

## A Social Neuroscience Mind Brain Perspective

Jay Evans Harris

**I know I'm real**, but I'm not sure about you, the audience. Winning you to my subjective position means turning you from an unfamiliar social group to self-extending others, a family grouping--almost tribal. Anyone who tries to find a fresh perspective on mind/brain process is vulnerable to a superego that divides identity between indwelling social judgment and a wish to communicate with would be allies. I think dividing the social world between self-extension and stranger manifests neural hubs that regulate the mind with innate ambivalence. Just as Freud conceived *heimlich* and *unheimlich* at the core of innate ambivalent drives, it seems inconceivable to me that the brain's structure of mind could fail to correspond to practical psychoanalytic principles. This presentation aims to synthesize psychoanalytic fundamentals with Edelman's Neural Darwinism.

Social neuroscience opens a portal into functional networks, showing mental process on the cusp of brain process. In fact, the US has begun a *Human Connectome Project* to explore the brain's functional connections. Mathematically enhanced MRI techniques show network hubs activating together during defined mental functions. Following Edelman's version of Hebbian dynamics --*neurons that play together stay together*--Van Dijk et al. in Buckner's Harvard group portray *Human Connectomics* as a structural frame for Hebbian ego functions. They locate several ego-function hubs in mathematical confluences of nodes of communication as in the left illustration.



emotional and cognitive identifications between self and other. Conceivably, billions of VMPFC/DMPFC excitatory links comprise identificatory building blocks of character.

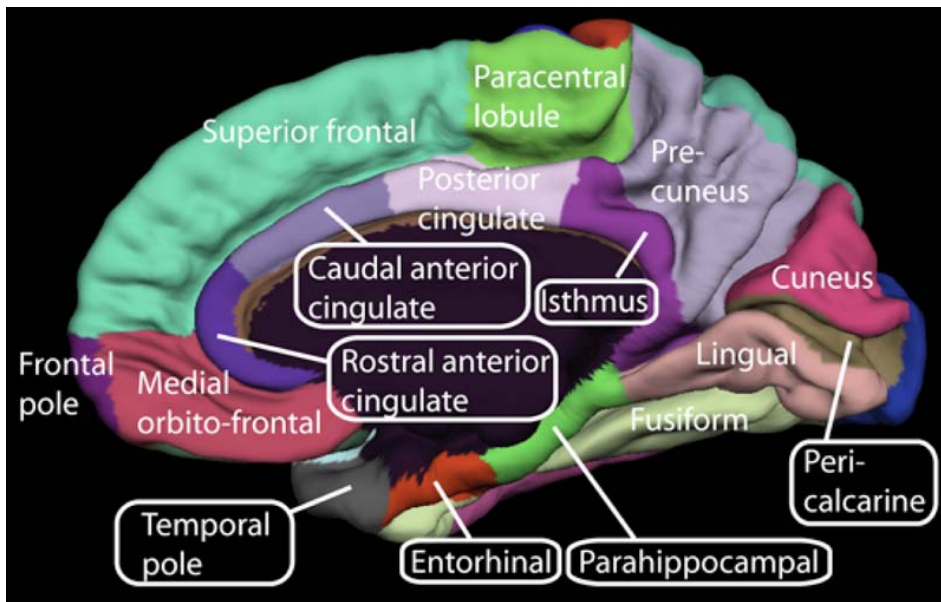
### **Discovering the Default Domain**

Marcus Raichle at Washington University pioneered the discovery of the *default domain*, medial cortical linked hubs of neocortical networks. Exploring functional characteristics of cortical networks of interest by measuring their metabolic activation levels, Raichle's group found that networks that were supposedly at rest showed higher levels of activation than networks functioning during task performance. They found a set of networks using more than 90% of the brain's metabolic energy day and night, even under anesthesia. People at rest in the fMRI chamber, supposedly thinking of nothing in particular, often daydream of future social situations; in fact Raichle connects that mental activity to what David Ingvar called *memory of the future*.

