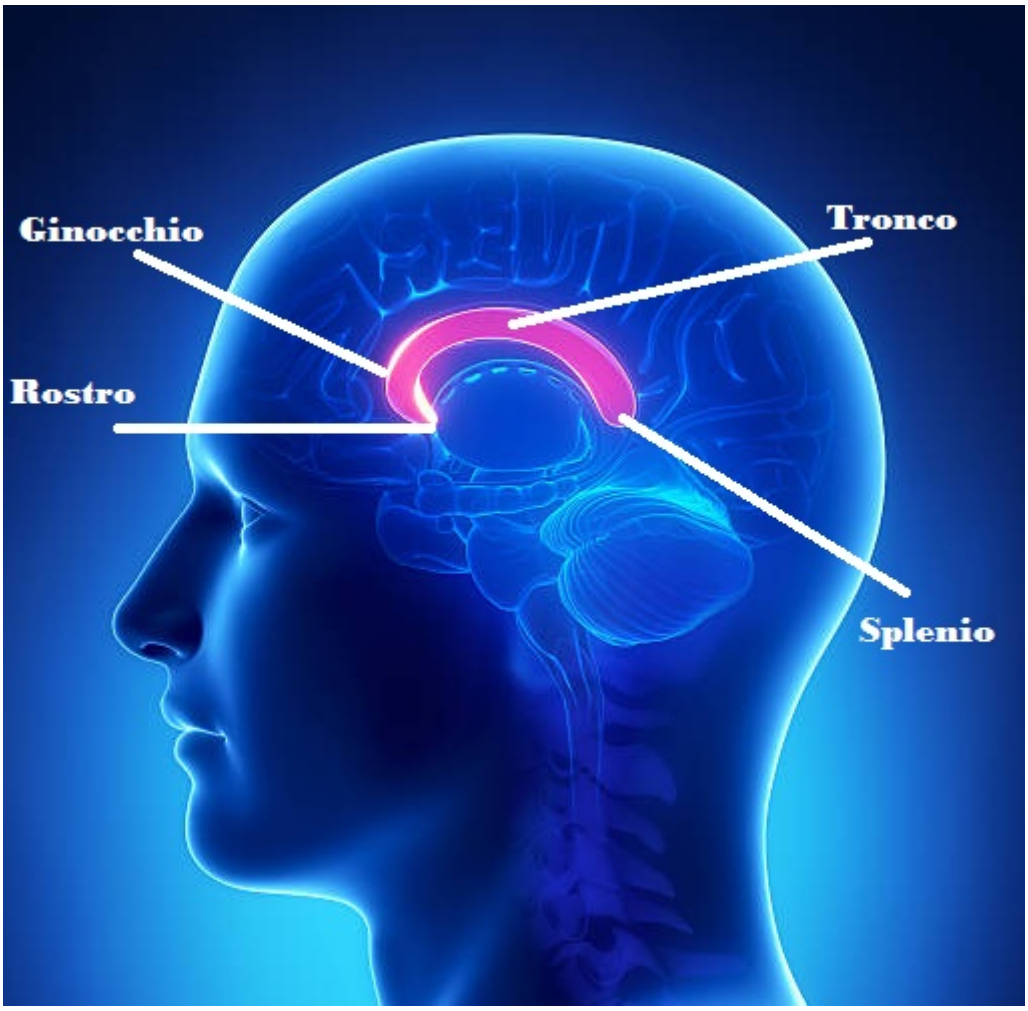
Corpo calloso

CORPO CALLOSO



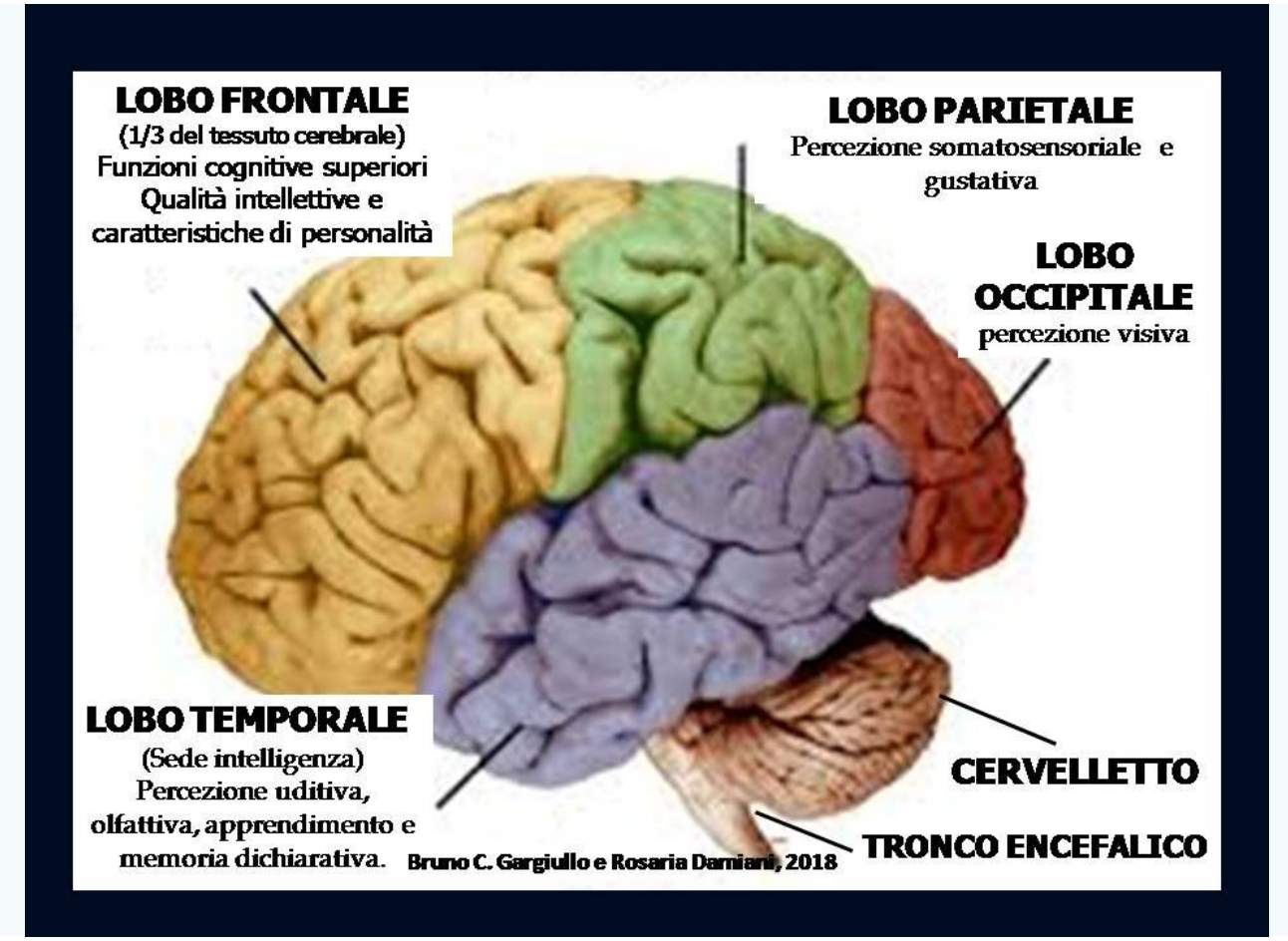
Fasci di fibre mieliniche che collegano tra loro aree corrispondenti nei due emisferi (tranne le parti anteriori dei lobi temporali)
Deficiente o scarso collegamento tra i due emisferi cerebrali, rende una persona emotivamente fredda e totalmente disinibita





