Instructions for the Handy Brain Model™

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The HANDY BRAIN MODEL was created to help clinicians and educators show which areas of the brain are activated during various emotional states. Nonprofessional terminology is used as much as possible but may need to be further simplified, depending on the target audience. Because the HANDY BRAIN MODEL turns 2-dimensional drawings on the mitten into a 3-dimensional object, it lacks anatomical accuracy, but shows the connection between structures. More accurate graphics of the brain are included as necessary.

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NOTE: To properly wear the mitten, cut off top of the poster-board insert and leave it under the Orbital Frontal Cortex to help that part of the mitten maintain its shape. Your fingers should go between the insert and the back of the mitten. Then, curl your fingers around your thumb to make a fist that represents the appearance of the cortex of the right brain hemisphere.

Acronyms for Brain Features

• ACC: anterior cingulate cortex
• dACC: dorsal ACC
• vACC: ventral ACC
• CNS: central nervous system
• GABA: gamma-amino butyric acid
• HC: hippocampus
• H.Th: hypothalamus
• LC: locus coeruleus
• NA: nucleus accumbens
• OFC: orbital frontal cortex
• PFC: prefrontal cortex
• PNS: parasympathetic nervous system
• R: Raphe’s nuclei
• SNRI: serotonin-norepinephrine reuptake inhibitors
• SSRI: selective serotonin reuptake inhibitors
• SNS: sympathetic Nervous System
• VTA: ventral tegmental area
**Explain the Brain Cortex**

Cross your fists with the Mitt on one hand to show the general layout of the two brain hemispheres. When the fists are held in this position, the brain appears more horizontal than it actually is, but the temporal lobes are on the sides, as they should be. The brain is more like the shape and size of a medium head of cauliflower.

1. This view shows the frontal lobes with the motor and sensory cortexes across the top on either side of the central sulcus. The frontal lobes are located at the front of both hemispheres. They promote attention, short-term memory, planning, and motivation.

2. The temporal lobe is beneath the lateral sulcus and is involved in receiving, organizing, and disseminating anything to do with sound and with the integration of visual, auditory, and spatial information, especially faces. The insula is folded deep within the lateral sulcus and can be seen on the HANDY BRAIN MODEL by opening the hand. It monitors all sensations coming from the body, integrates thoughts and feelings, and balances fight-flight, sympathetic (SNS) and rest-connect, parasympathetic (PNS) nervous systems.

3. The prefrontal cortex (PFC) is below the knuckles of the fingers. It is associated with planning complex cognitive behavior, personality expression, decision making, social behavior, and coordinating thoughts and actions with internal goals. The area in red (below the fingertip knuckles) is the orbital frontal cortex (OFC), behind the eye orbits. It detects danger and connects directly with the amygdala.

4. The parietal lobe is seen above the frontal lobe on the back of the hand. It perceives or maps surroundings, measure experience, and attends to spatial details. Input from the skin (touch, temperature, and pain) are relayed through the thalamus to the parietal lobe.

5. The occipital lobe (meaning behind [Oc] the head [caput]) is the visual cortex. It receives, organizes, and distributes visual information.

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**Explain the Limbic System**

1. The **limbic system** (meaning boarder or edge) is below the thumb knuckle on the mitt. It is revealed by “peeling back” the cortex. It is primarily responsible for emotions, and has much to do with forming memories. Two important structures are shown on the tip of the thumb: (a) The C-shaped **caudate** is involved in initiating movement, muscle tone, and disregarding irrelevant information. It may malfunction in OCD. It is wider at the head and has a long tail. The word “caudate” means something that has a tail. Together the caudate and **putamen** are called the striatum due to the striped appearance. (b) The **nucleus accumbens** (NA) is responsible for drive and motivation. It is shown as a purple area on the head of the caudate. It is the reward center and releases dopamine which is similar to cocaine.