Andrews-Hanna et al. in Buckner's Harvard group, surveying the default domain in context of the connectome, found two systems: A DMPFC subsystem observing self other interaction and a VMPFC subsystem fantasizing future self based on its cingulate cortex links to hippocampal episodic memory. The function of projecting self into the future uses most of the default domain's metabolic energy during a normative stream of consciousness at rest. Then we emerge from rest to prioritize our needs and safety issues, which leads to working memory's conscious pragmatic tasks necessary to fulfill the goals and subgoals we set. Endel Tulving describes the self's time travel as uniquely human.

Randall O'Reilly surmises that throughout the neocortex, ventral functions process the sensory *what*; dorsal functions process the motoric *how*. Based on actual genetic distinctions between the brain's dorsal and ventral cortex, Fanselow and Dong

show that the ventral hippocampus regulates emotional process within the VMPFC subsystem; dorsal hippocampus provides cognitive context to the DMPFC subsystem.



### Heteromodal Cortex

We can learn more about brain-mind transition by considering default domain *heteromodal* cortex in the anterior/posterior dimension. Heteromodal cortex responds to multiple sensory modalities. Posterior networks process objects in multiple modalities, while anterior networks only need one or two sensory cues to recognize what is sought. Posterior objects need not be completely assembled as data, only as cues and context. The motoric ego function *perception* seeks salient cues, enough data to indicate the presence of what is needed for conditioned need satisfaction. Hierarchical generalizations in the most anterior hubs of medial prefrontal form ego functions by linking to their distributed networks. In Edelman's view, when connected links of a hub are activated together they form a joined consciousness. If affect consciousness is a form of endogenous perception it is conceivable that the medial forebrain bundle also brings the whole realm of midbrain sources of feeling into a heteromodal context for the VMPFC self's reception. At the top

of the heap, social identity as amalgam of self and object forms the highest level of ego functions, cognized in a verbal form.

Luria and Vygotsky established that after age four, when a neocortical second signal system matures, any higher cortical function (ego-function) can be replaced when underlying networks are damaged, by using alternative networks. That is because a top-down, identity-based set of ego functions makes reflective (verbal) consciousness a monitor of brain process. Indwelling social sense feels like it arises verbally inside and outside mind. Corresponding to Freud's topographic approach, preconscious (experience) comes from self-feeling, while full consciousness requires reflection, knowing you know.

In a third dimension to mental topography, one's sense of identity includes left brain agency (subjective) and right brain representation (objective). Our left brain seeks need satisfaction; our right brain pursues safety with vigilance. The left brain regulates motor intentions, by reducing need pressure through satisfying sexual and metabolic needs. The right brain regulates safety by avoiding traumatic situations. Hilz, et al. found that the right VMPFC inhibits sympathetic cardiac activation when affect is too intense. VMPFC's top-down lateralized autonomic regulation—left brain parasympathetic and right brain sympathetic—has ramifications in psychosomatic process. Left brain pleasure processing keeps one's metabolism in equilibrium; right brain pain processing controls over-arousal to novelty or threat. Un-modulated affect disturbs the calm that regulates the sympathetic system. Type A persons suffer situational high blood pressure when the feeling of handling social situations is out of control.

### **Lateralized Network Origins of Self-Observation**

The right brain mode of objectifying identity as a physical object can lead to a sense of dehumanization. The right brain tends to identify objects (including one's own body) with a sense of external reality. Among the social vicissitudes of dehumanization we may include feelings of being forced into a social role (obedience) or being unable to protect our reputation (status). Self-observation is partly a developmental byproduct of observing other's social signals. STS (Superior Temporal Sulcus) is a default domain posterior network that provides social data by observing other's social signals. Pelphrey and Morris found right-sided STS activation during visual detection of others' biological motion: mouthing, eye-gaze direction, facial expression, even gait. For instance, in the *McGurk effect*, seeing a person on TV whose mouth movements are desynchronized from speech sounds produces a sense of dissonant social observation.

