When the Brain's 'Who Is This About?' Gate Leaks: A Testable Neural Hypothesis of Autogynephilia

How small, likely prenatally set shifts in self–other tagging and interoceptive drive could make self-female imagery arousing—with little or no conditioning

By Dr James Morandini (guest post) • September 17, 2025

Autogynephilia (AGP) may emerge when two neural "dials" are set a bit differently from birth: a slightly leaky self–other gate in the right temporoparietal junction (rTPJ/posterior parietal cortex), and a stronger self→arousal line from the anterior insula (AIC) into the hypothalamus. Those settings can make self-as-female imagery arousing even on first exposure. Conditioning may amplify it later, but isn't required to create it.

The cast (what each area does, in one line each)

- **EBA/FFA (ventral visual stream):** fast detectors for bodies and faces—many neurons tuned to female cues in gynephilic men.
- **rTPJ & PPC:** the self–other tagger; helps decide 'is this about me or someone else?'
- **mPFC & AIC:** the self-model—top-down self-representation plus interoceptive feeling of the body.
- **Amygdala → BNST → Hypothalamus (MPOA/VMH):** the arousal engine, projecting to PAG → S2–S4 for reflex implementation.
- **VTA → Nucleus Accumbens (NAc):** motivation/gain—turns signals into drive.
- **White-matter highways:** IFOF/ILF (occipito-temporal → frontal), SLF (parietal ↔ frontal), UF (frontal ↔ limbic), MFB (VTA ↔ hypothalamus), stria terminalis (amygdala/BNST ↔ hypothalamus).

How typical gynephilia works

Female visual cues activate EBA/FFA, flow to the right parietal self–other tagger, and are labeled other. From there, signals engage the arousal engine in the hypothalamus, with VTA→NAc providing motivation. The self-model (mPFC/AIC) is involved in many things, but here it is kept separate—female = someone else.

The core idea for AGP

AGP can appear if two parameters—call them dials—are set a little differently from birth:

1. **Gate strictness (θ_1):** the rTPJ/PPC gate is slightly leaky, so some female representations carry a self tag into the self-model.
2. **Self→arousal drive (θ_2):** the AIC → hypothalamus connection is strong, so being in a "self" state directly nudges sexual drive.

With θ_1 a bit lower and/or θ_2 a bit higher, self-as-female imagery becomes intrinsically arousing without special learning. Experience later can magnify what wiring already permits.

Several paths, one outcome

- **Gate-leak route ($\theta_1\downarrow$):** the self–other gate is permissive. Perspective-taking/agency tasks that tax rTPJ should strongly modulate self-female pull.
- **Interoceptive-drive route ($\theta_2\uparrow$):** the self state (AIC) has unusually direct access to hypothalamus. Embodiment/interoceptive focus ramps arousal.
- **Synchrony route (connectivity bias):** stronger long-range coupling (IFOF/SLF) makes female detectors co-fire with the self-model—fast "pre-binding."
- **Gain route (VTA→NAc):** higher tonic motivation amplifies any of the above (not sufficient by itself).

Why autism traits could raise risk (but don't determine outcomes)

Common autistic neurofeatures—local hyperconnectivity, altered excitation/inhibition, noisier self–other attribution—map onto the same dials: they can make θ_1 more permissive (gate leak) and θ_2 stronger (self→drive). This is a risk lens, not destiny: many autistic people won't have AGP; many with AGP aren't autistic.

What the model predicts (how to test it)

Behavioural/cognitive:

- rTPJ-loaded perspective-taking/agency tasks should change self-female pull more in gate-leak profiles.
- Interoceptive accuracy should track intensity in interoceptive-drive profiles.

Imaging/physiology:

- fMRI/DTI: enhanced right-lateralised coupling along SLF/IFOF (rTPJ↔mPFC/AIC) and UF/stria terminalis (frontal/limbic↔hypothalamus).
- MEG/EEG: stronger phase-locking between FFA/EBA and rTPJ/mPFC during female cues.
- MRS: shifted GABA/glutamate balance in rTPJ (gate) and insula (drive).
- Task fMRI: imagining self-as-female (vs female other) recruits hypothalamus/insula even at first exposure.

Causal tests:

- Inhibitory TMS/cTBS to right TPJ reduces self-female imagery (gate-leak).
- Insula-targeted neuromodulation increases it (drive).
- Dopaminergic manipulations alter intensity, not presence (gain).

What this is not

- Not a moral theory and not a judgment about identity.
- Not a claim that AGP is learned from scratch.
- Not a claim that autism 'causes' AGP—only that certain neurodevelopmental features may tilt the dials.

Bottom line

A small, likely prenatal tilt in two neural dials—how strictly the brain keeps "me" separate from "others" (θ_1) and how strongly the self state can drive arousal circuits (θ_2)—can make self-female imagery intrinsically arousing. Different combinations of those dials (plus general motivational gain) explain why AGP varies across people. Most importantly, this account is specific and testable.