2. The head of the hippocampus (meaning sea horse) is found deep in the temporal lobe where the thumb joins the hand on the underside of the mitt. The body of the hippocampus swings back towards the caudate and thalamus. It stores and retrieves memories and turns short-term into long-term memory.

3. The amygdala (meaning almond shaped) appears just above the head of the hippocampus deep in the temporal lobe. In the right hemisphere it is associated with detecting threats and raising alarms, but in the left hemisphere it is associated with arousal of both positive and negative emotions.

4. The cingulate cortex on the HANDY BRAIN MODEL is under the ring finger. It is on the bottom layer of the cortex but is considered part of the limbic system. Cingulate means “band” and it forms a band around the limbic system (shown by curling the fingers around the thumb). The front (anterior) of the cingulate cortex (ACC) curves dorsally over the limbic system where it processes thoughts. Activation of the dACC dampens amygdala activity. The part of the cingulate (tip of the ring finger) that curves ventrally under the limbic system processes emotions. The back (posterior) cingulate lies under the parietal lobe and handles general awareness.
Explain Features on the Palm of the HANDY BRAIN MODEL (shown below)

1. **Corpus collosum** (or tough body) on the mitt starts on the pinky finger. It is a flat bundle of myelinated fibers that connects the left and right hemispheres and is below the cingulate.

2. **Cerebellum** (little brain) is shown on the bottom of the mitt extending behind the brain stem. It is involved in coordination, fine tuning and timing of movement, and balance.

3. **Thalamus** (meaning chamber) is just above the mid brain. Acting as a “grand central station,” all sensory input from the central nervous system (CNS) comes through the thalamus and is directed to appropriate areas on the cortex.

4. **Hypothalamus** (H.Th.), below the thalamus and beside the brainstem, connects the nervous system to the endocrine system and is involved with drives for food, drink, sleep, and sex.

5. **Pituitary gland** is pea sized and hangs below the hypothalamus. It secretes hormones related to growth, blood pressure, pregnancy, sex, thyroid function, and pain relief.

6. **Insula cortex** is explained in the section on the temporal lobe previously.

7. **White matter** makes up much of the interior of the brain. It is a fatty (myelinated) substance covering nerve fibers. The unmyelinated cell bodies make up grey matter.

Explain the Brain Stem

The brainstem, shown on the palm side of the HANDY BRAIN MODEL, consists of the **Mid Brain** (associated with vision, hearing, motor control, sleep/wake cycles, and temperature regulation); **Pons** (relays signals from the forebrain to the cerebellum associated with sleep, dreaming, breathing, swallowing, bladder control, hearing, equilibrium, taste, and posture), and **Medulla** (controls involuntary functions of breathing, heart rate and blood pressure).

**Cranial Nerves in the Brain Stem**: The Sympathetic nervous system (SNS) is sympathetic or reactive to the environment (fight-flight). The para-sympathetic nervous system (PNS) is on the side (para) above or below the SNS. It complements the SNS and returns the body to rest-connect when danger is past. It conserves energy and does maintenance tasks during rest. There are three spinal sacral PNS nerves on the lower back (S-2, S-3, and S-4) and four cranial PNS nerves (c-10, c-9, c-7, c-3):

- **C-3**: Oculomotor (rising from the base of the mid brain) controls pupil dilation and constriction and eye movement that occurs during REM sleep.

- **C-5**: Trigeminal nerve is the largest cranial nerve. It has three branches that extend from the middle of the Pons and transmit sensation from the face. Although it is not a PNS nerve, various PNS nerve bundles (ganglia) use one of the trigeminal’s branches to reach their destination.

- **C-7**: Facial and intermediate (rising from the bottom of the Pons) control secretions of tear glands, mucus production, and facial expression.

- **C-9**: Glossopharyngeal (rising from the top of the Medulla) controls mucus and salivation in the mouth and extends from the trigeminal nerve (C-5) to the nose, uvula, upper lip, gums and pharynx).