Il **corpo calloso (CC),** costituito da un fascio di fibre nervose (circa 200 milioni di assoni), mette in connessione i due emisferi.
Il corpo calloso si sviluppa tra la 5° e la 18° settimana; pertanto, la diagnosi di agenesia del corpo calloso non dovrebbe essere fatta prima della 19° settimana.
L'assenza, parziale o completa, del corpo calloso (*agenesia*) può rendere una persona fredda e totalmente disinibita.
L'agenesia del CC deriva da errori genetici a carico delle molecole che guidano la crescita degli assoni; vi possono essere, inoltre, dei casi in cui il corpo calloso viene chirurgicamente reciso (*callosotomia*), per il trattamento di forme molto gravi di epilessia. La **callosotomia** non presenta serie conseguenze cognitive, ad eccezione di comportamenti bizzarri (" *come se si possedessero due cervelli separati*").

Lobi cerebrali



Il cervello, con le relative funzionalità, è suddiviso in 4 lobi:

- **lobo frontale (1/3 del tessuto cerebrale)** deputato alla formulazione, all'esecuzione e al controllo di azioni, comportamenti e processi cognitivi (*funzioni esecutive*). Disfunzioni del lobo frontale provocano impulsività, discontrollo del comportamento e dell'umore, disinibizione, rigidità cognitiva, scarse abilità di pianificazione, con mancanza di consapevolezza di tali alterazioni.

- **lobo temporale (sede dell'intelligenza)** coinvolto nella percezione uditiva, olfattiva, nonchè nell'apprendimento e nella memoria dichiarativa. Al suo interno si trova il *sistema limbico (archivio emozionale)*, un circuito di strutture profonde, connesso anche al lobo frontale, deputato all'elaborazione delle emozioni e dei processi di memoria.
  La disfunzionalità del lobo temporale può produrre disturbi linguistici e mnesici (es., disturbi del neurosviluppo), nonchè disregolazione emozionale, con particolare variabilità data dall'esatta sede e dalla lateralità del danno. È spesso coinvolto, ad esempio, da lesioni post ictus, e bersaglio di atrofia nella malattia di Alzheimer.
- **lobo parietale (percezione somatosensoriale e gustativa)**, posto tra il frontale e l'occipitale, è coinvolto nell'attenzione e nelle abilità matematiche.
  Le disfunzionalità della corteccia somatosensoriale
  a. primaria producono parestesie (formicolii e intorpidimento) sul lato del corpo opposto alla lesione e deficit attentivi. Come per il lobo temporale, è spesso coinvolto da lesioni post ictus;

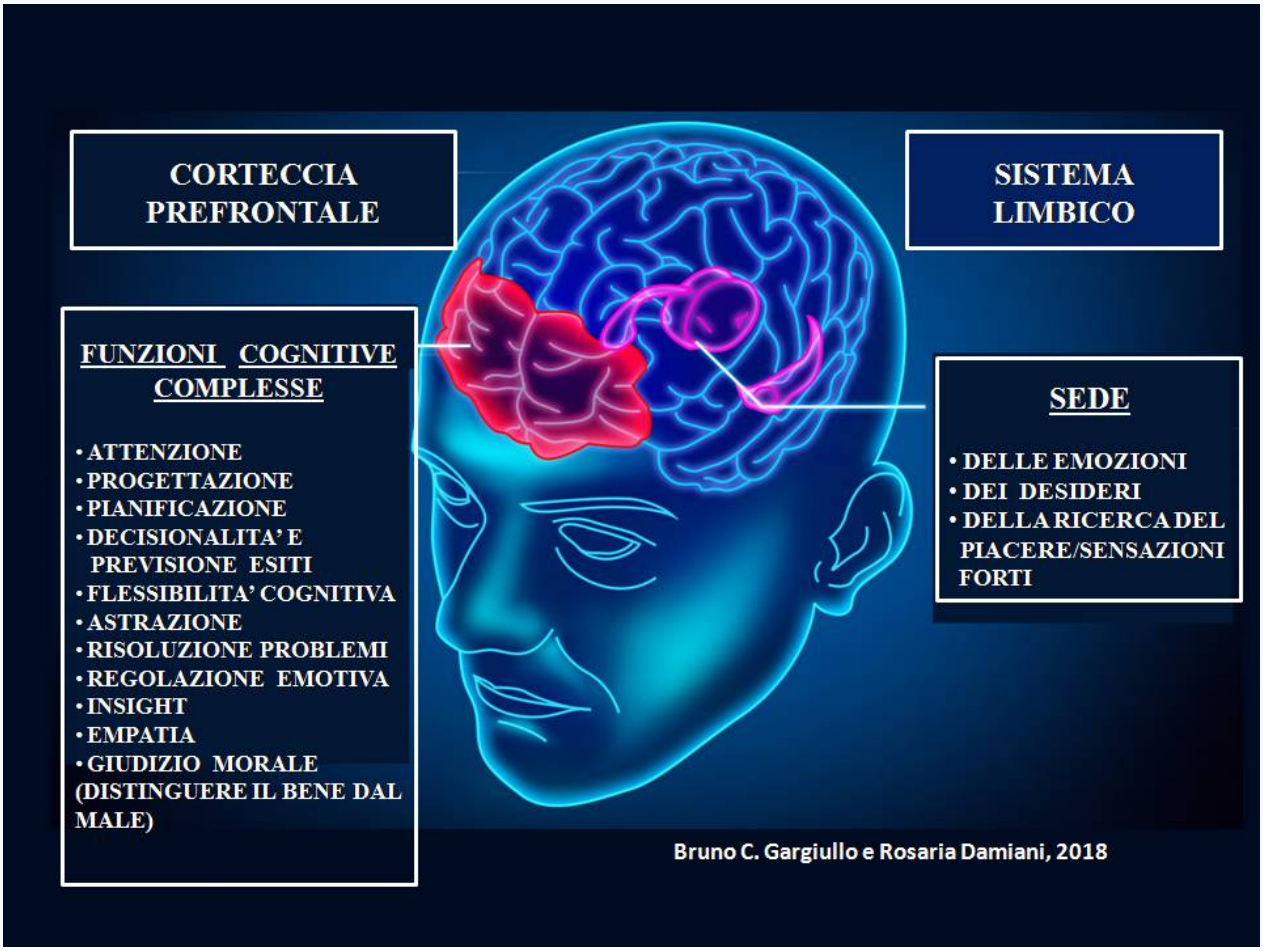
  b. posteriore conducono all'agnosia (incapacità o difficoltà nel riconoscere oggetti) che comprende diverse tipologie (es., stereoagnosia, riconoscimento di un oggetto toccato con la mano controlaterale rispetto alla lesione – anosognosia, ovvero negazione o scarsa consapevolezza del disturbo);

  del lobulo parietale inferiore, (o Sindrome di Gerstmann), che si "traducono" in agnosia delle dita (incapacità a riconoscere le diverse dita della propria mano e di quelle degli altri), disgrafia (disturbo del tratto grafico), discalculia (disturbo del calcolo) e dislessia (disturbo della lettura).