Olaf Blanke reports that STS damage or malfunction leads to *out of body experiences*: looking at oneself from outside perspective. Struck by the correspondence between looking at others biologically, and looking at oneself from the top-down during vital introspection, I conceive that the DMPFC uses the consciousness of embodied biological perspective, gleaned from its social construct domain as its sensate source of reflection. The OED begins its use of *sensate* with a 1652 attribution to a J. Smith: "These corporate motions, as they seem to arise from nothing else but merely from the *machina* of the body itself; so they could not at all be *sensated* but by the soul." Looking at self in a social mirror, we see self objectified as one soul (social identity) among others.

### **Geist, Ghosts and Social Spirit**

The biological body ego exists as an almost imaginary social being imbedded like a homunculus in the mind's eye. Many functional networks do not distinguish self from

other in their attributions of quality to consciousness. Collectively projecting social images into the external social world we create *geist*, which philosophers and historians have treated as social spirit. There are two kinds of *geist* corresponding to two kinds of empathy: left brain ethnic or tribal self-extension as joined *agents*; and right brain nation states, using bureaucratic power to confer *representative*, objectified, documented, status. Edelman debunks anything like a homunculus occupying the mind as actual neural structure. But for all practical purposes people assume that social influence is the major organizer of their inner world. Social transference to a king (kin) or president *resides and presides* in our collective minds. With *geist* and ghosts, generations of social continuity, we embed our social mythology in personal narratives.

### **The Cingulate Cortex Anxiety System**

Integrating and synthesizing identity at the mind brain juncture, the VMPFC and DMPFC determine which identity aspect runs the brain's show of consciousness: the initiative of agency or the objectivity of one's role. Masterminded by these hubs, default domain networks support many ego functions underlying psychoanalytic concepts. Thus, workhorse of pragmatic working memory, the *precuneus* integrates sensory and motor expectations (attention) with behavioral outcome. When expectations break, cingulate cortex triggers anxiety signals felt in the VMPFC-self. Limbic cingulate cortex underlies the medial default domain. Freud's putative anxiety system, it resets consciousness during data collection disparity. Cingulate cortex signals distinct anxiety quality when sensory unfamiliarity (vigilance), motor error (anticipatory), or social dissonance erupts.

Concepts of social transference and dissonance are fundamental to psychoanalytic practice. Consider Freud's work on the primal dichotomy between the *heimlich* and the

*unheimlich*, in which the familiar and the unfamiliar both contain the other in cognitive ambiguity and emotional ambivalence. In *The Uncanny*, Freud illustrated the primal ambivalence between self and other with the story of the rail car making a jolt and as the door swung open with its mirror reflecting Freud, he felt an antipathy to the supposed stranger. A primal social illusion that fuses self and other as self-extension had flourished in Freud's developing mind, because baby brother Julius died before Sigmund could clearly know him as other. Maintaining life conversation with his double, Freud made Julius a literary device, a straw man who had to be deposed in many of Freud's essays. Love/hate ambivalence traces back to self-other exchange and dissonance in infancy.

### **Clinical Implications of Anxiety versus Fear**

VMPFC gathers the preconscious feeling of experience; DMPFC provides full cognitive consciousness. Daydreaming in private thought we imagine future experience, while aligning our survival priorities. In clinical practice free association inevitably gives way to transference because, as Freud said in his *Negation* paper, repressed associations are innately private. When fantasy triggers anxiety, we may become fully conscious in order to process the past experience as a dangerous template for behavior. Anxiety leads us away from new fear conditioning, which can lead to PTSD. With interpretation and reconstruction, an analysand may feel safe enough to reframe past fear conditioning. But, when our lives present insoluble problems, we may resort to reliving dangerous situations that gave rise to experience and images that were relegated to a dynamic unconscious.