- **C-10**: Vagus nerve (just under C-9 on the Medulla) wanders like a vagabond to every organ in the body but the adrenals.

Explain Brain Stem Neurotransmitter Pathways

**Neurotransmitters** are chemicals that transfer impulses from one nerve across the space (synapse) between nerves to the dendrite of another nerve.

**Vesicles** on the nerve terminal release neurochemicals into the synapse.

**Receptors (=)** on the dendrite receive neurochemicals that “fit” them.
**Major neurotransmitters that affect mental states**

- **Serotonin** affects mood, reduces pain, and more. Raphé’s nuclei (R) is the primary source of serotonin in the brain (90% is produced in the gut) and projects pathways outward from the cingulate cortex.

- **Norepinephrine** affects wakefulness, energy, concentration, motivation, etc. It is produced in the locus coeruleus (LC) and projected along pathways similar to serotonin circuits and to the cerebellum.

- **Dopamine** affects alertness, motivation, learning, and fine motor tuning.

  It is mainly produced in the ventral tegmental area (VTA) and project along pathways to the forebrain. The VTA connects with the nucleus accumbens (NA) for pleasurable sensations and reward, common in addictive behaviors.

**Explain Neurophysiological Causes & Cures for Emotional Problems**

1. **Anxiety** is related to a hyperactive amygdala (the brain’s alarm system) that continue fight-flight reactions long after they are needed. It sends signals to the hypothalamus that tell the adrenal glands to release **adrenalin**. This fires up the sympathetic (fight-flight) nervous system. High stress levels and inherited deficiencies in serotonin and/or GABA also contribute to anxiety spectrum disorders like social phobia or panic attacks. Panic causes people to feel trapped and be overly aware of their bodies. Neurological tricks can help calm adrenalin surges.

   - **Cingulate brake**: The front part of the cingulate cortex above the limbic system is activated by deliberate thinking. Thinking tasks immediately reduce amygdala activity. Counting backwards from 100 by 3, naming states north, south, east, and west of you, and reciting bible verses are all excellent ways of using the Cingulate brake to dampen the amygdala. Cognitive Behavior Therapy (CBT) uses the cingulate brake when questions are asked to challenge negative automatic thoughts.

   - **Insula brake**: The Insula is activated by focusing attention on body sensations. One of insula’s main functions is to balance sympathetic (SNS) reactivity that makes thoughts race with parasympathetic (PNS) calm. As the PNS comes “on line,” the amygdala disengages. Somatic Experiencing, Brainspotting, and other therapies use this insula brake to slow down negative automatic thoughts so new insights can come.

**Activate the calming parasympathetic nervous system (PNS)**

- **a. Vagal brake** (C-10): The Vagus nerve wanders like a vagabond to every organ in the body, except the adrenals. Longer exhalations activate the Vagus nerve. It releases **acetylcholine**, a neurotransmitter that reduces inflammation and enhances learning and cell growth. Acetylcholine reduces heart rate and further slows the breath. Simply observing your breath will produce longer exhalations. Count your exhalations for 30 seconds. Practice again after making yourself yawn while staring at a spot and see if you take fewer breaths.

- **b. Yawning reflex**: Yawning cools overactive frontal lobes by taking in outside air that is cooler than 98.6. It relaxes and increases alertness faster than any many meditation methods (Newberg, 2010, pp. 158-159).

- **f. Oculomotor brake** (C-3): Intentionally making rapid eye movements activates PNS C-3. This is used in a treatment called Eye Movement Desensitization and Reprocessing Therapy (EMDR) that creates a “window of tolerance” when thinking about past traumas. C-3 can also be activated with a convergence exercise used by eye doctors. Hold your pen about a foot from your face and look at the tip for 2 slow exhales and then look past the tip to a point about 12 feet away for 2 slow exhales. Repeat this about 15 times. Eye muscles converge when looking at the closer point and diverge at the far point. This should be practiced daily to reduce panic attacks.