- **lobo occipitale (percezione visiva)** è coinvolto nell'elaborazione degli stimoli visivi grazie all'area visiva primaria e secondaria. Inoltre, come per gli altri lobi, non agisce autonomamente, ma in relazione funzionale con le regioni cerebrali soprariportate.
  La disfunzionalità del lobo occipitale conduce, ad esempio, a emianopsia (o perdita della metà del campo visivo), a prosopagnosia (difficoltà nella percezione di volti familiari, altrimenti riconoscibili dalla voce o dall'andatura) e acromatopsia (perdita selettiva del colore, ovvero visione della vita in "bianco e nero".

Fonte: Allan Siegel e Hreday N. Sapru, Fondamenti di neuroscienze, Piccin Editore, 2019.

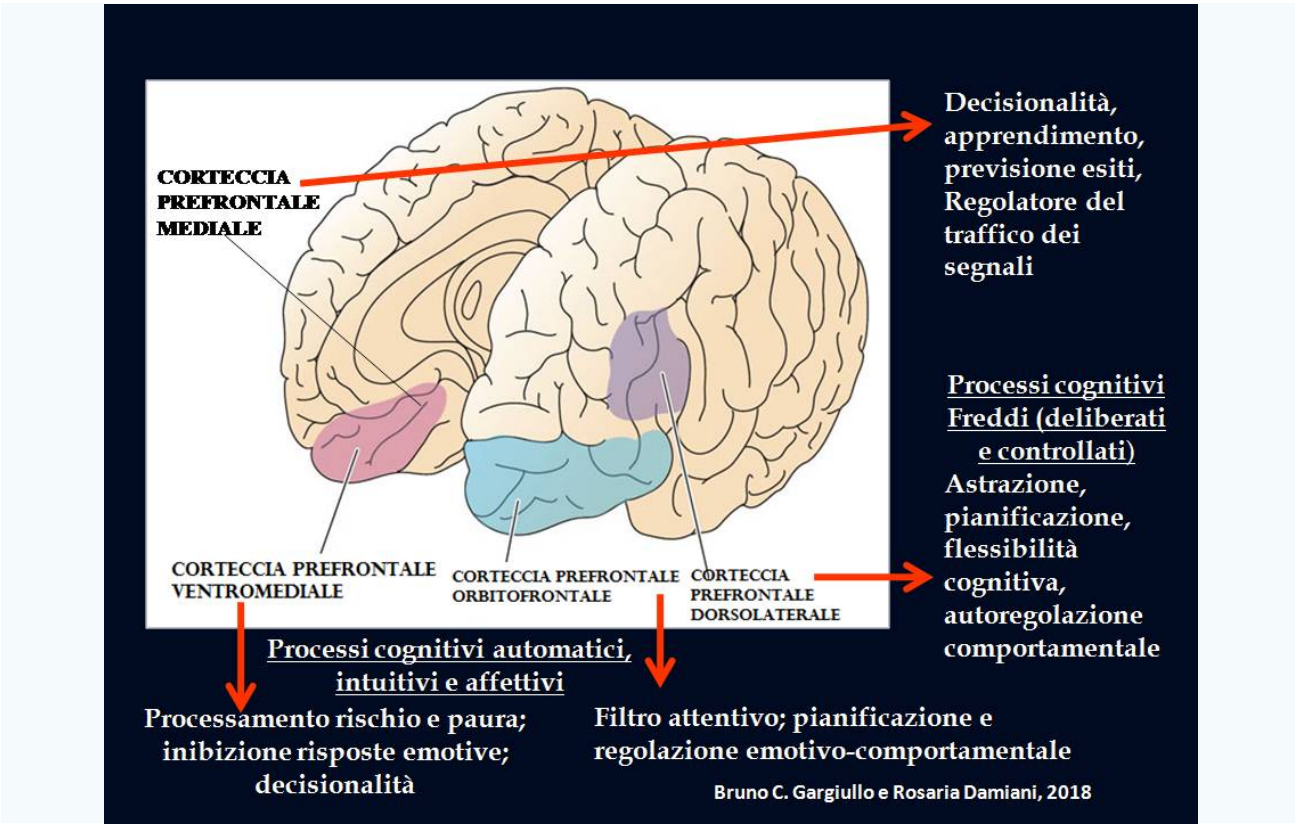
Area prefrontale e limbica



Soffermiamoci su due aree: corteccia prefrontale e sistema limbico.

La *regolazione degli stati emotivi*, che rappresenta un aspetto fondamentale per l'adattamento dell'individuo all'ambiente e per il suo funzionamento sociale, viene realizzata dalla **corteccia prefrontale.** Questa regione, collocata nella sezione anteriore del lobo frontale (quella che abbracciamo quando poniamo la mano sopra la fronte), oltre ad essere il centro della volontà, è responsabile delle cosiddette funzioni esecutive del cervello (funzioni cognitive superiori), particolarmente sviluppate nei primati ed, in special modo, nell'essere umano: attenzione, pianificazione, decisionalità, progettualità, giudizio, inibizione della risposta, regolazione e modulazione emotiva, apprendimento.

Questa corteccia, per essere sintetici, ci rende individui sociali, tant'è che se viene danneggiata, per lesioni o per malattie (es. demenza frontotemporale, trauma cranico), quello che viene a mancare è la capacità di mantenere un normale comportamento sociale (rispetto delle regole e delle norme sociali).
La corteccia prefrontale, inoltre, svolge l'importante funzione di coordinamento e di equilibrio tra cervello e corpo (es., segnali provenienti dal cuore, dall'intestino, dai muscoli) e fra reti interne e mondo sociale.