### **Screen Memories Veil the Trauma of Ontological Passages**

Freud posited that unbearably intense feelings trigger the formation of a dynamic unconscious; he equated overly high drive pressure with affect pain. Reframing that

concept, we can say that over-stimulated NMDA pathways can trigger apoptosis-- death of post-synaptic neurons; whereas moderate stimulation adds experience, as NMDA pathways become dedicated to their Hebbian pathways. Mark Johnson found that these cellular life and death mechanisms account for stage change in human ontogeny—and I think in endogenous PTSD, often veiled in screen memory. One-third of the brain's neurons die in the first postnatal year, most through apoptosis, which leaves surrounding contacts insulated from over-stimulation. Ontogenesis forms pathways for experience that are vital to our survival functions.

Psychologically, stage changes manifest new routines of consciousness. Prior consciousness feels too hot to handle. Perhaps exogenous trauma triggering PTSD uses the same mechanism as endogenous trauma during stage changes. It is safe to say that insulation from overly intense experience applies to the psychological and the neural world. There is a complete reorganization of the social-domain during the three to six year old period that brings most children into a world of Freudian dynamics. The stage change due to prefrontal maturation, which Freud called *Oedipal* and A. R. Luria (from Pavlov) called a *second signal system*, produces top-down consciousness that overrides the world of earlier childhood experience making detachment into reflection possible.

### **Forming the Dynamic Unconscious**

Panzer et al. present a cogent review of the relationship between unconscious and fully conscious processing that is useful to psychoanalytic thinkers. Fear conditioning shows how bottom-up behaviorism and top-down neuropsychology merge. Fear associates a painful, conditioned stimulus (*CS*) with an unconditioned stimulus (*UCS*), which triggers unpleasure instinctually. Excitatory inflow to the lateral and basolateral

amygdala disinhibit the central amygdala output to the hypothalamic-pituitary-adrenal (HPA) axis, which triggers instinctual fear responses and cascading hormonal stress responses. Hippocampal feedback may stop the cascade and protect the VMPFC from losing its behavioral regulation. It takes multiple neural triggers activated in precise order to overcome normal GABA inhibition. But set in motion, an intracellular cascade in the central amygdala leads to permanent long-term potentiation (LTP) of the fear experience.

During acute stress, VMPFC neurons respond to massive cortisone inflow by losing plasticity—a semi permanent emergency response. Foundations of self-continuity are stored in hippocampal long-term memory. Newly conditioned fear associations conflicting with stable long-term emotional memories are shunted to the unconscious, segregated from an increasingly inflexible self. The extent to which hippocampal circuits shunt these CS to unconscious processing determines how flexibly (consciously) one deals with new survival threats. Normally, warning imagery and signal anxiety protect one from the activation of the central amygdalar emergency response system. But in the helplessness of severe acute stress that leads to PTSD, the CS and its attendant imagery become dire threats. Permanently conditioned left hippocampal circuits render narrative memory of the trauma unavailable, breaking the self's continuity.

### **A Confusion of Tongues: Speaking the Abuser's Language**

Clearly, PTSD induces pathological identifications. One angle on how these identifications are formed comes from Ferenczi's paper: *A Confusion of Tongues Between Adults and Children*. Like the Stockholm syndrome that paper portrays *identification with the aggressor*. Identifying with the sexually abusing adult, to the extent of feeling responsible for the sexual activity between them, the child assimilates

aspects of the adult as self-extension. As adult stimulation encompasses the child's self with intense sexual experience, the child's premorbid self loses its familiar qualities; others—even strangers-- are turned into potential sexual partners. Ironically, as the abuser becomes familiar, the stranger quality dissipates in pathological identification.

*In Regional differences of the prefrontal cortex in pediatric PTSD*, Richert, Carrion, and Karchemskiy found that compared to normal children, grey matter in the abused child's VMPFC over-expands in years after the abuse; while that in the DMPFC contracts. I take this as a neural result of the effect of over-identification with the abuser. The stranger (and other future strangers) becomes self-extension. Stranger in this context includes a once familiar kin who became a stranger in the abusive situation. Sexually over-stimulated children may become perverse adults. In abuse—or loss—qualities of otherness are processed to the point of becoming a familiar part of the self. Patricia Hearst identified with captors who kidnapped her; later she married a bodyguard, a self-extending identification with the aggressor.