**Gamma-Amino Butyric Acid** (GABA) slows down the transmission of nerve impulses. It is produced and distributed all over the brain. Drugs like Xanax, Ambien, barbiturates, and alcohol add to the efficiency of GABA by increasing the frequency or length of time its receptor channels remain open.

2. **Obsessive Compulsive Disorder** (OCD) causes people to repeat a thought or ritual over and over to prevent “catastrophes.” Four areas of the brain are involved: (a) The **orbital frontal cortex**, OFC, (behind the eye orbits) signals danger. The degree of over-activity in the OFC is thought to predict of how effective treatment for OCD will be (Maia, 2008). (b) The OFC activates the **cingulate** (tip of the ring finger on mitt) involved in error detection. (c) The head of the **caudate** (back of the thumb on mitt) normally disrupts faulty danger/error signals, but with OCD this “gear” is stuck and the danger/error message goes on to the (d) **thalamus** (grand central station on the palm of the mitt). It may already be
over stimulated in people with OCD who have finely tuned nervous systems. Without the caudate to “turn the page,”
faulty signals cycle over and over between the OFC, cingulate, caudate, and thalamus, creating a worry circuit. Mild OCD
can be helpful in jobs that require organization and attention to detail. Three steps strengthen a malfunctioning caudate
that must be practiced repeatedly (Schwartz, 2012).

- **RE-LABEL** repetitive thoughts and rituals as OCD—*This is not me; it’s OCD*. Question anxiety caused by *germs*
  and *disorder; inability to check* locks, ovens, your appearance, your work, other’s safety or faithfulness, or body
  symptoms; *inability to repeat thoughts or behaviors* that “can prevent” misfortune; *fears of harming* yourself or
  loved ones; *lack of “perfection;”* and trouble *throwing away* something you might need in the future. Practice until
  you completely believe that rumination and rituals are caused by a faulty caudate.

- **RE-DUCE** anxiety caused by thoughts or rituals you now know are OCD by noticing exactly where the sensation is in
  your body and what it feels like—tightness, pressure, heaviness, etc. Go back and forth between awareness of anxiety
  sensations and the breath. This uses both the insula brake and the vagal brake (p. 4). Due to brain placidity, using
  calming strategies while deliberately activating OCD thoughts and/or preventing OCD rituals will reduce hyperactivity
  in the OCD worry circuit.

- **RE-FOCUSE** your attention on an enjoyable activity—exercise, crafts, dancing, listening to music, or
  looking at a lovely image. Although many ways are listed to reduce a surge of adrenalin (p. 4), with
  OCD it is especially important to activate the nucleus accumbens (NA), sitting on the head of the
  caudate, that releases the pleasure neurotransmitter, dopamine. This is like putting oil on a stuck gear.

**SSRIs** like Prozac and **SNRIs** like Effexor (see p. 6) are thought to eventually increase serotonin
receptor sites in the OFC and reduce over-activity.

**Cingulotomy** surgeries for OCD make a slight wound in the front of the cingulate to improve OCD in
people who are resistant to behavior therapy and medication.

### 3. Post-Traumatic Stress Disorder

PTSD is an intense, repetitive response to physical or emotional trauma with
heightened anxiety, flashbacks, and attempts to avoid reminders of what happened. During the event the amygdala
becomes highly activated and triggers the hypothalamus and pituitary to release adrenalin (HPA axis) and cortisol.
Cortisol dampens the immune system and enhances recall of the disturbing event. Chronic high levels of cortisol
damages, destroys, and shrinks the **hippocampus** (HC), which leads to increased cortisol levels. This damage causes too
fast and unspecific recall of the trauma.