Il **lobo frontale**, precedentemente descritto, può suddividersi in:
– **corteccia prefrontale laterale**, implicata in funzioni quali la memoria di lavoro (working memory), può essere ulteriormente suddivisa in
a) **corteccia prefrontale dorso-laterale** (capacità di astrazione e di pianificazione delle azioni, flessibilità cognitiva, processi di autoregolazione comportamentale tra cui impulsività e scarso controllo, tipici dell'antisocialità) e
b) **corteccia prefrontale ventro-laterale/orbito-frontale** (coinvolta principalmente nella regolazione delle emozioni, nell'apprendimento dai premi o dalle punizioni e nei processi decisionali).
Si precisa che la corteccia orbito-frontale, fra le diverse aree della corteccia frontale (prefrontale dorso-laterale, frontale interna), riveste un ruolo particolarmente importante nella pianificazione e nella regolazione del comportamento in generale (motor level).
La sua ridotta funzionalità è associata a disturbi del controllo emotivo (es. instabilità dell'umore) e comportamentale (es., ostinazione, impulsività, iperattività, ipersessualità, oppositività).

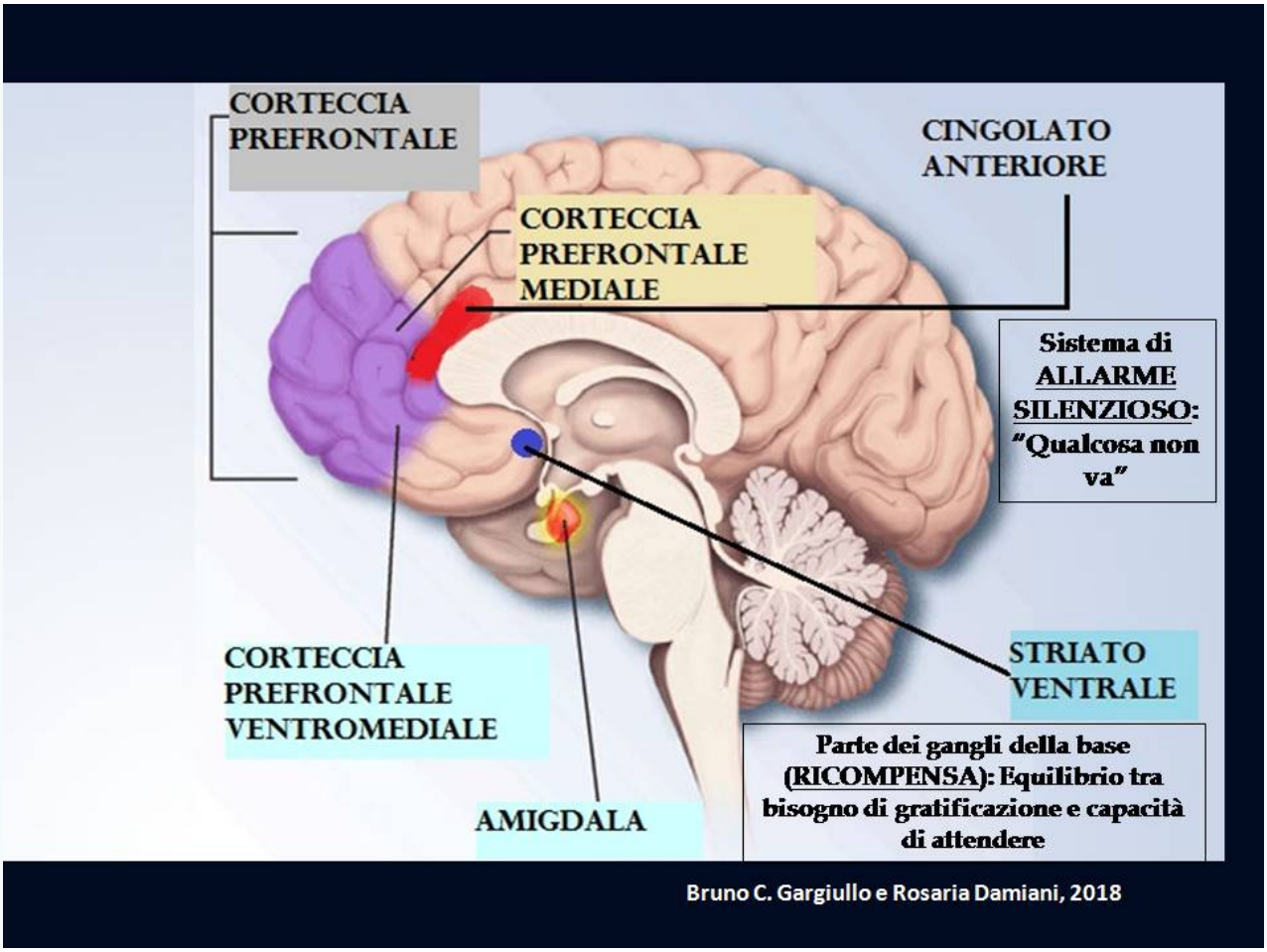
– **Corteccia prefrontale mediale**, ovvero la **corteccia cingolata anteriore** (identificazione degli errori di elaborazione e comportamentali, monitoraggio dei conflitti, apprendimento) ed il **giro frontale superiore** (selezione e flessibilità di un compito da eseguire, ovvero task switching).

Coloro che presentano dei danni nel cingolato anteriore sono più disinibiti ed aggressivi e presentano difficoltà nel controllo inibitorio e nel processo di elaborazione emozionale (Bellamoli E., Zoccatelli G. et al., 2014).

La **parte dorsale** della corteccia prefrontale, e le sue aree subcorticali interconnesse, è associata con la **cold cognition**, ovvero processi cognitivi (pensieri, percezioni, memoria a breve termine o esecutiva, pianificazioni e decisionalità) privi di coloritura emozionale ("fredde").
La **parte ventrale**, o bassa, della corteccia prefrontale, in gran parte costituita dalla corteccia orbitale e corteccia prefrontale ventromediale, è coinvolta in funzioni simili a connotazione automatica/affettiva (**hot cognition,** ovvero calde)». In altre parole, le hot cognition si riferiscono a quei processi mentali che sono guidati dai nostri desideri e sentimenti (Kunda Z., 1999).

Corteccia cingolata, striato ventrale, amigdala, ippocampo, insula

Proseguendo il viaggio all'interno del nostro cervello, senza soffermarsi troppo sui vari aspetti più tecnici, sotto la crosta civilizzata e superficiale rappresentata dalla corteccia prefrontale, arriviamo all'area limbica, sede delle nostre emozioni … Ed è qui che troviamo alcune delle importanti aree fra le quali l'ippocampo e l'amigdala che contribuiscono a realizzare l'equilibrio emotivo e i processi della memoria.
Presentiamoli brevemente.