### **A Unified Theory of Mental Syndromes**

Autism, PTSD, psychosis, major depression, and Alzheimer's dementia produce incapacity for empathy and a less vivid sense of the social world. Self and other enter a syndromatic divide when unrelenting stress shifts our survival mode from judgment to emergency. Thereafter, VMPFC passively conveys apathy to a DMPFC that provides rote frame for each syndrome. In PTSD, the prototype syndrome, hippocampal and VMPFC stress responses reduce secondary process thinking as NMDA transmitter circuits lose their plasticity through excitotoxicity and apoptosis. As more fear-conditioned experience enters the dynamic unconscious, the DMPFC narrows its field of pragmatic judgment.

The prodroma to psychosis includes all of the experiences that can lead to PTSD. In fact, sometimes a PTSD-inducing experience proceeds to psychosis. Psychosis follows the formation of primary delusions: losing distinction between the emotional self and the judgmental other. One enters psychosis through fear and terror that undermine belief in a theory of mind. When the appraisal of self-feeling loses reliability, the sense of individual and social integrity—of essential human identity—fails. The usual recourse is appeal to higher level social domain generalizations: a belief in god as final arbiter of all decisions and judgments. For example, the switch point to manic psychosis manifesting change from the pragmatic to the emergency survival mode is often religious.

The course of psychosis replaces individual, self-feeling identity with a sense of embracing the most generalized other--tantamount to a massive social identification with the aggressor. Institutional transference is typically profound in chronic schizophrenia, because the caring institution feels as socially inclusive as god. I assume that Hebbian association nodes dedicate most VMPFC neuron groups to DMPFC groups. Life becomes a daily ritual of the VMPFC-self piling up objects to fill the empty social space.

Supekar et al. with Greicius show that neural atrophy replaced by amyloid in Alzheimer's encompasses the dense Hebbian hubs of the default domain. Amyloid precursor protein concentrates in synapses, where it turns to amyloid as neurons die. Since old folks depend on maintaining higher cortisone levels to deal with stress, their NMDA neurons show hyper-metabolism in dying hubs of default domain processing. Breakdown in hippocampal medial prefrontal cortex linkage characterizes the fcMRI of Alzheimer patients. Clinically, these patients fail to distinguish self from other in social

interactions. They live in a self-other *mélange* like bewildered children, as kin become strangers—all the *sine quo non* of VMPFC and DMPFC failure to function normally.

### **Some Notes on Therapy**

Koenig et al. found that veterans with VMPFC or amygdala *damage* were protected from developing PTSD, because their fear responses could not amplify. People with PTSD feel broken identity. A patient of mine who exited one of the twin towers as it was collapsing during the 9/11 attacks was unable to return to his high level work as a financial analyst. Losing many friends and colleagues, he felt that his fast track identity was destroyed in the attack. I agreed: his prior identity was gone. We vest our love in familiar others. When they are traumatically lost, we feel our love capacity is lost with them. To keep his love alive, he decided to become a high school English teacher—to help repair America. After that decision, which restored his sense of a potentially benign social world, he felt free to love his wife again. Gaining confidence in pursuing his new life, his anxiety and sense of stress diminished. I reinforced his anxious decision to make a complete career change, despite the loss of earning power.

PTSD patients want to know, ‘Where’s my self?’ I explain that their past self has been permanently injured, but they can create a new, serviceable self. Even in psychosis, when self feels altered or lost, the therapist can point out that the person created a new self, because their past self no longer dealt with their world. There is clinical advantage in explaining mechanisms a patient uses: constructs buttress identity from the top down. Feeling that the therapist understands the nature of the loss helps the transference contain trauma. But pretending to understand, the therapist only amplifies the feeling of social abandonment. In left brain engendered PTSD, a person feels lost agency, because the

capacity to love disappears with the lost love one. Explaining this provides a prosthesis that binds trauma until new self-objects can be trusted—perhaps the therapist. Just as the patient who lost colleagues in the twin towers attack suffered survivor shame, veterans who lost war buddies feel humiliated when they try to love.