The good news is that the cells of the HC are able to reproduce, making it the center for new learning.
Additionally, the pathways for **acetylcholine** (that boosts immune function and promotes new learning) terminate or
pass through the prefrontal cortex and the HC. In the HC, acetylcholine actually prevents the retrieval of old memories
and helps make new memories. To grow a healthy HC remember:

- **Acetylcholine** is the main neurotransmitter used by the PNS.
- **Oculomotor brake** (used in EMDR) sends a spurt of acetylcholine, blocking old memories and creating
  new ones when eye movements are made.
- **Insular brake** (used in Brainspotting and Somatic Experiencing) balances the SNS and PNS, also increasing acetylcholine. These therapies create a “window of tolerance.” Alarming memories are revisited after the PNS “kicks in” reducing
  reactivity to the event.
- **Amygdala retraining** is used in Prolonged Exposure Therapy (PE). The traumatic memory is retold, written and re-read over and over. This activates
  the amygdala, but eventually it “learns” the event is past and there is no need
  for alarm.

### 4. Impulse Control Disorders

(ICD) involve a failure to resist a temptation, urge or impulse that may harm oneself or
others; such as, compulsive shopping, gambling, pornography use, sexual acts, angry outbursts, and more. The initial
experience with a particular activity causes a dopamine release (feel-good neurotransmitter). Much like cortisol in PTSD,
dopamine enhances long-term memory. People who naturally have lower levels of dopamine (as in Attention Deficit
Disorders) are at greater risk of engaging in behaviors that cause dopamine release.

Robert Miller (2010) describes a dopamine link that ties a positive feeling with a specific behavior that forms
a unit called a feeling state. The person is seeking the feeling state—not the behavior itself. Urges and desires are not
feelings but the wanting of a particular feeling. Unlike in OCD, people are not trying to avoid “catastrophes,” they are
seeking pleasure. Intense and repetitive responses to pleasurable events and attempts to avoid triggers, make impulsive
behavior a direct opposite of PTSD. Therefore, the treatment of PTSD suggests approaches for ICDs:
a. **Dopamine release** is promoted by identifying, describing, and imagining the exact behavior that generates the most intense positive feeling.

b. **The prefrontal and anterior cingulate cortices** are activated by having the person stare at a spot. This causes a state of concentration rather than distractibility.

c. **The insula** is used to focus on sensations created by the positive feeling. Because the insula promotes homeostasis, it reduces exaggerated positive feelings.

d. Or, the **Oculomotor nerve** is activated with eye movements to promote acetylcholine release that pre-empts old memories with new learning and reduces heart rate and excitability.

These steps are repeated until the person can think of the behavior without having an urge to do it and can identify events that caused the need to feel powerful, desirable, etc. Training in Miller’s protocol is recommended!

5. **Depression** is different from sadness. Difficulty concentrating, indecisiveness, loss of goal-directed behavior, inability to experience pleasure, inability to control negative thoughts, and social withdrawal are all symptoms. Planning, decision-making, and initiating action happen in the **frontal lobes** and the **cingulate** prioritizes relevant factors. They under-function in depression. Because the cingulate is on the border between the cortex and the limbic system, it enables connection of thought and feeling integral to empathy, affection, and attachment. Chronic and extreme stress destroys brain cells in these areas. Depression related to changes in brain structures and neurochemistry may require medication to “jump-start” the brain so people can engage in talk-therapy or meditation. Serotonin, norepinephrine, and dopamine are all key players in depression.

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**Selective Serotonin Reuptake Inhibitors** (SSRIs) like Prozac, Paxil, Zoloft, Celexia and Lexapro increase levels of serotonin by blocking its absorption at receptor sites. More serotonin helps brain cells send and receive messages, which boosts mood. Over time serotonin may kick off the production of new brain cells. This reverses the effect of stress that destroys brain cells.

- **Hallucinogens** target a serotonin receptor associated with vision. Images that would not normally be perceived become vivid and make us think they are real.

- **Ecstasy** releases large amounts of serotonin and triggers the release of the bonding hormone, oxytocin. The surge of serotonin later depletes its availability, causing depression, anxiety, and more.