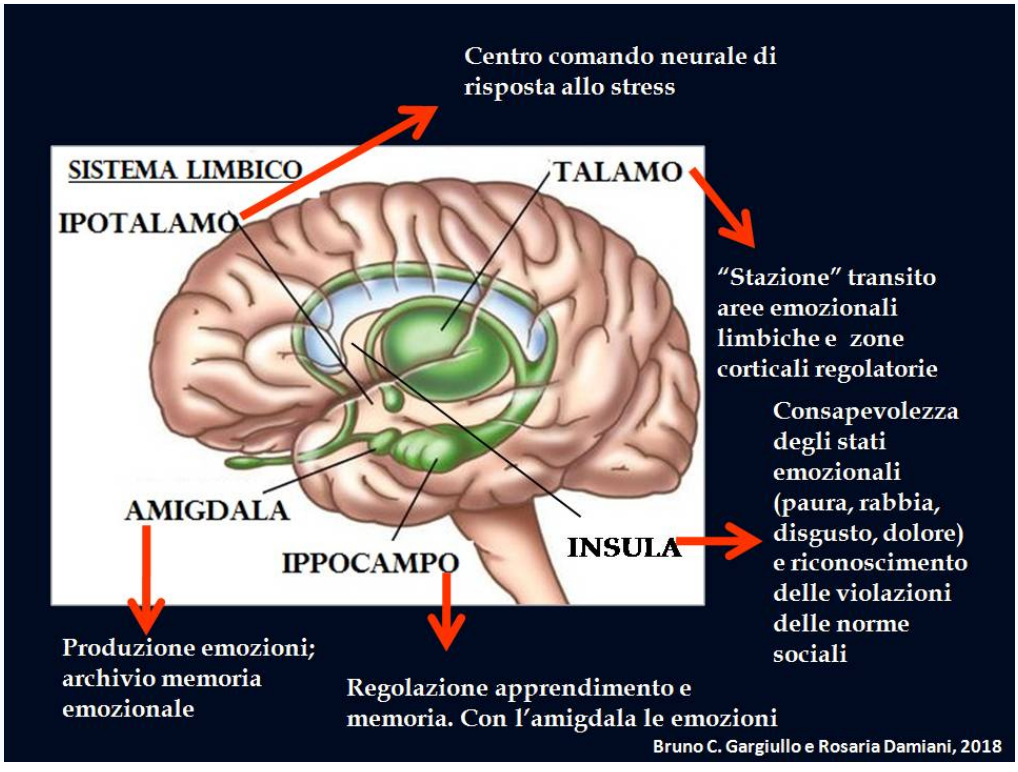
Corteccia Cingolata (C.C)

La C.C. è coinvolta nell'identificazione degli errori comportamentali e di elaborazione, nel monitoraggio dei conflitti e nell'apprendimento.
Coloro che presentano dei danni nel cingolato anteriore (centralina silenziosa di allarme) sono più disinibiti ed aggressivi e presentano difficoltà nel controllo inibitorio e nel processo di elaborazione emozionale.

Striato Ventrale

Struttura sottocorticale, che si trova in profondità nel cervello, segnala le gratificazioni e si attiva quando proviamo piacere.
Detta area cerebrale gioca un ruolo chiave nel mantenere l'equilibrio tra il bisogno di gratificazione e la capacità di attendere, tra le azioni impulsive e le scelte prudenti.
E' importante notare come un'anomalia dello striato ventrale produca una percezione alterata degli appagamenti.

Amigdala



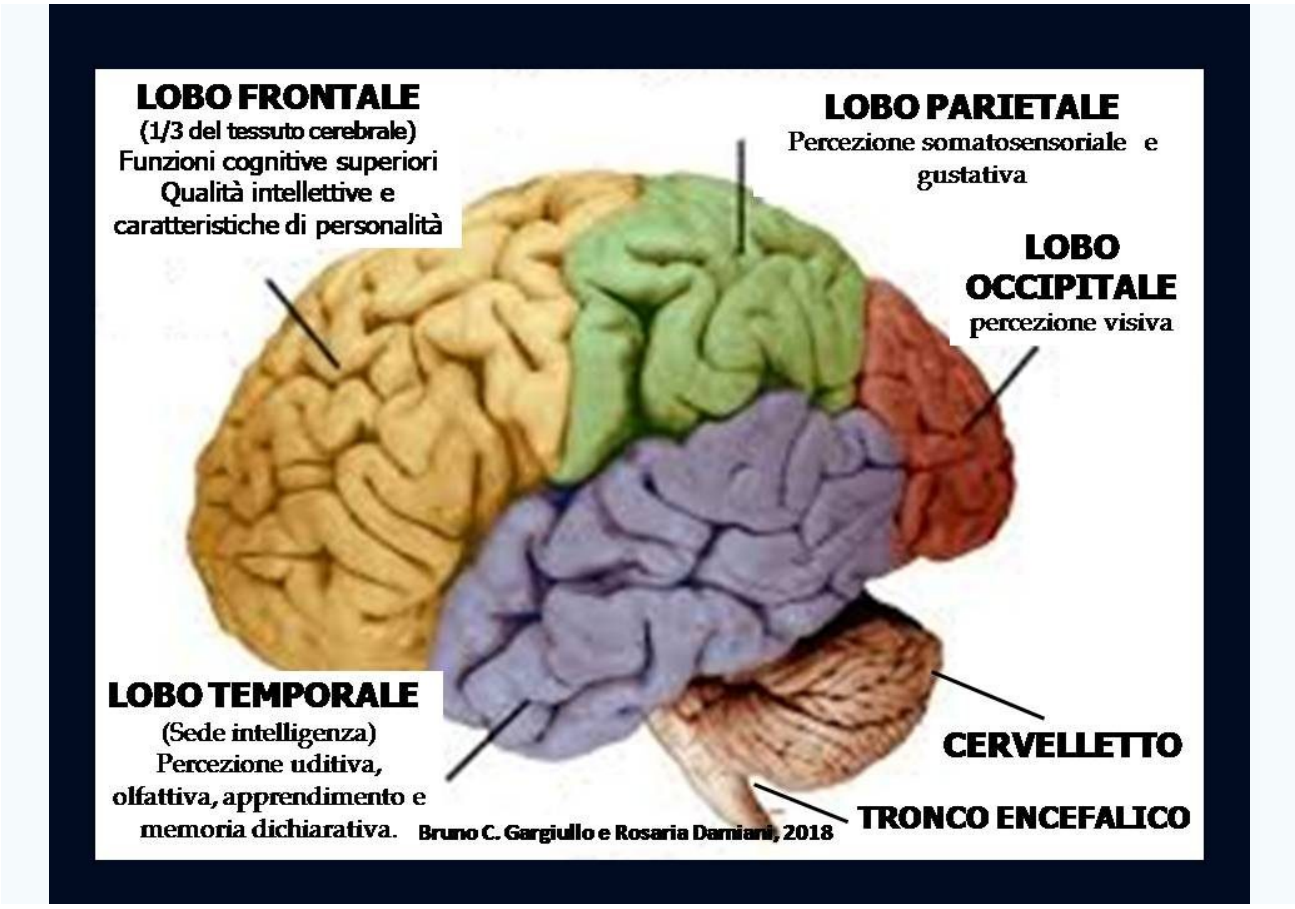
L’**amigdala**, insieme di nuclei a forma di mandorla, situata nella porzione mediale del lobo temporale anteriore, è coinvolta nella produzione delle emozioni ed è depositaria del significato stesso degli eventi (archivio della memoria emozionale).
Gli adulti ed i giovani con tratti psicopatici, che presentano risposte emotive fredde e forme di aggressività pianificata (proattiva), mostrano un ridotto volume dell’amigdala e della sua funzionalità, mentre gli individui più impulsivi, con una forma di aggressione reattiva, palesano un’esagerata reattività dell’amigdala.
Da notare, che i pazienti che presentano lesioni all’amigdala presentano una ridotta percezione del pericolo, sono meno timorosi ed evidenziano deficit nel riconoscimento delle varie espressioni facciali della paura (assenza di empatia).