### **Bibliography (Social Neuroscience References)**

Andrews-Hanna, Jessica R, Reidler, Jay S, Sepulcre, Jorge, Poulin, Renee & Buckner, Randy (2010) Functional-Anatomic Fractionation of the Brain's Default Network.

*Neuron*. 2010 February 25; 65(4): 550-562. doi: 10.1016/j.neuron.2010.02.005.

Blanke, Olaf and Arzy, Shahar (2005). The Out of Body Experience: Disturbed Self-Processing at the Temporo-Parietal Junction. *NEUROSCIENTIST* 11(1):16-24, DOI: 10.1177/1073858404270885.

Fanselow, Michael S & Dong, Hong-Wei (2010). *Neuron* 65, January 14, 2010.

Hilz, Max J, Devinsky, Orrin, Szczepanska, Hanna, Borod, Joan C, Marthol, Harald, and Tutaj, Marcin (2006). Right ventromedial prefrontal lesions result in paradoxical cardiovascular activation with emotional stimulation. *Brain* 2006 129(12):3343-3355.

Johnson, Mark H. (2005). Sensitive Periods in Functional Brain Development: Problems and Prospects. ([www.interscience.wiley.com](http://www.interscience.wiley.com)) DOI 10.1002/dev.20057.

Koenigs, Michael, Huey, Edward D., Raymond, Vanessa, Cheon, Bobby, Solomon, Jeffrey, Wasserman, Eric M. and Grafman, Jordan (2007). Focal brain damage protects against post-traumatic stress disorder in combat veterans. *Nature Neuroscience* 11, 232-237.

Luria, A.R., *Higher Cortical Functions in Man*, Basic Books, New York, 1966 (1980).

Northoff, Georg and Panksepp, Jaak (2008). The trans-species concept of self and the subcortical-cortical midline system. *Trends in Cognitive Sciences* Vol. 12 No. 7.

O'Reilly, Randall C (2010). The What and How of prefrontal cortical organization. *Trends in Neuroscience* 33 (2010) 355-61.

Panzer, A., Viljoen, M., and Roos, J.L. (2007) The neurobiological basis of fear: a concise review. *S Afr Psychiatry Rev* 2007:10:71-75.

Pelphrey, Kevin A & Morris, James, P (2006). Brain Mechanisms for Interpreting the Actions of Others From Biological-Motion Cues. *Curr Dir Psychol Sci.* 2006 June; 15(3): 136-140.

Raichle, Marcus, E. and Snyder, Abraham Z. (2007). A Default Mode of Brain Function: A Brief History of an Evolving Idea. *NeuroImage*:doi:10.1016/j.neuroimage.2007,02.O41.

Richert, KA, Carrion, VG, Karchemskiy, A, & Reiss, AL, Regional differences of the prefrontal cortex in pediatric PTSD: an MRI study (2006). *Depress Anxiety*: 2006;23(1):17-25.

Supekar, Kaustubh, Menon, Vinod, Rubin, Daniel, Musen, Mark & Greicius, Michael D. (2010). Network Analysis of Intrinsic Functional Brain Connectivity in Alzheimer's Disease. *PLoS Computational Biology*, 11/15/2010.

Van Dijk, Koene RA, Hedden, Trey, Venkataraman, Archana, Evans, Karleyton C, Lazar, Sara W & Buckner, Randy L. Intrinsic Functional Connectivity As a Tool For Human Connectomics: Theory, Properties, and Optimization (2010). *J Neurophysiol.* 103: 297-321. 2010. doi: 10.1152/jn.00783.2009.