**Serotonin Norepinephrine Reuptake Inhibitors** (SNRIs) like Prestiq, Effexor, and Cymbalta increase levels of both serotonin and norepinephrine by blocking their absorption. The dual action is supposed to improve energy and alertness in addition to mood. Effexor, especially, has been found more effective when depression has a strong anxiety component.

**Norepinephrine-Dopamine Reuptake Inhibitors** like Wellbutrin partially block absorption of these chemicals.

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6. **Attention Deficit (Hyperactive) Disorder** (ADD and ADHD) are caused by low levels of dopamine in the frontal lobes because over-active receptors make the neurotransmitters clear the synapse too quickly. People with the disorder have problems with organization, impulse control, procrastination, concentration, and working memory, but are equally intelligent as those without the disorder. Heightened energy and novelty seeking may be ADHD benefits and the reason why its rates are higher in America than in countries where people did not leave their homeland to start a new life (Hartman, 2005). Due to increasing demands for planning and managing life as people age, ADHD may not fully show itself until adolescence or adulthood. Behavior strategies can manage symptoms but do not address the cause.

**Prescribed Stimulants** either increase the release of dopamine and norepinephrine (Adderall type) or keep them from being reabsorbed (Ritalin type), normalizing levels of these neurotransmitters in people with ADHD.

- **Non-prescribed stimulants** like Methamphetamines and Cocaine are stronger, faster, and shorter acting than medicines that release dopamine in a slow, steady, natural manner.

- **Marijuana** temporarily increases dopamine levels. Its most potent chemical (THC) reduces firing of neurons in the caudate, putamen and other areas that initiate movement (reducing hyperactivity). The problem with self-medicating is that “doses” may be too high and cause other problems.

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7. **Schizophrenia** is a thought disorder recognized by delusions, hallucinations, and bizarre behavior due to excessive dopamine in the VTA-NA (mesolimbic) pathway. Loss of imagination, responsiveness, and motivation may be due underactivity of dopamine in the VTA-PFC (mesocortical) pathway.

Older anti-psychotic medications block dopamine in both pathways, reducing bizarre thoughts, but increasing lack of responsiveness. Newer medications both block dopamine and act on serotonin to maintain frontal lobe activity.
8. **Relationship problems** affect and are affected by the brain. Love is not an economic arrangement or unpredictable chance emotion. It is an attachment bond that provides a sense of belonging, support, and confidence that helps people cope with the ups and downs of life.

- **Oxytocin** is the neurotransmitter that fosters attachment in nursing, childbirth, and orgasm. Gentle touch, massage, soft eye contact, and other social behaviors generate Oxytocin that reduces activity in the amygdala.

- **The Hypothalamus** (H.Th) produces oxytocin. It is stored and secreted by the pituitary gland that hangs below the H.Th. An oxytocin bond triggers dopamine release from the VTA in response to anything pleasurable and endorphins, like opiates, (from the pituitary) in response to satisfaction. The first blush of love, driven by dopamine and endorphins, creates an obsession that makes people feel like all their needs are being met. Jack Panskeep (2012) explains several affective pathways that drive relationships and our very survival:

  a. The Panic pathway is activated when partners feel fleeting disconnection as loved ones push for their own needs to be met. The potential loss of the love bond is coded by the brain as primal panic. Endorphins are rapidly reduced, leaving people in withdrawal to face agonizing feelings of abandonment. The panic pathway is pro-survival, causing people to actively solicit help and social support. The sympathetic (fight-flight) nervous system is mobilized.

  b. The Rage pathway energizes the body to defend itself and provoke fear in others. The hypothalamus triggers the pituitary and adrenal glands to release cortisol and adrenalin. Adrenalin diverts blood away from the frontal lobes to organs and muscles. Under the influence of the Rage pathway partners become demanding and clingy to regain comfort. Bids for attention are laced with anger and experienced as pursuing, attacking, and defending.