Ippocampo e insula

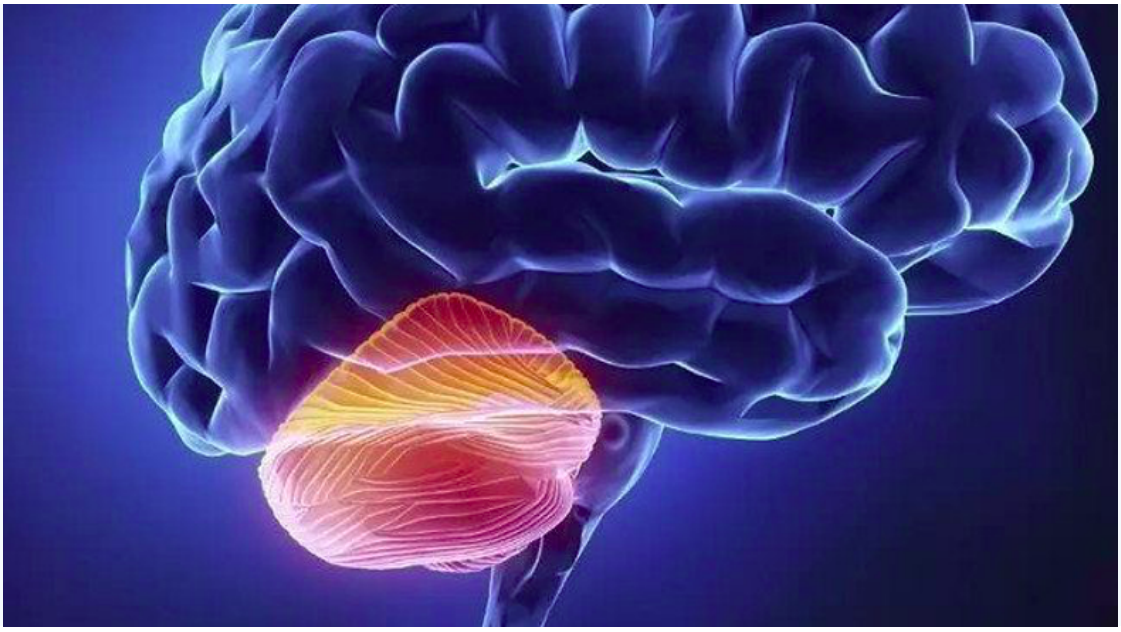
L’i**ppocampo**, porzione del lobo temporale anteriore a forma di “cavalluccio marino”, regola, per le ampie interconnessioni con aree associative corticali, apprendimento e memoria e, insieme all’amigdala per le ampie interconnessioni con le strutture limbiche proencefaliche, le emozioni.
L’ippocampo, in condizioni di deficit strutturali (parte destra più grande della sinistra), rende una persona incapace di distinguere il “bene dal male” e “insensibile” agli stimoli ambientali (mancanza di coscienza).
L’**insula** è la struttura che collega i processi dell’organismo alle aree prefrontali, rendendo possibile l’autoconsapevolezza.

Tronco cerebrale

Infine, la regione posta alla base del cranio, il **tronco cerebrale o tronco encefalico** (parte assiale dell'encefalo suddiviso, dal basso verso l'alto, in bulbo o midollo allungato, ponte e mesencefalo), costituisce un vero e proprio "centro di smistamento" bidirezionale (**encefalo ⇆ cervelletto ⇆ midollo spinale**) dei segnali nervosi, mediante fasci di fibre. Fra le funzioni principali del tronco encefalico rientrano il mantenimento dello stato di coscienza, la regolazione del ciclo sonno-veglia e, in interazione con la regione limbica, l'attivazione di stati emozionali come rabbia e paura.
In breve, tronco cerebrale e area limbica (amigdala, ippocampo, insula, corteccia cingolata, lobo superiore temporale) vengono definite aree o regioni sottocorticali poiché sono "avvolte" dalla corteccia cerebrale.



Il Cervelletto



Il **cervelletto** (*cerebellum*), che richiama alla memoria quanto "scoperto" da Cesare Lombroso nel cranio del brigante Villella, è collocato nella parte inferiore dell'encefalo (apparato nervoso centrale contenuto nel cranio).

Sebbene costituisca solo il 10% della massa cerebrale, contiene più della metà dei neuroni del cervello umano, occupando la maggior parte della fossa cranica posteriore (Kandel E.R. et al., 2000). In passato, il cervelletto non ha ricevuto molte attenzioni negli studi scientifici sugli aspetti non motori del comportamento umano, quali la cognizione e le emozioni.

Tuttavia, alcuni scienziati, alla fine degli anni '70, hanno iniziato a dimostrare l'importanza del cervelletto nei disordini emozionali (Snider R.S. e Maiti A., 1976; Heath R.G., 1977). A questa conclusione era giunto, due secoli prima, Franz Joseph Gall (1758-1828) che si spinse a considerare il cervelletto la "sede dell'amore". Una svolta significativa la si deve agli studi condotti da Jeremy Schmahmann che estese il ruolo funzionale del cervelletto ai domini della cognizione e delle emozioni (Schmahmann J.D., 1991). Successivamente, il legame cervelletto-emozioni venne confermato da diversi studi clinici che posero in evidenza una significativa presenza delle anomalie

cerebellari nei disturbi emozionali, inclusa la schizofrenia e la depressione (Schmahmann J.D., 2004). Ulteriori studi, sugli aspetti neurali delle emozioni, hanno confermato l'importanza del cervelletto nella regolazione e nel controllo degli stati emotivi, oltre alla coordinazione motoria, alla postura e al linguaggio (Baillieux H. et al., 2008).
E' da sottolineare che il cervelletto presenta innumerevoli connessioni bidirezionali con diverse aree della corteccia cerebrale, o neocorteccia (frontale, parietale e temporale) che modulano le emozioni (Turner B.M. et al., 2007; Middleton F.A. et al., 2001; Clausi S. et al., 2009).
Ad anomalie strutturali del "cervelletto limbico" (verme e nucleo del fastige) in adulti e bambini con disfunzioni congenite (agenesia cerebellare, displasia, ipoplasia) o acquisite (ictus cerebellare, tumore, cerebelliti, trauma e disturbi degenerativi) ne consegue una disregolazione degli affetti, definita da Schmahmann J.D. e Sherman J.C. (1998) **Sindrome Cognitivo Affettiva Cerebellare (*Cerebellar Cognitive Affettive Syndrome* – CCAS)**. I comportamenti osservati da Schmahmann, Weilburg e Sherman (2007), e descritti dagli stessi pazienti e loro familiari, includevano: distraibilità, iperattività motoria, disinibizione, ansietà, comportamenti ritualistici e stereotipati, pensieri illogici, assenza di empatia, ruminazioni e ossessioni, disforia e depressione, atteggiamento difensivo (distanza fisica dall'altro), iperattivazione sensoriale, apatia, comportamenti regressivi e evidenti difficoltà socio-relazionali, così come aggressività e irritabilità. A ciò si aggiungevano deficit delle funzioni esecutive, quali: pianificazione, ragionamento astratto, memoria di lavoro, e indebolimento intellettivo.
Questi profili neuro-comportamentali sono stati raggruppati in cinque domini: disturbo del controllo dell'attenzione, disturbo del controllo emozionale, disturbo delle abilità sociali, disturbo dello spettro autistico e disturbo dello spettro psicotico.
Alterazioni del cervelletto sono state documentate in diversi disturbi psicopatologici:

- nella schizofrenia è stato evidenziato: riduzione del volume totale del cervelletto, del volume del verme, e alterazioni della comunicazione tra la corteccia e il cervelletto, soprattutto tra la corteccia frontale, il talamo e il cervelletto (Andreasen et al., 1996)
- nel disturbo bipolare è stata evidenziata atrofia del verme (Mills et al., 2005)
- nella depressione maggiore vi è una riduzione del volume del cervelletto, correlata alla gravità e alla resistenza al trattamento (Konarski et al., 2005)
- nel caso di Disturbo Post-Traumatico da Stress è stata documenta una riduzione del volume di entrambi gli emisferi cerebellari nei bambini con PTSD in seguito a maltrattamenti (De Bellis et al., 2006); vi è inoltre un'iperattività del cervelletto nel PTSD, nell' ansia sociale e nel Disturbo di Panico (Kilts et al., 2006; Driessen et al., 2004)
- nei Disturbi dello Spettro Autistico è stato sottolineato come alterazioni della sostanza grigia cerebellare possano aiutare a discriminare gli individui con autismo da individui con sviluppo tipico: vi è infatti un ridotto volume delle regioni posteriori della corteccia cerebellare nelle persone con autismo, e ridotta connettività con le regioni frontali e temporali della corteccia (Olivito et al., 2018). Viene quindi sottolineato il ruolo specifico del cervelletto nella cognizione sociale e nella Teoria della Mente (la capacità di immedesimarsi nell'altro).

La tabella sottostante riporta alcune delle ricerche che hanno dimostrato il coinvolgimento del cervelletto nei disturbi emotivi.

Aree di ricerca	Autori	Risultati
Neuroanatomia funzionale	Heath et al. (1977; 1978) Supple et al. (1993) Schutter et al. (2005) Sacchetti et al. (2004)	Risposte del sistema limbico e stato umorale in seguito alla stimolazione elettrica del cervelletto Rapporto tra verme e paura appresa La stimolazione magnetica transcranica nella zona del cervelletto produce stati affettivi positivi Cambiamenti sinaptici a lungo termine indotti nella corteccia cerebellare dalla paura condizionata
Danni cerebrali	Schmahmann e Sherman (1988) Levisohn et al (2000)	Lesioni della fossa posteriore: CCAS; disinibizione comportamentale, ottundimento degli affetti, psicosi e dismetria cognitiva CCAS dopo la resezione chirurgica del tumore cerebellare in bambini: danni al Verme che producono una disregolazione affettiva Atrofia olivopontocerebellare: disinizione comportamentale, malinconia, paura e allucinazioni uditive
Neuroimmagine funzionale	Liotti et al. (2000) Habel et al. (2005) Wiech et al. (2005)	Attività cerebellare durante la malinconia e l'ansia in un gruppo di volontari senza problemi di salute Attività cerebellare durante gli stati umorali positivi Correlazione positiva tra attività del cervelletto ed esperienza cronica del dolore
Psichiatria	Ichimiya et al. (2001) Loeber et al. (2001) Kwoseva (2004) Okugawa et al. (2005) Okugawa et al. (2003) Soares e Mann (1997) Beyer et al. (2002) Leroi et al. (2002) Neil et al. (2005)	Ridotto volume del verme cerebellare in pazienti schizofrenici trattati con neurolettici Ridotto volume del cervelletto inferiore nella schizofrenia Anomalie cerebellari in pazienti schizofrenici Anomalie della sostanza bianca nei peduncoli cerebellari mediani in pazienti schizofrenici Ridotto volume del verme nella schizofrenia cronica Depressione conseguente alla riduzione del volume cerebellare Ridotto volume del verme nei disturbi dell'umore Psicopatologia in pazienti con patologie degenerative cerebellari: una comparazione con il Morbo di Huntington Riduzioni del volume del verme nel disturbo maniaco-depressivo

Conclusioni
Plasticità neuronale

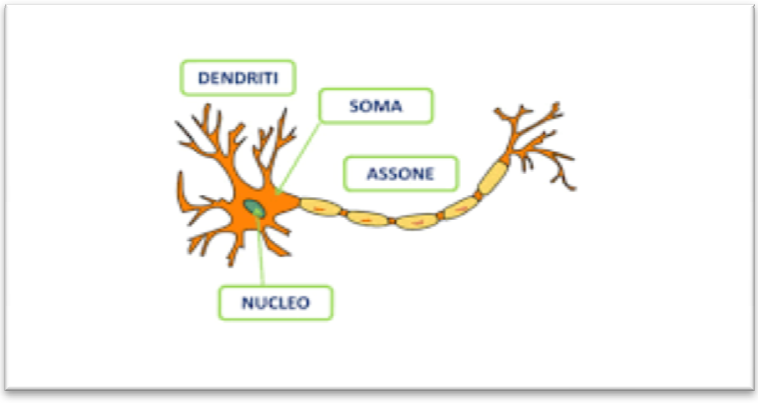
E' dal nostro cervello, un insieme di almeno 100 miliardi di neuroni (cellule nervose) interconnessi, che hanno origine bisogni (fame/sete, sonno/veglia, accoppiamento/procreazione), percezioni, emozioni, pensieri e azioni. L'essenza della nostra identità è racchiusa nella peculiarità di queste connessioni cerebrali e nell'interazione tra organismo ed ambiente. Sembra incredibile che da questo organo, che pesa circa 1400 grammi, il 2% in media del nostro peso corporeo, possano dipendere

«*… le gioie, i piaceri, le risa e gli svaghi, e i dispiaceri e le angosce, lo scoramento e le afflizioni. E grazie a ciò, in special modo, noi acquisiamo la saggezza e la conoscenza, e vediamo e udiamo e sappiamo ciò che è giusto e ciò che è infame, ciò che è buono e ciò che è cattivo, ciò che è dolce e ciò che è insipido … E per mezzo dello stesso organo diventiamo pazzi e deliriamo, e le paure e il terrore ci assalgono … Tutte queste cose sopportiamo dal cervello, quando esso non è in salute*» (Ippocrate - La malattia sacra – "De morbo sacro" – a cura di Roselli A., Marsilio Editore, 1996).

Vale la pena rammentare che il nostro cervello è continuamente esposto al flusso delle stimolazioni ambientali che influenzano, ad esempio, le nostre capacità di percepire gli stimoli, di compiere movimenti, di pensare, di apprendere, di ricordare e di pianificare strategie comportamentali. Nel corso della nostra vita, questo instancabile organo, nonostante sia contenuto all'interno di una scatola cranica ed avvolto da tre strati protettivi (partendo dall'esterno verso l'interno, dura madre, aracnoide e pia madre), si modifica continuamente (sia strutturalmente che funzionalmente) mediante un processo definito "neuroplasticità" (Gogtay N., Thompson P.M., 2010).
La parte del cervello che presenta maggiore plasticità è la corteccia frontale, sede delle funzioni cognitive ed esecutive.
La plasticità neuronale è presente solo durante lo sviluppo e a seguito di una lesione cerebrale, o è anche una caratteristica del cervello dei soggetti in età adulta?
Tenere bene a mente che la neuroplasticità è presente, anche se ridotta, nel cervello di un adulto e che la neurogenesi (processo di formazione di nuove cellule nervose) non si arresta inesorabilmente con il completamento dello sviluppo neuronale (a partire dalla prima settimana, dopo il concepimento, sino al superamento del ventesimo anno di età, periodo in cui la corteccia cerebrale raggiunge la sua piena maturità – Bricolo F., Zoccatelli G. e Serpelloni G., Elementi di Neuroscienze e Dipendenze 2° edizione, Dipartimento delle Dipendenze ULSS 20 – SerD, 2010); anche in età avanzata, infatti, il cervello ha la capacità di creare nuovi neuroni, di riprogrammare le proprie reti neurali e di superare danni derivanti da traumi, lesioni o malattie (Begley, 2007; Bartels, 2008). Inoltre, come afferma Stiles (2000), non bisogna pensare alla plasticità solo come una "risposta" del cervello a una lesione o a una condizione psicopatologica (situazioni in cui è più evidente), ma come una proprietà fondamentale del normale funzionamento neuronale e cognitivo.

In sintesi, secondo la visione plastica dell'attività cerebrale, un sistema di strutture e di meccanismi neurali, influenzato da fattori interni ed interattivo con gli stimoli dell'ambiente esterno, genera di continuo processi mentali che, a loro volta, modificano la struttura cerebrale stessa. Secondo la teoria della plasticità neuronale, quindi, i neuroni si modificano costantemente per effetto degli stimoli ambientali, dell'apprendimento, delle esperienze e del programma genetico. La vulnerabilità genetica di base è in grado di modulare (rinforzare e/o estinguere) la probabilità di sviluppare un dato comportamento. Tale effetto non è diretto, ma viene a sua volta modulato da alcune variabili socio-ambientali. La più importante di quest'ultime è l'esposizione ad eventi stressanti, in periodi critici dello sviluppo, che produce un effetto potenziante sulla originaria predisposizione genetica a sviluppare un dato comportamento. In breve, la plasticità declina in età adulta, ma la capacità del cervello di rispondere agli stimoli ambientali permane per tutta la vita (Stiles J., 2000; 2005). In proposito, si sottolinea che la psicoterapia, proprio grazie a questa neuroplasticità, produce dei significativi cambiamenti cognitivo-comportamentali nei soggetti in trattamento (LeDoux J., 1996; Schnell K. e Herpertz S.C., 2007; Beutel M.E. et al., 2010; Karlsson H., 2011).
Per completare questa sintetica panoramica sulla plasticità, è bene ribadire che essa rappresenta la capacità dei circuiti nervosi di sfuggire alle restrizioni imposte dal corredo genetico e di variare la loro struttura e funzione in risposta agli stimoli esterni, alle modificazioni ambientali, all'esperienza e anche ai fattori intrinseci del soggetto (Blundo, 2007; Ansermet e Magistretti, 2008). In breve, i circuiti cerebrali sono programmati geneticamente e dipendenti dall'esperienza (Siracusano A., Sarchiola L. e Niolu C., 2008). Ciò significa che i geni "guidano" le prime fasi dello sviluppo cerebrale e la formazione iniziale delle connessioni neurali, ma sono le interazioni con l'ambiente a completare, in maniera appropriata e specifica, la maturazione dei circuiti deputati al controllo della maggior parte delle funzioni cerebrali (Johnson, 2001; Thatcher, 1992; Stiles J., 2005). Infine, riprendendo Malabou (2007), vi sono tre "ambiti di azione" della plasticità: *di sviluppo* (genesi e conformazione della rete neurale nell'embrione e nel bambino), *di riparazione* (compensazione di una lacuna prodotta, ad esempio, da un trauma, e rinnovamento neuronale), *di modulazione dell'effetto sinaptico* (modificabilità delle connessioni neuronali nel corso della vita).

Unità centrale del sistema nervoso.



«Il neurone è costituito da tre importanti componenti: il corpo cellulare; il prolungamento principale in uscita, una fibra nervosa detta assone o cilindrasse; i prolungamenti in entrata, le fibre nervose dette dendriti. I neuroni sono collegati tra loro in circuiti in cui si riconoscono gli equivalenti dei fili conduttori (le fibre degli assoni) e dei connettori (le sinapsi, cioè i punti in cui gli assoni sono in contatto con i dendriti di altri neuroni). Quando i neuroni divengono attivi ("scaricano" o "sono eccitati" nel gergo delle neuroscienze), dal corpo cellulare si propaga lungo l'assone una variazione di potenziale elettrico o potenziale di azione. Arrivato ad una sinapsi, questo innesca l'emissione di particolari sostanze chimiche note come neurotrasmettitori. ... A loro volta, i neurotrasmettitori operano sui recettori. In un neurone eccitatorio, l'interazione in cooperazione con molti altri

neuroni le cui sinapsi sono adiacenti, e che possono liberare oppure no i propri neurotrasmettitori, determina se il prossimo neurone sarà eccitato oppure no, cioè se esso produrrà il proprio potenziale di azione, con la conseguente emissione dei propri neurotrasmettitori, e così di seguito» (Damasio A.R., 1995, pp. 64-65).

Per concludere questo viaggio nella mente criminale, si ribadisce quanto già detto all'inizio di questo lavoro: lo studio di un comportamento violento richiede l'esame delle molteplici cause (biologiche, psicologiche e socio-ambientali) che si accompagnano alla genesi ed allo sviluppo di ogni singolo episodio criminoso. Da ciò ne consegue che lo studio della eziologia del comportamento deviante è quanto mai complesso dal momento che la personalità del criminale è il risultato dello stretto rapporto esistente tra le variabili individuali e biologiche e quelle ambientali e socio-culturali.

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International Scientific Review:

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