  c. The Fear pathway (flight/freeze reflex) reduces the impact of anger from others to minimize the chance of bodily destruction. Threatening stimuli register in the amygdala. At first cortisol and adrenalin are released, but when too overwhelmed people (like reptiles) descend into primitive withdrawal to detach, soothe, control, and protect themselves with the back (dorsal) part of the Vagal PNS nerve. This “vegetative Vagus” (rest-digest) is uncoated (unmyelinated) and slow to respond. Under great stress people freeze and immobilize.

  d. The Seeking pathway comes to the rescue in this dismal picture. Dopamine activates arousal, focus, and a desire for meaning. The frontal cortex (involved with ‘fore’ thought) comes back on line after being shut down by rage and fear. Space is made for the social pathways of Care, Play, and Lust. The “smart (ventral) vagus” is coated (myelinated) to provide quick control in regulating fight-flight behavior. The vagus is closely connected to oxytocin receptors, the superstar of the attachment bond.

This cascade of events, when secure attachment is temporarily lost, suggests that all the neurological tricks listed on pp. 4-5 be used when couples find themselves in attack-defend-withdraw cycles. Johnson (2008) describes how to have accessible, responsive, expressive relationships in her book *Hold Me Tight*.

### Explaining Gender Differences and Sexual Orientation

**Gender differences and sexual orientation** are linked to variant brain structures and hormones, even ones not related to sexual arousal. Knowing these variations can help gay people and their families be more accepting of their orientation. A size difference in the corpus callosum that connects right and left hemispheres may be a myth. Findings show on average:

**Straight men and lesbians have:**

- A right hemisphere that is 1 – 2% larger than the left, particularly the parietal lobe that has to do with spatial awareness.
- Strong connections between the amygdala and the sensorimotor cortex and caudate-putamen that are involved in the fight-flight reactions.
- A longer ring finger than index finger due to more exposure to testosterone in utero.
- An *INAH-3 (tiny area) in the hypothalamus (H.Th) that is twice as large as the INAH-3 in heterosexual females.

**Straight women and gay men have:**

- Cerebral hemispheres that are the same size.
- Strong connections between the amygdala and the anterior cingulate and the sub-callosal areas that influence mood.

**Note:** Gay men also have a longer ring finger than index finger.

- An *INAH-3 in the H.Th. that is twice as small as the INAH-3 of straight men and lesbians.

*The 3rd interstitial nucleus of the anterior hypothalamus (INAH) influences secretions of testes, ovaries, & attraction to females.*

**For fascinating information on ring finger length, Google digit ratio, Wikipedia.**
Explaining Meditation

Various possible brain changes happen during meditation. Fixing the gaze activates the prefrontal cortex and anterior cingulate. The caudate and putamen are active throughout meditation to help detach from irrelevant information. This creates the attentional climate to achieve benefits in two phases:

**PHASE I:** *Self monitoring* involves switching from the ventro medial (underside) prefrontal cortex (vmPFC) “Me Center” that causes people to take things personally, worry, and feel depressed to the dorsomedial (back) prefrontal cortex (dmPFC) Me Center that increases empathy, maintains social connections, and views events from a balanced perspective. The lateral (side) PFC curbs the vmPFC and heightens the activity of the dmPFC, and most important, which grows new brain cells over time. This fights effects of stress and prevents depression!

**PHASE II:** *Thought monitoring* begins when the front (anterior) part of the temporal lobe connected to the Hippocampus controls the mental stream of thought and keeps the mind from wandering Cortico-thalmic-lymbic loops may also be activated that maintain meditation. Choiceless awareness, just noticing what is happening, and being in-the-present is achieved. Experienced meditators attain Phase II and, after years of practice, may not start with Phase I.

Neuroscientist Andrew Newberg (2009) believes that the above brain areas are key in many meditative practices including breath awareness, repeating mantras, choiceless awareness, guided imagery, candle gazing, and centering prayers. Some researchers have found that ventren practioners have reduce activity in their insulas and parialtal lobes during meditators. This may create feelings of transcendence.

References


